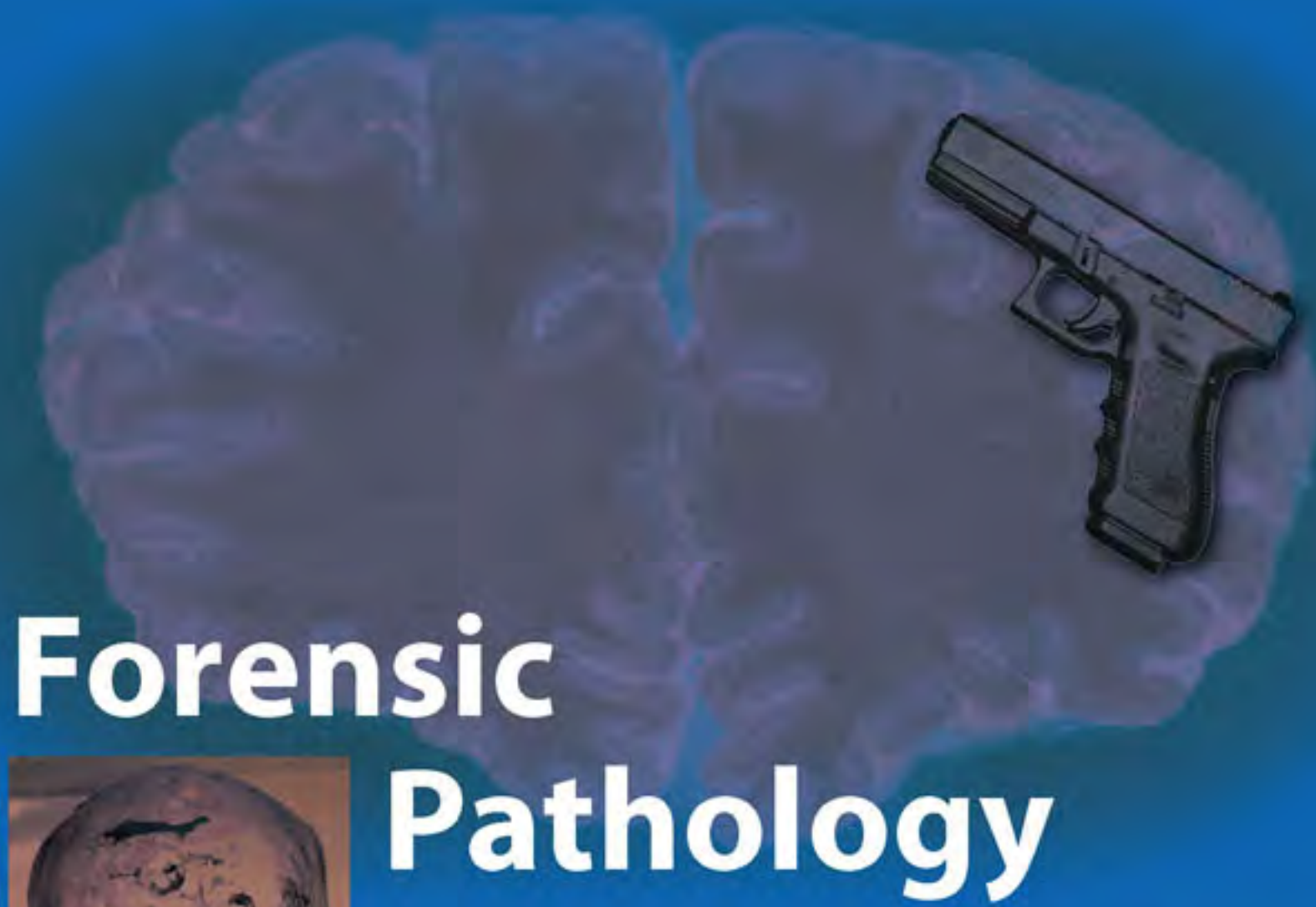
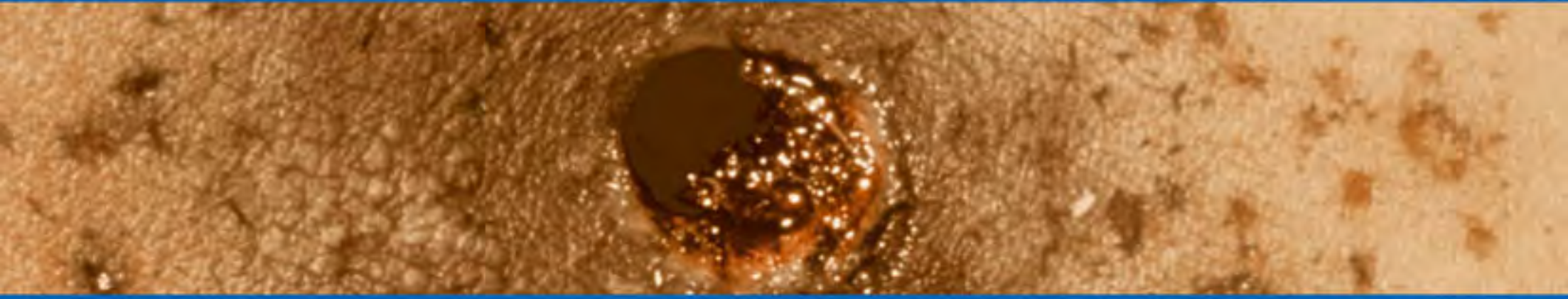


Joseph Prahlow



Forensic Pathology



for Police,
Death Investigators,
Attorneys, and
Forensic Scientists



 Humana Press

Forensic Pathology for Police, Death Investigators,
Attorneys, and Forensic Scientists

Joseph Prahlow

Forensic Pathology
for Police, Death
Investigators, Attorneys,
and Forensic Scientists



Humana Press

Joseph Prahlow
Indiana University School of
Medicine – South Bend and the South
Bend Medical Foundation, Inc.
530 North Lafayette Blvd.
South Bend, IN 46601
USA
jprahlow@sbfml.org

ISBN 978-1-58829-975-8 e-ISBN 978-1-59745-404-9
DOI 10.1007/978-1-59745-404-9
Springer New York Dordrecht Heidelberg London

Library of Congress Control Number: 2009937148

© Springer Science+Business Media, LLC 2010

All rights reserved. This work may not be translated or copied in whole or in part without the written permission of the publisher (Springer Science+Business Media, LLC, 233 Spring Street, New York, NY 10013, USA), except for brief excerpts in connection with reviews or scholarly analysis. Use in connection with any form of information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed is forbidden.

The use in this publication of trade names, trademarks, service marks, and similar terms, even if they are not identified as such, is not to be taken as an expression of opinion as to whether or not they are subject to proprietary rights.

While the advice and information in this book are believed to be true and accurate at the date of going to press, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

Dedication

This textbook was written to the glory of God and is dedicated to my Lord and Savior, Jesus Christ. I believe that it is appropriate to briefly share with the reader why such a dedication is in order, and how my life's work has helped me to come to a better understanding of things eternal. My job has helped to confirm for me three specific insights into life and death.

First, by having the unique privilege of being able to actually see inside the physical body of someone, and to then view their tissues under a microscope, I have come to the conclusion that the human body is an absolute marvel. From its anatomic structures visible to the naked eye, all the way down to its smallest molecules, as well as its complex physiology, the human body is nothing other than spectacular. To suggest that such a marvelous entity, along with all of the other incredible biological forms within our world, somehow randomly came into existence does not make sense to me. The incredible complexity of nature is proof to me that there is a higher power, a power able to create such complexity, a power who I know as God. I consider life on earth as a gift from this higher power. As a Christian, I believe that God's original intent was for us to live in harmony with Him and each other.

Second, as a forensic pathologist, I am confronted on an almost daily basis with the fact that physical death represents an unavoidable part of the entire process of life on earth. We cannot escape death. As a Christian, I believe that the human condition of unavoidable physical death results from the fact that we are sinful beings. I see all too frequently within my work the fact that human beings have a sinful free will. I believe that our sin separates us from God and His original intent to live in harmony with us.

The third insight that my job has provided for me involves the recognition of a spiritual component of life. Throughout the history of the world, many humans have been aware of and concerned about their mortality. Is there a "soul," a "spirit," or a "life-force?" If so, does it die along with the physical body? If not, what happens to it? Through my work as a forensic pathologist, I have come to understand and believe that, without a doubt, there is a soul (spirit, life-force). Dead bodies simply represent "empty shells." When I work on dead bodies, the true "person" is no longer present. As a Christian, I believe that the human spirit lives on beyond the death of the physical body. I also believe that what happens to the spirit depends on one thing. That one thing is whether or not, during life on earth, a person has

accepted the free gift of Jesus Christ as their Lord and Savior. If you believe that Jesus Christ suffered and died, taking your sin on Himself, and then rose from the grave, defeating sin, then you will live with God for eternity after your physical death on earth. This is my belief and faith as a Christian, and it is my prayer for everyone reading this.

Foreword

Forensic pathology is a unique profession. As a forensic pathologist, I am privileged to see, on an almost daily basis, the wonders of the human body and the value of life. At the same time, my job affords me the opportunity to see the fragility of life, the ravages of disease, and the unimaginable horrors that are perpetrated on some people by their fellow human beings.

In recent times, forensic pathology has become very popular within the public and various media outlets. To an extent, this popularity has been beneficial to my profession, in that more of society has a basic understanding of what we do. However, many misperceptions and myths persist. Occasionally, as a result of such misperceptions, forensic pathologists are faced with the task of educating the public (such as jurors), as well as other professionals, regarding the fallacies of such myths. My hope is that this textbook will help to correct these misperceptions. At the same time, it is my hope that this text will help to educate its readers about a truly unique profession.

There are several outstanding, up-to-date forensic pathology textbooks in existence today. For the most part, these texts are written for a specific audience, namely pathologists, or more specifically forensic pathologists. Although such texts may be suitable for many of the professionals with whom forensic pathologists interact, none of these fine textbooks has been written specifically for the non-forensic pathologist. When I was asked to write this text, it was for the purpose of writing specifically for the non-forensic pathologist professionals who frequently or occasionally interact with forensic pathologists. As such, the reader will note that several of the early chapters in the book address issues that are not ordinarily covered in the typical forensic pathology textbook, such as general overviews of medicine and pathology, as well as anatomy and physiology.

The overall goal of this textbook is to provide police officers, death investigators, attorneys, and other forensic scientists with a working knowledge of forensic pathology, in order to better enable these individuals to interact with forensic pathologists during their daily duties. Because forensic pathology is an incredibly visual discipline, it is necessary to include numerous photographs in this text. I am a firm believer that providing more photographs is better than providing fewer photographs . . . and color photos are better than black and white. As such, this entire book-writing endeavor has created somewhat of a challenge: how can we provide enough

quality photographs in a format that is affordable to the intended audience? My hope is that we have succeeded in this endeavor. We have attempted to include within the textbook itself the minimum number of black and white photographs necessary to provide a basic understanding of the concepts presented, but we have also included a computer disc containing color versions of each textbook image. In addition, the computer disc contains numerous extra color images of additional topics/issues, with references to these extra images within the textbook itself.

The photos in this text are largely taken from autopsy cases that I performed; however, there are numerous photos from cases performed by my colleagues in Indiana, North Carolina and Texas. In most of these instances, I actually saw the cases with my own eyes, as others performed the autopsies. Occasional images are from cases that were performed by fellow forensic pathologists, but were not personally witnessed by me. I have attempted to identify these latter cases throughout the text by giving appropriate recognition to the pathologist or office from which the photographs originated. Every attempt has been made to protect the identity of the individuals shown in the photographs, while attempting to illustrate a particular point of forensic interest.

An attempt has been made to provide as much information as possible within this book, recognizing that the text is meant to be an overview of forensic pathology, and not an all-encompassing review of each topic. Each chapter provides numerous references for additional reading, and an appendix at the end of the book provides a listing of additional resources. My hope is that this text fills a need for a forensic pathology text specifically geared toward those professionals who work alongside or interact with forensic pathologists.

Acknowledgements

I am indebted to many individuals and institutions for enabling me to produce this book. I extend my gratitude to my editor, Harvey Kane, and my publisher, Springer, for their patience, encouragement, and professionalism during the entire process.

Very importantly, I acknowledge and respectfully express my heartfelt thanks to my patients, for without them I would not be able to perform my job. Death is often a very tragic event, but I believe that it is possible to learn something from every death. To my patients, I offer my heartfelt thanks, and I offer my sincere condolences to the families and loved ones of those for whom I am privileged to work.

I am also indebted to the various institutions with which I have been associated throughout my education and professional career, including the following, which were each an important part of my formal education: Valparaiso University, Indiana University School of Medicine, including Indiana University School of Medicine – Northwest, the Department of Pathology of Wake Forest University School of Medicine, North Carolina Baptist Hospital, the Dallas County Medical Examiners Office, the Southwestern Institute of Forensic Sciences, and the University of Texas – Southwestern Medical Center. I am grateful to my current employer, associations, and affiliations, each of which have been supportive of me in my work: the South Bend Medical Foundation, Indiana University School of Medicine – South Bend at the University of Notre Dame, Elkhart General Hospital, Memorial Hospital of South Bend, and St. Joseph Regional Medical Center.

I would like to extend a special “thanks” to the administrative support I have had over the years, including Genoal, Melinda, Connie, Judy, Liz, Stacey, Diane, Melissa, Ann, Jim, Bob, Ann, Diane, Annette, Heather, Peggy, Sandi, and Diane and her crew. I also say “thanks” to my current partners at the South Bend Medical Foundation: Nicole, Fred, Al, Blair, Frank, Jim, Amobi, Derrick, Luis, Nita, Rick, Kristen, Dave, Bill, Kurtis, John, Odeta, Joyce, Bobbie, and Bob, as well as my colleagues at the medical school, including Rudy, Jack, Suzie, Carl, Ed, Ken, Molly, Rob, Tracy, Mike, Gary, David, George, Faye, Diane, and Mark.

I am privileged to have had the opportunity to work with and learn from many outstanding police officers, death investigators and coroners, attorneys, and forensic scientists over the years. In particular, I would like to thank those with whom I continue to work, including toxicologist Prentiss Jones, the coroners and deputy coroners of St. Joseph, Elkhart, Fulton, Pulaski, LaPorte, Marshall, and Porter

Counties, as well as police officers from South Bend, Mishawaka, Elkhart, Goshen, LaPorte, Michigan City, Valparaiso, Portage, St. Joseph County, Elkhart County, LaPorte County, Marshall County, Porter County, the Indiana State Police, and the Metro-Homicide Unit of South Bend/St. Joseph County. I would like to specifically thank the autopsy assistants with whom I have worked over the years. They perform incredible work for very little pay and very little thanks. I especially would like to thank Eddie, John, and Jim from North Carolina, Hamo, Andrew, Joe, Darren, and Sid from Texas, and Wayne, Andre, Chip, Sherri, Suzie, Jared, and Whitney in Indiana.

Throughout my career, I have been incredibly blessed by knowing, working with, and learning from many outstanding forensic pathologists. I am indebted to each of them, especially John Pless, Pat Lantz, Gregory J. Davis, Don Jason, Jeffrey Barnard, and Rick Hoover. I am also indebted to the following professional organizations: the American Academy of Forensic Sciences (AAFS), the American Society for Clinical Pathology (ASCP), the College of American Pathologists (CAP), and the National Association of Medical Examiners (NAME). By participating in various educational programs, committees, and organizational forums supported by these organizations, I have had the great fortune of meeting and working with some of the best people in medicine, forensic sciences, and forensic pathology. The following is just a sampling of the colleagues with whom I have had the good fortune of working in one capacity or another. Most are forensic pathologists. Sadly, some are no longer with us. Some I know well; others not so well. Some I see often. Others I see very rarely. Some are well-known within the forensic pathology community. Some may be considered “giants” in the field. Others are relatively unknown outside of their particular office. Each is an outstanding professional. Each is considered a friend. I apologize if I have overlooked anyone. T Andrew, M Anzalone, J Arden, A Baker, E Balraj, J Barnard, N Batalis, M Bell, L Biedrzycki, C Boden, J Carter, M Case, J Cavanaugh, S Cina, M Clark, S Clark, J Clouse, S Cohle, K Collins, S Colvin, S Comfort, S Conradi, T Corey, G Dale, J Davis, GJ Davis, GG Davis, J deJong, JS Denton, V DiMaio, G Di Vella, J Dix, M Doberson, D Dolinak, E Donoghue, J Downs, L Dragovic, MF Ernst, M Fierro, L Finelli, S Fiore, C Fligner, M Flomenbaum, D Fowler, R Froede, J Frost, J Gerns, M Gonsoulin, J Goodin, M Graham, M Greenwald, A Gruszecki, J Guileyardo, K Gunson, W Gunther, K Haden-Pinneri, R Hanzlick, A Hart, C Harvey, D Hawley, J Howard, J Hunsaker, D Jason, J Jentzen, P Jones, F Jordan, M Kalelkar, W Kemp, R Kohr, P Lantz, W Lavezzi, B Levy, D Little, J Luke, C Mallak, J McClain, P McFeeley, D McNally, F Miller, C Milroy, M Nashelsky, T Noguchi, K Nolte, J Oeberst, W Oliver, G Peterson, J Pless, JK Pinckard, R Prichard, R Quinton, B Randall, D Reay, R Reichard, K Ross, L Salzberger, L Sathyavagiswaran, G Schmunk, D Schultz, MA Sens, C Siebert, G Simmons, S Spotswood, C Stern, D Stewart, M Super, E Todd, J Townsend-Parchman, S Turner, J Urban, S Wagner, M Ward, V Weedn, C Wetli, D Wolf, R Zumwalt.

Besides being blessed by my teachers and colleagues, I have been blessed by and have learned a great deal from my students throughout my career. They help to

keep me humble. I have a great deal of respect for them, and I enjoy being able to participate in their education.

I would also like to thank my family. Starting with my childhood family, I would like to thank my parents, August and Lois, and my siblings for their love and support throughout my lifetime. Moving on to my own family, I would like to express my love and thanks to my children, Jacob, Samuel, Noah, Joseph, and Mary Anna. I would like to proclaim my most sincere appreciation and deep love for my wife, Tamara. Thanks for your love and support! Finally, I would like to publicly express my thanks for my Lord and Savior, Jesus Christ, for His saving grace, and His loving example of how to live my life.

Contents

Part I Introductory Topics

1	Introduction to Pathology	3
	Disc Image Legends	15
	Selected References	15
2	Introduction to Forensic Sciences	17
	Overview	17
	Chain of Custody	18
	Admissibility of Tests, Evidence and Testimony	18
	Expert Witness	18
	Forensic Science Disciplines	19
	Forensic Pathology	19
	Forensic Anthropology	19
	Forensic Odontology	19
	Forensic Entomology	20
	Forensic Toxicology	22
	Forensic Psychiatry	22
	Trace Evidence	22
	Firearms and Toolmarks Examiners	24
	Document Examination	25
	Fingerprint Evidence	26
	Serology/DNA	27
	Other Disciplines	29
	Disc Image Legends	32
	Selected References	32
3	Introduction to Forensic Pathology	35
	Overview	35
	Duties of the Forensic Pathologist	36
	Investigation	37
	Autopsy	37
	Verification of Identity	40
	Documentation of Findings	40

- Death Certification 41
- Testifying 41
- Consultation 42
- Training and Qualifications of Forensic Pathologists 43
- Disc Image Legends 46
- Selected References 47
- 4 Death Investigation 49**
- Introduction 49
- Death Investigation Systems Within the United States 50
 - Overview 50
 - Jurisdictional Issues 51
 - Coroner Systems 51
 - Medical Examiner Systems 52
 - Mixed Systems 53
- Duties of Death Investigators 53
 - Overview 53
 - Initial Investigation 54
 - Scene Investigation 55
 - Follow-up Investigation 60
- Grief Counseling 61
- Disc Image Legends 61
- Selected References 62
- 5 Death Certification 63**
- Introduction 63
- Death Pronouncement 66
- Cause of Death 68
- Manner of Death 71
- Disc Image Legends 79
- Selected References 79
- 6 Overview of Anatomy and Physiology 81**
- Introduction 81
 - Overview 81
 - Gross Anatomy 82
 - Histology 90
 - Physiology 91
- Body Regions and Compartments 92
- Specific Organ Systems 92
 - Integumentary System (Skin) 92
 - Musculoskeletal System 94
 - Nervous System 97
 - Cardiovascular System 104
 - Respiratory System 106
 - Gastrointestinal System 108

- Hepatobiliary System 111
- Reticuloendothelial System (Including the Immune System) 113
- Endocrine System 116
- Genitourinary System 120
- Special Sensory Structures 122
- Disc Image Legends 124
- Selected References 126

Part II General Topics in Forensic Pathology

- 7 The Postmortem Forensic Examination/Autopsy 129**
 - Introduction 129
 - Forensic Versus Hospital Autopsies 130
 - Investigation 133
 - External Examination 134
 - Internal Examination 137
 - Ancillary Procedures 153
 - Autopsy Report 154
 - External Examination Only (Without Autopsy) 156
 - After-the-Fact and In-Absentia Cases 157
 - Autopsy Assistants 158
 - Office Accreditation and Forensic Autopsy Standards 159
 - Disc Image Legends 159
 - Selected References 161
- 8 Postmortem Changes and Time of Death 163**
 - Introduction 163
 - Early Postmortem Changes 163
 - Livor Mortis 163
 - Rigor Mortis 166
 - Algor Mortis 168
 - Other Early Postmortem Changes 168
 - Decomposition 169
 - Postmortem Injuries 177
 - Time of Death Estimation 179
 - Disc Image Legends 182
 - Selected References 184
- 9 Identification of Human Remains 185**
 - Introduction 185
 - Policies for Identification of Bodies 188
 - Common, Non-scientific Methods of Identification 188
 - Hospital Identification 188
 - Visual Identification 188
 - Scientific Methods of Identification 190
 - Fingerprint Identification 190

- Dental Identification 191
- Radiologic Identification 192
- DNA Identification 194
- Identification Based on Other Unique Features 195
- Circumstantial Identification 198
- Unidentified Remains 198
- Disc Image Legends 200
- Selected References 201

Part III Major Causes/Mechanisms of Death

- 10 Natural Deaths 205**
 - Introduction 205
 - Infectious Disease 207
 - Cardiovascular System 208
 - Congenital Heart Disease 209
 - Congestive Heart Failure (Heart Failure) 210
 - Coronary Artery Atherosclerosis 211
 - Coronary Artery Dissection 214
 - Other Coronary Artery Disorders 214
 - Aortic Aneurysms (Abdominal and Thoracic) 215
 - Cerebrovascular Disease 216
 - Vasculitis 217
 - Fibromuscular Dysplasia 217
 - Myocarditis 217
 - Hypertensive Cardiovascular Disease 218
 - Valve Disorders 219
 - Hypertrophic Cardiomyopathy 220
 - Dilated Cardiomyopathy 220
 - Restrictive Cardiomyopathy 222
 - Deep Venous Thrombosis 222
 - Conduction System Abnormalities 222
 - Neoplastic Heart Disease 223
 - Central Nervous System 223
 - Congenital Anomalies 223
 - Cerebral Palsy 223
 - Infection 224
 - Seizure Disorders 225
 - Dementia 226
 - Cerebrovascular Disease 226
 - Hypertensive Disease 226
 - Spontaneous Intraparenchymal Hemorrhage 227
 - Ruptured Berry Aneurysm 227
 - Ruptured Arteriovenous Malformation (AVM) 227
 - Dural Sinus Thrombosis 228
 - Neoplasia 229

- Respiratory System 229
 - Congenital Anomalies 229
 - Neonatal Conditions 230
 - Upper Airway Conditions 230
 - Pulmonary Thromboembolism 231
 - Bacterial Pneumonia 231
 - Aspiration Pneumonia 232
 - Viral Pneumonia 232
 - Other Infections (Tuberculosis, Fungi, Parasites) 232
 - Asthma 233
 - Chronic Obstructive Pulmonary Disease (COPD) 234
 - Chronic Lung Disease 235
 - Pulmonary Hypertension 235
 - Neoplasia 235
 - Sarcoidosis 235
- Gastrointestinal and Hepatobiliary System 236
 - Congenital Anomalies 236
 - Gastritis 236
 - Peptic Ulcer Disease (PUD) 236
 - Cirrhosis/Esophageal Varices 237
 - Mallory–Weiss Tears 238
 - Other GI Abnormalities 238
 - Hemochromatosis 239
 - Pancreatitis 240
- Reticuloendothelial and Immune Systems 240
 - Autoimmune Disorders 241
 - Leukemia/Lymphoma 241
 - Immunodeficiency 242
- Endocrine System 243
- Genitourinary System 244
- Bones, Joints, and Soft Tissues 245
- Multisystem and Other Disorders 245
 - Amyloidosis 246
 - Chronic Alcoholism 246
 - Diabetes Mellitus 247
 - Sickle Cell Disease 248
 - Inborn Errors of Metabolism 249
 - Other Genetic Disorders 250
 - Sarcoidosis 251
 - Psychiatric Disease 251
- Disc Image Legends 252
- Selected References 254
- 11 Drug-Related and Toxin-Related Deaths 257**
 - Introduction 257
 - Investigation of Drug-Related Deaths 258

- Autopsy Findings in Drug-Related Deaths 261
- Toxicology Issues 263
- Death Certification 267
- Specific Drugs and Toxins 269
- Ethanol and Related Substances 269
 - Ethanol 269
 - Ethylene Glycol 272
 - Methanol 272
 - Isopropanol 273
- Amphetamines and Similar Substances 273
 - Other Stimulants 274
- Barbiturates 275
- Cocaine 275
- Opiates and Related Substances 277
 - Morphine 279
 - Heroin 279
 - Methadone 280
 - Fentanyl 281
 - Other Opiates 281
- Psychoactive Drugs of Abuse (Hallucinogens) 282
 - Marijuana 282
 - Lysergic Acid Diethylamide (LSD) 282
 - Phencyclidine (PCP) 283
 - Mescaline (Peyote) 283
 - Mushrooms (Psilocybin) 283
 - Other Hallucinogens 284
- Over-the-Counter (OTC) Drugs 284
 - Acetaminophen 285
 - Aspirin (Salicylate) 285
 - Ephedrine/Herbal Ecstasy 286
- Other Prescription Drugs 286
 - Antidepressants 286
 - Nonbarbiturate Sedative Hypnotic Drugs 286
 - Antipsychotic Drugs 287
 - Anabolic Steroids 288
 - Insulin 288
- Volatiles and Inhalants 289
 - Nitrous Oxide 291
 - Hydrocarbons 291
 - Helium and Other Simple Asphyxiants 293
- Heavy Metals 294
 - Arsenic 294
 - Cadmium 295
 - Iron 295
 - Lead 295

- Mercury 295
- Other Poisons 296
 - Organophosphates 296
 - Strychnine 296
- Naturally-Occurring Toxins 296
- Disc Image Legends 297
- Selected References 299
- 12 Blunt Force Injury Deaths 301**
 - Introduction 301
 - Classification of Blunt Force Injuries 302
 - Abrasions 302
 - Contusions 304
 - Lacerations 305
 - Fractures 308
 - Avulsions 310
 - Blunt Force Head and Neck Trauma 311
 - Skin and Mucosal Injuries 312
 - Subcutaneous Injuries 312
 - Skull and Facial Bone Injuries 313
 - Epidural, Subdural, and Subarachnoid Hemorrhage 315
 - Gross Brain Injuries 317
 - Microscopic Brain Injuries 320
 - Neck, Spinal Cord, and Vertebral Artery Injuries 321
 - Special Topics Related to Blunt Force Injuries 322
 - Mechanisms of Death in Blunt Force Trauma 322
 - Delayed Deaths Related to Blunt Force Injury 324
 - Patterned Injuries 325
 - Clothing Examination 327
 - Specific Subtypes of Blunt Force Injury 327
 - Disc Image Legends 332
 - Selected References 334
- 13 Gunshot Wound Deaths 337**
 - Introduction 337
 - Types of Weapons and Ammunition 337
 - Gunshot Wounds 343
 - Entrance Wounds 344
 - Range of Fire 346
 - Exit Wounds 351
 - Graze Wounds 351
 - Caliber 352
 - Miscellaneous Features of Handgun Wounds 354
 - Characteristics of High-Velocity Wounds 354
 - Shotgun Wounds 356
 - Miscellaneous Issues 362

- Internal Examination 362
- Mechanism of Injury 366
- Documentation 367
- X-Rays 368
- Clothing Examination 368
- Gunshot Residue 368
- Manner of Death Issues 369
- Special Weapons, Ammunition, and Circumstances 370
- Disc Image Legends 373
- Selected References 376
- 14 Sharp Force Injury Deaths 379**
 - Introduction 379
 - Stab Wounds 381
 - Incised Wounds 385
 - Chop Wounds 388
 - Special Issues 388
 - Internal Examination 388
 - Mechanism of Injury 390
 - Trace Evidence 390
 - Clothing Examination 390
 - Defensive Wounds 391
 - Hesitation Marks 391
 - Self-Inflicted Wounds/Suicide 392
 - “Self-Injurious Behavior” (“Cutting”) 392
 - Accidental Sharp Force Injuries 393
 - Direction of Incised Wound 393
 - Handedness of Attacker 393
 - X-Rays 394
 - Decomposition 395
 - Postmortem/Perimortem Wounds 395
 - Artifacts 397
 - Disc Image Legends 398
 - Selected References 399
- 15 Asphyxial Deaths 401**
 - Introduction 401
 - Suffocation 403
 - Simple Asphyxia (Environmental Asphyxia) 403
 - Smothering (External Airway Obstruction) 405
 - Choking (Internal Airway Obstruction) 406
 - Mechanical Asphyxia (Traumatic Asphyxia) 408
 - Positional Asphyxia 410
 - Combination Forms of Suffocation 410
 - Neck Compression (Strangulation) 411
 - Hanging 412

- Strangulation 415
- Ligature Strangulation 416
- Manual Strangulation 417
- Non-ligature, Non-manual Neck Compression 418
- Chemical Asphyxia 419
 - Carbon Monoxide 419
 - Cyanide 422
 - Hydrogen Sulfide 423
- Other Issues 423
 - Autoerotic Asphyxia 423
 - Choking Game 424
 - Restraint Asphyxia 425
 - Combination Asphyxial Deaths 427
 - Drowning 428
- Disc Image Legends 428
- Selected References 430
- 16 Drowning 433**
 - Introduction 433
 - Physiology and Mechanism of Death in Drowning 434
 - Scene Investigation 435
 - Autopsy Findings 437
 - Causes of Death Other Than Drowning 444
 - SCUBA Deaths 445
 - Manner of Death 446
 - Disc Image Legends 447
 - Selected References 447
- 17 Electrical Deaths 449**
 - Introduction 449
 - Electrocution 451
 - Mechanism of Death in Electrocutions 453
 - Scene Investigation 453
 - Autopsy Findings 455
 - Low Voltage 455
 - High Voltage 457
 - Other Features 459
 - Lightning 461
 - Non-Lethal Electronic Shock Devices 464
 - Death Certification and Manner of Death 465
 - Disc Image Legends 465
 - Selected References 466
- 18 Temperature-Related Deaths 469**
 - Introduction 469
 - Hypothermia 470

- General Features 470
- Scene Investigation 471
- Autopsy Findings 471
- Death Certification 475
- Hyperthermia 475
 - General Features 475
 - Scene Investigation 476
 - Autopsy Findings 477
 - Death Certification 478
- Disc Image Legends 479
- Selected References 479
- 19 Burns and Fire-Related Deaths 481**
 - Introduction 481
 - Burn Types 484
 - Dry Burns 484
 - Radiant Burns 484
 - Scald Burns 485
 - Chemical Burns 487
 - Fire-Related Burns 488
 - Fire Deaths 489
 - Questions to Address 489
 - Autopsy 492
 - Mechanism of Death 496
 - Death Certification 497
 - Fire Investigation 498
 - Cremations 498
 - Disc Image Legends 499
 - Selected References 500
- 20 Deaths in Infancy and Childhood 501**
 - Introduction 501
 - Discarded Fetuses/Infants and Fetal/Infant Deaths
in Unattended Births 502
 - Who is the Mother? 503
 - Was the Fetus/Infant Viable? 503
 - Was the Fetus/Infant Live-Born or Stillborn? 503
 - What Was the Cause of Death? 505
 - Birth-Related Infant Deaths 506
 - Infant Deaths 508
 - Natural Death in Childhood 513
 - Accidental Childhood Deaths 514
 - Suicidal Childhood Deaths 516
 - Homicidal Childhood Deaths 516
 - Classification of Childhood Homicides 526
 - Violent Outburst (Angry Impulse) 527

- Negligence/Neglect 527
- Sadistic Acts of Punishment 528
- Munchausen’s Syndrome by Proxy 529
- Unwanted Child 529
- Unwanted Pregnancy (Neonaticide) 529
- Spouse Revenge 530
- Postpartum Mental Disorder 530
- Acute Psychosis 530
- Altruism 530
- Euthanasia (True Mercy Killing) 531
- Sexual Abuse 531
- Violent Older Child 532
- Drug and Alcohol Abuse 532
- Seizure Disorder 532
- Innocent Bystander 532
- Pediatric Autopsy Considerations 533
- Disc Image Legends 535
- Selected References 537

21 Miscellaneous Topics 539

- Introduction 539
- Aircraft Crashes 540
- Allergic Reactions (Anaphylaxis) 542
- Animal Attacks 544
- Artifacts and Mimics 544
- Emboli 546
 - Pulmonary Thromboemboli 547
 - Systemic Thromboemboli 548
 - Bone Marrow and Fat Emboli 548
 - Amniotic Fluid Emboli 548
 - Gas/Air Emboli 549
 - Bullet and Other Foreign Body Emboli 550
- Exhumations 550
- Explosions and Blast Injuries 554
- High-Profile Cases 554
- Homicide by Heart Attack 555
- In-Custody Deaths 556
- Mass Fatality Incidents 560
- Multiple Causes of Death 561
- Nutrition and Hydration Disorders 562
 - Obesity 563
 - Malnutrition and Starvation 563
 - Vitamin Deficiencies 564
 - Anorexia 564
 - Bulimia 565

- Dehydration 565
- Overhydration 566
- Food Poisoning 566
- Occupational Deaths 567
- Organ and Tissue Procurement Issues 568
- Postmortem Chemistry Tests 572
- Postmortem Cultures 574
- Pregnancy-Related Maternal Deaths 575
- Product-Related Deaths 578
- Radiation 578
 - Ionizing Radiation 578
 - Non-ionizing Radiation 580
- Sexual Assault 580
- Terrorist Agents 581
 - Biologic Agents 581
 - Chemical Agents 582
- Therapy-Related Deaths 583
- Disc Image Legends 586
- Selected References 588
 - Aircraft Crashes 588
 - Allergic Reactions (Anaphylaxis) 588
 - Animal Attacks 588
 - Emboli 588
 - Exhumation 589
 - Explosions and Blast Injuries 589
 - High-Profile Cases 589
 - Homicide by Heart Attack 589
 - In-Custody Deaths 589
 - Mass Fatality Incidents 589
 - Nutrition and Hydration Disorders 590
 - Occupational Deaths 590
 - Organ and Tissue Procurement Issues 590
 - Postmortem Chemistry Tests 590
 - Postmortem Cultures 590
 - Pregnancy-Related Maternal Deaths 590
 - Product-Related Deaths 591
 - Radiation 591
 - Terrorist Agents 591
 - Therapy-Related Deaths 591
- Appendix: Additional Resources and Reference Books 593**
- Index 595**

About the Author

Joseph A. Prahlow, M.D. is from Valparaiso, Indiana. He attended college at Valparaiso University, where he received a Bachelor of Science in Biology and Chemistry. He attended medical school at Indiana University School of Medicine, where he received an M.D. degree. His Internship and Residency took place within the Department of Pathology of Wake Forest University and the North Carolina Baptist Hospital, where he completed Anatomic Pathology and Clinical Pathology training. His formal education was completed in Dallas, Texas, where he completed a fellowship in Forensic Pathology at the University of Texas – Southwestern Medical Center and the Dallas County Medical Examiners Office/Southwestern Institute of Forensic Sciences. He is Board-certified by the American Board of Pathology in Anatomic, Clinical, and Forensic Pathology.

Dr. Prahlow currently practices forensic pathology in South Bend, Indiana, where he is employed by the South Bend Medical Foundation, a large, not-for-profit regional laboratory that provides pathology, laboratory, blood bank, and forensic services to many communities within Indiana, Michigan, and other Midwestern states. Dr. Prahlow spends much of his time performing autopsies for local coroners. Another major part of his job involves teaching the entire basic science pathology course to second-year medical students at Indiana University School of Medicine – South Bend at the University of Notre Dame, where he is a professor of pathology. Dr. Prahlow enjoys presenting at various local, national, and international meetings, and he has published extensively within the medical/scientific literature.

Dr. Prahlow recently completed service to the National Association of Medical Examiners as Vice President, President, and Chairman of the Board. He has a similar history of service to the American Academy of Forensic Sciences, where he has served as the Pathology/Biology Section's Program Chair, Secretary, and Section Chair. He continues to serve both organizations as a member of various committees. He is an active participant in two Pathology organizations as well, the American Society for Clinical Pathology and the College of American Pathologists. In both, his primary role involves participation in various educational activities specifically related to forensic pathology.

Dr. Prahlow is married to his high-school sweetheart, Tamara. He and his beautiful wife reside in Southwestern Michigan, where they raise their five children.

Part I
Introductory Topics

Chapter 1

Introduction to Pathology

They will die of deadly diseases.
Jeremiah 16:4

Abstract This chapter provides a basic description of “pathology,” which literally means the “study of disease.” Pathology is unique in that the discipline represents one of the “basic sciences” taught in medical school, but it is also considered a medical specialty, a career path chosen by certain physicians. The reader is provided with a description of classic medical education within the United States. Much of the chapter deals with describing the specialty of pathology, including training requirements, the two major branches of pathology (anatomic and clinical pathology), and various subspecialty areas.

Keywords Pathology · Medicine · Medical school · Anatomic pathology · Clinical pathology

“Pathology” literally means “the study of disease” and is studied by all physicians as part of their medical school curriculum. It is considered one of medicine’s “basic sciences” (along with gross anatomy, microscopic anatomy (“histology”), physiology, biochemistry, pharmacology, etc.) and is usually studied during the second year of a student’s four years of medical school training. The typical medical school curriculum is shown in Table 1.1.

After graduating from medical school, a physician typically participates in additional “postgraduate” training, encompassing the internship and residency years. Physicians choose the type of medical specialty that they wish to pursue as a career and participate in a residency program for that particular specialty. Although pathology is an essential “basic science” (as described above) within the traditional medical school curriculum, it is also a medical specialty (like pediatrics, radiology, internal medicine, surgery, cardiology, etc.). In other words, a medical student can choose to become a pathologist, just as another medical student chooses to become an anesthesiologist, an endocrinologist or an obstetrician. Table 1.2 shows a list of various medical specialties available to physicians. A specialty can be considered one of three general types: (1) those that are “medical” (meaning “non-surgical”)

Table 1.1 An example of a traditional medical school curriculum (courses may vary depending on the medical school)

Basic sciences (first two years)	
1st year –	Gross anatomy Histology (microscopic anatomy) Physiology Biochemistry Microbiology Neuroanatomy Behavioral science
2nd year –	Pathology Pharmacology Introduction to medicine Genetics Physical diagnosis
Clinical sciences (last two years)	
3rd year –	Internal medicine General surgery Surgical subspecialties Obstetrics and gynecology Pediatrics Psychiatry Anesthesiology Neurology/neurosurgery Primary care
4th year –	Radiology Electives

(Disc Image 1.1); (2) those that are “surgical” (Disc Image 1.2); and (3) those that are “miscellaneous.” Pathology fits into the “miscellaneous” category; it is relatively unique within the world of medicine in that it represents a basic science but also a distinct medical specialty.

In the United States, there are two types of medical degrees, the MD (doctor of medicine) and the DO (doctor of osteopathy). In order to graduate from medical school, and then continue in formal postgraduate medical training (residency) and become licensed to practice medicine, there are certain “board examinations” which a medical student must take and pass. For MD training, the examinations, referred to as the United States Medical Licensing Examination (USMLE), encompass three steps, two of which are usually taken during medical school, and a third which is taken after at least one year of postgraduate training. The examinations are administered by the National Board of Medical Examiners (NBME). A similar set of examinations is administered to DO students/physicians by the National Board of Osteopathic Medical Examiners (NBOME). The use of the term “medical examiner” in the context of the NBME and the NBOME is totally different from the use of the term as it applies to forensic pathology and death investigation.

Table 1.2 Medical specialties (incomplete listing)

Specialty	Deals with . . .
“Medical specialties”	
Internal medicine	General medicine
Cardiology	Heart
Pulmonology	Lungs
Gastroenterology	Liver, pancreas, stomach, intestines
Endocrinology	Glands (pituitary, thyroid, etc.)
Hematology	Blood and bone marrow
Oncology	Cancer
Nephrology	Kidneys
Rheumatology	Joints and muscles
Family practice	General medicine
Emergency medicine	Emergencies and urgent care
Hospitalist	Hospitalized patients
Pediatrics	Children
Dermatology	Skin
Sports medicine	Sports injuries
Physical medicine and rehabilitation	Rehabilitation
Neurology	Nervous system
“Surgical specialties”	
General surgery	Surgical disorders
Cardiothoracic	Heart, lungs, and chest
Trauma	Trauma
Transplant	Transplant
Vascular	Blood vessels
Plastic	Cosmetic
Pediatric	Children
Orthopedic	Bones, joints, and muscles
Urology	Urinary and genital
Obstetrics and gynecology	Females
Ophthalmology	Eyes
Otorhinolaryngology	Ears, nose throat
Neurosurgery	Brain and spinal cord
“Miscellaneous specialties”	
Anesthesiology	Anesthesia and/or pain management
Interventional radiology	Non-surgical therapies
Occupational medicine	Workplace health
Pain management	Pain
Pathology	Laboratory and diagnosis
Preventive medicine	Disease/injury prevention
Psychiatry	Behavioral and psychological disorders
Radiation oncology	Radiation treatment of cancer
Radiology	Imaging

To become a pathologist, a student must first receive a medical degree (MD or DO) from a medical school. Following medical school, the physician must then successfully complete a residency in pathology. Currently, the typical pathology

residency training program lasts four years and involves two basic branches of pathology: clinical pathology and anatomic pathology.

Clinical pathology can be considered “laboratory medicine.” The various hospital and medical laboratories are under the direction of pathologists, while the bulk of the work is performed by laboratory technologists and technicians. Typical laboratories include the chemistry laboratory (where items such as electrolytes, cholesterol, and other substances are measured in the blood or other body fluids, such as urine) (Fig. 1.1), the microbiology laboratory (where various tests are performed in order to identify infectious organisms, such as bacteria, fungi, and viruses), the hematology laboratory (where “blood counts” are performed, determining various issues related to red and white blood cells and other blood factors) (Fig. 1.2), and the blood bank (where blood typing and matching allows for safe blood transfusions to occur). An emerging laboratory field involves molecular genetics, where identification of genes (DNA) and their products plays a role in disease diagnosis, prognosis (the expected outcome), and therapy. As directors of the clinical pathology laboratories, pathologists play a major role in attending to the medical needs of physicians and their patients.



Fig. 1.1 An automated chemistry machine in the clinical pathology laboratory

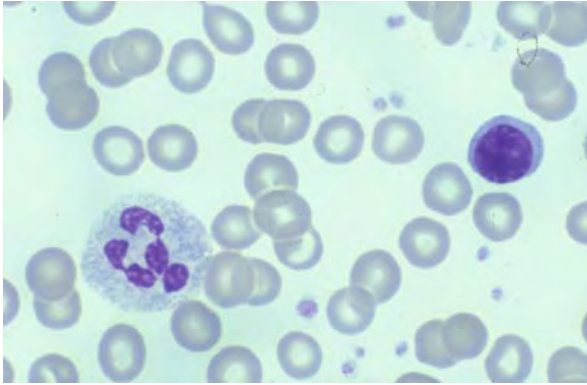


Fig. 1.2 A blood smear, as seen via a microscope. The *round cells* with no nuclei are *red blood cells*. The slightly larger cells with nuclei are *white blood cells*, including a neutrophil on the left and a lymphocyte on the right. The small structures are platelets

Anatomic pathology is the second of the two major branches within the medical specialty of pathology. The term “anatomic” refers specifically to the structure (or “morphology”) of tissues and organs. Chapter 6 provides an overview of normal anatomy. Pathologists, in their role as anatomic pathologists, deal with recognizing and diagnosing abnormal, or diseased anatomy. A tissue or organ that is afflicted with a disease undergoes various changes, such that its gross (naked-eye) appearance, microscopic (histologic) appearance, and function are altered. Pathologists rely on the gross and microscopic appearance of diseased tissues, as well as various other laboratory tests, to render diagnoses. There are three basic components within anatomic pathology: surgical pathology, cytology (also called cytopathology), and autopsy pathology.

Surgical pathology involves the gross and microscopic evaluation of tissues removed by surgical means. Whenever a person has a surgical biopsy performed, or an organ removed via surgery, the removed tissue is sent to the surgical pathology laboratory, where, after the specimen has become “fixed” in formalin (a preservative solution containing formaldehyde), pathologists or their assistants dissect the specimen (Fig. 1.3 and Disc Images 1.3 and 1.4) and submit samples of the tissue (Fig. 1.4) to the “histology laboratory.” Here the samples are “processed” by histotechnologists via a procedure utilizing various chemicals (Fig. 1.5), such that the samples end up embedded in paraffin wax (Figs. 1.6 and 1.7). The histotechnologists then cut extremely thin sections of the tissues (Fig. 1.8), place the sections on glass slides (Fig. 1.9), and stain them for viewing under the microscope. The usual stain that is used for histologic examination is hematoxylin and eosin (referred to as an H&E stain) although there are numerous other “special stains” that can be utilized. The pathologist views the glass slides via the microscope (Fig. 1.10) and renders diagnoses, based on the microscopic anatomy that is seen (Figs. 1.11, 1.12, 1.13, 1.14 and Disc Image 1.5). The microscope allows pathologists to visualize the tissues and cells at various magnification powers. By evaluating tissue specimens



Fig. 1.3 Gross examination and dissection of a surgical pathology specimen (a portion of lung)



Fig. 1.4 Samples of tissue placed into plastic “cassettes” for processing



Fig. 1.5 Processing machines in the histology laboratory



Fig. 1.6 A histotechnologist embedding a tissue section in paraffin wax

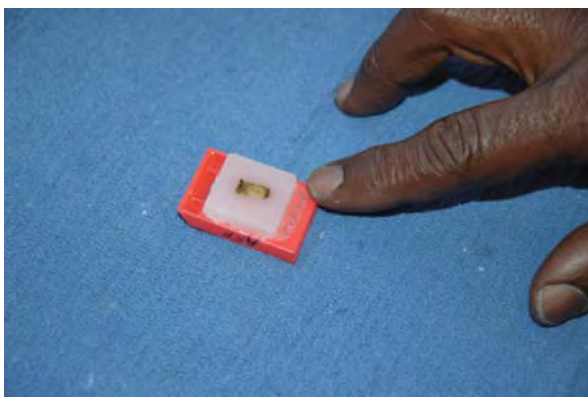


Fig. 1.7 A tissue section embedded in paraffin, ready to be sectioned (cut into extremely thin sections)



Fig. 1.8 A histotechnologist using a microtome to cut a section of tissue from the paraffin tissue block

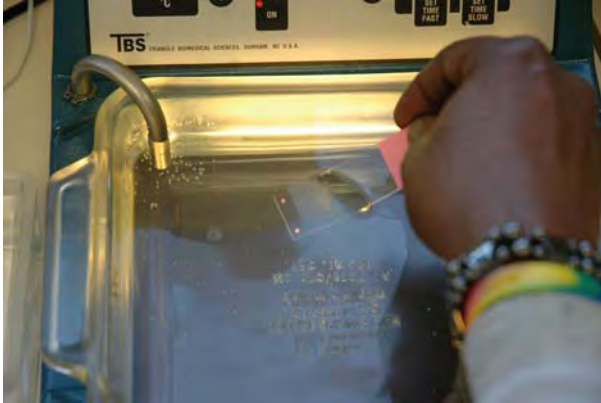


Fig. 1.9 A histotechnologist placing the tissue section onto a glass slide



Fig. 1.10 A pathologist viewing a slide via a microscope

grossly and under the microscope, surgical pathologists answer very important questions regarding patients' conditions: Is it an infection? Is it a tumor? Is it benign (non-cancerous) or is it malignant (cancer)? What kind of cancer is it? Is it low-grade (slow-growing) or high-grade (rapidly-growing)? Did the surgeon remove all of the cancer? Are the lymph nodes involved? Most pathologists spend much of their time performing their surgical pathology duties.

Cytology, or cytopathology, is similar to surgical pathology in that pathologists look at glass slides under the microscope in order to make diagnoses and answer questions similar to those listed above. In contrast to histology slides, where tissues have been processed, embedded in paraffin wax, and cut into thin sections, cytology



Fig. 1.11 An example of the appearance of a histology slide (normal fallopian tube) viewed under the microscope at low-power magnification (approximately 20×)

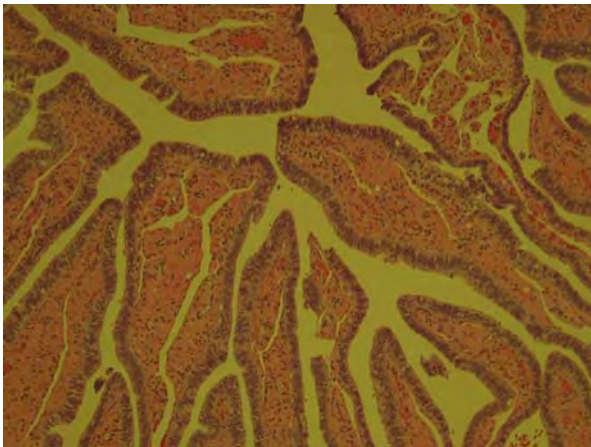


Fig. 1.12 A fallopian tube viewed under the microscope at medium-power magnification (40×)

slides are composed of cells that have been scraped off of a tissue’s surface or aspirated (sucked) out of a solid or fluid-filled tissue using a needle, and then stained (Fig. 1.15).

Autopsy is the third component of anatomic pathology. The autopsy can be considered a surgical procedure that is performed on a dead body and which involves cutting into the body, removing the organs, and dissecting the organs, with or without subsequent microscopic examination of tissue/organ sections. There are two basic types of autopsies: hospital (medical) autopsies and forensic (medicolegal)

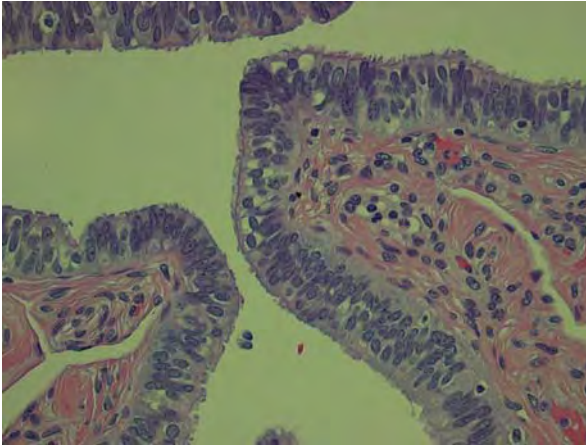


Fig. 1.13 A fallopian tube viewed under the microscope at high-power magnification (400 \times)

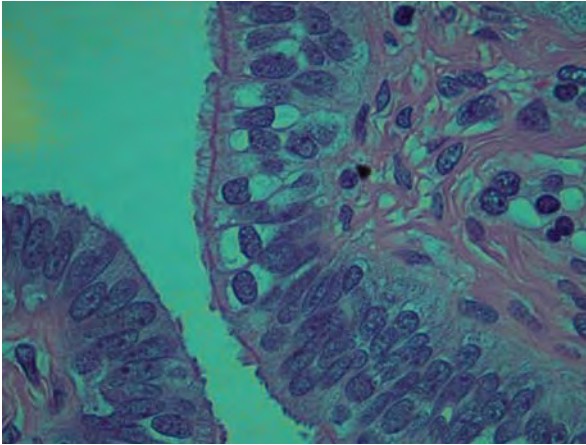


Fig. 1.14 A fallopian tube viewed under the microscope at very high-power magnification (1000 \times)

autopsies. A detailed description of the two types of autopsies is provided in Chapter 7; however, a short description is provided here.

Hospital (medical, non-forensic) autopsies are performed on persons who have died of natural diseases, typically in the hospital. Compared to several decades ago, the number of hospital autopsies that are performed annually is extremely low. As such, most hospital-based pathologists do not perform many autopsies, unless they happen to perform medicolegal autopsies (but this is the exception, rather than the rule). In fact, many pathologists do not particularly enjoy performing autopsies.

Medicolegal (forensic, medical examiner, or coroner) autopsies are performed on individuals whose deaths meet the legal requirements for being a death that is

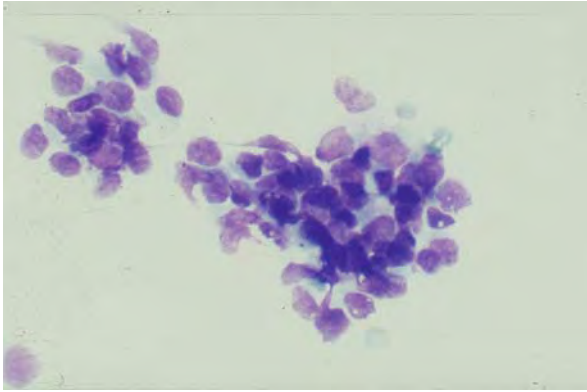


Fig. 1.15 An example of the appearance of a cytology slide (breast cancer) viewed under the microscope

to be officially investigated by the local governmental death investigation agency (medical examiner or coroner – refer to Chapter 4). Typically, this includes any death that is caused by something that is not considered a natural disease, as well as any death that is sudden, unexpected, or suspicious. Occasionally, medicolegal autopsies are performed by hospital-based pathologists who have received limited, little or no formal training in forensic pathology. In other jurisdictions, these types of cases are performed by forensic pathologists, who have special expertise in the investigation of sudden, unexpected, and violent deaths (see Chapter 3).

Most pathologists receive structured pathology residency training in both anatomic and clinical pathology (Disc Image 1.6), but occasional pathologists only participate in one area or the other. Whatever the case, to become “Board-certified” in pathology, a physician must first successfully complete a pathology residency at an institution that is appropriately accredited by the Accreditation Council for Graduate Medical Education (ACGME), and then pass a Board examination administered by the American Board of Pathology (ABP). Certificates are available for both anatomic pathology and clinical pathology. Pathologists are sometimes referred to as the “physicians’ physician,” a physician to which other physicians come to find answers to diagnostic problems. As such, an important aspect of pathology is the participation in educational conferences with other physicians (Disc Image 1.7).

In each of the two basic areas of pathology (anatomic and clinical), a pathologist can also participate in additional formal training within a “fellowship” program, usually after completing residency, to gain further expertise within any of a number of “subspecialties.” Table 1.3 shows the various basic components of anatomic and clinical pathology disciplines and Table 1.4 lists the Board-certifiable “subspecialties” available within each discipline. Fellowships are available in various other pathology subspecialty areas, such as surgical pathology, without an official subspecialty Board certification available. Such fellowships are used to gain further experience and training in a particular discipline. Following successful completion of the fellowship training and, if applicable, the passing of a subspecialty Board

Table 1.3 Pathology disciplines

Clinical pathology	Anatomic pathology
Chemistry	Surgical pathology
Hematology	Cytology
Microbiology	Autopsy pathology
Blood banking	
Molecular	

Table 1.4 Board-certified subspecialties within pathology

Clinical pathology	Anatomic pathology
Chemical pathology	Cytopathology
Hematology	Forensic pathology
Medical microbiology	Pediatric pathology
Blood banking/transfusion medicine	Dermatopathology
Molecular genetic pathology	Neuropathology

examination administered by the ABP, the pathologist is then Board-certified within that specific pathology subspecialty.

The certifying agencies for MD and DO degrees are the NBME and NBOME, respectively, while the certifying body for pathology and its subspecialty areas is the ABP. The reader should be aware that there are various international degrees that are considered analogous to the MD and DO medical degrees. Likewise, there are international credentials that are similar to the certifications in pathology granted by the American Board of Pathology. Some international degrees and credentials may be considered equivalent to those obtained in the United States; however, this is not always the case. The reader should also be aware that the term “Board certification,” as it applies to pathology and forensic pathology, should only be accepted when such certification is conferred by the American Board of Pathology. Further discussion of this issue will be presented in Chapter 3.

As with every medical specialty, there are numerous organizations that exist to promote the pathology specialty, offer continuing medical education opportunities, and act as an advocate agency for pathologists. Some of the larger organizations are the College of American Pathologists (CAP), the American Society for Clinical Pathology (ASCP), the United States and Canadian Academy of Pathology (USCAP), and the International Academy of Pathology (IAP). Subspecialties within pathology also have organizations geared toward their particular subspecialty. The leading forensic pathology and death investigation organization is the National Association of Medical Examiners (NAME). A larger organization which encompasses all forensic science disciplines, not just forensic pathology, is the American Academy of Forensic Sciences (AAFS).

Disc Image Legends

- Disc Image 1.1 “Medical” specialties include family practice, internal medicine, pediatrics, and other non-surgical specialties (Photo courtesy of Department of Pathology, Wake Forest University and North Carolina Baptist Hospital, Winston-Salem, NC).
- Disc Image 1.2 “Surgical” specialties include any specialty that involves the performance of surgery (Photo courtesy of Department of Pathology, Wake Forest University and North Carolina Baptist Hospital, Winston-Salem, NC).
- Disc Image 1.3 A pathologist dissecting a portion of colon removed for cancer.
- Disc Image 1.4 A portion of colon with cancer (note the mass that protrudes above the level of the surrounding tissue).
- Disc Image 1.5 A medium-power microscopic section showing normal colon (*left*) adjacent to colon cancer (*right*).
- Disc Image 1.6 Pathology residents using a multi-headed microscope during their training (Photo courtesy of Department of Pathology, Wake Forest University and North Carolina Baptist Hospital, Winston-Salem, NC).
- Disc Image 1.7 A pathologist presenting at an educational conference (Photo courtesy of Department of Pathology, Wake Forest University and North Carolina Baptist Hospital, Winston-Salem, NC).

Selected References

- The Accreditation Council for Graduate Medical Education. www.acgme.org
- The American Academy of Forensic Sciences. www.aafs.org
- The American Board of Pathology. www.abpath.org
- The American Society for Clinical Pathology. www.ascp.org
- The College of American Pathologists. www.cap.org
- The International Academy of Pathology. www.iaphomepage.org
- The National Association of Medical Examiners. www.thename.org
- The National Board of Medical Examiners. www.nbme.org
- The National Board of Osteopathic Medical Examiners. www.nbome.org
- Prahlow JA, Vogel DG. Pathology: Myths and Truths. *The New Physician*. 1994; September: 51–52.
- The United States and Canadian Academy of Pathology. www.uscap.org

Chapter 2

Introduction to Forensic Sciences

Hate evil, love good; maintain justice in the courts.
Amos 5:15

Abstract While most of the book deals with forensic pathology, this chapter provides readers with an overview of other forensic science disciplines. After a brief description of the chain of custody, test admissibility, evidence and testimony, the chapter provides brief descriptions of a variety of forensic science careers, including forensic anthropology, toxicology, forensic entomology, firearms examination, and DNA testing.

Keywords Forensic science · Evidence · Testimony

Overview

According to one definition, the word “forensic” means the application of scientific knowledge to legal problems. The term “forensic science” refers to a group of scientific disciplines which are concerned with the application of their particular scientific area of expertise to law enforcement, criminal, civil, legal, and judicial matters. It is beyond the scope of this book to provide detailed descriptions of the various forensic science disciplines so the reader is therefore referred to the many excellent textbooks and reviews of the forensic sciences for more detailed descriptions. This chapter provides only a very broad overview of the disciplines, with an emphasis on how forensic pathologists might interact with each. Where possible, a specific organization within each discipline, that is the recognized certifying agency for that profession is provided. Readers should beware that there are other organizations and/or “boards” that present themselves as legitimate. Such entities may or may not be reputable.

Chain of Custody

Before providing a short description of the forensic disciplines, it is necessary to discuss three concepts that are important in all forensic sciences. The first involves maintaining the proper “chain of custody” when dealing with evidence. Evidence of whatever type must be carefully and properly documented and evaluated. Because of the nature of certain types of evidence it cannot all be collected and preserved indefinitely. An example is a human corpse that is evaluated at autopsy. In such instances, proper documentation is essential in order to re-evaluate the evidence (the body) at a later date. In the case of an autopsy, such documentation is performed via diagrams, photographs, and an autopsy report. There are many other types of evidence that also require collection and preservation; for example, trace evidence such as hairs or fibers discovered at a crime scene. Some forms of evidence are actually consumed or destroyed during evaluation (for example, blood samples being tested for drugs). Maintaining a proper “chain of custody” involves producing and maintaining written documentation which accompanies the evidence and provides an uninterrupted timeline showing the secure location of the evidence from the time that it was discovered until the present time. Any transfer of evidence from one person or secure location to another must be documented. Maintaining this chain of custody helps to ensure that the evidence has not been contaminated or compromised in any way. If the proper “chain of custody” is not maintained, the breaking of the chain may well provide a potential reason for such evidence to be inadmissible in court.

Admissibility of Tests, Evidence and Testimony

The second issue of concern that crosses all fields of forensic science involves the existence of legal standards for the admissibility of forensic tests and expert testimony. One legal standard for the admissibility of a forensic test is *Frye v United States*, which states that the forensic technique in question must have “general acceptance” by the scientific community. Rule 702 of the Federal Rules of Evidence regulates the admissibility of expert testimony in regard to a test or discipline. *Daubert v Merrell Dow Pharmaceutical, Inc* states that the decision about the admissibility of scientific evidence resides with the judge hearing the case.

Expert Witness

The third issue that relates to all forensic science disciplines is the concept of the expert witness. In contrast to a “fact witness,” who is usually only able to relate the facts of the issue at hand as he/she observed them, an “expert witness,” because of his/her specific expertise within a particular discipline, is also able to offer opinions regarding issues that relate to the specific discipline. In order to be recognized as an expert witness, the witness must be officially qualified, or recognized as an expert, by the court. Usually this involves a legal process referred to as *voir dire*,

wherein the credentials, training, experience, etc. are presented to the court via questions/answers between an attorney and the witness. So long as this presentation is acceptable to both sides and the judge, a witness may be qualified to testify as an expert in a particular field.

Forensic Science Disciplines

Forensic Pathology

Forensic pathology represents a subspecialty within the medical specialty of pathology (see discussion of pathology in Chapter 1), dealing specifically with the investigation of sudden, unexpected, and/or violent deaths. The autopsy is central to the practice of forensic pathology. An overview of forensic pathology is provided in Chapter 3.

Forensic Anthropology

Forensic anthropology is a subspecialty within the scientific field of physical anthropology (the study of human beings in relation to their physical character), in which forensic anthropologists examine skeletal remains (bones). Forensic anthropologists attempt to answer questions about bones, including questions regarding species of origin (human versus nonhuman), gender, age, race, stature, nutritional status, existence of disease processes, and the presence and character of skeletal trauma. A forensic pathologist may consult with a forensic anthropologist when attempting to address any of the questions above. A frequent instance of consultation occurs when the forensic pathologist is presented with a badly decomposed or skeletonized corpse that is unidentified (refer to Chapter 9) (Fig. 2.1 and Disc Image 2.1). Board certification in forensic anthropology is conferred by the American Board of Forensic Anthropology.

Forensic Odontology

Forensic odontology is a subspecialty within dentistry in which a dentist has specialized expertise in using dental examination to assist in the identification of human remains, and in the evaluation of bite-marks, wherein a bite-mark on a victim may be “matched” to a suspect (Fig. 2.2 and Disc Image 2.2). The majority of a forensic odontologist’s involvement in forensic casework involves assisting forensic pathologists in the identification of bodies. Assistance is usually provided in cases where the body is not visibly identifiable, and identification by fingerprint comparison or other means is not possible. The most common types of cases are persons who are badly burned and those who are badly decomposed (Fig. 2.3). Chapter 9 provides

Fig. 2.1 Unidentified skull at autopsy. Consultation with a forensic anthropologist can aid in determination of gender, race, and approximate age and stature



Fig. 2.2 A bite-mark on the skin surface at autopsy

more details about dental identification. Board certification in forensic odontology is conferred by the American Board of Forensic Odontology.

Forensic Entomology

Forensic entomology is a subspecialty within the biological science discipline of entomology (the study of insects) that primarily deals with insect succession patterns in decomposing human bodies. Evaluation of insects (including larval stages,



Fig. 2.3 Postmortem examination of the teeth of a badly burned body in order to assist in the positive identification of the decedent. Note that the soft tissues of the face (lips and cheeks) have been cut away in order to maximize the visibility of the teeth

or maggots) found on decomposing bodies can permit scientific estimation of the time of death (Fig. 2.4). In certain circumstances, information regarding the location of death may also be ascertained. Forensic pathologists do not consult with forensic entomologists in all decomposed cases, but will consult with them on select cases where estimating the time of death may be very important (for example, in homicides with decomposition and insect activity). Chapter 8 provides more details on how forensic entomologists may estimate the time of death. Board certification in forensic entomology is under the direction of the American Board of Forensic Entomology.



Fig. 2.4 Fly maggots on a decomposing body

Forensic Toxicology

Forensic toxicology is a discipline that involves the identification and quantification of drugs and other poisons or toxins in body tissues, including blood. “Screening tests” are said to be “qualitative,” where a test is either positive (indicating that the drug/toxin is present) or negative (indicating that the drug/toxin is not present). When specific levels of drugs or toxins are determined, the tests are said to be “quantitative.” For a result to have forensic significance, two separate methodologies are required, an initial (screening) test, and a confirmatory (quantitative) test. Another function of some toxicology laboratories is drug identification. For example, if a bag of white powdery substance is found in the pocket of a dead person, the substance can be submitted to the laboratory for identification. Forensic pathologists rely a great deal on the forensic toxicology laboratory. In many jurisdictions, toxicology testing is performed on a majority of the autopsy cases. In a significant percentage of forensic autopsy cases, the cause of death is related to the toxicology results. Forensic toxicology laboratories should be appropriately accredited by an officially recognized agency, such as the American Board of Forensic Toxicology. Toxicologists may have varying levels of education. Board certification in forensic toxicology is conferred by the American Board of Forensic Toxicology.

Forensic Psychiatry

Forensic psychiatry represents a discipline dealing with the evaluation of the mental state of criminals. Occasionally, forensic pathologists will interact with forensic psychiatrists and police investigators to form a “psychiatric profile” of a suspect in a particular murder or series of murders. A “psychological autopsy” is sometimes necessary when attempting to determine the state of mind of a suicide victim. Board certification in forensic psychiatry is available via the American Board of Psychiatry and Neurology.

Trace Evidence

Trace evidence is a general term used to describe various relatively small pieces of evidence that can be evaluated scientifically. Such evidence can include such items as hair (Figs. 2.5 and 2.6), fibers, paint chips, glass fragments, soil, gunshot residue (primer components, gunpowder, etc.), accelerants, and explosives. With certain types of evidence, the mere identification of the trace evidence may have significance in a particular case, for example, the identification of gunpowder on the clothing of a shooting victim. With other types of trace evidence, crime laboratory scientists may be able to “match” or “associate” a particular piece of evidence collected from a crime scene to a source, including the alleged perpetrator

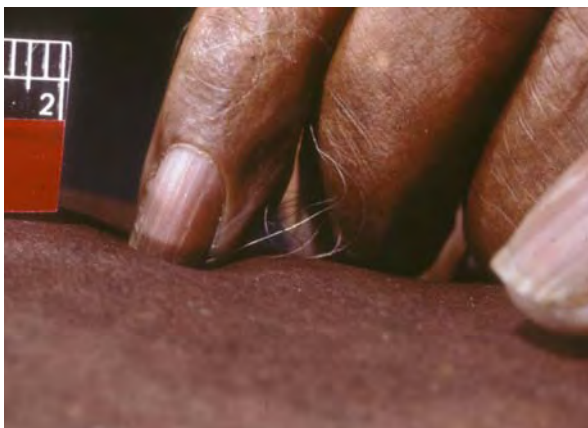


Fig. 2.5 A hair present on the fingers of a homicide victim

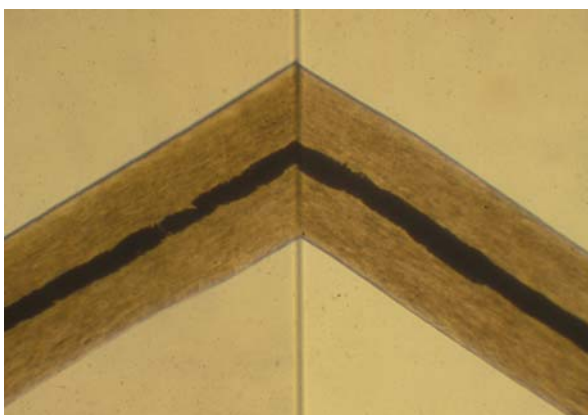


Fig. 2.6 Microscopic comparison of a strand of evidence hair, compared with a hair standard obtained from a suspect

of a crime. For example, a paint chip collected from the clothing of a hit-and-run pedestrian victim may be matched to a specific vehicle. Forensic pathologist interaction with trace evidence analysts is usually via the recognition and collection of pertinent trace evidence on a body by the forensic pathologist with subsequent submission of the evidence to the crime laboratory. Trace evidence evaluations typically occur within the larger setting of a crime laboratory. Crime laboratories must be accredited by the American Society of Crime Laboratory Directors (ASCLD). Trace evidence analysts can be certified via the American Board of Criminalistics (ABC).

Firearms and Toolmarks Examiners

Firearms and toolmarks examiners are forensic scientists with a specific expertise in the evaluation of firearms and ammunition. As part of their job, these scientists are able to match an evidence bullet (such as that collected from a body at autopsy) to a particular suspect weapon. The first step in attempting to determine whether or not a suspect weapon fired an evidence bullet is to determine the “class characteristics” of each. In regard to firearms having a rifled barrel (rifles and most handguns), the class characteristics include the caliber (diameter) of the barrel (and bullet), the number of lands and grooves (alternating raised and lowered areas within the inner surface of the barrel that spiral along the course of the barrel) of the barrel and imprinted on the sides of the bullet, and the direction of the spiraling (the “twist”) within the barrel and imprinted on the sides of the bullet (Fig. 2.7). If an evidence bullet has different class characteristics than a particular suspect weapon, the weapon can be excluded as the one that fired the bullet.

Fig. 2.7 A deformed bullet collected at autopsy. Note the land and groove impression marks on the sides of the bullet



If the class characteristics are the same, then the firearms examiner has to make a more precise comparison. One way of doing this is to have the examiner fire a bullet from the suspect weapon (often into a watertank [Disc Image 2.3]). The examiner then collects the bullet (the exemplar) and compares the exemplar to the evidence bullet, using a “comparison microscope” (Disc Image 2.4). Besides creating marks that correspond to the lands and grooves of the barrel, rifled firearms produce unique, weapon-specific, microscopic marks (“striations”) on the sides of bullets as the bullets travel through the barrel. These marks are created by the physical machine-produced make-up of the inside of the barrel. They are unique to an individual weapon (no two weapons are identical), and they produce essentially

identical microscopic markings on every bullet fired from the same weapon. By comparing these “toolmarks” that are inscribed by the inside of the barrel on the sides of the exemplar bullet to those present on the evidence bullet, an examiner can determine if the evidence bullet was fired from the suspect weapon (Fig. 2.8).

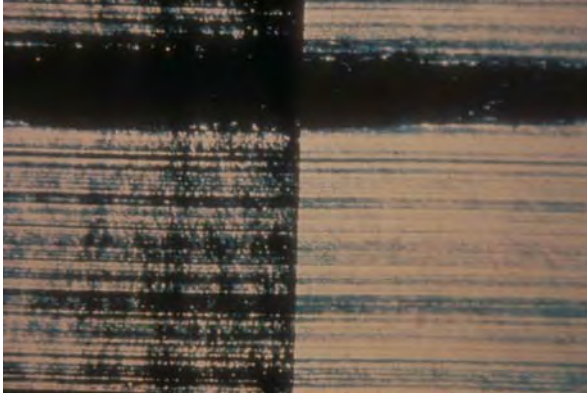


Fig. 2.8 An evidence bullet compared to a standard bullet fired from a suspect weapon. By aligning the striations, an evidence bullet can be matched to a specific weapon

Other toolmark patterns can allow examiners to “match” bullet casings to specific weapons (firing pin impressions, ejector marks, extractor marks, etc.). Marks left by other items, such as knife blades or blunt instruments, may also be specific enough to allow toolmarks examiners to match a suspect weapon to a crime scene or even occasionally to a body (via marks produced in cartilage or bone). Firearms and toolmarks examiners are also able to evaluate firearms regarding functionality, answering questions related to whether or not a firearm functions appropriately, etc. Forensic pathologists frequently collect and submit evidence from bodies for firearms and toolmarks examination, mostly in the form of firearm projectiles recovered during autopsy. The official certifying organization within this forensic discipline is the Association of Firearm and Tool Mark Examiners (AFTE).

Document Examination

Document examiners evaluate handwriting or machine-produced printing (typewriters, computer printer, copiers, etc.) and other documents. As such, these forensic scientists play a very important role in a variety of crimes, including forgeries, fraud cases, and counterfeit operations. Most interaction with forensic pathologists occurs in suicide cases, where document examiners can compare suicide notes with exemplars of the suicide victim’s known handwriting to establish whether or not the victim actually wrote the suicide note (Disc Image 2.5). Document examiners occasionally perform other forensic tests, such as fingerprint analysis, impression

analysis, or voice analysis. Board certification within this forensic discipline is available through the American Board of Forensic Document Examiners (ABFDE).

Fingerprint Evidence

Ever since fingerprints were discovered as a valuable means for identifying people, the discipline has been an important part of police and forensic investigations. Fingerprints represent unique patterns of the ridges on the pads of the fingers (including the thumbs). The ridges occur in the epidermis (the part of the skin closest to the surface) but extend into the dermis (the deeper part of the skin). Barring changes related to scar formation, which can obliterate portions of a fingerprint, fingerprints remain the same throughout an individual's life. The presumption, which has essentially been proven by decades of experience and casework, is that each individual has their own unique set of fingerprints. No two fingerprints have ever been found to be exactly identical even between identical twins. Therefore, a fingerprint represents a specific, individual characteristic of a particular person. Fingerprint examiners rely on various class characteristics (loops, whorls, and arches) as well as individual characteristics ("ridge characteristics" or "minutiae") of fingerprints in their examinations. An evidence fingerprint (such as a "latent" or invisible fingerprint) at a crime scene can be matched to a known print in a database. A variety of methods are used to collect and preserve evidence fingerprints. Several automated fingerprint identification systems (AFIS) are available: these are computerized databases of fingerprints that are on-file within various law enforcement agencies. For forensic pathologists, fingerprint comparison can be extremely useful in identifying an unknown corpse. In many offices, it is a standard operating procedure to create a fingerprint record of all bodies (Fig. 2.9). Certification of fingerprint analysts can be obtained via the International Association for Identification.

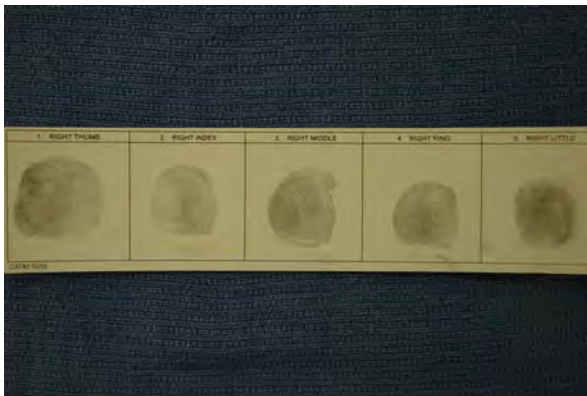


Fig. 2.9 A fingerprint card collected from a body in the morgue

Serology/DNA

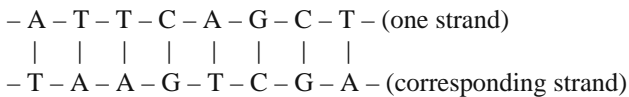
Blood and other bodily fluids can be transferred from one person to another, or identified at crime scenes. Forensic serologists can perform tests to determine if a suspicious fluid or stain is saliva, semen or blood. Tests are available to determine if the evidence, such as blood, is of human origin. Once it is identified as human, then DNA (deoxyribonucleic acid) testing can be attempted. Forensic DNA testing is sometimes referred to as “DNA fingerprinting.” With the exception of identical twins, no two individuals’ DNA is exactly alike. Like other comparison tests within the forensic sciences, a standard sample from a suspect is required in order to make a comparison. Alternatively, if a known standard is not available (for example, when an unidentified body is thought to be a particular person, but no standard of that person is available for comparison), then samples from close relatives (parents, siblings) can be used. It is beyond the scope of this text to adequately explain DNA testing, but an attempt will be made to provide the basic concepts.

Our genetic information is contained within structures called genes, which are made-up of DNA. Each gene can be considered a template for the production of a specific protein. Proteins are substances that perform all sorts of functions within our bodies; without them, life would be impossible. Our genes are mostly contained within structures called chromosomes, which reside within the nucleus of our cells. A few genes are present within mitochondria, a subcomponent of cells that reside outside of the nucleus within the cytoplasm of our cells. All humans (excluding those with certain genetic abnormalities) have a total of 46 chromosomes within each of their nucleated cells (cells that have a nucleus). It should be mentioned here that most blood cells (normal red blood cells) do *not* have nuclei, although white blood cells do have nuclei, so the DNA testing performed on blood relies on the DNA contained in the white blood cells. Almost all other cells of the body *do* have nuclei. The 46 chromosomes are composed of 22 pairs of “autosomes” (numbered 1–22) and a pair of “sex chromosomes.” In females, there are two X chromosomes, while in males, there is a single X chromosome and a single Y chromosome. Each person receives 23 chromosomes (22 autosomes and one sex chromosome) from their mother and 23 chromosomes from their father.

Each chromosome has its own complement of genes. For a given gene, its location is always on a specific chromosome at a specific location. We refer to this specific location as the gene’s “locus” (plural: “loci”). In females, each specific gene actually occurs in two places, one on each of the 23 pairs of chromosomes. For males, each gene present on the autosomes (chromosomes 1–22) occurs twice, while the genes occurring on the X and Y chromosomes have only one copy. Genes can have different subtypes. The specific subtypes are referred to as “alleles.” Some genes only have a couple of possible alleles. Others have many possible alleles. The differences in gene alleles are what contribute to the variations that exist amongst individual persons. The gene subtypes are determined by the specific sequence of molecules that make up the genes. A chromosome is a very long double-chain of DNA molecules (a “double helix”) that represents many genes connected to each

other but separated by other DNA molecules that can be thought of as “spacers” between the DNA that makes up the genes. DNA segments that are part of specific genes are said to be “coding” regions of the DNA, since they “code for” a protein. The spacer areas are referred to as “non-coding” regions.

Each specific DNA molecule is called a nucleotide and is composed of three parts, a sugar (deoxyribose), a phosphorus molecule, and a base. Nucleotide bases come in four possible types: adenine (A), thymine (T), cytosine (C), and guanine (G). Each strand of DNA is a chain of nucleotides joined end-to-end. In the double-helix that makes a chromosome (composed of two chains or strands of bases), a base on one strand is always paired (connected) to a complimentary base on the other strand. Adenine is always paired with thymine, while cytosine is always paired with guanine. So, for a given segment of a chromosome (whether it is within a specific gene or in one of the non-coding regions), the base-pair sequence might look like:



The “base-pair” configuration and the variations that exist between one person’s sequence of base pairs and another person’s sequence is the basis by which DNA testing works, whether for medical diagnosis of disease (identifying a “defective gene”) or for forensic DNA testing. Forensic DNA experts rely on the fact that in many different locations within human chromosomes, there are sequences of DNA base-pairs that demonstrate a great deal of variation from one individual to another. If a forensic scientist is able to evaluate and identify the exact base-pair sequences from a sufficient number of these variable regions, then from a statistical standpoint, the forensic scientist is able to determine a unique “DNA profile” for any individual evaluated.

The reader is referred to other resources regarding the descriptions of the evaluation methods used to detect the DNA variations that exist between different people. Terms such as restriction fragment length polymorphism (RFLP), variable number of tandem repeats (VNTR), short tandem repeats (STR), Southern blot analysis and polymerase chain reaction (PCR) refer to some of the types of variable DNA regions that have been or are currently tested, as well as the methods used to identify and characterize the individual variations within these regions. DNA testing can be an incredibly discriminating tool for identification. Forensic pathologists may rely on the technology to determine the positive identification of a particular decedent. In other cases, DNA analysts rely on the pathologist collecting standard DNA samples (usually in the form of “blood spot” cards) at autopsy (Fig. 2.10) so that other crime scene evidence (like blood spatter) can be matched to or excluded from the decedent. Certification of DNA analysts occurs via the American Board of Criminalistics.

Fig. 2.10 A “blood spot” card from an autopsy, used to preserve a blood sample for potential DNA testing



Other Disciplines

Blood Spatter Analysis: Blood spatter analysis encompasses the evaluation of blood drops at a crime scene (or sometimes on a body or elsewhere). Based on the size, shape, character, density, and location of blood spatter, forensic scientists are able to determine possible causes of, or scenarios responsible for creating the particular pattern. Specific features that are able to be determined include the direction of travel, the relative velocity, and the angle of impact of the blood droplets. High velocity, low velocity, arterial, cast-off, and other patterns have specific characteristics. Persons performing blood spatter analysis are of varying backgrounds, including crime scene police officers. Some are crime laboratory personnel who have other responsibilities, such as trace evidence examiners. Occasionally, it may be important to evaluate blood spatter on the body or on the clothing of a dead person (Fig. 2.11 and Disc Image 2.6). In these cases, the pathologist is responsible for identifying, documenting, and, in the case of clothing, preserving such evidence for subsequent blood spatter analysis. Certification in blood spatter analysis is available via the International Association for Identification.

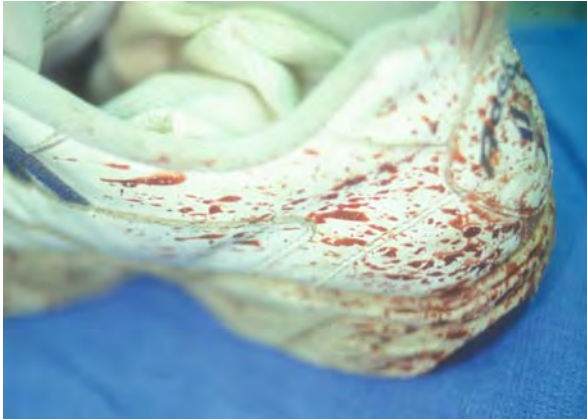


Fig. 2.11 Blood spatter on a shoe

Impression Analysis: Impression evidence includes items such as tire tracks, footprints (Disc Image 2.7), and shoeprints, although many different objects may cause impression evidence. Toolmark evidence as described above is a form of impression evidence. As with firearms examiners being able to include or eliminate a particular weapon as being the source of a particular bullet based on class characteristics, investigators evaluating other types of impression evidence can frequently include or exclude a suspect item (like a shoe) based on class characteristics. For example, if a shoe print found at a crime scene is consistent with a size 12 athletic shoe, a suspect shoe that is a size 9 work boot can be excluded. Unlike the situation with bullet examination, where it is relatively common for a firearm examiner to conclusively “match” or exclude a weapon as the source of a bullet, it is less common for shoe or tire imprint evidence to contain enough individual characteristics to absolutely match the evidence to the object. Some firearms and toolmarks examiners perform analysis of other imprint evidence. Sometimes, other forensic scientists, such as document examiners or other crime laboratory employees, perform these examinations. Certain police crime scene personnel may also do this. Depending on the case being evaluated, a forensic pathologist may discover imprint evidence at autopsy, either on the clothing or on the body. In such cases, the pathologist must document the evidence and preserve it, if possible. Examples include a bloody footprint on a body or a tire tread imprint on a pedestrian’s clothing (Fig. 2.12). Certification in footwear impression analysis is available via the International Association for Identification.

Computer Forensics: Computer forensics is a rapidly evolving discipline that is concerned with computer and electronic crimes. Examples of the types of crimes investigated include identity theft, credit card and other financially motivated fraud, child pornography, etc. Forensic pathologists may interact with computer forensic specialists in a variety of situations, especially with regard to specific death



Fig. 2.12 A tire track mark on the clothing of a pedestrian

investigations. For example, if a death is determined to be a suicide using an unusual method, an analysis of the person's personal computer may disclose how the individual learned of that particular method of suicide.

Forensic Artistry: Forensic artistry relies on an artist's ability to draw or sculpt an image of how a suspect or victim might appear. The appearance may be created on the basis of witness recollections of a suspect or on other criteria, some of which may have more scientific basis, such as occurs with computer assistance when an attempt is made to show how a missing person or suspect might appear several years later. The most common situation for forensic pathologist interaction with forensic artists is when the pathologist has an unidentified skeleton. Anthropological evaluation can provide estimates of race, gender, and age. A forensic artist can attempt to reconstruct the facial appearance by literally sculpting a face of clay over the skull. A picture of the reconstructed face can then be published to the public, with the hope that someone might recognize who the person might be. Chapter 9 will provide more details regarding this method of identification. Forensic art certification is available via the International Association for Identification.

Forensic Engineering: Forensic engineering is a discipline that can encompass virtually any type of engineering. Examination of collapsed buildings, bridges, and other structures is one area of forensic engineering. Other topics include vehicular collisions and accident reconstruction, evaluation of engines or other devices for defects responsible for injuries/death, and evaluation of electrical appliances or sources of electricity in cases of suspected electrocutions. Forensic pathologists may call upon a forensic engineer when a specific death is related to any of these or other engineer-related issues. Board certification in forensic engineering is regulated by the International Institute of Forensic Engineering Sciences, Inc.

Disc Image Legends

- Disc Image 2.1 A partially skeletonized corpse at autopsy (seen from the back). Following autopsy, with careful evaluation of the soft tissues, consultation with a forensic anthropologist in such a case can provide valuable information.
- Disc Image 2.2 A dental model of a suspect's teeth used by a forensic odontologist to compare with a bite-mark on a victim.
- Disc Image 2.3 A firearms examiner firing a bullet into a watertank at the Southwestern Institute of Forensic Sciences in Dallas, Texas. The bullet is collected from the tank and compared to an evidence bullet.
- Disc Image 2.4 A firearms examiner at the Southwestern Institute of Forensic Sciences in Dallas, Texas using a comparison microscope to compare an evidence bullet to an exemplar bullet fired from a suspect weapon.
- Disc Image 2.5 A suicide note collected at the scene of an alleged suicide. A document examiner can compare the handwriting in the note to known standards from the victim to determine if the person actually wrote the note.
- Disc Image 2.6 Blood spatter on the foot of a decedent, as seen at autopsy.
- Disc Image 2.7 A footprint found at the scene of a crime.

Selected References

- American Academy of Forensic Sciences. <http://www.aafs.org>
- American Board of Criminalistics. <http://www.criminalistics.com>
- American Board of Forensic Anthropology, Inc. www.csuchico.edu/anth/ABFA/
- American Board of Forensic Entomology. www.research.missouri.edu/entomology/
- American Board of Forensic Odontology. www.abfo.org
- American Board of Forensic Toxicology. www.abft.org
- American Board of Psychiatry and Neurology, Inc. www.abpn.com
- American Society of Crime Laboratory Directors. www.asclcd.org
- Bernitz H, Owen JH, van Heerden WFP, Solheim T. An integrated technique for the analysis of skin bite marks. *J Forensic Sci* 2008;53:194–8.
- Bevel T, Gardner RM. *Bloodstain Pattern Analysis – With an Introduction to Crime Scene Reconstruction*, 3rd ed. Boca Raton, FL: CRC Press; 2008.
- Byrd JH, Castner JL eds. *Forensic Entomology – The Utility of Arthropods in Legal Investigations*. Boca Raton, FL: CRC Press; 2001.
- Cattaneo C. Forensic anthropology: developments of a classical discipline in the new millennium. *Forensic Sci International* 2007;165:185–93.
- Forensic Specialties Accreditation Board. <http://www.thefsab.org>
- Hawthorne MR. *Fingerprints – Analysis and Understanding*. Boca Raton, FL: CRC Press; 2009.
- International Association for Identification. <http://www.theiai.org/certifications/index.php>
- International Institute of Forensic Engineering Sciences, Inc. <http://www.iifes.org>
- Krogman WM, Iscan MY. *The Human Skeleton in Forensic Medicine*, 2nd ed. Springfield, IL: Charles C. Thomas; 1986.
- Neale BS, ed. *Forensic Engineering – Diagnosing Failures and Solving Problems*. London, England: Taylor & Francis; 2005.
- Newman R. *Computer Forensics – Evidence Collection and Management*. Boca Raton, FL: Auerbach Publications; 2007.

- Ogle RR, Fox MJ. *Atlas of Human Hair – Microscopic Characteristics*. Boca Raton, FL: CRC Press; 1999.
- Pinckard JK. Overview of Other (Non-Pathology) Forensic Sciences (Chapter 19). In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. College of American Pathologists, Northfield, IL; 2006.
- Pinckard JK, Memorial Eckert Paper for 2007. Forensic DNA analysis for the medical examiner. *Am J Forensic Med Pathol* 2008;29:375–81.
- Rudin N, Inman K. *An Introduction to Forensic DNA Analysis*, 2nd edition. Boca Raton, FL: CRC Press; 2002.
- Sapir GL. Qualifying the Expert Witness: A Practical Voir Dire. *Forensic Magazine*. February/March 2007. <http://www.forensicmag.com/articles.asp?pid=132>
- Stimson PG, Mertz CA, eds. *Forensic Dentistry*. Boca Raton, FL: CRC Press; 1997.
- Stone JH, Roberts M, O’Grady J, Taylor AV, O’Shea K. *Faulk’s Basic Forensic Psychiatry*, 3rd ed. Oxford, UK: Blackwell; 2000.
- Warlow TA. *Firearms, the Law and Forensic Ballistics*. Bristol, PA: Taylor & Francis; 1996.

Chapter 3

Introduction to Forensic Pathology

What man can live and not see death, or save himself from the power of the grave?

Psalm 89:48

Abstract Chapter 3 provides the reader with an overview of forensic pathology, including the duties of a forensic pathologist, the required training, and various qualifications. Specific duties of forensic pathologists include death investigation, autopsy performance, identification of dead bodies, documentation of findings, death certification, testifying and consultation.

Keywords Forensic pathology · Training requirements · Board certification · Testifying · Consultation

Overview

Note: The reader is referred to Chapter 1 (Introduction to Pathology) and Chapter 2 (Introduction to Forensic Sciences) as prerequisite reading prior to this chapter.

Forensic pathology represents a subspecialty area within the larger field of pathology that specifically deals with the investigation of sudden, unexpected, and/or violent death. As “pathology” literally means “the study of disease,” the subspecialty of “forensic pathology” may be considered the study of “diseases” (taken to include natural disease as well as “injury”) that can cause death. Another useful definition of “forensic pathology” is the application of forensic science and pathology to the investigation of death.

It is worth noting that forensic pathology can also be considered a part of a somewhat larger division within medical terminology called “forensic medicine.” “Forensic medicine” can be defined as the application of forensic sciences to medicine. As such, forensic medicine does not limit its scope to death investigation. In fact, the practice of so-called “clinical forensic medicine” specifically deals with examination of living patients. Depending on the jurisdiction, local resources,

and the existence of cooperative efforts within a given medical and law enforcement community, forensic pathologists are sometimes asked to perform various “clinical forensic medicine” examinations.

Duties of the Forensic Pathologist

Forensic pathologists have a variety of duties (see Table 3.1). These involve various aspects of the investigation of certain types of deaths that, according to various state laws, fall under the jurisdiction of the local medicolegal death investigation agency. In some jurisdictions, such as certain county-based medical examiners systems and most coroner systems, the official, legally-mandated responsibility for the investigation of such deaths is designated to someone other than the forensic pathologist. For coroner systems, this individual is the coroner. For certain county-based medical examiner systems, this individual is the “medical examiner,” who is usually a physician, but rarely a pathologist, let alone a forensic pathologist. In such settings, the forensic pathologist serves primarily as the person responsible for performing autopsies when called upon to do so by the local medical examiner or coroner, although in many instances, the county medical examiners and coroners rely a great deal on the experience of the forensic pathologist in assisting with difficult cases and decisions. In these systems, the ultimate responsibility of certifying death rests on the county medical examiner or coroner. Coroners and county medical examiners may also hire assistants to help in their death investigation duties. In other jurisdictions, including various state, regional, and metropolitan medical examiners systems, the duty of performing the official investigation of such deaths, including death certification, is the responsibility of the forensic pathologist. In such settings, the forensic pathologist (or agency) may employ lay death investigators to assist in investigations (essentially fulfilling the investigative role that coroners, county medical examiners and their assistants perform in those systems), but the ultimate responsibility rests with the forensic pathologist(s). In the ensuing discussion of forensic pathology and death investigation, it is presumed that the forensic pathologist is involved with each step in the process, recognizing that individual jurisdictional differences exist.

Table 3.1 Duties of the forensic pathologist

-
- Investigate sudden, unexpected, and violent deaths
 - Perform forensic autopsies
 - External examination
 - Internal examination
 - Ancillary procedures
 - Ensure positive identification of decedents
 - Document injuries and diseases
 - Determine cause of death
 - Determine manner of death
 - Testify to findings
 - Consultation
-

Investigation

In general terms, a complete forensic death investigation may be divided into 3 parts: (1) the initial investigation, which includes the scene investigation; (2) the examination of the body, which may or may not involve the performance of an autopsy. The autopsy can be further subdivided into the external examination, the internal examination, and additional (ancillary), specialized procedures; and (3) the subsequent follow-up investigation. The reader is referred to Chapter 4 for details regarding the initial and follow-up investigations, and to Chapter 7 for a more detailed description of the forensic examination of dead bodies, including autopsy performance. The responsibility for the initial and follow-up investigations depends to a great extent on the legally-mandated criteria for a particular state or jurisdiction, as briefly described above. As such, forensic pathologists may or may not be officially responsible for these aspects of death investigations; however, forensic pathologists will certainly interact with those who are, and the forensic pathologist's ultimate conclusions regarding a particular death may rely heavily on the work product of those who are officially mandated to perform such investigations. It is very important to realize that the initial death investigation and the subsequent follow-up investigation (following autopsy performance) are frequently critical factors involved in the conclusions reached by the forensic pathologist. Proper training and communication with police, emergency medical and fire personnel, coroners, county medical examiners, and medical examiner death investigators is of paramount importance in establishing and maintaining a high-quality death investigation system. In some jurisdictions, forensic pathologists actually participate in scene investigations fairly frequently, while in others, participation is limited (Fig. 3.1). In general, forensic pathologists attempt to know as much about a particular death investigation as possible prior to performing the autopsy. Depending on the case, information obtained on initial investigation, particularly that related to death scene investigation, can help forensic pathologists focus the autopsy examination to a particular concern.

Autopsy

Chapter 7 provides a more detailed description of the forensic examination of dead bodies, including autopsy performance. Only the basic facts are presented here. First, it must be emphasized that not every death that is referred to a death investigation agency will require an autopsy (see below). The autopsy can be divided into 3 parts: (1) external examination; (2) internal examination; and (3) ancillary procedures. The external examination involves the examination and documentation of anything on the external surface of the body (Fig. 3.2), and is important in many cases in establishing the identity of the decedent (the dead person). The external examination includes documentation of clothing, medical treatment, injuries, postmortem changes, and various individual characteristics, such as height, weight, racial features, gender, hair color and length, eye color, tattoos, scars,

Fig. 3.1 Scene investigation is one of the most important aspects of a death investigation. Depending on the jurisdiction, forensic pathologists may or may not participate in this aspect of the investigation, although most will visit at least an occasional scene, particularly if asked to do so by police or investigators. In this case, a forensic pathologist supervises as an elevator shaft accident victim is extricated by fire department personnel



Fig. 3.2 The external examination is a very important part of a forensic autopsy. In some cases, particularly in homicide cases with multiple injuries, the external examination requires much more time than the internal examination. In the example shown, a case with multiple injuries, including blunt force, sharp force, and gunshot wounds, the external examination took several hours to complete

etc. Photography is an important means of documentation, particularly regarding identity and injury issues.

The internal examination involves surgically opening the body, removing the organs, and dissecting the organs in order to document the presence and/or absence of disease and/or injury (Fig. 3.3). Traditionally, a “complete” autopsy includes removal and examination of the brain, the neck structures, and the organs of the trunk (chest, abdomen and pelvis). In some instances, a “limited” autopsy may be performed, with only certain organs being examined.

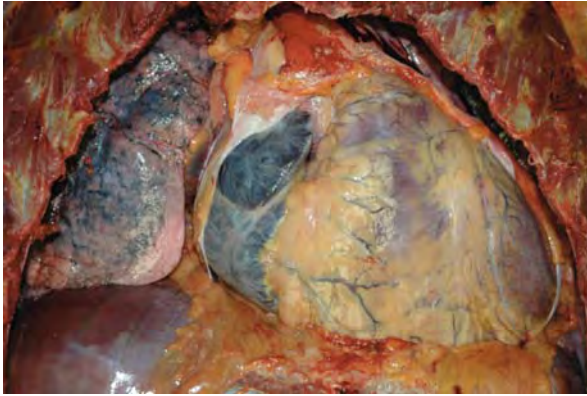


Fig. 3.3 The internal examination allows the forensic pathologist to identify diseases and injuries at autopsy, and to collect tissue samples and certain types of evidence. In this case, showing the internal appearance of the chest, after removal of the chest plate and the anterior (front) pericardial sac, a markedly enlarged heart is evident. The structure to the left of the heart in the photograph is the right lung. Immediately below the lung is the right side of the diaphragm

Various ancillary procedures may take place as part of a forensic autopsy. Depending on the specific procedure, these may occur either during the external or internal examination, or following the internal examination. Some of the procedures, such as toxicology testing (evaluating blood or other tissues for drugs and toxins) are part of the majority of forensic autopsies while other procedures are required in only a few select cases. Ancillary procedures that may be included in a forensic autopsy include toxicology testing, histology (microscopic) examination (Fig. 3.4 and Disc Image 3.1), retention of organs (brain, heart) (Disc Image 3.2) or other tissues for further study, chemistry testing, microbiology (cultures for bacteria, fungi, etc.) and other laboratory testing. Other common procedures include radiologic examination (X-rays) (Disc Image 3.3), specialized dissections, such as removal of the eyes, the middle and inner ears, or the spinal cord, and dissection of the legs or other areas of the body. In many cases, evidence must be collected, including trace evidence, sexual activity evidence, bullets, etc. In some offices, fingerprints are taken and retained on all bodies. In some forensic autopsies, additional consultation with other forensic specialists such as forensic odontologists and forensic anthropologists is warranted.



Fig. 3.4 Microscopic examination may be a very important part of a forensic autopsy, particularly in cases where the cause of death is a natural disease process. In this photograph, a forensic pathologist is viewing slides under a light microscope

As noted above, not all deaths that are referred to a death investigation system will be autopsied. Depending on the type of system (coroner versus medical examiner), the established protocols (or traditions), the available resources, and sometimes the expectations of the public, it is not uncommon for a large percentage of cases to pass through the death investigation process without being autopsied. In such instances, the death investigation can proceed as described above for select cases, including the performance of an external examination and various ancillary procedures, such as toxicology testing. There are various criteria that an office should establish in order to determine whether or not an autopsy should be performed, as well as whether or not an official “external examination” (without autopsy) should be performed. A more detailed discussion of this is presented in Chapter 7.

Verification of Identity

Whether or not an autopsy is performed on a particular body that falls under the jurisdiction of a death investigation agency, it is imperative that the body is positively identified. A variety of methods are used to ascertain a positive identity and these are described in detail in Chapter 9. Depending on the type of death investigation system, the forensic pathologist may have the legal responsibility for ensuring positive identity, or he/she may simply assist in ascertaining positive identification.

Documentation of Findings

A very important part of the practice of forensic pathology involves the documentation of findings. Autopsy notes, photographs, tissue samples, evidence collection, and autopsy reports are all considered parts of the documentation process. Most

autopsy reports will provide objective descriptions of the positive findings as well as a section that provides an opinion as to the cause of death and the manner of death. A more thorough discussion regarding the autopsy report is presented in Chapter 7.

Death Certification

Death certification essentially refers to the completion of an official “death certificate.” Death certificates include various items of personal information (name, birthdate, social security number, etc.) along with official documentation of the “cause of death” and the “manner of death.” The cause of death refers to the disease, injury, or combination of these responsible for death. The manner of death refers to the means or circumstances by which the cause of death occurred. The typical choices for manner of death include natural, accident, suicide, homicide, and undetermined. It is important to remember that, strictly speaking, the cause and manner of death are considered medical opinions. As such, there may be disagreement between forensic pathologists regarding these opinions, particularly when considering complex cases or certain manner of death rulings.

Each state within the United States requires a properly completed death certificate to be filed whenever a person dies. The individuals allowed to legally certify deaths are delineated by various state laws. In general terms, deaths that are considered solely due to an underlying natural disease can be certified by a physician having knowledge of the deceased. Any death that is due to something other than a natural disease *must be* referred to the local death investigation agency (coroner or medical examiner) for certification. When the forensic pathologist is working within a system that confers on him/her the responsibility of certifying deaths, such as occurs in many medical examiner systems, then the forensic pathologist is the person who must complete the death certificates. In other systems, such as most coroner systems and some types of medical examiner systems, the forensic pathologists’ primary responsibility is the performance of autopsies, while the official duty of completing death certificates falls on the coroner or the county medical examiner. In such situations, it is very common for the forensic pathologists to provide cause and manner of death determinations within their autopsy reports, with subsequent official certification by the coroner/medical examiner based on these autopsy report rulings.

Testifying

Forensic pathologists are frequently called upon to provide official testimony within the legal system regarding deaths that they have investigated. For many forensic pathologists, most testimony is within the confines of the criminal court system; however, testimony in civil proceedings is not uncommon. Most common is the participation within specific legal proceedings such as Grand Jury hearings, depositions, and courtroom hearings and trials (Figs. 3.5, 3.6 and Disc Image 3.4).

While the forensic pathologist can be considered a fact witness in cases in which the pathologist played an official role in a particular death investigation, he/she is usually also recognized as an expert witness within the field of forensic pathology, thus allowing the forensic pathologist to offer opinions as a part of the testimony.



Fig. 3.5 A forensic pathologist participating in a videotaped deposition. A court reporter documents the proceedings



Fig. 3.6 Testifying in court proceedings, including criminal and civil trials, is an important aspect of being a forensic pathologist

Consultation

Some forensic pathologists who work within a death investigation system are, on occasion, consulted by someone (police, family, attorney, etc.) who is looking for an expert opinion regarding an injury, a medical issue, or a death that was not originally investigated as part of that forensic pathologist's normal daily duties. However,

it should be noted that some forensic pathologists are restricted by their employer (often a county or other government), and are not allowed to perform such consultations. Depending on office policies, resources available, and other concerns, some forensic pathologists actually spend a relatively large amount of their time performing forensic consultations. There are also forensic pathologists who do not have an official position within, or association with, an official death investigation agency, but instead, spend most of their professional time performing forensic consultations and other activities related to such consultations. Frequently, these individuals have previously gained extensive experience working within an official death investigation system.

Training and Qualifications of Forensic Pathologists

Currently, the training required to become a forensic pathologist in the United States is as follows: successful participation in and completion of medical school (MD or DO degree), successful participation in and completion of an anatomic pathology residency program (typically 3–4 years), successful participation in and completion of a forensic pathology fellowship program (1 year). It should be noted that many choose to participate in a combined anatomic and clinical pathology residency even though the only requirement is anatomic pathology. In 2007, there were 150 accredited pathology residency training programs within the United States, with over 2500 positions available per year. In contrast, there were 37 accredited forensic pathology programs with a total of 70 fellow positions available. To be an American Board of Pathology (ABP)-Board-certified forensic pathologist, one must first be ABP-Board-certified in anatomic pathology, and then in forensic pathology. Table 3.2 provides a synopsis of the education, training, and examination requirements to become a Board-certified forensic pathologist.

Table 3.2 Education, training, and examination requirements for Board-certified forensic pathologists (USA)

-
- Successful completion of medical school (MD or DO degree)
 - Successful completion of an anatomic pathology residency (with or without clinical pathology residency) at an ACGME-accredited program
 - Successful completion of a forensic pathology fellowship at an ACGME-accredited program
 - Fulfillment of the requirements of the American Board of Pathology (ABP) and officially declared a diplomate and certified in anatomic pathology (via taking and passing the AP Board examination)
 - Fulfillment of the requirements of the ABP and officially declared a diplomate and certified in forensic pathology (via taking and passing the FP Board examination)
 - Recertification by the American Board of Pathology (recently required for newly-certified persons)
-

Simply defined, the term “forensic pathology” may be used to describe a subspecialty within the world of pathology that deals specifically with the investigation of sudden, unexpected, or violent deaths via the performance of forensic autopsies. As

such, forensic pathology can be considered a branch of pathology, and therefore, a discipline that is performed by pathologists. All pathologists are required to receive some training in forensic pathology during their residency training; however, the training varies greatly between different residency programs. The National Association of Medical Examiners has endorsed, and in association with the College of American Pathologists, has published Guidelines for AP residency training in forensic pathology (Table 3.3). In summary, therefore, forensic pathology is a subspecialty area that is practiced by pathologists.

Table 3.3 Guidelines for AP residency training in forensic pathology

During training in an accredited anatomic pathology residency training program, each AP resident should receive training in forensic pathology. During this training, it is recommended that each resident will . . .

- (1) Assist in, or perform under direct forensic pathologist supervision, medicolegal autopsies.
 - (2) Learn the general principles of autopsy and biosafety.
 - (3) Understand the statutory basis for the medicolegal death investigation systems, and the requirements to serve as medical examiner or coroner or forensic pathologist.
 - (4) Learn how the medicolegal death investigation system interacts with the criminal and civil legal systems and public health and safety agencies.
 - (5) Learn the three core elements of a medicolegal autopsy: scene/death investigation, autopsy, and toxicology.
 - (6) Learn to recognize the common injury patterns seen in blunt force trauma, sharp force injury, firearms injury, motor vehicle fatalities, asphyxial injuries, temperature and electrical injuries, and suspected child and elder abuse.
 - (7) Learn to recognize common postmortem changes, including decomposition patterns.
 - (8) Understand the causes and autopsy findings in cases of drug- and toxin-related fatalities.
 - (9) Understand the causes and autopsy findings in cases of sudden, unexpected, natural death.
 - (10) Understand the importance of proper documentation in medicolegal autopsies.
 - (11) Understand the concept of evidence recognition, collection, preservation, transport, storage, analysis, and chain of custody.
 - (12) Understand the basic disciplines of forensic science and their relevance to death investigation systems.
 - (13) Understand the concept and application of clinical forensic medicine.
 - (14) Learn the importance of professional interaction with families and the public.
 - (15) Understand the proper method of death certification.
 - (16) Understand and correctly apply the terms “cause,” “manner,” and “mechanism” of death.
-

What then is the definition of a “forensic pathologist?” The answer to this question is not so simple. Unfortunately, there are examples of physicians claiming to be forensic pathologists who are not even trained in pathology. Indeed, there are individuals who are not even physicians who have claimed to be forensic pathologists. Whether it be a non-pathologist performing autopsies, a non-licensed physician performing autopsies, or even a *non-physician* performing autopsies, such situations are a disservice to the profession of forensic pathology and the general public and should not be tolerated. Therefore someone claiming to be a forensic pathologist must, at least, be a physician and a pathologist. As the performance of autopsies is generally considered the practice of medicine, a practicing forensic pathologist

should be licensed to practice medicine in the particular jurisdiction within which he/she works.

Using the simple definition of “forensic pathology” above, a “forensic pathologist” would be a physician who is trained in pathology who specifically practices “forensic pathology.” However, since forensic pathology is a recognized subspecialty within the world of pathology, it is best to limit the designation “forensic pathologist” to those who have specifically received formal subspecialty training within forensic pathology, currently in the form of a one-year fellowship. Still others prefer to limit the designation to those who have not only received specialized training (either via fellowship or experience), but who have also taken and passed the forensic pathology Board Examination administered by the American Board of Pathology, as mentioned above and clarified below.

For those desiring the most qualified individuals to perform medicolegal autopsies, it is advisable to select a forensic pathologist who is Board-certified in forensic pathology by the American Board of Pathology (ABP). The ABP began certifying forensic pathologists in 1959, requiring satisfactory completion of a Board examination in order to become Board-certified. Historically, there were two routes by which a person could become eligible to take the Board examination. One was based on experience, wherein a pathologist could document sufficient experience within the field of forensic pathology in order to satisfy the requirements set forth by the ABP. This has traditionally been referred to as the “experience route,” but for many years, this route has no longer been accepted as a means by which a pathologist can become eligible to take the forensic Board examination.

The other route by which a pathologist can qualify to take the forensic Board examination is the only route currently available. It requires a person to have successfully completed a one-year fellowship (additional year of training) in forensic pathology at a forensic pathology graduate training program that is accredited by the Accreditation Council for Graduate Medical Education (ACGME) and to have previously successfully passed the Anatomic Pathology (AP) Board examination. The AP Board examination is an examination administered by the ABP to persons who have successfully completed training in an ACGME-accredited pathology residency program. Another type of board examination that is frequently administered to persons who have completed their pathology residency is called the Clinical Pathology (CP) Board examination. In order to be allowed to take and successfully pass the Forensic Pathology (FP) Board examination, an individual *must* have previously passed the AP examination, although many Board-certified forensic pathologists have also taken and passed the CP examination. As the AP (and CP) Board examinations are multiple-day examinations that are generally regarded as extremely difficult, there are a certain percentage of individuals who have successfully completed pathology residency training but are unable to pass their pathology Board examinations. Some of these individuals participate successfully in a forensic pathology fellowship following their pathology residency, but still, even after multiple attempts, are unable to pass their pathology Board examinations. These individuals, having been trained in both pathology and forensic pathology, may

otherwise be considered competent, but they *may not* truthfully present themselves as Board-certified forensic pathologists.

The reader should be aware of the fact that there are some organizations, including some that promote themselves as an official “board,” which offer “board certification” in various disciplines. This is especially common within the forensic world. The criteria for certification by such groups is not necessarily rigorous, and should not be considered equivalent to Board-certification by the ABP. Even if a person is Board-certified by the ABP in anatomic pathology, they might not be Board-certified in forensic pathology, although they might imply that they are. In a like fashion, the reader may recall from Chapter 1 that, in order for a physician to progress through his or her medical education, he/she must take and pass a series of “board examinations.” In other words, a physician who is able to be licensed and practice medicine in a given state *must be* board-certified by the National Board of Medical Examiners (NBME) or similar Board. Consequently, there are persons who practice forensic pathology who are *not* certified by the ABP in pathology *or* forensic pathology but who deceptively claim to be “board-certified forensic pathologists,” when the “board-certification” to which they refer applies to their NBME certification. Recall that the use of the term “medical examiner” in the NBME name has absolutely nothing to do with death investigation, although it could certainly be portrayed in this fashion by someone falsely claiming to be a board-certified forensic pathologist. In the United States, the term “Board-Certified Forensic Pathologist” should be reserved for persons certified by the ABP in forensic pathology, and the reader is advised to inquire further when a person claims to be a “board-certified forensic pathologist.” In 2007, The National Association of Medical Examiners (NAME) approved the following position statement: “NAME defines a ‘Board Certified Forensic Pathologist’ in the USA as a Pathologist certified in FP by the American Board of Pathology. The NAME does not recognize Board certification by any other body. Any unqualified representation of Board Certification not by the ABP is considered a misrepresentation of professional standing”.

Disc Image Legends

Disc Image 3.1 Microscopic section of normal thyroid gland.

Disc Image 3.2 An infant brain that has been “fixed” in formalin for several weeks following autopsy. The brain is markedly swollen, or “edematous.”

Disc Image 3.3 The ability to perform and evaluate X-rays is a necessary part of the forensic autopsy. X-rays are primarily utilized to locate projectiles (bullets), to evaluate injury, and to aid in identification. This is an example of a normal postmortem chest X-ray.

Disc Image 3.4 A forensic pathologist participating in a deposition along with attorneys from both sides of the case.

Selected References

Accreditation Council for Graduate Medical Education. www.acgme.org

American Board of Pathology. www.abpath.org

Dix J, Calaluce R. *Guide to Forensic Pathology*. Boone County, MO: Jay Dix, MD; 1998.

Graham MA, Hanzlick R. *Forensic Pathology in Criminal Cases*. Carlsbad, CA: Lexis Law Publishing; 1997.

Guidelines for AP Residency Training in Forensic Pathology (Chapter 2). In: Basic Competencies in Forensic Pathology – a Forensic Pathology Primer. College of American Pathologists, Northfield, IL; 2006. pp 3–4.

Hanzlick R, Prahlow JA, Denton S, Jentzen J, Quinton R, Sathyavagiswaran L, Utley S. Selecting forensic pathology as a career – a survey of the past with an eye on the future. *Am J Forensic Med Pathol* 2008;29:114–22.

Position statement re: “Board-certified forensic pathologist.” National Association of Medical Examiners, Board of Directors 2007 Annual Meeting, October 12, 2007.

Chapter 4

Death Investigation

And while they were in the field, Cain attacked his brother Abel and killed him. Then the Lord said to Cain, 'Where is your brother Abel?' 'I don't know,' he replied. 'Am I my brother's keeper?' The Lord said, 'What have you done? Listen! Your brother's blood cries out to me from the ground.'

Genesis 4:8–10

Abstract This chapter deals primarily with the non-autopsy aspects of death investigation. It provides an overview of the various types of death investigation systems within the United States and describes the various responsibilities of death investigators, including initial investigation, scene investigation, and follow-up investigation. A brief description of grief counseling is also provided.

Keywords Death investigation · Coroner · Medical examiner · Scene investigation · Grief counseling

Introduction

In its simplest description, forensic pathology is about the investigation of death, and the forensic autopsy is but one aspect of the entire death investigation. As was presented in Chapter 3 (introduction to forensic pathology), the overall death investigation may be divided into the initial investigation, the examination of the body (which may or may not include a complete autopsy), and the follow-up investigation. In this chapter, we will focus on the different types of death investigation systems and then concentrate on a death investigator's duties regarding the non-autopsy aspects of the death investigation, namely the initial investigation, including the scene investigation, and the subsequent follow-up investigation. The reader is referred to Chapter 7 for a more in-depth discussion concerning the body examination and forensic autopsy.

Death Investigation Systems Within the United States

Overview

Death investigation systems within the United States represent a mixed-bag, ranging from coroner systems to medical examiner systems to mixed systems. Whatever the system, those who work within that system are responsible for investigating deaths that fall under their jurisdiction as detailed by state law. This typically includes any sudden, unexpected, or violent death. It always includes deaths that are not related solely to natural disease and it may include other specific death types, such as all infant deaths, deaths in custody, deaths that represent a public health hazard, deaths occurring during a medical procedure, etc. The reader is referred to the specific state laws in any given state for the most detailed explanation of which cases are to be referred to the death investigation system.

The following paragraphs describe each type of death investigation system (coroner, medical examiner, mixed). It should be emphasized that even when comparing specific jurisdictions within one of the aforementioned system types (for example, comparing one medical examiner system to another medical examiner system), and certainly when comparing one system type to another, there can be differences between the types of cases investigated, protocols utilized, interaction with or utilization of forensic pathologists or others involved in death investigation, issues related to death certification, determination of which cases are autopsied, and just about any other aspect of death investigation that one might consider, including the overall quality of the system. While it is tempting for some individuals to proclaim that one system type is better than another system type, the reality is that there can be excellent death investigation systems of whatever type and there can be not-so-good systems of whatever type. Having said this, it is the opinion of many within the forensic community that an excellent system headed by a Board-certified forensic pathologist tends to be the best.

Before beginning a detailed discussion of the various system types, it is necessary to discuss the importance of the ability of a death investigation system (of whatever type) to operate in a manner that is unbiased and uninfluenced by other entities. It is important that death investigation agencies function as independent agencies that are not unduly influenced by any other agency (governmental or otherwise), such as the police, the prosecutors office, a particular hospital or other non-governmental institution. The appropriate placement of the death investigation office within the governmental organizational structure varies from one place to another. In some jurisdictions, it operates well within the auspices of public safety (a law enforcement arm of government) while in others, it might not do so well within this arm of government. Another option is within the realm of public health, as a division of the health department, for example. Again, this seems to work adequately for some, but not for others. The important point is that, wherever a particular office is located within the organizational structure of government, the office must have the resources, the autonomy, the independence *from*, yet the

support of all other parts of government so that it can operate in an unbiased, professional manner, without undue influence from any other entity. In some situations, forensic pathologists or the entire death investigation office function quite well as private business entities that provide services to the county or state government via a contractual arrangement.

Jurisdictional Issues

For most deaths, the death investigation agency responsible for death investigation is determined by where the dead body is found, or where it is officially pronounced dead. As such, a county coroner or medical examiner will take jurisdiction of any dead body that meets the statutory rules within their jurisdiction. It is not unusual for the police agency responsible for investigating a particular death to be from a geographic location distinct from the death investigation agency, particularly in deaths that occur following transportation of an injured patient to a major medical center, but also in cases where a body has been moved after death. Cooperation and communication are of utmost importance in these situations. Deaths occurring in airplanes or on ships at sea become the jurisdiction of the death investigation agency within the next port.

Special jurisdictional circumstances exist with certain Federal Government cases (for example, the President of the United States and military personnel killed in the line of duty), wherein federal agencies and investigators have legal jurisdiction. Another special circumstance occurs with deaths of Native Americans occurring on Indian Reservations.

Coroner Systems

The coroner system can be traced back to England, when representatives of the crown (the royal family), called “crowners,” were responsible for making sure that the king received his rightful portion of a person’s property after the death of that person. This transformed into the crowner (coroner) being responsible for investigating (via inquest) a person’s death. This system was used by all states until the advent of the medical examiner system (see below) in the nineteenth century. Many states remain coroner states, but occasionally, a state will convert to a medical examiner system or a mixed system. Currently, 10 states have only coroners as the official death investigation system, while 18 have mixed systems. In certain states (Texas), Justices of the Peace function as coroners, although in Texas, large cities have medical examiners.

The coroner system is typically a county-based death investigation system, such that each county (or parish or occasionally a district) in a coroner state has a coroner, who is usually elected and may or may not have assistants (deputy coroners). In a majority of coroner jurisdictions, there are very few requirements which must be

met in order to run for coroner; however, occasional jurisdictions have some strict requirements, such as a medical degree, although having a medical degree does not necessarily mean that someone has been trained in death investigation. Recently, more coroner systems require coroners and deputy coroners to receive some type of formal training in death investigation, and some require continuing education, although formal training is still lacking in many states. Ultimately, there remains a great degree of variability within the organizational structure as well as the quality of the coroner systems within the United States.

In coroner systems, it is the coroners (and sometimes the deputy coroners) who are officially responsible for performing death investigations. In most coroners' jurisdictions, the decision as to whether or not an autopsy should be performed also rests with the coroner (or deputy) – if the coroner deems that an autopsy is necessary, then they must arrange for it to be performed. In some places, these autopsies are performed by forensic pathologists while in other places, the coroners must rely on non-forensic pathologists to perform the autopsies. In some counties within certain coroner states, policies have been instituted such that the decision regarding whether or not an autopsy is necessary rests with the forensic pathologist rather than the coroner. Similar arrangements exist by law within other states; however, these would fall under the category of “mixed systems” (see below), rather than pure coroner systems.

Official death certification is normally the responsibility of the coroner, although in some settings, the pathologist may also be able to certify deaths. Whether or not the latter is allowable, in coroner systems, the coroners will frequently rely on the pathologist's autopsy findings (cause and manner of death determinations) in order to complete the death certificate.

Medical Examiner Systems

Medical examiner (ME) systems first came into existence within the United States in 1877. They usually involve an appointed individual who serves as the official death investigator for a state, region or district, or county, depending on the system. In most of the 23 states that currently have medical examiner systems, the ME is required to be a physician, although there are occasional exceptions to this. In some jurisdictions, the ME must be a pathologist or a forensic pathologist. Most ME systems that exist in large cities are headed by a forensic pathologist and have several assistant MEs that are also forensic pathologists. Such metropolitan offices usually employ several non-physician, non-pathologist, so-called *lay* death investigators who are responsible for performing a bulk of the non-autopsy aspects of the death investigation. These death investigators have various titles, depending on the office, including “death investigator,” “field agent,” and “medical examiner investigator.” Statewide ME systems vary in their organizational structure. Some have a definite hierarchy, with a forensic pathologist overseeing the entire state, and death investigators such as exist in metropolitan offices. Others have regions or districts, each with one or more forensic pathologists, as well as lay death investigators. Others are

county-based, without much statewide oversight, with the county MEs functioning in a similar fashion as coroners. In many cases, statewide county MEs are physicians who have little or no formal training in death investigation.

Because of the lack of consistency from one ME system to the next, there is a great deal of variability in the quality of the system and in the meaning of the word “medical examiner.” For example, in certain jurisdictions a “medical examiner” is a non-physician who functions as a death investigator (just like a coroner) while in other places, a “medical examiner” is a physician, with very little or no training in death investigation, who functions primarily as an administrator overseeing the county’s death investigators. There are other systems who reserve the term “medical examiner” for Board-certified forensic pathologists.

Within ME systems, the decision regarding whether or not to perform an autopsy and the official certification of death are the responsibility of the medical examiner. If the ME is a forensic pathologist, then the forensic pathologist performs these tasks. If the ME is not a forensic pathologist, then the forensic pathologist may or may not be formally involved with either of these tasks.

Mixed Systems

Of the 50 states and the District of Columbia, 18 are considered mixed systems, and are usually one of two general types. In one type, the medical examiner offices exist within metropolitan areas and the coroner (or similar) offices exist in rural counties. In the second type, each county is served by a coroner, but the state has one or more medical examiner offices staffed by forensic pathologists in order to perform autopsies for the coroners. In some jurisdictions, the coroners make the decision of whether or not to perform an autopsy. In others, that decision is delegated to the forensic pathologists.

Duties of Death Investigators

Overview

As the preceding paragraphs have indicated, there is no universally accepted title for an individual responsible for performing death investigations. The generic term “death investigator” will be used here to include coroners, deputy coroners, justices of the peace responsible for death investigation, non-pathologist medical examiners, medical examiner office field agents, death investigators, or any number of other titles assigned to individuals who work for a death investigation system and are charged with performing the initial investigation, including scene investigation, and any subsequent investigation related to the overall investigation of a death. Depending on the office, the jurisdiction, or the state, the specific duties of a death investigator vary from one jurisdiction to another. Common duties include scene investigation, notification of the next-of-kin, gathering information

leading to identification, handling personal property, and follow-up investigation. Other responsibilities may include photography, providing autopsy support, and court testimony. The amount of training required to be a death investigator is quite variable around the country and there are numerous death investigation training programs in existence. There is also a certifying agency which grants official Board-Certification in death investigation. The American Board of Medicolegal Death Investigators (ABMDI) is a national, not-for-profit, independent professional certification board that strives to maintain high-quality death investigation by administering a certification examination and requiring continuing education requirements for recertification.

Initial Investigation

When a death occurs and the circumstances indicate that it falls under the jurisdiction of the death investigation agency (coroner or medical examiner), then it is incumbent upon those who are aware of the death to report the case to the death investigation agency. In most cases, those who do such reporting are law enforcement officers, fire department employees, emergency medical services personnel, or healthcare workers. Occasionally, funeral home personnel notify the coroner/medical examiner when the funeral home employee recognizes that the case was not properly referred to the office. If a family member or acquaintance or other person discovers a dead body or witnesses a death, they usually notify the police, who then contact the coroner or medical examiner.

From the perspective of the death investigation agency, a death investigation begins when they are notified of a person's death. The first question that must be answered is whether or not the agency will accept jurisdiction of the death. State laws vary regarding which deaths must be investigated. If the death is not due to natural disease, then the agency must accept jurisdiction. If the death is obviously due to natural causes, the death investigation agency may or may not have to accept jurisdiction. Often, in these situations, if the decedent's (dead person's) physician is willing to sign the death certificate, and there are no indications of foul play, then the death investigation agency will release jurisdiction and not accept the case. If there is no physician willing to sign the death certificate, or if there are investigational issues that suggest possible foul play, or if it is important to rule-out foul play, the agency will typically take jurisdiction of the body and perform an official investigation.

Important information to obtain as early as possible in every death includes the decedent's name, birth date, gender, race, social security number, address, and legal next-of-kin. The responsibility for notifying the next-of-kin of a decedent's death may or may not rest with the death investigator. During this initial investigation period, death investigators must also be working to establish positive identification of the body, although protocols for identification vary from one office to the next. The topic is discussed further in Chapter 9. Information regarding past medical

history, drug use history, psychiatric history, employment status, recent activities and state of mind, relationship issues, etc. should also be obtained. Other items of importance include the time that the death was officially pronounced, where the person died, whether or not an injury occurred, the position of body, the condition of the body, evidence of postmortem changes, environmental information, and when and where the person was last known to be alive. In order to obtain many of these items of information, investigators must interview family members, health-care providers, witnesses and first responders. Obtaining copies of medical records, including Emergency Medical Service documents, is necessary (Disc Image 4.1). If possible, a scene investigation should be performed.

Scene Investigation

If resources are sufficient and the circumstances of death so dictate, it is ideal for death investigators to perform a scene investigation (Figs. 4.1, 4.2). This is particularly relevant if the body remains at the scene of death, and has not been transported to the hospital during attempts at resuscitation; however, a scene investigation can be vitally important and provide valuable information even if the body has been transported to the hospital. If a body is pronounced dead at the scene (as opposed to after transport to the hospital), many death investigation systems require a scene investigation. Others have various protocols as to which case types absolutely require a scene investigation (whether or not the body is present at the scene). Case types that should always have a scene investigation include all confirmed or suspected homicides, suicides, accidents, child deaths, traffic-related deaths, in-custody deaths, and workplace-related deaths.



Fig. 4.1 A very important part of the initial investigation of a death is the scene investigation. In this scene photograph, the exact location of the decedent's body is documented prior to moving the body



Fig. 4.2 Another scene photo showing the condition of a motor vehicle following a fatal collision. Photographs such as this can be very helpful in understanding the forces involved and the injuries sustained

The first rule in performing a death scene investigation is to make certain that the scene is safe and secure. Usually, this requires police involvement but in some instances, it will require other professionals, such as fire department personnel or utility workers. The second rule is to not contaminate or disturb the scene. At the very least, death investigators should wear disposable examination gloves and it is also advisable to wear shoe covers and hair nets. Occasionally, full body covering is desirable. If an investigator needs to move the body or anything else at the scene, it is best to first consult with the police crime scene investigation officers prior to doing so, to make sure that all photographs and other procedures have been completed. Likewise, the police should not disturb anything related to the body until consulting with the death investigator, who typically has legal authority over the body. When touching items at a scene, examination gloves should always be worn and care should be taken not to sit on furniture or lean against or brush against walls or furniture.

Death investigators should note the general conditions at the scene (indoors versus outdoors, temperature, wet versus dry, etc.) (Fig. 4.3), the specific location of the decedent, the body position of the decedent, the presence or absence of rigor mortis, livor mortis, and body cooling (see Chapter 8), the presence or absence of important trace evidence on or near the body, the presence or absence of drugs, weapons, electrical devices, or other pertinent objects or materials (Fig. 4.4). Placing paper bags on the decedent's hands can help to preserve trace evidence for subsequent collection at autopsy (Fig. 4.5). Depending on the circumstances, the evidence, and protocols, some investigators may choose to collect certain evidence at the scene. If possible, witnesses should be interviewed. It is important to interview persons who initially discover the body, as well as emergency first responders, in order to determine if the body or clothing have been moved (Fig. 4.6 and Disc Image 4.2). Besides focusing on the body, it is also important to note the remainder of the scene as well. In certain cases, some very valuable information can be gained by



Fig. 4.3 General overall scene photographs are useful in providing documentation of the general conditions of the scene of death. Even though the death associated with the photograph shown occurred inside the home, the photograph of the outside of the home provides important information about the death scene



Fig. 4.4 This photograph shows the decedent before his body was moved. Also visible is a container of a toxic substance on the top of the bookshelf. Toxicologic analysis for the specific substance within the blood of the decedent confirmed that the death was due to the poison

closely examining the scene *away from* the body (Fig. 4.7 and Disc Images 4.3–4.5). Medications and/or drugs (Fig. 4.8), as well as weapons thought to have been used in the incident (Fig. 4.9), should be seized and secured (this may require police support). Medications may eventually be analyzed, but are more often documented and subsequently destroyed in the presence of a witness. Photographs (including photographs of the body prior to touching or moving the body, as well as overall scene photos), a written report, and diagrams, as indicated, are all important aspects of a thorough scene investigation.

Many offices use a death investigation form (Disc Image 4.6). Other resources are also available for death investigators to use as aids in performing scene investigations. An excellent resource produced through the efforts of the National Institute



Fig. 4.5 Paper bags were placed on the hands of this homicide victim by the death investigator at the scene in order to preserve any trace evidence present on or about the hands



Fig. 4.6 The presence of a blood-soaked area on the pavement adjacent to the decedent (seen at the top of the photograph) indicates that, at some point, the gunshot wound victim was probably face-down on the pavement at that location. The death investigator was able to confirm with emergency medical services (EMS) personnel that, when they arrived, the victim was in fact face-down at that location. The decedent is in his present location as a result of EMS workers rolling him onto his back to pronounce death

of Justice under the auspices of the U.S. Department of Justice is a booklet entitled, “Death Investigation: A Guide for the Scene Investigator.” It is beyond the scope of this chapter to provide all the details contained in the booklet, so the reader is referred to the booklet as a more detailed guide of what is required during a death scene investigation. It provides detailed information regarding six aspects of a comprehensive scene investigation: (1) investigative tools and equipment, (2) arriving at the scene, (3) documenting and evaluating the scene, (4) documenting and evaluating the body, (5) establishing and recording decedent profile information, and (6) completing the scene investigation. The booklet is available for free download at



Fig. 4.7 When at a death scene, it is important for investigators to observe areas away from the body. This is a photograph of the bathroom of a decedent found dead in another room. The dried red fluid in the bathroom was not blood, but was consistent with vomit (gastric contents) containing abundant medication, leading investigators to suggest the possibility of an overdose

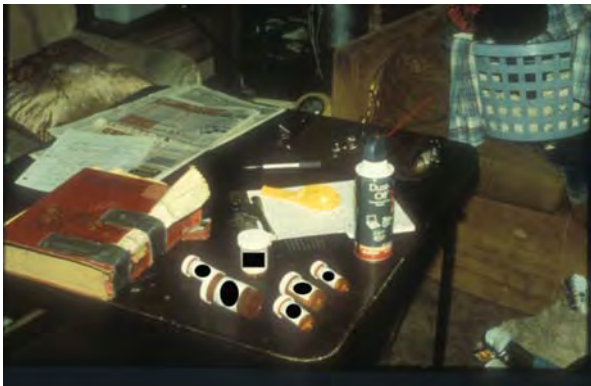


Fig. 4.8 The discovery of medications at a death scene should prompt the death investigator to collect the medication containers, record the information on the containers, count and record the contents of each container, and secure the containers until final disposition. In some cases, final disposition will involve analysis of the contents by a drug laboratory. In most cases, final disposition involves eventual safe, proper, witnessed and documented destruction of the contents

the Occupational Research and Assessment, Inc website at www.orainc.com under the “Reports” tab.

Other valuable resources include guidelines and a form that are specifically created to aid in the evaluation of infant deaths. Known as the “Sudden, Unexplained Infant Death Investigation” (SUIDI) Guidelines and Reporting Form, they are also available for free download at www.orainc.com under the “Reports” tab. Many other resources are available for the death investigator at this website.



Fig. 4.9 If a weapon, such as a gun, is present at a death scene, it is important that the weapon be secured and made safe, while maintaining any evidence that may be present on or near the weapon

In virtually every jurisdiction, other governmental agencies are likely to be involved when a death occurs. This includes, but is not limited to, the police, emergency medical services, the fire department, the health department, social services, child protective services, the prosecutors office, etc. It is extremely important for death investigators to recognize that, although investigations performed by any of these other agencies may provide useful information to the overall investigation of a particular death, these other investigations should *never* replace the investigation performed by the official death investigation system (coroner or medical examiner).

Follow-up Investigation

While it is vitally important to perform a comprehensive initial investigation, as well as a scene investigation when possible, it is equally important to recognize that, in many cases, additional follow-up information is also required. In many cases, all of the relevant information surrounding the death is not known prior to autopsy, therefore it is always important to obtain all available information, even if it may take several days or even weeks. This can become particularly important if the pathologist or investigator needs to review old medical records (Disc Image 4.7). Following the performance of an autopsy, a death investigator may be asked to gather additional information from family, friends, police, medical professionals, etc. because of specific issues or findings that became evident during the autopsy. In fact, it is not unusual for much of an investigator's time regarding a specific death to occur after the autopsy is performed. This certainly is not the rule, but it is a frequent occurrence. Death investigators should remain aware of this fact and pursue the continued investigation of a case with the same degree of professionalism and importance as they do when performing an initial death investigation.

Grief Counseling

When a mass fatality incident occurs within the United States, it is common for various agencies to provide grief counseling for friends and relatives of the victims. Everyday within the death investigation agencies across the nation, thousands of individual and sometimes multiple fatality incidents occur. The surviving family members and friends of these decedents face many of the same issues as those associated with the more publicized mass fatality incidents. Unfortunately, in most of the non-mass fatality incidents, grief counseling is not routinely available. Any such counseling is typically left to the family and friends. A few death investigation offices have deliberately initiated the development of grief counseling services for the survivors of decedents whose deaths are investigated at their office. When able to be implemented, such services can provide important and valuable services to the community. Unfortunately, grief counseling services are usually viewed as a luxury rather than a necessary part of the death investigation process, and when an office is under-funded or shorthanded, grief counseling does not seem very important. As the entire death investigation process moves into the future, addressing issues such as under-funding and lack of staffing, the implementation of grief counseling should remain a priority.

Disc Image Legends

Disc Image 4.1 Investigators should obtain copies of all medical records associated with a decedent's death. These can be particularly helpful to the forensic pathologist when medical therapy introduces artifacts that can be confused with true injuries. In the example shown, an emergency department diagram shows the exact location of injuries as seen prior to implementation of medical therapy, which introduced numerous artifacts.

Disc Image 4.2 A death investigator's photograph showing the appearance of a decedent as first seen by the investigator at a death scene. In this case, it was important for the death investigator to interview the family members who discovered the body, as well as police and emergency medical workers in order to determine if the body had been moved, if the shirt was already raised when the decedent was found, and if the pants were partially down as they appear in this photograph.

Disc Image 4.3 A young female decedent found dead in bed with her dead mother (disc image 4.4).

Disc Image 4.4 A woman found dead in bed with her daughter who was also dead (disc image 4.3).

Disc Image 4.5 Initially, it was thought that the mother and her daughter (shown in disc images 4.3 and 4.4) were killed by another person; however, there were no signs of injury on either body and no signs of forced entry or disruption at the scene. Another opinion was that they may have succumbed to carbon monoxide or some other environmental hazard, which would possibly put others in the

apartment complex at risk for injury or death. However, examination of the refrigerator contents at the scene revealed the presence of a “milkshake” in a glass jar, with the contents demonstrating a medicinal odor. Further examination of the mother’s computer revealed that she had concocted the “milkshake” and given it to her daughter to kill her, prior to committing suicide. Autopsy toxicology results revealed lethal levels of barbiturates in each decedent.

Disc Image 4.6 An example of a portion of a four-page death investigation form used in one jurisdiction.

Disc Image 4.7 An example of a sizeable medical record that required review by a forensic pathologist.

Selected References

- Coburn MU, Borges MC, Knake E, Harper M. The multidisciplinary approach to dealing with families: a model for medical examiners. *J Forensic Sci* 2000;45(6):1278–9.
- Death Investigation: A Guide for the Scene Investigator. National Institute of Justice. 1999. Available at www.orainc.com/files/guidelines.pdf
- Haglund WD, Ernst MF. The lay death investigator: in search of a common ground. *Am J Forensic Med Pathol* 1997;18:21–5.
- Hanzlick R. *Death Investigation System and Procedures*. Boca Raton, FL: CRC Press; 2007.
- Hanzlick R, Parrish RG. Death investigation report forms (DIRFs): generic forms for investigators (IDIRFs) and certifiers (CDIRFs). *J Forensic Sci* 1994;39:629–36.
- Hanzlick R. On the need for more expertise in death investigation (and a National Office of Death Investigation Affairs?). *Arch Pathol Lab Med* 1996;120:329–32.
- Hanzlick R, Combs D. Medical examiner and coroner systems: history and trends. *JAMA* 1998;279:870–4.
- Hanzlick R. Death Investigation Systems. In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. College of American Pathologists; Northfield, IL; 2006.
- Hanzlick R. The conversion of coroner systems to medical examiner systems in the United States: a lull in the action. *Am J Forensic Med Pathol* 2007;28:279–83.
- Jentzen JM, Ernst MF. Developing medicolegal death investigator systems in forensic pathology. *Clinics Lab Med* 1998;18:279–322.
- Prahlow JA, Lantz PE. Medical examiner/death investigator training requirements in state medical examiner systems. *J Forensic Sci* 1995;40:55–8.
- Randall B, Randall L. Initiation of formal death investigation procedures among the Northern Plains Indians: a necessary adjunct in the study of American Indian sudden infant deaths. *Am J Forensic Med Pathol* 1999;20:22–6.
- Shemonsky NK, Reiber KB, Williams LD, Froede RC. Death investigations on military installations. *Military Medicine* 1993;158:585–7.
- Sudden, Unexplained Infant Death Investigation Reporting Form. www.orainc.com/files/SUIDIRF2-9-06.pdf
- Sudden, Unexplained Infant Death Investigation Guidelines. www.orainc.com/files/SUIDI_Guidelines.pdf
- The American Board of Medicolegal Death Investigators. <http://www.slu.edu/organizations/abmdi/>
- Vanezis P, Busuttill A (eds). *Suspicious Death Scene Investigation*. London, England: Arnold; 1996.

Chapter 5

Death Certification

There is a time for everything, and a season for every activity under heaven: a time to be born and a time to die.

Ecclesiastes 3:1–2

Abstract Chapter 5 deals specifically with the official certification of death and gives a description of the death certificate. Two major concepts are discussed in detail: the cause of death, and the manner of death with an important distinction made between a “cause” of death and a “mechanism” of death. Numerous issues related to manner of death determination are reviewed, and many example cases are provided.

Keywords Death certification · Death certificate · Cause of death · Mechanism of death · Manner of death

Introduction

As part of a worldwide effort to collect mortality statistics, headed by the World Health Organization (WHO), the United States, via the efforts of the National Center for Health Statistics (NCHS) (which is part of the Centers for Disease Control and Prevention, CDC), requires states to collect and provide data on every death that occurs. As the U.S. Standard Certificate of Death (Fig. 5.1) is used by all states as the basis for their death certificate, all state certificates are similar. There are approximately 2.6 million deaths per year within the United States, and the NCHS collects information regarding each of them. The death certificate, therefore, is a means by which data can be collected regarding mortality statistics.

The various vital statistics agencies within the states are responsible for collecting the data and forwarding it on to the NCHS. There are rules which dictate who is allowed to certify a death, with only certain individuals being allowed to complete death certificates. As long as a death is considered “natural” (occurring as a result of an underlying natural disease), and a physician has cared for the decedent (dead

U.S. STANDARD CERTIFICATE OF DEATH

LOCAL FILE NO. _____ STATE FILE NO. _____

1. DECEDENT'S LEGAL NAME (Include AKA if any) (First, Middle, Last) _____ 2. SEX _____ 3. SOCIAL SECURITY NUMBER _____

4a. AGE-Last Birthday (Years) _____ 4b. UNDER 1 YEAR _____ 4c. UNDER 1 DAY _____ 5. DATE OF BIRTH (Mo/Day/Yr) _____ 6. BIRTHPLACE (City and State or Foreign Country) _____

7a. RESIDENCE-STATE _____ 7b. COUNTY _____ 7c. CITY OR TOWN _____

7d. STREET AND NUMBER _____ 7e. APT. NO. _____ 7f. ZIP CODE _____ 7g. INSIDE CITY LIMITS? Yes No

8. EVER IN US ARMED FORCES? Yes No 9. MARITAL STATUS AT TIME OF DEATH Married Married, but separated Widowed Divorced Never Married Unknown 10. SURVIVING SPOUSE'S NAME (If wife, give name prior to first marriage) _____

11. FATHER'S NAME (First, Middle, Last) _____ 12. MOTHER'S NAME PRIOR TO FIRST MARRIAGE (First, Middle, Last) _____

13a. INFORMANT'S NAME _____ 13b. RELATIONSHIP TO DECEDENT _____ 13c. MAILING ADDRESS (Street and Number, City, State, Zip Code) _____

14. PLACE OF DEATH (Check only one: see instructions)
 IF DEATH OCCURRED IN A HOSPITAL: Inpatient Emergency Room/Outpatient Died on Arrival Hospice facility Nursing home/Long term care facility Decedent's home Other (Specify) _____
 15. FACILITY NAME (If not institution, give street & number) _____ 16. CITY OR TOWN, STATE, AND ZIP CODE _____ 17. COUNTY OF DEATH _____

18. METHOD OF DISPOSITION: Burial Cremation Donation Entombment Removal from State Other (Specify) _____ 19. PLACE OF DISPOSITION (Name of cemetery, crematory, other place) _____

20. LOCATION-CITY, TOWN, AND STATE _____ 21. NAME AND COMPLETE ADDRESS OF FUNERAL FACILITY _____

22. SIGNATURE OF FUNERAL SERVICE LICENSEE OR OTHER AGENT _____ 23. LICENSE NUMBER (Of Licensee) _____

ITEMS 24-28 MUST BE COMPLETED BY PERSON WHO PRONOUNCES OR CERTIFIES DEATH

24. DATE PRONOUNCED DEAD (Mo/Day/Yr) _____ 25. TIME PRONOUNCED DEAD _____

26. SIGNATURE OF PERSON PRONOUNCING DEATH (Only when applicable) _____ 27. LICENSE NUMBER _____ 28. DATE SIGNED (Mo/Day/Yr) _____

29. ACTUAL OR PRESUMED DATE OF DEATH (Mo/Day/Yr) (Spell Month) _____ 30. ACTUAL OR PRESUMED TIME OF DEATH _____ 31. WAS MEDICAL EXAMINER OR CORONER CONTACTED? Yes No

CAUSE OF DEATH (See instructions and examples)

32. PART I. Enter the chain of events—disease, injury, or complications—that directly caused the death. DO NOT inter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary. Approximate interval: Onset to death

IMMEDIATE CAUSE (Final disease or condition resulting in death) _____ Due to (or as a consequence of): _____

Sequentially list conditions, if any, leading to the cause listed on line a. Enter the UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST

b. _____ Due to (or as a consequence of): _____

c. _____ Due to (or as a consequence of): _____

d. _____

PART II. Enter other significant conditions contributing to death, but not resulting in the underlying cause given in PART I _____ 33. WAS AN AUTOPSY PERFORMED? Yes No

34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? Yes No

35. DID TOBACCO USE CONTRIBUTE TO DEATH? Yes Probably No Unknown

36. IF FEMALE: Not pregnant within past year Pregnant at time of death Not pregnant, but pregnant within 42 days of death Not pregnant, but pregnant 43 days to 1 year before death Unknown if pregnant within the past year

37. MANNER OF DEATH Natural Homicide Accident Pending investigation Suicide Could not be determined

38. DATE OF INJURY (Mo/Day/Yr) (Spell Month) _____ 39. TIME OF INJURY _____ 40. PLACE OF INJURY (e.g., Decedent's home, construction site, restaurant, wooded area) _____ 41. INJURY AT WORK? Yes No

42. LOCATION OF INJURY: State _____ City or Town _____ Street & Number _____ Apartment No. _____ Zip Code _____

43. DESCRIBE HOW INJURY OCCURRED: _____ 44. IF TRANSPORTATION INJURY, SPECIFY: Driver/Operator Passenger Pedestrian Other (Specify) _____

45. CERTIFIER (Check only one): Certifying physician-To the best of my knowledge, death occurred due to the cause(s) and manner stated. Pronouncing & Certifying physician-To the best of my knowledge, death occurred at the time, date, and place, and due to the cause(s) and manner stated. Medical Examiner/Coroner-On the basis of examination, and/or investigation, in my opinion, death occurred at the time, date, and place, and due to the cause(s) and manner stated.

Signature of certifier: _____

46. NAME, ADDRESS, AND ZIP CODE OF PERSON COMPLETING CAUSE OF DEATH (Item 32) _____

47. TITLE OF CERTIFIER _____ 48. LICENSE NUMBER _____ 49. DATE CERTIFIED (Mo/Day/Yr) _____ 50. FOR REGISTRAR ONLY-DATE FILED (Mo/Day/Yr) _____

51. DECEDENT'S EDUCATION-Check the box that best describes the highest degree or level of school completed at the time of death. 8th grade or less 9th - 12th grade, no diploma High school graduate or GED completed Some college credit, but no degree Associate degree (e.g., AA, AS) Bachelor's degree (e.g., BA, AB, BS) Master's degree (e.g., MA, MS, MEd, MEd, MSW, MBA) Doctorate (e.g., PhD, EdD) or Professional degree (e.g., MD, DDS, DVM, LL.B., JD)

52. DECEDENT OF HISPANIC ORIGIN? Check the box that best describes whether the decedent is Spanish/Hispanic/Latino. Check the "No" box if decedent is not Spanish/Hispanic/Latino. No, not Spanish/Hispanic/Latino Yes, Mexican, Mexican American, Chicano Yes, Puerto Rican Yes, Cuban Yes, other Spanish/Hispanic/Latino (Specify) _____

53. DECEDENT'S RACE (Check one or more races to indicate what the decedent considered himself or herself to be): White Black or African American American Indian or Alaska Native (Name of the enrolled or principal tribe) _____ Asian Indian Chinese Filipino Japanese Korean Vietnamese Other Asian (Specify) _____ Native Hawaiian Guamanian or Chamorro Samoan Other Pacific Islander (Specify) _____ Other (Specify) _____

54. DECEDENT'S USUAL OCCUPATION (Indicate type of work done during most of working life. DO NOT USE RETIRED) _____

55. KIND OF BUSINESS/INDUSTRY _____

Fig. 5.1 A photograph of the U.S. Standard Certificate of Death

person) and has knowledge of the person’s medical history, then that physician is allowed to certify the death. If no physician is available or willing to certify death, or if the death is the result of anything other than natural disease, then the death must typically be certified by the official death investigation agency that has jurisdiction in the location where the death occurred. By state statute, the coroner or medical examiner must investigate certain types of death, including sudden, unexpected, violent, and suspicious deaths. This includes any non-natural death, as well as many natural deaths. The specific types of deaths that must be investigated may differ slightly from one state to the next. For example, deaths occurring during a medical procedure or deaths occurring while in-custody, may or may not be specifically referred to within the statutes of a given state. In certain jurisdictions, the autopsy pathologist may be allowed to certify a death. Even in jurisdictions that do not allow this practice, the official certifier (coroner or non-pathologist medical examiner) will frequently rely on the pathologist’s autopsy findings in order to properly certify the death.

Each death certificate provides blank spaces to fill in various identification items regarding the decedent, such as name, age, birth date, etc. (Fig. 5.2) Certifiers must provide information regarding the date and time of death (Fig. 5.3). In addition, there are spaces to enter the cause of death (COD; see below) (Fig. 5.4), the manner of death (MOD; see below) (Fig. 5.5), and the circumstances of death (Fig. 5.6). Many death certificates have additional questions to answer such as “did tobacco use contribute to death?” (Disc Image 5.1) All require the certifying individual to sign the certificate. Funeral directors also have sections to answer on the certificate (Disc Images 5.2 and 5.3). Once a death certificate is completed, it is forwarded to

FEDERAL FILE NO.		STATE FILE NO.	
1. DECEDENT'S LEGAL NAME (Include AKA's if any) (First, Middle, Last)		2. SEX	3. SOCIAL SECURITY NUMBER
4a. AGE-Last Birthday (Years)	4b. UNDER 1 YEAR Months Days	4c. UNDER 1 DAY Hours Minutes	5. DATE OF BIRTH (Mo/Day/Yr)
6. BIRTHPLACE (City and State or Foreign Country)		7a. RESIDENCE-STATE	
7b. COUNTY		7c. CITY OR TOWN	
7d. STREET AND NUMBER		7e. APT. NO.	7f. ZIP CODE
7g. INSIDE CITY LIMITS? <input type="checkbox"/> Yes <input type="checkbox"/> No		8. EVER IN US ARMED FORCES? <input type="checkbox"/> Yes <input type="checkbox"/> No	
9. MARITAL STATUS AT TIME OF DEATH <input type="checkbox"/> Married <input type="checkbox"/> Married, but separated <input type="checkbox"/> Widowed <input type="checkbox"/> Divorced <input type="checkbox"/> Never Married <input type="checkbox"/> Unknown		10. SURVIVING SPOUSE'S NAME (If wife, give name prior to first marriage)	
11. FATHER'S NAME (First, Middle, Last)		12. MOTHER'S NAME PRIOR TO FIRST MARRIAGE (First, Middle, Last)	
13a. INFORMANT'S NAME		13b. RELATIONSHIP TO DECEDENT	13c. MAILING ADDRESS (Street and Number, City, State, Zip Code)
14. PLACE OF DEATH (Check only one: see instructions)			
IF DEATH OCCURRED IN A HOSPITAL <input type="checkbox"/> Inpatient <input type="checkbox"/> Emergency Room/Outpatient <input type="checkbox"/> Died on Arrival		IF DEATH OCCURRED SOMEWHERE OTHER THAN A HOSPITAL <input type="checkbox"/> Hospice facility <input type="checkbox"/> Nursing home/Long term care facility <input type="checkbox"/> Decedent's home <input type="checkbox"/> Other (Specify):	

Fig. 5.2 A closer view of the top portion of the U.S. Standard Certificate of Death, showing the various demographic information that is required for each case

ITEMS 24-28 MUST BE COMPLETED BY PERSON WHO PRONOUNCES OR CERTIFIES DEATH		24. DATE PRONOUNCED DEAD (Mo/Day/Yr)	25. TIME PRONOUNCED DEAD
26. SIGNATURE OF PERSON PRONOUNCING DEATH (Only when applicable)		27. LICENSE NUMBER	28. DATE SIGNED (Mo/Day/Yr)
29. ACTUAL OR PRESUMED DATE OF DEATH (Mo/Day/Yr) (Spell Month)		30. ACTUAL OR PRESUMED TIME OF DEATH	
31. WAS MEDICAL EXAMINER OR CORONER CONTACTED? <input type="checkbox"/> Yes <input type="checkbox"/> No			

Fig. 5.3 A closer view of the section of the U.S. Standard Certificate of Death dealing with the date and time of death

CAUSE OF DEATH (See instructions and examples)		Approximate interval: Onset to death
<p>32. PART I. Enter the <u>gross of events</u>—diseases, injuries, or complications—that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary.</p>		
<p>IMMEDIATE CAUSE (Final disease or condition resulting in death)</p>	<p>a. _____ Due to (or as a consequence of):</p>	<p>_____</p> <p>_____</p> <p>_____</p>
<p>Sequentially list conditions, if any, leading to the cause listed on line a. Enter the UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST</p>	<p>b. _____ Due to (or as a consequence of):</p>	
	<p>c. _____ Due to (or as a consequence of):</p>	
	<p>d. _____ Due to (or as a consequence of):</p>	
<p>PART II. Enter other <u>apparent conditions contributing to death</u> but not resulting in the underlying cause given in PART I</p>		<p>33. WAS AN AUTOPSY PERFORMED? <input type="checkbox"/> Yes <input type="checkbox"/> No</p>
		<p>34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? <input type="checkbox"/> Yes <input type="checkbox"/> No</p>

Fig. 5.4 A closer view of the section of the U.S. Standard Certificate of Death dealing with the cause of death

Fig. 5.5 A closer view of the section of the U.S. Standard Certificate of Death dealing with the manner of death

COMPLETE THE CAUSE OF DEATH
<p>37. MANNER OF DEATH</p> <p><input type="checkbox"/> Natural <input type="checkbox"/> Homicide</p> <p><input type="checkbox"/> Accident <input type="checkbox"/> Pending Investigation</p> <p><input type="checkbox"/> Suicide <input type="checkbox"/> Could not be determined</p>

the state’s vital statistic bureau. In many of the cases that come under the jurisdiction of the coroner or the medical examiner, a specific cause (or manner) of death is not known immediately following completion of the gross portion of the autopsy. The results of further testing (toxicology, histology) or follow-up investigative information is required before the death can be officially certified. In such cases, a certificate is considered “pending.” In some instances, the word “pending” can be entered in the COD and MOD blanks of the certificate, so that various legal issues requiring a death certificate can occur. When the final autopsy results are known, a final certificate can be issued. However, even if a certificate is finalized, if additional relevant information comes to light and the COD or MOD needs to be changed, it is possible to issue an amended death certificate. It is also important to realize that, since the COD and MOD statements represent opinion statements, no one is necessarily bound by these official rulings. In other words, the courts, insurance companies, and even the vital statistics agencies that rely on the certificates to tabulate data are not necessarily required to agree with, or abide by, what is listed on the certificate.

Death Pronouncement

Before certifying a cause and manner of death, it is first necessary to ensure that death has occurred in a specific individual, and it is essential to ensure the positive identity of the dead person (the “decedent”). The reader is referred to Chapter 9 for further discussion of body identification.

<input type="checkbox"/> Unknown if pregnant within the past year			
38. DATE OF INJURY (Mo/Day/Yr) (Spell Month)	39. TIME OF INJURY	40. PLACE OF INJURY (e.g., Decedent's home, construction site, restaurant, wooded area)	41. INJURY AT WORK? <input type="checkbox"/> Yes <input type="checkbox"/> No
42. LOCATION OF INJURY: State:		City or Town:	
Street & Number:		Apartment No.:	Zip Code:
43. DESCRIBE HOW INJURY OCCURRED:			44. IF TRANSPORTATION INJURY, SPECIFY: <input type="checkbox"/> Driver/Operator <input type="checkbox"/> Passenger <input type="checkbox"/> Pedestrian <input type="checkbox"/> Other (Specify)

Fig. 5.6 A closer view of the section of the U.S. Standard Certificate of Death dealing with the circumstances of death

Pronouncement of death is usually relatively straight-forward, although in the hospital setting and a few other settings, certain factors may confuse the issue. At death scenes, the lack of a pulse (heartbeat) and the lack of respiration (breathing) are the usual findings that suggest that death has occurred. Depending on the circumstances, if there are no other signs of death, it is frequently advisable to attempt resuscitation in such cases. If, however, there are other signs of death, including evidence of rigor mortis (postmortem body stiffening), livor mortis (postmortem settling of blood), cooling of the body, or decomposition changes, then one can be confident that death has indeed occurred. An exception to these general comments involve persons who have been submerged in very cold water. In such cases, the body temperature may be markedly reduced, but resuscitation can sometimes still be successful, thus explaining the emergency medical mantra “they’re not dead until they’re warm and dead.”

In the clinical setting, nurses and doctors have the benefit of various tests and studies to aid in the diagnosis of death. Besides feeling for a pulse and looking and listening for breathing, medical personnel can use a stethoscope to listen for a heartbeat and respiration. Sometimes, medical personnel will use some type of “noxious stimulus,” such as a “sternal rub” (rubbing one’s knuckles hard over the sternum/chest); however, the absence of a response is not absolutely indicative of death, although the presence of a response rules it out. Electrocardiograms (ECG or EKG; electronic monitoring of the heart rate) can confirm that there is no electrical activity within the heart and an electroencephalogram (EEG) can be used to determine the lack of brain waves. Brain blood flow studies can show that blood flow to the brain is absent.

The term “brain death” deserves special mention. From a legal standpoint, once someone has been officially declared brain dead, then death has officially occurred. This is of great importance for organ transplantation, since organs cannot legally be removed from a living person (unless they are a “living” donor, such as someone choosing to donate a kidney). The official declaration of brain death involves a set of specific clinical criteria that must be met and documented. Brain death can be declared while the heart remains beating on its own and breathing is maintained artificially, via a ventilator.

Of note is the fact that there are all sorts of definitions for the word “death.” We have already addressed two of the most common definitions: (1) death defined as permanent absence of cardiac and respiratory function; and (2) brain death. In the

latter, there is no question that the other (non-brain) organs remain “alive,” in that many of them are able to be transplanted into a different individual and continue to function (live) for many more years. There is evidence that, at least in the early hours after permanent cessation of heart and lung function, certain organs can still be transplanted and continue to “live.” It is also well-documented that certain cells can be collected many hours, even over a day after death, and grow in “culture” in the laboratory, so that various biochemical tests can be performed to aid in the diagnosis of certain conditions. In other words, certain cells continue to *live* for many hours after “death” of the body. Therefore, “time of death” can be different for cells, organs, the brain, and the entire organism or body, not to mention how the absence of one’s “spirit” or “soul” might enter into the discussion. As such, when we speak of the “time of death,” to be absolutely specific, we are speaking of death as defined in one of the two ways initially described above. It is important for death certifiers to recognize that, when organ transplantation occurs, the legal time of death is the time that the donor was officially declared brain dead. This typically occurs many hours prior to the actual organ recovery surgery, during which time the ventilator is eventually turned off.

Cause of Death

The cause of death (COD) can be defined as the disease(s) or injury(ies), or combination of disease(s) and injury(ies) that initiate(s) an uninterrupted chain of events that leads to the cessation of life. On death certificates, the COD section allows certifiers to include one or two parts. In many instances, only part I is completed. Part II may be used if there are two unrelated causes of death, such as an injury and a disease, or two unrelated disease or injury processes. Occasionally, there are more than two unrelated processes (injuries and/or diseases) that cause death and in these instances, multiple causes can be listed in part I and/or part II, separated with a semicolon or the word “and” or “with.” The “proximate” COD is defined as the “underlying” COD, that which initiates the chain described above. Various “intermediary” causes as well as an “immediate” COD may also be described, but are ultimately the result of the underlying cause. Hence, a cause of death statement format may read as follows: immediate COD due to intermediary COD due to intermediary COD due to underlying COD. Conversely, it can also be appropriate to simply list the proximate (underlying) COD, or to list an immediate COD due to an underlying COD. For statistical purposes, as well as manner of death determinations (see below), the underlying COD is what is considered most important, and it must be included in the COD portion of the death certificate. Cause of death statements can be quite simple or somewhat complicated, depending on the case type and circumstances.

When making COD determinations, it is important to remember that listing only *mechanisms of death* is not acceptable. A “mechanism of death” can be considered a physiologic, metabolic or anatomic alteration that is produced by the underlying cause of death, but is not, in itself, an adequate explanation of what

initiated the sequence of events that caused death. Sometimes, it is acceptable to include a mechanism of death within a COD statement, but only if a definite underlying disease or injury (which represents the underlying COD) is included. Examples of mechanisms of death include exsanguination (bleeding to death), a cardiac dysrhythmia (irregular heart rhythm), air-embolism (gas bubbles blocking blood vessels), cardiac tamponade (when the heart is surrounded and compressed by something, usually blood), sepsis (overwhelming infection within the body), and dehydration (not enough water). Other terms, such as cardiac arrest (the heart abruptly stops beating), cardiopulmonary arrest (the heart and lungs stop functioning), and multi-organ failure (many organs stop functioning) are even less specific than the mechanisms described above and their use for death certification should be minimized. Never should they (or mechanisms of death) stand alone, without an underlying disease or injury as the ultimate COD.

Another important point to remember is that the timing of the initiating event has no bearing on whether or not it may be considered the underlying COD. Consider the following example. A 15-year-old person sustains a spinal cord injury when he is shot by another person in a gang-related shooting and as a result of this injury, he is a paraplegic (paralyzed from the waist down) and experiences multiple urinary tract infections (a known complication of being a paraplegic) throughout the rest of his life. If he dies at age 32 as a result of sepsis (a blood infection) related to a urinary tract infection, then the underlying COD is the gunshot wound. An appropriate COD statement in this case might be as follows: sepsis due to urinary tract infection due to paraplegia related to a remote spinal cord injury due to a gunshot wound of the back. The “but for” rule is sometimes helpful in determining whether or not a potential cause should be considered *the* cause of death. In the example just given, the “but for” rule would be stated as follows: *but for* the gunshot wound of the back sustained 17 years earlier, the person would not have died at the time that he did.

When determining the COD, forensic pathologists and others attempt to take into consideration any and all possible contributing factors. In many cases, there are specific anatomic or laboratory (toxicology, microbiology, chemistry) findings at autopsy that enable the forensic pathologist to definitively determine a COD. Examples of definite anatomic lesions that can explain death include cases of multiple gunshot wounds, blunt force injuries sustained in a motor vehicle collision, and various natural disease processes, such as a ruptured aneurysm or an acute myocardial infarct. An example of definite laboratory evidence of a COD is a case with postmortem toxicology evidence of cocaine intoxication in combination with an absence of an anatomic explanation for death. In cases where there are more than one contributing anatomic and/or laboratory factor in death, determining an appropriate COD is often straight-forward, such as a case where someone dies of severe heart disease in combination with severe lung disease, or a case related to the toxic effects of cocaine with an underlying badly-diseased heart. Occasionally, it becomes somewhat challenging for a forensic pathologist to determine the most appropriate COD ruling when there are numerous potential diseases, injuries, and/or laboratory findings sufficient to explain death. Cases with multiple injury types are not

infrequent and it is reasonable in such cases to list all contributing injury types in the COD section of the death certificate.

At the other end of the spectrum where there are cases with no anatomic or laboratory findings sufficient to explain death, there are two possibilities. In the first, there is sufficient information regarding the circumstances of the case such that a definitive COD can be determined. These types of cases are sometimes referred to as “diagnoses of exclusion,” and include drowning deaths, certain electrocutions, certain asphyxial deaths (particularly in infants), deaths related to extremes in temperature (hypothermia and hyperthermia), and certain natural deaths, including deaths related to seizure disorders and certain deaths related to presumed cardiac dysrhythmias (irregular heart rhythms). The second possibility with cases that have no anatomic or laboratory explanation for death is when there is no or insufficient information regarding the circumstances of death such that one of the “diagnoses of exclusion” is not considered an option. It is in these types of cases that the forensic pathologist must acknowledge the fact that a definite COD could not be ascertained. The most common example of this type of case is the death that is designated “sudden infant death syndrome” (SIDS), or “sudden unexplained infant death” (SUID) and the reader is referred to Chapter 20 for more discussion about these deaths. Another circumstance where a definite COD might not be able to be determined is when there is extensive decomposition. Finally, there are a small percentage of non-decomposed, non-infant cases where a complete autopsy, including laboratory testing and investigation, fails to provide an explanation for death. In these cases, the cause of death is typically considered “undetermined” and if the circumstances are such that foul play is ruled-out, the term “undetermined natural causes” is sometimes used.

In between the two extremes (cases with a definite anatomic or laboratory COD and cases without a clear anatomic or laboratory COD), there are a variety of case types where the anatomic and laboratory findings, in combination with the circumstances of death, allow the forensic pathologist to determine the COD. Within this “middle ground” there are case types that are relatively straightforward, such as a death occurring as an anesthesia-induced cardiac dysrhythmia (irregular heart rhythm) in a person undergoing surgery for severe underlying heart disease.

Other cases within this “middle ground” can present tremendous challenges to the forensic pathologist. An example would be a young adult male under the influence of cocaine who is confronted by police, resists arrest, and is forcibly restrained by half a dozen officers, and dies while lying facedown with a police officer applying pressure on his back and one applying a “neck hold.” In such a case, cocaine-induced excited delirium is no doubt a factor in death; however, the fact that a police officer was applying force to the man’s back and another was applying a choke hold, cannot and should not be ignored and it is reasonable to conclude that the forcible restraint contributed to this man’s death. In cases such as these, it is imperative that forensic pathologists are provided with all available information regarding the death, including witness statements, videotapes of the incident, detailed time lines of what happened and when it happened, etc. The reader is referred to Chapter 21 for additional discussion regarding in-custody deaths.

Another case type that presents a challenge is when an individual with severe, potentially lethal, natural disease sustains some type of trauma in the days, weeks, or even months prior to death. Further discussion of this type of case occurs below in the “manner of death” section. In these and other complex cases, forensic pathologists need to evaluate each case on an individual basis and make a ruling based on the autopsy findings, the facts regarding the circumstances and timing of the event, and pertinent and applicable scientific data regarding the specific issue at hand.

Manner of Death

The manner of death (MOD) is considered the means or circumstances by which the cause of death occurred. In most jurisdictions there are five choices for the MOD: natural, accident, suicide, homicide, and undetermined. When a death is solely related to underlying natural disease, the appropriate MOD is *natural*. If death results from an unintentional, unforeseen or unpredictable injurious event, *accident* is an appropriate MOD ruling. When a death results from an intentional act to severely harm or kill one’s self, *suicide* is the correct MOD. There are various definitions of *homicide* as a MOD but a simple version is “death at the hands of another individual.” Another definition is as follows: death resulting from a volitional act of commission or omission by another individual that is meant to cause injury, harm, fear, or death, or resulting from the wanton disregard for human life. Making a ruling regarding the MOD can depend considerably on the circumstances of a particular death. Therefore, initial investigation, including scene investigation, as well as follow-up investigative information, are frequently very important in making the proper MOD ruling. Autopsy findings can also be very important in MOD rulings. If, after thorough investigation, including autopsy performance, a definite ruling of natural, accident, suicide, or homicide cannot be confidently rendered, then it is appropriate to rule the MOD in such a case as *undetermined*. The following paragraphs provide some additional commentary on natural, accidental, suicidal, and homicidal MOD rulings.

Natural – Deaths that result solely from an underlying natural disease process or multiple diseases are considered natural deaths. A more complete discussion of natural deaths is presented in Chapter 10. With most disease processes, it is recognized as fairly logical that the death is natural; however, there are some instances where the “natural” death ruling doesn’t seem to make too much sense. This tends to be related to the fact that people often consider “natural” diseases to be entirely “internal” (or “endogenous”), without any or much influence from “external” (or “exogenous”) forces. Although many natural disease processes are influenced significantly by external factors, the exogenous influence on a particular disease process may not be sufficient to move a death out of the “natural” category. For example, a death that is related to chronic alcoholism is considered a natural death even though an external, “non-natural” factor (the ethanol) is largely responsible for the pathologic

changes in the body that lead to death. Part of the rationale for calling such deaths “natural” involves the recognition of chronic alcoholism as a disease in itself and the fact that the external influence has its effects over an extended time period (years to decades). As such, it is reasonable to consider deaths due to chronic alcoholism as natural deaths and similar reasoning also justifies the ruling of “natural” for deaths due to chronic drug abuse.

Another confusing area involves certain deaths due to infectious diseases, because the typical scenario involves the transmission of the micro-organism from an external source into the body. If this transmission occurs in a way that is considered usual for that particular organism and death occurs, the death is typically considered natural. For example, if someone dies of malaria (a parasite infection transmitted by mosquito bites), or if someone dies of West Nile virus encephalitis following a mosquito bite, the death is considered “natural.” If someone dies of bacterial meningitis after being exposed at school to someone else with the disease or if someone dies of influenza, after contracting the disease in the community, the death is natural. Despite these examples, infectious disease cases are not always so straightforward. If someone dies of an infectious disease that develops as a complication of some sort of traumatic event, it is reasonable to rule the MOD based on how the trauma occurred. For example, if a person is stung by a wasp and subsequently develops a bacterial infection at the sting site, with eventual sepsis and death, some would consider this an accidental death, because bacterial infection does not typically occur via a wasp sting. Likewise, if a laboratory worker accidentally sticks themselves with a blood-contaminated needle and contracts hepatitis C (a viral infection of the liver) and eventually dies from it, this death would also be considered accidental.

Accident – Accidental deaths result from unforeseen events that typically involve some sort of external factor. Most common are traumatic events, including blunt force, asphyxia, drowning, and fire-related injuries. Toxin exposure is relatively common as well. Sometimes death occurs relatively soon after the traumatic event; however, in other cases, death may not occur until many years after the event. A common type of accidental death that causes confusion regarding MOD determination is a death related to recreational drug abuse. If a person chooses to abuse potentially lethal drugs, despite knowing that they should not do so, how can one consider a subsequent death from this activity as an “unforeseen event?” While most agree with the logic of this question, the unfortunate fact is that, in most jurisdictions, death certificates only allow five choices for MOD. It is obvious that a typical death from recreational drug abuse does not represent a natural or a homicidal death and the question of MOD then comes to suicide, accident, or undetermined. As discussed below, the term “suicide” is reserved for cases where the victim is thought to have intended to end his or her life. Participating in a reckless or dangerous activity such as drug abuse is not sufficient to rule such deaths as suicides. However, if the reckless or dangerous activity carries with it a high propensity to cause sure death (such as playing “Russian roulette” as described below), then, for many forensic pathologists, it is reasonable to rule such deaths as suicidal. The fact that many persons repeatedly participate in recreational drug use without dying makes the “odds”

of death so small compared to the Russian roulette scenario that an “accidental” ruling seems justified. In certain jurisdictions, another MOD option, “unclassified,” is available and is typically used to describe recreational drug deaths (see below).

Other potentially confusing MOD issues are cases involving animals, specifically those related to predator attacks and those related to allergic reactions to stings or bites. These are discussed more completely in Chapter 21. With regard to allergic reactions, some pathologists argue that an underlying hypersensitive immune system is responsible, so the deaths should be ruled “natural” while others argue that the exogenous sting makes these cases “accidental.”

Suicide – A suicide is a death that results from the intentional action of an individual to harm himself/herself. Perhaps no other case type causes as much difficulty for the death investigation community as do suicides, particularly regarding surviving family members who will not accept the MOD ruling. In clear-cut cases, the victim’s intentions are obvious, including the presence of one or more suicide notes; however, the majority of suicide victims do not leave notes – only one quarter to one third will have a suicide note. As such, it should be obvious that a suicide note is not required to rule a death as a suicide. In many cases, the method of suicide combined with the scene and overall death investigation provides ample evidence of the victim’s suicidal intent. For example, if a person has gone to the trouble of placing a vacuum cleaner hose over the tailpipe of his car with the other end inserted through the window of the car, and has sat in the running car waiting for the carbon monoxide within the exhaust to kill him, and there is no evidence of foul play, then the case is obviously a suicide, whether or not there is a suicide note.

Other cases are not necessarily so obvious, although they may appear to be at first glance. If a person goes to the trouble of finding and setting-up a stepladder, tying a rope to the garage rafters, placing a loop of rope around their neck, and then kicking the stepladder out from under their feet, the case may well represent a suicide, but further information must be sought out. If *all other investigation reveals no other possible explanation for death*, then the death is clearly a suicide; however, it cannot be overemphasized that a very thorough death investigation must take place. The investigation should include questioning friends, family members, co-workers, neighbors, and possibly others, specifically questioning the person who found the body about the scene and whether or not it was altered, determining when the victim was last seen alive, what they were doing, and how they were acting, finding-out if they had ever been depressed or previously suicidal, determining whether or not they had experienced any recent major life events, asking about their home life, social life, finances, health, mental status, and work, investigating whether or not there might be someone who wanted to harm them, and asking about any history of autoerotic activity.

A thorough investigation is necessary for all cases, but if it is *not* performed in a case that is ruled a suicide, the case can quickly become an ongoing nightmare for all involved. Surviving family members may become very upset and even bring allegations of a “cover-up.” It is strongly advised, therefore, that death certifiers make certain that a thorough and competent investigation be completed for all

suicides. In some cases, it may be advisable to “pend” the final MOD ruling until such investigation has been finalized.

“Russian roulette” involves placing a single cartridge in the cylinder of a revolver, spinning the cylinder, placing the gun to one’s own head, and pulling the trigger. Depending on the revolver, the person typically has anywhere from a 1 in 5 to a 1 in 9 chance of killing themselves. These odds are so great that, for many forensic pathologists, a death from Russian roulette is considered a suicide. In other words, if a person willingly participates in an activity that is considered reckless and dangerous and carries a significant risk of lethal injury, and that person deliberately carries out an action (pulling the trigger) that may cause that lethal injury, it is appropriate to rule such a death as a suicide. The person intentionally loaded the gun, they intentionally spun the cylinder, they intentionally put the gun to their head, and they intentionally pulled the trigger. They deliberately participated in a highly lethal activity using a device that is intended to kill. Those that argue that Russian roulette cases should be ruled as accidents argue that the person did not really intend to die and that it is analogous to someone who knows that they are drunk getting behind the steering wheel of a car and then crashing and dying.

A subset of suicides that is sometimes difficult to identify are those where the suicide victim chooses to end his or her life using a motor vehicle collision. As with other suicide cases, a thorough investigation is often required in order to rule these as suicides. Obviously, if the person was suicidal and/or left a suicide note, the case becomes relatively easy to solve. Other cases are more challenging. Scene findings and circumstances that suggest the possibility of suicide by motor vehicle collision include a single driver, single vehicle versus fixed object (tree, bridge abutment), with no obvious explanation (intoxication, bad road conditions, etc.) and no evidence of braking, a vehicle that has been noted to be stopped at a crossing for many minutes (waiting for oncoming cross-traffic or a train) prior to pulling-out in front of the traffic or train, or a vehicle stopped on train tracks. Obviously, any of these situations may also occur as an accidental occurrence, so a thorough investigation is necessary to identify those that truly represent suicides.

Another subset of suicides is interesting in that the person employs more than one method of injury infliction. Some refer to these as “complex suicides” and they may be planned or unplanned. In planned complex suicides, the person intentionally uses more than one method, presumably in order to ensure death. An example would be an individual who shoots himself in the head after placing his neck in a noose (Fig. 5.7). In unplanned complex suicides, the person chooses another method after the first method fails. An example would be an individual who cuts their wrists but not deep enough to die so they then attempt to overdose on drugs. After the overdose fails, they jump from a third-story window to ensure their demise.

Homicide – Unlike suicides, where the intent of the individual plays a significant role in determining the MOD, a ruling of homicide *does not necessarily require* evidence that the individual who committed the homicide actually intended to do so. Therefore, if an armed robber fires a “warning shot” at the floor and the bullet ricochets, strikes, and kills a person, the proper ruling is “homicide.” It does not matter that the robber didn’t really intend to kill someone – the fact is that they

Fig. 5.7 A planned complex suicide. Note the gun on the floor near the decedent's feet. He shot himself in the temple. The hanging ensured that he did not survive the gunshot wound



did. As with many “rules,” this rationale does not necessarily apply to all situations (see drunk driving description below). Despite the fact that evidence of intent is not necessary in homicides, there are occasional cases that are ruled “homicide” where the intent of the assailant actually *does* play a role in determining the MOD. For example, if a person driving a motor vehicle intentionally chases another vehicle with the intent to run that vehicle off the road, and the other vehicle crashes and the driver dies, the case is properly ruled a “homicide.” Another example involves deaths that result from arson; a person may intentionally start a fire, but not intend to kill anyone, yet someone ends up dying from the fire. Such cases are appropriately ruled “homicides.”

If a person has severe underlying natural disease (usually heart disease, with an increased susceptibility to develop a fatal arrhythmia), and that person is the victim of a criminal act, such that the stress of the criminal activity induces a mechanism of death that is ultimately related to the underlying natural disease, it is appropriate to rule such a death as a homicide. The term “homicide by heart attack” is sometimes used to describe such a case. This topic is discussed in greater detail in Chapter 21.

Occasionally, a case is encountered in which the condition of the body is such that a definite cause of death cannot be established, but the circumstances surrounding the death are such that the case is obviously homicidal. For example, a female body is found in a building that is determined to have been intentionally set on fire. The body is on its back and is so badly burned that virtually no soft tissue remains in the anterior (front) neck region; however, a ligature remains tightly binding the wrists together behind the decedent's back. It is determined that the victim was dead prior to the fire (see Chapter 19 – Burns and Fire-Related Deaths), but no cause of

death is identified at autopsy. The victim may have been strangled or may have had their neck slashed or perhaps some other mechanism of neck trauma had been used to kill them. Because of the condition of the body, a definite cause of death cannot be determined; however, the case is obviously a homicide. In such cases, it is appropriate to rule the cause of death as “homicidal violence,” and the manner of death as “homicide.” Besides burns, decompositional changes and sometimes massive postmortem body trauma may mask the cause of death in such cases.

Cases that are particularly difficult to make MOD rulings on include cases where acts of omission or carelessness result in the death of another person. In many of these case types there exists a reasonable argument for making a ruling of homicide; however, in some, by convention, “accident” is used. The most common example is a motor vehicle collision fatality related to someone else’s drunk driving. Many of these deaths unquestionably represent “deaths at the hands of others,” yet, for a variety of reasons, such cases are considered accidental. Despite the MOD ruling in such cases, most states have laws which specifically deal with fatalities related to drunk driving. In other case types, arguments may be made for ruling a death an accident or a homicide. Consider the case of an infant who is accidentally left unattended in a car seat in a very hot automobile who succumbs to hyperthermia. Some would argue that the case represents a homicide due to negligence while others would argue that the case represents an unfortunate accident.

Pedestrians who die after being hit by a motor vehicle, wherein the motorist flees from the scene are commonly referred to as “hit and run” deaths. By convention, some forensic pathologists rule these deaths as “homicides” while others, by convention, rule such cases as “accidents” unless it can be shown that the driver intentionally hit the pedestrian.

Other MOD Issues – The National Association of Medical Examiners (NAME) has published guidelines regarding MOD rulings. Some general considerations are described here, but the reader is referred to the NAME guidelines for a more detailed explanation.

- If more than one COD is listed, and each has a different MOD, a non-natural MOD takes precedence over a natural MOD. In a like fashion, a suicide MOD should take precedence over accident, and a homicide MOD should take precedence over all others.
- A death related to the acute (quick) toxic effects of drugs taken during recreational drug use activity is typically considered an accident.
- A death related to the chronic (long term) effects of drug abuse is typically considered a natural death.

Another issue that occasionally arises occurs when one is attempting to decide whether or not a specific event played a role in a particular death when death occurs some time after the original event. There are several possibilities with regard to this issue. First, if a person was directly injured by the event, and subsequently dies as a result of the injury, even if it is years or decades later (as in the example above within the “cause of death” section where a person died many years following a gunshot

wound of the spine), the MOD ruling should reflect the circumstances of the original injury (in the example, therefore, the death would be considered a homicide). Long-term survival following a suicidal act, with death ultimately related to the remote act, represents a difficult situation for some regarding MOD ruling. Logically, to remain consistent, if it is appropriate to rule cases as “homicides” many years after the event, it is also appropriate to rule cases as “suicides” many years after the event.

If there is a sufficient intervening process that “breaks the chain” connecting the original event from death, then it is *not* appropriate to conclude that death was a result of the original event. For instance, if the paraplegic man described above died of sepsis but had no kidney infection, and instead had infected heart valves resulting from his use of illegal intravenous drugs, then the death is more appropriately considered as resulting from complications of drug use, and the manner of death should not be homicide. Many would rule such a death as a natural death, since it was due to the long term effects of drug abuse. In another example, a pedestrian is struck by a car and sustains minor injuries but 10 days later, he is found dead in bed. The autopsy is negative, but toxicology shows lethal levels of ethanol in his blood. The injuries sustained in the motor vehicle versus pedestrian event had nothing to do with death. The acute ethanol intoxication represents a sufficient explanation for death.

In some cases, the timing of the injury in relation to the actual event itself may play a role in MOD determination. In other words, if a person is injured *after* the original event has ended, yet the injury is indirectly related to the event, some forensic pathologists consider this time lag a sufficient intervening event. Consider the following example. If a criminal intentionally sets a house fire in an attempt to kill those residing in the home, and a firefighter perishes while attempting to extinguish the fire (or if the firefighter is badly burned and eventually dies from his injuries months later), it is reasonable to consider such a death a homicide, as the death/injury actually occurred during and as a result of the event (the fire). If, several days later, after the fire is extinguished, a demolition worker falls through the floor of the burnt home and suffers lethal injuries, the MOD may be better ruled as an accident (although some might still rule this a homicide). In this example there was a sufficient intervening period of time between the event and the injury/death such that the two events could be considered separate, despite the fact that one could argue that “but for” the fire, the demolition crew would not have been working on the home. Depending on the event that is responsible for a death or a future death, it may be difficult or debatable as to when an acute event officially ends and when a sufficient amount of intervening time has elapsed in order to separate a particular injury or death from the original event.

It is relatively common for forensic pathologists to investigate cases where an injury occurs in someone who has significant underlying natural disease, and the person eventually dies, perhaps days, weeks, or even months after the injury. In such cases, the question is whether or not the injury played a contributory role in death. If it is determined that the injury contributed to death, then the MOD would be accident (or homicide or suicide); however, if the injury did not contribute, then the MOD would be natural. The basic rule of thumb that many forensic pathologists

use involves an attempt to determine the “baseline” health and activity level of the individual prior to the injury. Establishing this baseline involves interviewing health care workers and perhaps others, and reviewing relevant medical records. The forensic pathologist then attempts to determine whether or not the individual improved following the injury such that he/she made it back to his/her baseline status prior to death. If the person returned to baseline status prior to death, then it is reasonable to conclude that the injury did not play a role in the death. On the other hand, if the person never returned to baseline status, but ended up dying before fully recovering from the injury, then it is reasonable to conclude that the injury did, in fact, contribute to death, if not directly, then certainly as an added stressor for someone with underlying significant natural disease. When a person’s baseline status is steadily headed downward prior to the injury, and the person showed definite signs of improvement from the injury prior to death, but not back to the baseline that existed immediately before the injury, then it becomes very difficult to make a definitive ruling, because one cannot be sure of where the person’s baseline would have been if there had not been an injury.

In certain jurisdictions, other options are available for MOD rulings. In some regions, a *therapeutic complication* MOD is available to use for deaths that result from the adverse effects of medical therapy. Readers are referred to Chapter 21 for further discussion of death related to medical therapy. In some jurisdictions, there is an *unclassified* MOD, which is used for recreational drug abuse deaths, since neither *suicide* nor *accident* (an unforeseen event) are very good descriptors of these deaths. In the majority of jurisdictions, these other MOD options are not available for use on death certificates.

Another important point regarding MOD rulings deserves reiteration. Manner of death rulings should be considered medical opinion statements used for statistical purposes. No entity (insurance companies, prosecutors, courts, etc.) is legally bound to agree with such rulings, and the terms *accident* and *homicide* should not be equated with various legal terminology. For example, a MOD ruling of *homicide* does not necessarily equate to the legal term *murder*. Specifically with regard to the example provided above within the “cause of death” section wherein a man was forcibly restrained by police officers, if the restraint is considered contributory to death, even if underlying cocaine-induced excited delirium is considered the major factor in death, it is appropriate to rule such a death a “homicide.” This does not necessarily mean that the police were “out of line,” but only that the police officers’ actions were considered contributory to death. Certainly, in a similar scenario, wherein a person resisting arrest is subsequently shot and killed by police officers, there is no question that such a case represents a homicide. Whether or not it represents a “murder” or whether or not such a shooting is “justified” is not for the medicolegal death investigation community to decide. Such questions and their subsequent answers are best left to the courts.

In addition to the fact that no one is required to necessarily agree with or be bound by a MOD ruling, another fact regarding MOD rulings is that a variety of case types do not “fit nicely” into a particular MOD category. As such, there can be considerable debate regarding how to appropriately rule the MOD in certain case types. For

these reasons, some within the forensic pathology community have suggested that the MOD part of the death certification process be eliminated.

Disc Image Legends

Disc Image 5.1 Examples of other types of medical questions asked on death certificates.

Disc Image 5.2 Funeral homes must provide specific information about the funeral home itself, as well as the disposition of the body.

Disc Image 5.3 Examples of other information required to be completed by the funeral home.

Selected References

- Cingolani M, Tsakri D. Planned complex suicides: report of three cases. *Am J Forensic Med Pathol* 2000;21:255–60.
- Davis GG. Mind your manners. Part I: history of death certification and manner of death classification. *Am J Forensic Med Pathol* 1997;18:219–23.
- Davis JH. Can sudden cardiac death be murder? *J Forensic Sci* 1978;23(2):384–7.
- Gill JR, Goldfeder LB, Hirsch CS. Use of “therapeutic complication” as a manner of death. *J Forensic Sci* 2006;51:1127–33.
- Goodin J, Hanzlick R. Mind your manners. Part II: general results from the National Association of Medical Examiners manner of death questionnaire, 1995. *Am J Forensic Med Pathol* 1997;18:224–7.
- Hanzlick R, Goodin J. Mind your manners. Part III: individual scenario results and discussion of the National Association of Medical Examiners manner of death questionnaire, 1995. *Am J Forensic Med Pathol* 1997;18:228–45.
- Hanzlick R, Hunsaker JH, Davis GJ. A guide for manner of death classification. St. Louis, MO: National Association of Medical Examiners, 2001.
- Hanzlick RL Medical Certification of Death and Cause-of-death Statements. In: Froede RC, editor. *Handbook of Forensic Pathology*, 2nd Edition. College of American Pathologists, Northfield, IL; 2003.
- Hanzlick R Death Certification. In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. College of American Pathologists; Northfield, IL; 2006.
- Hirsch CS, Flommenbaum M. Problem-solving in death certification. *ASCP Check Sample* 1995;FP95-1:1–31.
- U.S. Standard Death Certificate. <http://www.cdc.gov/nchs/data/dvs/DEATH11-03final-acc.pdf>. Viewed on 7/23/08.

Chapter 6

Overview of Anatomy and Physiology

For you created my inmost being; you knit me together in my mother's womb. I praise you because I am fearfully and wonderfully made.

Psalm 139:13–14

Abstract Many persons consulting this book may not possess a working knowledge of basic human anatomy and physiology. Chapter 6 provides a basic overview of these important concepts and begins with some general information regarding gross anatomy, microscopic anatomy (histology), and physiology. The remainder of the chapter provides a more in-depth presentation of various body regions and compartments, as well as specific organ systems.

Keywords Anatomy · Physiology · Histology · Human body

Introduction

Overview

There are many excellent reviews, textbooks, and resources available regarding human anatomy and physiology. It is certainly beyond the scope of this textbook, let alone a single chapter within the textbook, to provide a thorough explanation of these very important topics. Most current forensic pathology texts are written specifically for pathologists or forensic pathologists, and therefore do not include “basic” information. However, since this textbook is specifically written for non-pathologists, it is appropriate to include a brief description of human anatomy and physiology, in order to provide a basic knowledge base for readers who have little or no medical background. This basic knowledge should allow the reader to better understand subsequent chapters within this textbook. The chapter provides basic descriptions related to the fields of anatomy and physiology, as well as basic anatomy and physiology descriptions of the human body, based on various organ systems.

Gross Anatomy

The term “gross” has at least two meanings when applied to medicine, anatomy, and pathology. The most appropriate definition, and the one that is applicable here, refers to anything that is visible to the naked eye. Hence, “gross anatomy” refers to the various parts of the human body, both externally and internally, that can be seen with the naked eye. The second definition is a relatively slang usage of the word, where the word is meant to describe something that is disgusting or nasty. The field of anatomy deals with the examination and study of the structure, or *morphology*, of things. How is something put together? What is its shape? How does it fit and work together with other things? Most people have at least some amount of knowledge regarding gross anatomy; however, as with many terms within medicine, there are often medical or scientific names that are applied to common names of anatomical structures. For example, what most people refer to as the skull is also known as the “cranium”, and an arm is an “upper extremity.” Just as there is a scientific language related to the naming of body parts, physicians frequently use other descriptive scientific terms when discussing or describing patients, or in the case of forensic pathology, injuries or dead bodies. For example, the “anterior” part of something means the front part, while “posterior” refers to the back. Table 6.1 provides a list of scientific and common names or descriptions for various anatomic structures while Table 6.2 provides definitions for many other medical/scientific terms.

Table 6.1 Scientific names and common names or descriptions

Scientific name	Common name or description
Abdomen	The part of the trunk below the chest and above the pelvis
ACA	Anterior cerebral artery; arteries supplying the front of the brain
ACF	Anterior cranial fossa; the anterior-most part of the basilar skull
Actin	A myofilament; allows muscles to contract
Adipose	Fat
Adnexa	Specialized parts of the skin or female organs
Alveoli	Tiny air sacs within lungs; where oxygen from air enters blood and carbon dioxide exits blood into air
Ampulla of Vater	An area within the duodenum where bile and pancreatic enzymes are excreted into the intestine
Amylase	An enzyme that digests carbohydrates; produced by salivary glands and the pancreas
Antecubital fossa	Inner bending part of elbow
Anterior iliac crest	Front of hip bone, at the belt line
Anus	The exit point of the GI system, located between the buttocks
Aorta	Large artery arising from the heart
Aortic valve	A heart valve with three semilunar-shaped valves, separating the left ventricle from the aorta
Appendix	A small, worm-like extension attached to the cecum
Arachnoid	A thin, translucent membrane overlying the brain
Arterioles	Small arteries, just before capillaries
Artery	A blood vessel that takes blood away from heart
Ascending colon	The second part of the large intestine

Table 6.1 (continued)

Scientific name	Common name or description
Atrium	An upper chamber of the heart (plural: atria)
AV node	Atrioventricular node; an internal pacemaker of the heart
Autonomic	An involuntary part of the nervous system
Axilla	Armpit
Axon	Extensions from neurons, allowing signals to be transferred between neurons
Basal ganglia	A deep gray matter structure within the brain
Basement membrane	A structure to which epithelial surfaces bind
Basilar artery	An artery formed by the merging of the two vertebral arteries adjacent to the brainstem
Basophil	A type of white blood cell
Bile	Product of the liver; stored in the gallbladder; assists with digestion
Biliary	Having to do with bile or the biliary tract
Brainstem	Connects the cerebrum, cerebellum, and spinal cord; has many primary functions, including respiration
Bronchiole	Small air tubes in the lungs, connecting bronchi to alveoli
Bronchus (bronchi)	Air tubes in lungs, connecting the trachea to the alveoli
Capillary	The smallest blood vessel type, between arteries and veins
Carina	Where the trachea divides into the right and left mainstem bronchi
Carotid artery	A neck artery, carrying blood from heart toward face and brain
Carpals	Wrist bones
Cartilage	A rubbery component of the skeletal system; frequently occurring where bones join one another (at joints)
Cecum	The first part of the large intestine
Celiac artery	An artery that arises from the aorta and supplies blood to the stomach and other parts of the GI tract
Cell membrane	The border of a cell
Cerebellum	A small part of the brain located in the lower back region, involved in balance and other lower functions
Cerebrum	The largest, highest-functioning portion of the brain
Cervical	The neck portion of the vertebral/spinal column and spinal cord
Cervix	The opening of the uterus; where the uterus joins the vagina
Chole	Prefix having to do with the biliary system (liver and gallbladder)
Chromosome	A structure within a cell's nucleus that contains DNA (genes)
Clavicle	Collar bone
CNS	Central nervous system
Colon	Large intestine (large bowel)
Columnar	A type of epithelial cell; example – trachea, intestine
Conduction system	The part of the heart responsible for controlling heart rhythm
Connective tissue	Structural substances; examples: collagen, elastin
Corpus callosum	The white matter connection between both cerebral hemispheres
Cortex	The outer part or layer of an organ
Cortical	Having to do with the cortex (outer portion of an organ)
Cranium	Skull/head
CSF	Cerebral spinal fluid
Cutaneous	Skin (cutis; dermis; epidermis; integument)
Cutis	Skin (cutaneous; dermis; epidermis; integument)
Cx	Cervix

(continued)

Table 6.1 (continued)

Scientific name	Common name or description
Cytoplasm	The contents of a cell, excluding the nucleus
DNA	Deoxyribonucleic acid; the genetic code
Dermis	Skin (cutaneous; cutis; epidermis; integument)
Descending colon	The fourth part of the large intestine
Diaphragm	Umbrella-shaped muscle that separates the chest organs above from the abdominal organs below; its up and down movement allows expiration and inspiration
Diaphysis	The central, long part of a long bone
Diastole	The part of the heartbeat in which the ventricles relax and fill with blood
Digits	Fingers
Duodenum	The first part of the small intestine
Dura mater	A tough membrane adjacent to the inner aspect of the skull
Endocrine pancreas	The portion of the pancreas involved in hormone production
Eosinophil	A type of white blood cell
Epidermis	Skin (cutis; cutaneous; dermis; integument)
Epiglottis	A flap of tissue that covers the opening to the larynx/air passages in order to allow food to enter the esophagus during swallowing
Epiphysis	The part of a long bone that separates the end (metaphysis) from the central long region (diaphysis)
Epithelial	Having to do with the epithelium
Epithelium	Cells that line a surface of something
Erythrocyte	Red blood cell
Esophagogastric junction	Where the esophagus and stomach connect (gastroesophageal)
Esophagus	The “food tube” connecting the throat to the stomach
Ethmoid	A skull bone
Exocrine pancreas	The portion of the pancreas involved in GI enzyme production
Extremities	Arms and legs
Falx cerebri	An extension of dura mater that separates right and left cerebral hemispheres
Femur	Thigh bone
Fibula	One of the two bones of the lower leg (other: tibia)
Foramen magnum	The “hole” in the basilar skull where the spinal cord traverses
Frontal	The front portion of the scalp, skull and brain
Gallbladder	Storage reservoir for bile; attached to lower part of liver
Ganglion	A group of neurons, usually located outside the CNS (Pl: ganglia)
Gastric	Having to do with the stomach
Globe	Eyeball (ocular; orbit)
Gyrus	A portion of cerebral cortex as viewed externally (Pl: gyri)
H&E	Hematoxylin and eosin; the usual stain used for histology slides
Hemisphere	One half of the brain
Hepatic	Having to do with the liver
Hepatocyte	Liver cells
Hilum	The central portion of an organ, where vessels, etc. enter/exit
Histiocyte	A cell of the immune system derived from blood monocytes
Histology	The anatomy of tissue, as viewed with a light microscope
Humerus	Upper arm bone
Hypothalamus	A deep structure within the brain; connects to the pituitary gland

Table 6.1 (continued)

Scientific name	Common name or description
Ilium	The third and final part of the small intestine
IMA	Inferior mesenteric artery; supplies blood to lower GI tract
Inguinal region	Groin
Integument	Skin (cutis; cutaneous; dermis; epidermis)
Interatrial septum	Separates right and left atria of heart
Intercostals	Muscles between the ribs
Internal capsule	A portion of deep white matter within the brain
Interventricular septum	Separates right and left ventricles of heart
IVC	Inferior vena cava; returns blood from lower body to heart
Jejunum	The second part of the small intestine
Jugular vein	A neck vein, carrying blood from brain toward heart
Keratin	The outer covering of the skin
Keratinocyte	The name used to describe skin epithelial cells (epidermis)
LA	Left atrium of heart
LAD	Left anterior descending coronary artery
Large intestine	Cecum, ascending colon, transverse colon, descending colon, sigmoid colon, rectum
Larynx	Voicebox
LCA	Left coronary artery
LCx	Left circumflex coronary artery
Leukocyte	White blood cell
Ligament	A structure that connects one bone to another bone
Lipase	An enzyme that breaks down fat; produced by pancreas
Lymphatics	Vessels that drain lymph from soft tissues back into blood vessels
Lymphocyte	A type of white blood cell; two types: B and T
Lumbar	The lower-most part of the spinal column and cord
LV	Left ventricle of heart
Macrophage	A type of inflammatory cell; arise from blood monocyte
Mandible	Jaw bone
MCA	Middle cerebral artery
MCF	Middle cranial fossa; the middle portion of the basilar skull
Medulla	(1) The inner portion of an organ; (2) the lowermost part of the brainstem
Medullary	Having to do with the medulla of an organ
Melanin	Skin pigment
Melanocytes	Specialized skin cells that produce melanin pigment
Meninges	Brain membranes or coverings (dura, arachnoid and pia mater)
Metacarpals	Hand bones
Metatarsals	Foot bones
Midbrain	The uppermost part of the brainstem
Mitral valve	A two-cusp heart valve separating the left atrium and ventricle
Monocyte	A type of white blood cell; gives rise to tissue macrophage
Motor nerves	Nerves that transmit signals away from the CNS
Mucous membrane	Certain internal epithelial surfaces
Myocyte	A muscle cell
Myofilament	The molecules that allow muscles to contract (actin and myosin)
Myosin	A myofilament; allows muscles to contract
Nares	Nostril

(continued)

Table 6.1 (continued)

Scientific name	Common name or description
Nasal ala	Outer edge of nose, outside of nostril
Nasopharynx	Internal portion of nose and throat
Neuron	Brain parenchymal cell
Neutrophil	A type of white blood cell; also called a granulocyte or a PMN
Nuclear membrane	The border surrounding the nucleus of a cell
Nuclei	More than one nucleus; groups of neurons in the spinal cord
Nucleus	The part of a cell that contains DNA (chromosomes; genes)
Occipital	The back portion of the scalp, skull and brain
Ocular	Having to do with the eye (globe; orbit)
Orbit	Eyeball (globe; ocular)
Organ	A group of tissues and cells organized into a grossly evident structure having a specific function
Oropharynx	Mouth and throat
Osteoblast	Bone-forming cells
Osteoclast	Bone-resorbing cells
Osteocyte	Bone cell
Parasympathetic	The “rest and relaxation” part of the autonomic nervous system
Parenchyma	The major functioning cells within an organ
Parietal	The upper side portion of the scalp, skull and brain
Patella	Kneecap
PCA	Posterior cerebral artery
PCF	Posterior cranial fossa; the rear-most portion of the basilar skull
Pelvis	The lowest part of the trunk; where the legs attach; also used to describe the bones of this region
Perineum	The area in front of the anus
Peritoneal cavity	Abdominal cavity
Phalanges	Finger bones and toe bones
Pharynx	Throat
Phrenic nerve	A nerve that innervates the diaphragm
Pia mater	A thin membrane adherant to the outer brain
Pinna	Outer part of ear
Pituitary	The master gland
Plasma cell	A mature B-lymphocyte; produces antibodies
Platelet	A type of blood cell involved in the clotting process
Pleural	Having to do with the outer lining of the lungs
PMN	Polymorphonuclear cell; a type of white blood cell; neutrophil
PNS	Peripheral nervous system
Pons	The middle part of the brainstem
Popliteal fossa	Inner bending part of knee
Portal triad	Microscopic part in liver, composed of branches of hepatic artery, portal vein, and bile duct
Portal vein	A blood vessel that transports nutrient-rich blood from the intestines to the liver
Proprioception	Brain’s recognition of body part position
Protease	An enzyme that breaks down proteins
Pubic symphysis	Front attachment of the two sides of the bony pelvis
Pulmonary	Having to do with the lungs (respiratory)
Pulmonary artery	Large artery taking blood from heart to lungs

Table 6.1 (continued)

Scientific name	Common name or description
Pulmonary valve	A valve of the heart containing three semilunar-shaped valves, separating right ventricle and pulmonary artery
Pulmonary vein	A blood vessel that transports blood from the lungs to the LA
Pylorus	The connection between the stomach and duodenum
Radius	One of the two bones of the forearm (other: ulna)
RCA	Right coronary artery
Rectum	The final part of the large intestine, just before the anus
Respiratory	Having to do with the lungs (pulmonary); breathing
Reticuloendothelial	Having to do with lymph nodes, spleen, and bone marrow
Retroperitoneal	Behind (posterior to) peritoneal cavity
SA node	Sinoatrial node; one of the heart's internal pacemakers
Sarcomere	The ultrastructural functional unit of skeletal muscle
Scapula	Shoulder blade
Sella turcica	The depression in the basilar skull that contains the pituitary
Sensory nerves	Nerves that transmit toward the CNS
Sigmoid colon	The fifth part of the large intestine, just before the rectum
Sinusoids	Vascular spaces that run adjacent to hepatocytes in the liver
Small intestine	Duodenum, jejunum, and ileum
Sphenoid	A bone of the skull; contains a sinus cavity
Spine	Backbone
Spleen	An abdominal organ located in the upper left peritoneal cavity
Squamous	A type of epithelial cell; example: skin, esophagus
Sternum	Breastbone
Stroma	The supporting, background, structural cells within an organ
Subarachnoid space	Underneath meninges, but outside of brain; contains CSF
Subclavian	Underneath (behind) the clavicle (collarbone)
Subcutis	Adipose tissue under the skin
Subcutaneous	Under the skin
Subgaleal	The soft tissue of the scalp, under the skin but outside of the skull (subscalpular)
Subscalpular	The soft tissue of the scalp, under the skin but outside of the skull (subgaleal)
Sulcus	A depression that separates one cerebral gyrus from an adjacent gyrus (Pl: sulci)
SMA	Superior mesenteric artery; supplies blood to GI tract
SVC	Superior vena cava; returns blood from upper body to heart
Sympathetic	The "fight or flight" part of the autonomic nervous system
Systole	The part of the heartbeat in which the ventricles contract
T-tubules	Allow electrical signal transduction in heart
Tarsals	Ankle/foot bones
Temporal	The lower side portion of the scalp, skull and brain
Tendon	A structure that connects a muscle to a bone
Tentorium	A portion of the dura mater that separates the cerebrum from the underlying cerebellum
Thalamus	A deep gray matter structure within the brain
Tibia	Shin bone (one of two bones of lower leg; other: fibula)
Tissue	A group of several cell types
Thoracic	Having to do with the chest region; spinal column attached to ribs
Thorax	Chest

Table 6.1 (continued)

Scientific name	Common name or description
Thromboembolus	A thrombus that breaks away and travels within the blood to another location
Thrombus	A blood clot
Trabeculae	Scaffold-like connections; seen in various structures (bone, spleen)
Trachea	Windpipe
Tract	Another name applied to various organ systems; a group of axons
Transverse colon	The third part of the large intestine
Tricuspid valve	Heart valve with three cusps separating right atrium and ventricle
Trunk	The main part of the body, including chest, abdomen, and pelvis
Ulna	One of the two bones of the forearm (other: radius)
Ulnar nerve	The “funny bone”
Ultrastructure	Structures visible only by electron microscopy
Umbilicus	The “belly button”
Upper extremity	Arm
Vagus nerve	A nerve that attaches to various organs of the trunk
Vein	A blood vessel that carries blood toward the heart
Vena cava	Large veins (superior v.c. and inferior v.c.) attached to heart
Ventricles	Cavities within the brain or heart
Venules	Small veins, just after capillaries
Vertebral artery	An artery that supplies blood to the brain; travels along the vertebral column
Vertebral column	Backbone
Zona fasciculata	The middle zone of the adrenal cortex; produces cortisol
Zona glomerulosa	The outermost zone of the adrenal cortex; produces aldosterone
Zona reticularis	The innermost zone of the adrenal cortex; produces sex steroids

Table 6.2 Other medical/scientific terms

Term	Definition
Abberant	Abnormal
Abdominal	Having to do with the abdomen
Abduction	Moving a limb or portion of limb away from the body
Acute	Occurring quickly; rapid (opposite of “chronic”)
Adduction	Moving a limb or portion of limb toward the body
Antegrade	Forward flow or movement (opposite of “retrograde”)
Anterior	Front
Bilateral	Occurring on both sides
Caudal	Toward the bottom
Cephalad	Toward the head
Chronic	Occurring slowly; drawn out (opposite of “acute”)
Coronal plane	A plane that divides the body into front and back parts (also called the “frontal” plane)
Cross-section	A cut through something, usually across its width
Distal	Further away from (opposite of “proximal”)
Evert/eversion	Rotating a limb outward
Extend/extension	Straightening a joint, including bending beyond straight
Fixation	Preservation of tissues, usually with formalin
Fixed	Having undergone fixation (with formalin)

Table 6.2 (continued)

Term	Definition
Flex/flexion	Bending a joint into a more acute angle
Frontal plane	A plane that divides the body into front and back parts (also called the “coronal” plane)
Gross	Able to be seen with the naked eye
Histology	A method for microscopically viewing tissues
Inferior	Below
Inflammation	A process involving many cells, especially white blood cells, whereby the body responds to various stressors or injuries
Invert/inversion	Rotating a limb inward
In situ	Within the body (as opposed to after removal from body)
In vitro	Occurring outside the body (as opposed to in vivo)
In vivo	Occurring within the body (as opposed to in vitro)
Lateral	Away from midline (toward the side)
Longitudinal	Referring to the long axis of something
Lumen	The hollow part of a tube-shaped structure
Midline	A line drawn through the center of something, up to down, dividing it into two equal sides (right and left)
Midsagittal	A plane that divides the body equally into right and left sides
Medial	Toward the midline
Microscopic	Seen using a microscope
Midaxillary line	A line drawn through the middle of the body, dividing in into front and back sections
Morphology	Structure
Mural	Having to do with the wall of an organ or blood vessel
Necrosis	Dead tissue within an otherwise viable (living) organ
Occlusion	Blockage
Physiology	Function
Posterior	Back
Proximal	Closer to (opposite of “distal”)
Regurgitation	Backward flow
Retrograde	Backward flow or movement (opposite of “antegrade”)
Sagittal plane	A plane that divides the body (or part) into right and left parts
Section	A “slice” of an organ; the act of cutting an organ; a portion of very thin tissue on a microscopic slide.
Stenosis	Narrowing or partial occlusion
Subcutaneous	Under the skin
Superficial	Close to the surface (opposite of deep)
Superior	Above
Thoracic	Having to do with the chest region
Transverse	A cross-section

There are several general regions of the body. The head includes the cranial cavity, within which the brain resides, and the oral and nasal cavities. The anterior neck contains the esophagus, larynx, upper trachea, thyroid and parathyroid glands, blood vessels (carotid arteries, jugular veins), and various muscles. The posterior neck includes the cervical vertebral column, the cervical spinal cord, and muscles. The trunk includes the thorax (chest), abdomen, and pelvis. The thorax has two

pleural (chest) cavities, within which the lungs reside, the pericardial cavity, within which the heart resides, and the “mediastinum,” where the thymus, lymph nodes, and portions of the trachea and esophagus reside. The aorta and distal esophagus travel behind the heart toward the abdomen. The diaphragm separates the chest cavities from the abdominal (peritoneal) cavity. The peritoneal cavity contains the stomach, small and large intestine, liver, spleen, and pelvic organs. The “retroperitoneal” area is located behind the peritoneal (abdominal) cavity and contains the pancreas, the kidneys, and the adrenal glands. Pelvic organs in males include the upper aspect of the urinary bladder. In women, the uterus, fallopian tubes, and ovaries are in the pelvic portion of the peritoneal cavity. In males, the prostate gland and seminal vesicles exist between the bladder and the perineum (the area between the legs), right in front of the anus. In females, the vagina connects the uterus and perineum.

Histology

Histology is a term that refers to microscopic anatomy, or the structure (morphology) of something as it appears via a microscope. Specifically, the term “microscope” here refers to the typical “light” microscope, as opposed to an electron microscope, which is used to visualize “ultrastructural anatomy,” meaning that which is smaller than what we can see with a normal light microscope. Under the light microscope, we are able to visualize the cells of the body and, in most instances, we are able to see individual cells, including the cell borders (cell membranes). Most cells in the body contain a single “nucleus” (plural: “nuclei”) which contains DNA and is surrounded by a nuclear membrane (border), while the remainder of the cell substance surrounding the nucleus is referred to as the cytoplasm. Under the microscope, we are able to see cells, including the nuclei and the cytoplasm. There are many different types of cells within our bodies. Anatomists and doctors categorize cell types based on their microscopic appearance and their function. A pathologist is able to determine the type of cell that is seen under the microscope based on the cell’s shape, size, character, location, arrangement with other cells, and various staining characteristics. Pathologists use many different types of stains in order to visualize the cells; some can be very helpful in identifying cell types; the “usual” stain is called hematoxylin and eosin (“H&E”).

When certain cell types are present together in a particular arrangement with one another, we call this configuration a “tissue.” Hence, we can describe various tissue types, such as adipose (fat) tissue, or muscle tissue, or bone, etc. When a variety of tissue types are arranged within a distinct, grossly-evident structure that has specific functions, we call that structure an “organ.” A general term for cells that make up the major functioning cells of a particular organ is “parenchyma cells.” In contrast, a general term to describe the structural, background, supporting cells within a particular organ is “stroma cells.” The term “epithelium” or “epithelial cells” refers to cells that cover the surface of something. Frequently, epithelial surfaces are bound to the underlying tissue via a “basement membrane.” Epithelial surfaces in some locations, such as the oral and nasal cavities, are frequently referred to as

“mucous membranes” and can be made of cells of differing character. Examples include “squamous” and “columnar” epithelium.

Another name that is sometimes applied to a specific organ and any related tissues is the term “system.” Hence, we say that the *cardiovascular system* is composed of the heart (an organ) and the blood vessels (which are composed of vascular tissue). Each organ and tissue has a specific gross as well as histologic (microscopic) appearance. Pathologists rely on their ability to recognize and distinguish normal and abnormal microscopic (and gross) appearances of tissues and organs in order to render diagnoses. When specific recognizable microscopic (and gross) changes are seen, a corresponding specific diagnosis of a disease process can be made.

Physiology

Physiology is a term used to describe how a living organism functions, not only from a structural or anatomic standpoint, but also from a biochemical, nutritional, enzymatic, hormonal, electrical and molecular standpoint. It describes how our cells, tissues, and organs “communicate” with one another, how they interact with each other and their environment, and how they work. While anatomy and histology describe what a particular organ, tissue, or cell looks like (its “morphology”), physiology describes what that organ, tissue, or cell actually does (its function).

While the anatomical appearance of a tissue or organ can sometimes give us an idea of how the particular structure is functioning, this is not always the case. Many diseases have specific anatomic (gross and microscopic) features, as well as physiologic derangements; however, there are some diseases that are essentially purely physiologic. The cells are not functioning normally, but there is no anatomic abnormality that can be identified grossly or microscopically. For this reason, not every cause of death (natural or non-natural) can be identified by gross or microscopic findings at autopsy.

In life, we are frequently able to perform various laboratory tests (on blood, urine, or other fluids or tissues), or other types of tests (electrical), in order to evaluate the physiologic function of an organ. For example, we can perform blood and urine tests to evaluate the function of the kidneys. If the tests are abnormal, we know that something is wrong with the kidneys. We can perform electrocardiograms (ECGs) on living individuals, in order to evaluate the electrical function of the heart. An abnormal ECG may indicate a physiologic abnormality of the heart, even if the heart is structurally (grossly and microscopically) normal. While some laboratory tests remain valid after death, many of them do not, and none of the electrical tests can be performed after death. Consequently, it is much more difficult to ascertain a physiologic abnormality after death than it is to diagnose an anatomic abnormality. From the standpoint of death investigation, it is important to remember that there are some causes of death where there is no “anatomic correlate” (no gross or microscopic structural findings) and no available test to make a definitive diagnosis of the underlying cause of death. For example, in cases of certain cardiac conduction system abnormalities (which cause death by producing abnormal heart rhythms) and some forms of central nervous system (brain) seizure disorders (epilepsy), we

have no postmortem laboratory test available to make a diagnosis. Determining the cause of death in these cases requires knowing the medical history of the person (for example, knowing that the person had a history of epilepsy) and then ruling out all other causes of death.

Body Regions and Compartments

From an anatomic standpoint, the human body can be divided into various regions, each of which contains structural supporting elements (bone, cartilage, and connective tissue), muscles, joints and ligaments that allow for movement, and blood vessels that supply oxygen and nutrition. The head region includes the intracranial cavity, within which the brain resides, as well as the sinuses, the nasal cavity, the oral cavity, and the pharynx (the back of the throat, where nasal and oral cavities merge). The neck contains numerous structures, including blood vessels, structures that are part of the gastrointestinal system and the respiratory system, and a portion of the vertebral column and spinal cord. The remainder of the body can be divided into the extremities (two upper extremities or arms; two lower extremities or legs) and the trunk region.

The trunk includes everything other than the head and neck and the extremities. There are four body cavities contained within the trunk (Disc Images 6.1 and 6.2). The dome-shaped diaphragm muscle separates the chest cavities above from the abdominal, or peritoneal, cavity below. The chest cavities include two pleural, or thoracic, cavities, within which the lungs reside, and the pericardial cavity (the heart sac), within which the heart resides. The peritoneal (abdominal) cavity contains most of the remainder of the trunk organs, including the liver, spleen, stomach, and a large portion of the intestines. The lowermost portion of the peritoneal cavity is referred to as the pelvis region.

Not all of the organs within the trunk region actually reside within the body cavities. Some, in fact, are behind the actual cavities themselves. Within the chest, the “mediastinum” is an area above the pericardial sac and medial (towards the middle) to the pleural cavities. The mediastinum contains the aorta, pulmonary arteries, trachea, mainstem bronchi, thymus, and a portion of the esophagus. Within the abdomen and pelvis, the kidneys, adrenal glands, most of the pancreas, portions of the intestines, and the aorta and inferior vena cava are actually behind the peritoneal cavity, in what is considered the “retroperitoneum” (which literally means behind the peritoneum).

Specific Organ Systems

Integumentary System (Skin)

Although it may seem a bit unusual to consider the skin (the integument) as an organ, the skin can certainly be considered an organ, since it represents a specific grouping of cells and tissues that exists in a specific location and performs specific

functions. In fact, the skin is the largest organ system in our bodies and the term “cutaneous” is sometimes used to describe it. Grossly, we are all familiar with skin. There are different types of skin: thick skin occurs on the palms of the hands and the soles of the feet and hair-bearing skin occurs in many locations, particularly on the scalp, in the axilla (armpit), and in the groin region, but also in other locations, with some differences between male and female hair growth patterns.

Microscopically, skin has two basic parts, separated by a basement membrane. The most superficial (close to the surface) part is called the “epidermis” which is an epithelial surface made-up of multiple layers of squamous cells referred to as keratinocytes. At the very upper surface of the skin, the keratinocytes convert into a protective layer of keratin. Skin cells are constantly growing, with lower layers of cells (just above the basement membrane) moving upward, becoming keratinized, and then sloughing-off. The epidermis also contains various other cells, including melanocytes, which produce melanin pigment, which is responsible for giving skin its pigmentation.

Underneath the epidermis is the “dermis.” This contains connective tissue (collagen, elastin, etc.), blood vessels, nerve fibers and sensing devices, and structures referred to as the skin “adnexa,” which are actually parts of the epidermis that have grown downward into the dermis. The adnexal structures include things like hair follicles, sweat glands, and other glands. The “subcutis” refers to the soft tissues (primarily fat, or adipose, tissue) that underlie the dermis. Strictly speaking, this is distinct from the skin, and it is frequently referred to as “subcutaneous tissue.” It can be thought of as an interface between the overlying skin and the underlying musculoskeletal tissues. Figure 6.1 shows the microscopic appearance of the epidermis and part of the underlying dermis.

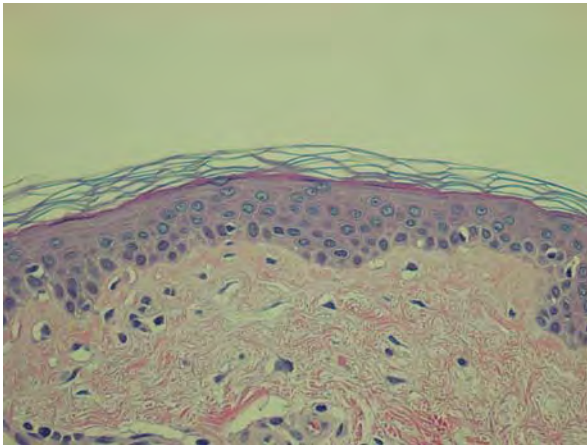


Fig. 6.1 A high-power microscopic view of the skin

The skin carries out many important functions. It is a protective barrier against all sorts of external elements (sun, germs, toxins, other injurious agents) and it

is an important aid in temperature and body water regulation. Various metabolic processes, such as vitamin D metabolism, require sunlight via the skin. The skin is also vital for various bodily functions that many of us take for granted, such as the sense of touch and the ability to move and interact with others and our environment.

In the practice of forensic pathology, the skin is extremely important. Much of what we refer to as the “external examination” portion of an autopsy involves evaluation of the skin. We may use permanent features of the skin, such as pigment characteristics, scars, and tattoos, in identifying a body. There are postmortem changes that involve the skin, such as livor mortis or lividity (settling of blood), and various decompositional changes. The skin is frequently the site of specific injuries and its response to various injurious forces results in specific injury patterns. Recognition of these patterns allows forensic pathologists to categorize the injury into a specific type. Examples include blunt force injuries (abrasions, lacerations, contusions), thermal injuries (burns), electrical injuries, gunshot wounds, etc.

Musculoskeletal System

Many textbooks consider the musculoskeletal system as two separate entities, the muscular system and the skeletal system, but for this introductory presentation, they will be considered together. The bones represent the skeletal component of the musculoskeletal system. They provide support and protection for the rest of the body, and they allow movement via the presence of moveable joints that separate yet connect one bone to another. Bones are very active metabolically, particularly when it comes to calcium metabolism; they also contain bone marrow, where blood cells are produced. Certain specialized bones are necessary for our sense of hearing.

From a gross standpoint, bones can be “long bones,” such as the bones of our arms and legs, or they can be “flat bones,” such as the bones of our skull. The end of a long bone is referred to as a metaphysis, the long central part is called the diaphysis, and where the two connect is called the epiphysis (Fig. 6.2). It is within the epiphysis (the epiphyseal plate) that long bone growth occurs during childhood and into adolescence (Disc Image 6.3). In growing bones, the epiphyseal plates remain “soft” and are evident grossly as well as by X-ray. However, once a bone stops growing, the epiphyseal plates become calcified (hardened) and incorporated into the rest of the bone. With certain flat bones, such as those that occur in the skull, several bones actually fuse as a person grows older to form a single skull (the skull is actually made up of frontal, temporal, parietal, occipital, sphenoid, ethmoid, and other bones) (Fig. 6.3). Whether they are long bones or flat bones, a bone’s outer solid, hard surface is referred to as the cortex (cortical bone), and the inner part of a bone is referred to as medullary bone. Medullary bone is composed of a scaffold-like arrangement of thin but hard structures called trabeculae that surround the bone marrow spaces. It is also called “spongy” bone, since it is reminiscent of what a sponge looks like. Bone marrow (containing blood-producing cells and fat) fills the spaces that exist within medullary bone.



Fig. 6.2 An example of a long bone

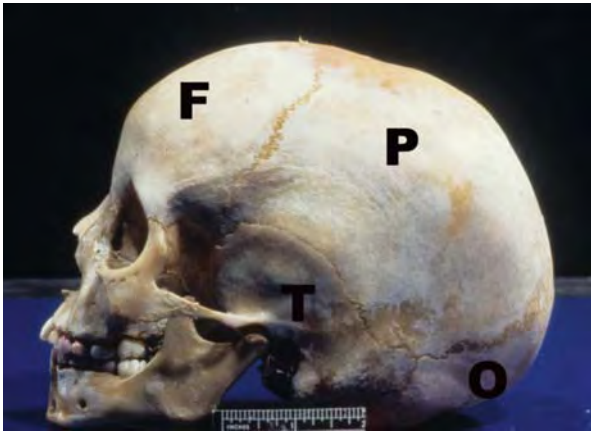


Fig. 6.3 A child's skull, showing the bones that fuse with one another

Pathologists are able to examine bones under the microscope, however, because bones are so hard as a result of their calcium content, a bone must first undergo a chemical “decalcification” process before microscopic slides can be made. Cortical bone and medullary bone have specific appearances under the microscope (Disc Image 6.4). As mentioned earlier, bone is very active metabolically, with constant “turnover,” meaning that existing bone is constantly being “absorbed” and new bone is constantly being formed. The cells that exist within existing bone are called osteocytes, the cells that are actively absorbing bone are called osteoclasts, and the cells that are actively producing new bone are called osteoblasts.

Bones may be injured in certain cases that are examined by forensic pathologists. The typical injury is referred to as a fracture (a broken bone) and in some forms of injury, the specific characteristics of the fracture can provide information regarding the direction of force that produced the injury, or perhaps the circumstances that resulted in the fracture. A healing fracture can be evaluated microscopically in an attempt to determine how old it is. Occasionally, bones can contain weapon-specific toolmark impressions and subsequent comparison of these marks with a suspect

weapon may allow for a toolmark examiner to conclude that the weapon was used in an attack. Another area where bone examination is important within forensic pathology involves the identification of an unknown individual. The typical scenario involves a body that is badly decomposed, or perhaps even skeletonized (having little or no remaining soft tissues). In such cases, anthropologic examination can aid in attempting to determine the gender, age, and racial/ethnic nature of the bones (refer to Chapter 9).

Bone is not the only part of the skeletal system. Cartilage is a rubbery tissue, somewhat similar to bone, that is present in certain locations requiring firmness but also flexibility (ears, nose, trachea, larynx). It is also a major component of joints (where bones “connect” with one another) and provides a rubbery, “shock-absorber”-like function (Disc Image 6.5). Injured cartilage is occasionally collected for subsequent examination when various toolmark impressions from a weapon might exist (for example in stabbing deaths where a stab wound cuts through the cartilaginous portion of a rib).

Firm, connective tissue bands that connect one bone to another are called “ligaments.” They are flexible enough to allow movement, yet firm, or tight enough to prevent too much movement. When a ligament stretches too far, it results in a “sprain” of the particular joint involved.

The muscles of the musculoskeletal system are specifically designated as “skeletal muscles,” to distinguish them from two other muscle types that occur in our bodies, cardiac (heart) muscle and smooth muscle (which occurs in blood vessels and other internal organs). Skeletal muscles function to allow movement of the skeletal system, and thus, the various parts of our bodies. Muscles are attached to bones by structures called “tendons” and when a muscle contracts, it shortens, thus moving the bones around a particular joint. Usually there are muscles with opposing actions around each joint. With regard to the extremities, muscles that function to bend a joint are called “flexors,” while those that function to straighten a joint are called “extensors.” Grossly, skeletal muscles have a red, meat-like appearance (Fig. 6.4).



Fig. 6.4 Gross appearance of skeletal muscles at autopsy. The lighter-colored areas represent fat

Microscopically, muscles consist of fascicles, which are bundles of muscle fibers. Each muscle fiber can be thought of as many cells that have coalesced together into one long fiber containing numerous nuclei along its periphery. Within the center of the fiber, there are numerous end-to-end ultrastructural elements called sarcomeres, which contain alternating bands of myofilaments and other structures. The myofilaments, “actin” and “myosin,” are able to slide next to each, allowing shortening (contraction) of the entire muscle fiber. When numerous muscle fibers within an entire muscle contract, the entire muscle itself contracts. Pathologists are able to visualize “striations” (under the microscope) that are characteristic of muscle (Fig. 6.5); these correspond to different zones of actin and myosin within the end-to-end sarcomeres which make-up the muscle fibers.

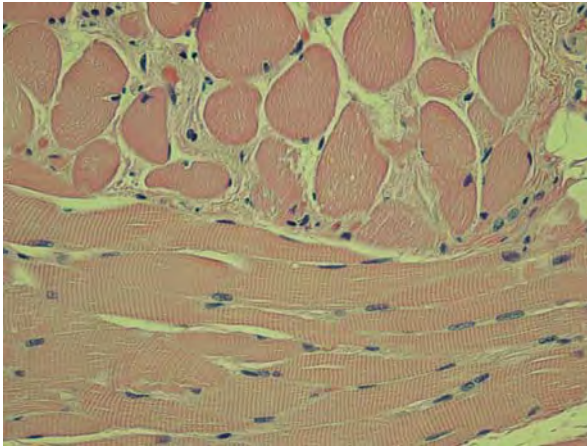


Fig. 6.5 Microscopic appearance of skeletal muscle. Cross sections of multiple fibers are seen towards the *top of the photo*, while longitudinal sections are seen toward the *bottom*

As with the skeletal system, the muscular system is frequently injured in certain case types that forensic pathologists examine. Injuries are characterized grossly by the presence of hemorrhage within the muscles which is evident as an area of dark to bright red, bloody muscle, distinct from the less bloody areas surrounding it.

Nervous System

The nervous system can be divided into the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system (PNS), which includes the nerves of the body. Virtually every part of the body is innervated (meaning that each area of the body has nerves attached to it). Innervation allows our various body parts to function normally. In general, there are two basic forms of nerve signals: those that travel from an area of the body toward the CNS, providing information to the CNS (these may be called sensory nerve signals), and those that

travel from the CNS toward a body part and cause that part to perform a particular function (these may be called motor nerve signals). Another useful categorization of the nervous system divides the system into voluntary and involuntary pathways. The voluntary pathways are those nerve signals that we can purposefully control, such as arm and leg movement, eye movement, speech, etc. The involuntary pathways are those that function without us having to think about them, such as reflexes, gastrointestinal tract function (movement of intestines), diaphragm function (breathing), etc. Part of the involuntary nervous system is referred to as the autonomic nervous system, and can be further divided into the sympathetic and parasympathetic systems. In very general terms, the sympathetic system can be considered the “fight or flight” system, increasing heart, skeletal muscle, and lung capabilities, while the parasympathetic system relates to rest and relaxation, slowing or inhibiting the aforementioned systems and stimulating other systems, such as the gastrointestinal and genitourinary systems. There is obviously some degree of overlap between the voluntary and involuntary systems, such that with some functions, we can voluntarily “override” the involuntary, at least to an extent (holding our breath).

From outside to inside, the head is composed of the scalp, including skin and underlying fat and muscle, the skull (bone), and the dura mater, which is a thin but tough membrane that is normally adherent to the inside of the skull (Fig. 6.6). Underlying the dura is the subdural space (really a potential space), which is traversed by “bridging veins,” the disruption of which can cause a “subdural hemorrhage” (Fig. 6.7). Immediately overlying the brain is a thin membrane known as the arachnoid membrane (Figs. 6.8 and 6.9) and cerebral spinal fluid circulates under the arachnoid membrane. A microscopic layer of cells called “pia mater” is the next layer and then comes the brain itself.

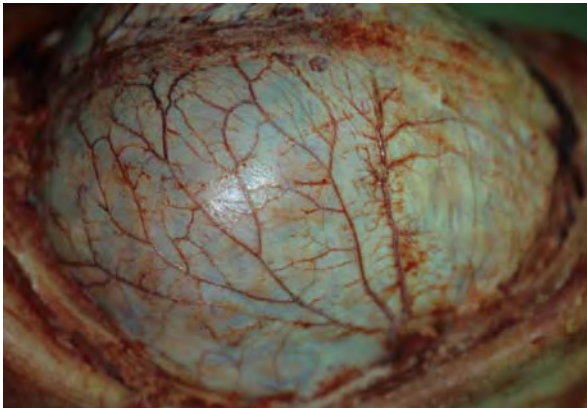


Fig. 6.6 A photo of the inside of the skull, after scalp reflection and skull cap removal. During skull removal, the dura mater peeled away from the inner aspect of the skull, leaving the dura covering the brain

Grossly, the brain can be divided into 3 basic parts: the cerebrum, the cerebellum, and the brainstem (Fig. 6.10). The cerebrum is the largest part and is divided



Fig. 6.7 In this photo, the skull is being reflected away from the brain. The dura remains tightly attached the inside (underside) of the skull. Note the “bridging veins” that connect the dura mater to the underlying meninges/brain

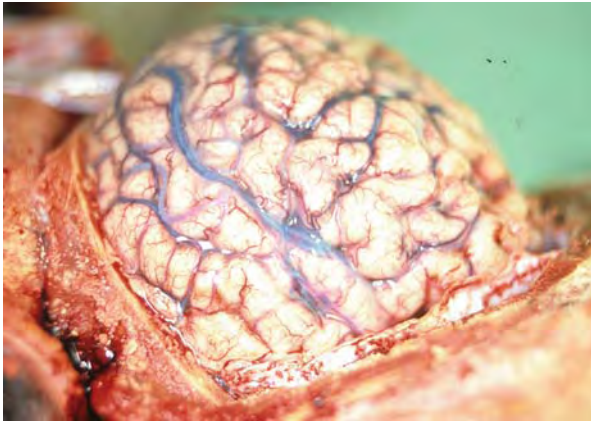


Fig. 6.8 A photograph showing the brain after reflection of the scalp and removal of the overlying skull and dura mater. A thin, translucent arachnoid membrane covers the brain

into two equal halves or hemispheres, the right and the left. Each hemisphere has four lobes visible from the outside: the frontal lobe (occurring in the front), the parietal lobe (upper side), the temporal lobe (lower side), and the occipital lobe (back) (Fig. 6.11). The outer aspect of the cerebrum is referred to as the cerebral cortex and has multiple foldings of brain tissue, such that it mimics a series of hills and valleys. The hills are referred to as gyri (singular: gyrus), while the valleys are called sulci (singular: sulcus). If you cut a brain in half, you will see areas with different colors. The very outer part of the cerebral cortex has a dark tan color, while the tissue immediately underlying it has a very light tan color (Fig. 6.12). The darker tan is referred to as gray matter, while the lighter tan area is called white matter. Deeper within the cerebrum, there are additional areas of gray matter (including the basal

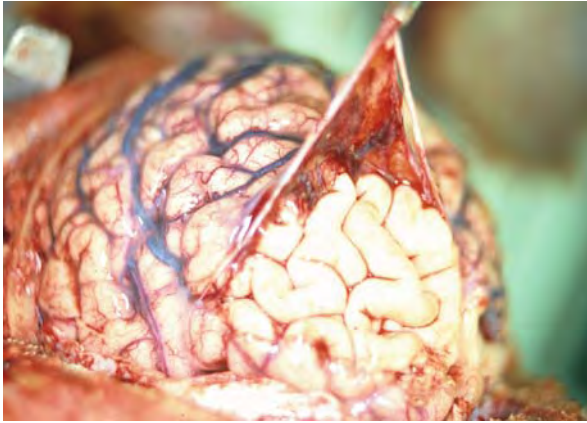
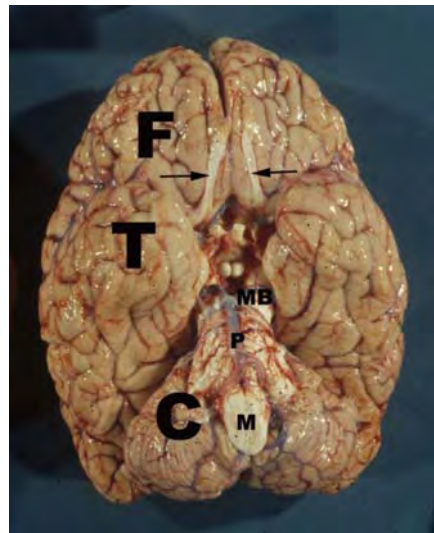


Fig. 6.9 A photograph of the brain depicted in Fig. 6.8, while the arachnoid membrane is being peeled away from the underlying brain

Fig. 6.10 A “fresh” (unfixed) brain after removal from the cranial cavity at autopsy. Visible are the inferior aspects of the frontal lobes (F), the temporal lobes (T), and the cerebellum (C), as well as the medulla (M), pons (P), and midbrain (MB) of the brainstem. The arrows indicate the olfactory tracts (nerves for the sense of smell)



ganglia, the thalamus, and the hypothalamus), as well as other areas of white matter (including the internal capsule and the corpus callosum). The gray matter contains neurons, which can be thought of as the functional “brain cells,” whereas the white matter contains axons, which are actually extensions from the neurons that function to transfer nerve impulses from one neuron to another (Disc Image 6.6). Other supportive cells also exist within the gray and white matter, including blood vessels. At the central lower portion of the cerebrum, there is a connection to the brainstem. The cerebrum is also connected via numerous axons that travel through the brainstem, to both the cerebellum and the spinal cord. Also present within the cerebrum

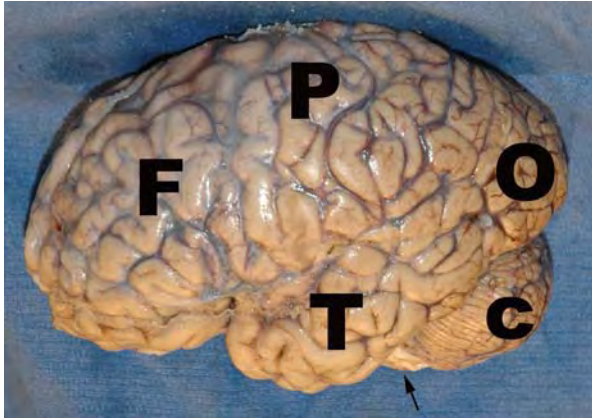


Fig. 6.11 Normal, formalin-fixed brain viewed from the side, showing the cerebrum (composed of the frontal (F), parietal (P), temporal (T), and occipital (O) lobes), the cerebellum (c), and a small portion of the brainstem (*arrow*)

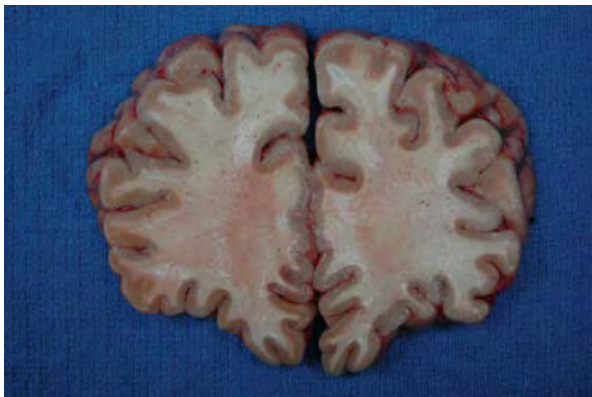


Fig. 6.12 Gross appearance of a cross-section of a fresh (unfixed) cerebrum at autopsy. The outer darker areas are the gray matter, while the lighter inner areas are white matter

are a series of cavities called ventricles (lateral ventricles, third ventricle) and it is within these ventricles that cerebral spinal fluid (CSF) resides. The CSF is produced by specialized cells that line the ventricles and it is constantly produced and flows out of the ventricular system of the brain, into the subarachnoid space (see below), where it is ultimately reabsorbed into the blood. The cerebrum is the part of the brain responsible for various “higher level” functions, including motor and sensory functions, thought, consciousness, memory, and other cognitive abilities.

The cerebellum is a separate part of the brain that exists underneath the occipital lobes of the cerebrum, in the back lower portion of the brain (Disc Image 6.7). It is attached to the upper portion of the brainstem. Its function includes various important subconscious activities such as balance, proprioception (keeping track

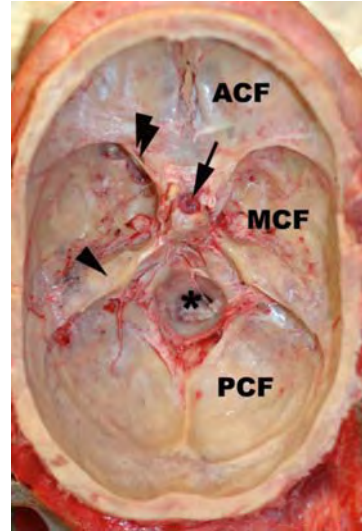
of where various body parts are), etc. Like the cerebrum, the cerebellum has gray matter (neurons) and white matter (axons).

The brainstem occurs underneath the central portion of the cerebrum, in front of the cerebellum, and above and attached to the spinal cord. The brainstem can be divided into three parts, the uppermost midbrain (Disc Image 6.8), the middle pons (Disc Image 6.9), and the lowermost medulla. It is important in providing connections between the cerebrum, cerebellum, and spinal cord, in providing various life-preserving basic neurological functions (breathing, autonomic nervous system function), and in being the site of origin of many of the cranial nerves that innervate the head, face, and neck, and other areas. As with the cerebellum and cerebrum, the brainstem contains neurons as well as axons.

Before continuing downward to the spinal cord, it is important to note some other structures associated with the CNS. When it comes to documenting injuries of the brain, it is very important to understand the various skin, scalp, bone (skull), and other injuries that may be associated with the actual brain injuries. For that reason, it is important to note here the anatomy of the scalp, skull and intracranial cavity. Working from outside to inside, we first see the skin of the scalp and immediately underlying the skin is the subscalpular (subgaleal) soft tissues, composed of subcutaneous fat and skeletal muscle. Next, we have the skull bones. Immediately inside the skull, there is a relatively dense membrane called the dura mater, or simply the dura. Normally, the dura is firmly attached to the inner surface of the skull, including the skull that overlies the sides, front, back, and top of the brain, as well as the base of the skull (basilar skull), which separates the bottom of the brain from the deep structures of the face/head (eyes, sinuses, nasal cavity, throat). The dura also has two parts that extend further into the cranial cavity and function to physically separate various parts of the brain. One part is called the falx cerebri and it arises from the upper midline of the dura and extends downward, from front to back, separating the right and left cerebral hemispheres. It does not totally separate the two halves of the brain, just the outer aspects. The second part is the tentorium, which arises from the back of the dura and separates the occipital lobes above from the cerebellum below. Immediately underlying the dura (toward the brain) is an area referred to as the subdural “space.” Blood vessels traverse this space, connecting the dura to the underlying brain coverings. The arachnoid membrane is a very thin, translucent membrane containing small blood vessels that covers the entire brain and underlying the arachnoid is a space that contains cerebral spinal fluid. This is referred to as the subarachnoid space. Finally, we get to the brain itself, which has a very thin layer of cells covering it called the pia mater. Together, the dura, arachnoid, and pia are referred to as the meninges.

The base of the skull has many undulations, projections, depressions, and “holes.” The holes are actually “foramina” (singular: “foramen”), where various structures (spinal cord, nerves, blood vessels) traverse the skull. The largest hole is the “foramen magnum,” through which the spinal cord passes. The base of the skull can be roughly divided into three general areas: the anterior cranial fossa (ACF), the middle cranial fossa (MCF), and the posterior cranial fossa (PCF) (Fig. 6.13); each is divided into right and left sides. The frontal lobes of the brain overlie the

Fig. 6.13 Normal appearance of the basilar skull, following brain and dura mater removal. Note the anterior cranial fossa (ACF), the middle cranial fossa (MCF), and the posterior cranial fossa (PCF). The arrowhead indicates the petrous ridge of the temporal bone, while the double arrow indicates the lesser wing of the sphenoid bone. The arrow indicates the location of the pituitary gland. The asterisk denotes the location of the foramen magnum, where the brainstem connects to the spinal cord



ACF, the temporal lobes overlie the MCF, and the cerebellum overlies the PCF. A depression in the midline of the basilar skull, behind the ACF and in front of the foramen magnum, is called the “sella turcica.” The pituitary gland resides in this location and is connected to the overlying hypothalamus of the brain.

Like blood vessels elsewhere in the body, arteries supply oxygen and nutrient rich blood to the brain, and veins take blood away from the brain, back towards the heart. There are two pairs of arteries that supply the brain, the internal carotids, which come up through the deep soft tissues of the neck, go through the base of the skull and enter the cranial cavity, and the vertebral arteries, that ascend toward the brain along the vertebral column and enter the cranial cavity. Once inside the cranial cavity, each internal carotid artery divides into the middle cerebral artery (MCA), the anterior cerebral artery (ACA), and the posterior communicating artery, with the left and right anterior cerebral arteries connected by a single anterior communicating artery. The two vertebral arteries combine to form a single, midline, basilar artery, which exists immediately in front of the brainstem. As the basilar artery approaches the base of the cerebrum, it gives rise to the cerebellar arteries and then divides into right and left posterior cerebral arteries (PCA), to which the posterior communicating arteries from the internal carotids join. The resulting circular connection of arteries at the base of the brain is referred to as the “circle of Willis” (Disc Image 6.10). Numerous veins traverse the meninges, with some veins traversing the subdural space to enter the dura. These veins are referred to as “bridging veins.” A large central sinus (vein) resides within the midline of the dura.

The spinal cord represents a continuation of the lower medulla, extending through the spinal canal within the vertebral column (Disc Image 6.11). The spinal cord can be loosely divided into the cervical (upper) region, the thoracic (middle) region, and the lumbar (lower) region. It contains neurons in groups referred to as

“nuclei,” as well as numerous axons. Neuroscientists use the term “tract” to define a specific group of axons that connect various parts within the CNS, whether it is within the cerebrum, cerebellum, brainstem, or spinal cord. Ultimately, the spinal cord gives rise to the spinal nerves, which exit the spinal canal and become the nerves of the peripheral nervous system. Ganglion cells or ganglia represent neurons which exist in groups outside of the CNS. They may exist in localized structures distinct from an organ, or they may reside within various organs or tissues.

Cardiovascular System

The cardiovascular system encompasses the heart (Fig. 6.14) and the blood vessels, including arteries, veins, and capillaries. The heart is composed of a special type of muscle tissue called cardiac muscle. Like skeletal muscle, it is striated, and contains actin and myosin filaments. However, unlike skeletal muscle, the cells of cardiac muscle contain central nuclei (instead of peripheral nuclei) (Fig. 6.15). Heart muscle also contains a specialized system for allowing electrical impulses to be easily transmitted through the entire organ (the T-tubules). In addition, there are specialized cells that can be thought of as being a combination of nerve and cardiac cells. These specialized conduction cells include cells that make-up the sinoatrial (SA) node and the atrioventricular (AV) node, which are internal electrical pacemakers, as well as other cells that connect the nodes and distribute electrical impulses to the heart muscle. Together, these cells form the “conduction system” of the heart.

Grossly, the heart is essentially a pump that is composed of a pair of two-chambered pathways that exist next to one another. Blood low in oxygen content



Fig. 6.14 Gross appearance of the heart

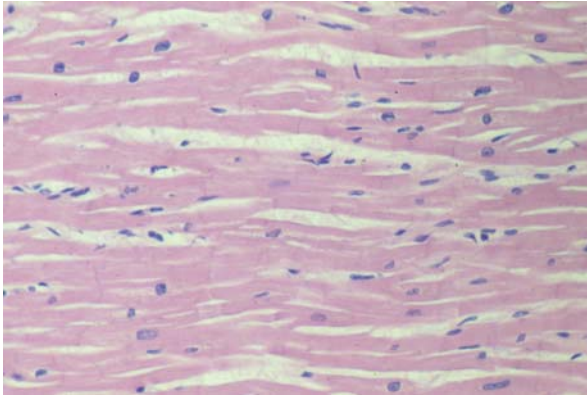


Fig. 6.15 Microscopic appearance of the heart, composed of numerous cardiac myocytes (heart muscle cells)

but high in carbon dioxide content returns to the heart from the majority of the body via veins which empty into the superior vena cava (SVC) and the inferior vena cava (IVC); blood from the head, neck and arms returns via the SVC and blood from the trunk and legs returns via the IVC. Both the SVC and IVC empty blood into the right atrium (the first of four chambers of the heart). Once in the right atrium, blood is pumped across the tricuspid valve into the right ventricle (the second chamber) (Disc Image 6.12) and is then pumped across the pulmonary valve into the pulmonary artery (Disc Image 6.13). The pulmonary artery then divides into right and left sides and distributes blood to both lungs, where the blood gets rid of excess carbon dioxide and takes on a fresh load of oxygen. After blood is oxygenated in the lungs, it exits the lungs via the pulmonary veins and enters the left atrium (the third chamber of the heart). The oxygen-rich blood is then pumped across the mitral valve into the left ventricle (the fourth and final heart chamber) (Disc Image 6.14), where it is then pumped across the aortic valve into the aorta (Disc Image 6.15). From the aorta, blood travels throughout the arteries of the body, entering smaller and smaller branches until the blood reaches small arterioles and then the microscopic capillaries, where oxygen, carbon dioxide, and nutrient and waste products are able to diffuse across the capillary walls into and out of tissues and organs. Blood travels from the capillaries into small venules and then successively larger veins until it ultimately enters either the SVC or IVC and returns again to the heart to repeat the cycle. The veins of the body actually have valves interspersed along their course, to prevent retrograde (backward) flow. The heart itself is supplied with oxygenated blood via the left and right coronary arteries, which arise from the beginning part of the aorta, just past the aortic valve. The openings into the coronary arteries are referred to as “ostia” (singular: “ostium”). The left coronary artery divides into the left anterior descending coronary artery and the circumflex coronary artery. A final set of vessels that exists throughout the body are the lymphatic vessels. These are discussed further in the reticuloendothelial system section, but morphologically, they are similar to blood vessels.

The pumping action of the heart is controlled by the internal pacemakers described previously (SA and AV nodes), with considerable input from the autonomic nervous system and other regulating mechanisms. The heart pumps in two phases, one called systole, where the ventricles forcibly contract and push blood into the pulmonary artery (from the right ventricle) and the aorta (from the left ventricle), and one called diastole, where the ventricles relax, and blood leaves the atria and re-fills the ventricles. During the pumping action of the heart, the heart valves open and close. When open the valves allow blood to pass through unimpeded and when closed, they prevent blood from flowing backwards across the valves. The walls of the left ventricle of the heart are much thicker than the right ventricle, since the pressures within arteries are much greater than the pressures within veins and the lungs. In other words, it takes more pressure to push blood to the entire body than it does to push blood into the lungs to become oxygenated. The inner walls of the heart that separate the right and left sides are referred to as the interatrial septum and the interventricular septum.

Basically, the heart and blood vessels are responsible for providing blood flow to all organs, tissues and cells of the body. This provides oxygen and nutrients to the cells, and also allows cells to get rid of waste products, including carbon dioxide. Pathologists frequently see the results of damage to the cardiovascular system, whether it be through natural disease processes or as the result of traumatic injuries.

Respiratory System

The respiratory or pulmonary system encompasses the lungs and the breathing passages (Disc Images 6.16 and 6.17 and Fig. 6.16). Strictly speaking, therefore, the nose, nasal cavity, sinuses, mouth and throat (pharynx), and larynx (voicebox area), as well as the trachea and the mainstem bronchi may all be considered part of the

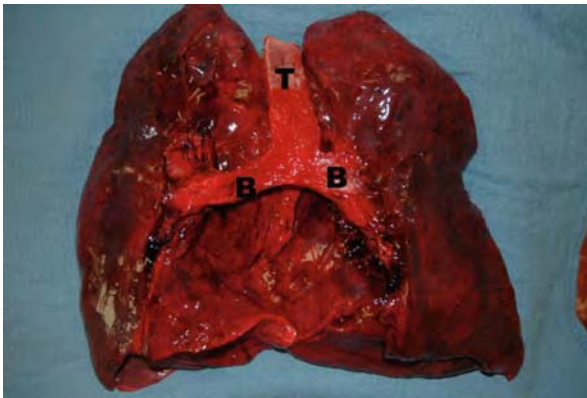


Fig. 6.16 Trachea (T), right and left mainstem bronchi (B), and lungs (posterior view (from behind))

respiratory system. This fact becomes particularly important when we discuss the various forms of asphyxial injuries that can cause death (Chapter 15). When a person breathes in (inspiration), air enters the nose (and/or mouth), travels through the pharynx (back of throat), and downward to the larynx, where the respiratory and gastrointestinal systems divide, with food entering the esophagus and air passing by the vocal cords of the larynx and entering the trachea. Within the pharynx, there are various tissues (the tonsils) that play a role in fending off pathogens (infectious micro-organisms). The epiglottis is a structure that folds over and covers the air passage when we swallow, preventing food from entering the trachea and lungs. After entering the trachea, the air continues downward, past the carina, where the trachea divides into the right and left mainstem bronchi. The mainstem bronchi enter the lungs centrally and toward the middle, in an area referred to as the hilum of the lung (the same location where the pulmonary artery enters and the pulmonary veins exit). The bronchi continue to branch into ever-smaller bronchi until they divide into very small air tubes called bronchioles. The bronchioles deliver air into microscopic, blind-ended air sacs called alveoli (Fig. 6.17). Blood travels in capillaries within the walls of the alveoli. Through diffusion, carbon dioxide from blood crosses the capillary and alveolar walls into the air contained within the alveolar spaces. At the same time, oxygen within the inhaled air contained in the alveoli diffuses across the alveolar and capillary walls into the blood. After the exchange of gases between blood and alveolar air, the carbon dioxide-rich alveolar air is then exhaled, traveling in reverse direction during the process of expiration. The lungs, therefore, act in a fashion similar to a bellows. When the lungs expand, air is drawn inward and when the lungs deflate, air is forced outward. The inflation and deflation actions are created by the combined action of the diaphragm, which is an umbrella-shaped, relatively thin, muscle that separates the lungs from the abdominal organs, and the chest wall (made up of the mobile rib cage and the associated muscles, referred to

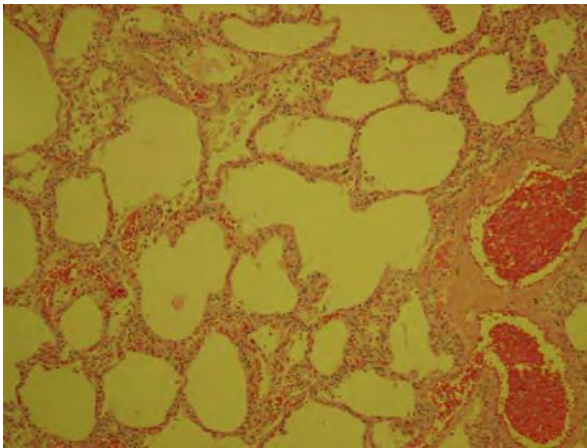


Fig. 6.17 Microscopic appearance of a lung. The air sacs are called alveoli. Two blood-filled vessels are evident on the right

as “intercostal” muscles). The phrenic nerve innervates the diaphragm and allows “automatic” breathing to occur. A variety of activities or processes can initiate a hunger for air, including elevated carbon dioxide levels in blood and a variety of things that increases our body’s demand for oxygen.

Microscopically, each part of the upper airway has a specific appearance. Much of the lining cells are made up of what pathologists refer to as “respiratory epithelium” (Disc Image 6.18) and this respiratory epithelium also lines the trachea and bronchi. The walls of the trachea and bronchi also contain smooth muscle cells, mucus-producing glands, as well as cartilage, to provide a firm yet flexible structural support. The alveolar walls are very thin and surround alveolar spaces that appear clear under the microscope. Blood vessels are present throughout the lungs. Lymph nodes occur near the hilum.

Gastrointestinal System

The gastrointestinal (GI) system begins at the oral cavity and ends at the anus (Figs. 6.18, 6.19, 6.20). It can be thought of as one long tube starting at one end of the body and ending at the other end; however, there are also various other organs and glands that play a crucial role in the proper functioning of the GI system and some of those will be discussed in this section. Others will be covered in other systems. The main purpose of the GI system is to provide sustenance for the body. To that end, water and nutrients (protein, carbohydrates, fats, vitamins, trace minerals) are ingested, broken down (digested) into absorbable particles, and absorbed into the blood. Any residual substances, along with certain waste products that are excreted into the GI system, are eventually expelled via the feces.

Fig. 6.18 Initial view of the gastrointestinal system at autopsy, as viewed following initial autopsy incision and removal of the anterior chest plate. The arrowheads indicate the location of the diaphragm, while the arrow indicates the liver. A portion of the stomach is visible immediately adjacent to the liver, while the fatty omentum prevents visualization of the intestines

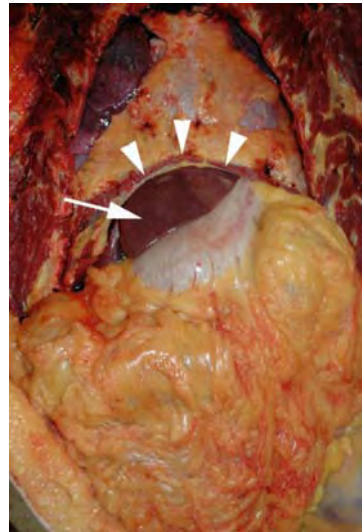


Fig. 6.19 In this photograph, which corresponds to Fig. 6.18, the omentum has been flipped upwards. The arrowheads denote the transverse colon (part of the large intestine). Multiple loops of small intestine are visible in the lower half of the photo

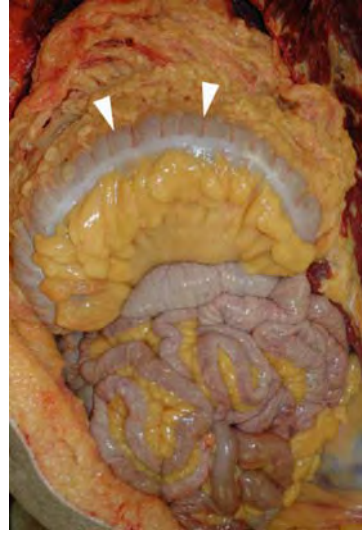
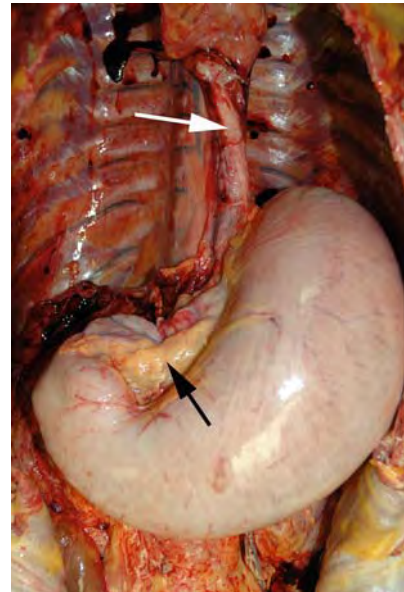


Fig. 6.20 A photograph taken at autopsy following removal of the heart, lungs, diaphragm, liver, omentum, large intestine, and a majority of the small intestines. Remaining intact are the esophagus (*white arrow*), the stomach, which is distended by gas, the duodenum (hidden from view), and the pancreas (the head of the pancreas is denoted by the *black arrow*)



The oral region includes the teeth, which break apart food via the chewing process, the tongue, which helps in the chewing and swallowing processes, and the salivary glands, which produce enzymes that initiate enzymatic digestion. The throat region (pharynx), larynx, and esophagus come next and essentially act as transport passages for food. The tongue and the muscles of the pharynx and larynx, with the

especially important function of the epiglottis as described above, act together to produce swallowing. The food then enters the esophagus, where a progressive wave of muscular contraction called “peristalsis” propels the food towards the stomach. The upper portion of the esophagus is behind the trachea, while the lower portion is behind the heart. Crossing the esophagogastric junction, the food enters the stomach, where stomach acid and digestive enzymes are mixed with the food to continue the digestive process. Eventually, the food crosses the pyloric sphincter (pylorus), exiting the stomach and entering the first of three parts of the small intestine (or small bowel), the duodenum. Within the duodenum, digestion continues, and bile from the liver/gallbladder and enzymes produced by the pancreas enter the duodenum via the ampulla of Vater (the opening of the ducts carrying bile and pancreatic enzymes). During the remainder of the food’s journey through the small intestines, first through the jejunum and then the ileum, digestion continues and the resulting nutrients are absorbed across the wall of the intestine into the blood stream. The fatty, fan-shaped section of tissue that attaches the lengthy loops of the intestines to the back of the peritoneal cavity is called the “mesentery” (Fig. 6.21). It is through this fatty structure that the blood vessels that supply and drain the intestines travel. The ileum empties into the first part of the large intestine (or large bowel), the cecum. This is located in the lower right abdomen, and it is the part of the bowel to which the appendix attaches. For years, it was thought that the appendix had no particular function; however, a recent study suggests that it may provide a reservoir for normal intestinal bacteria. From the cecum, the bowel contents travel up the ascending colon (on the right side of the abdomen), across the transverse colon (upper abdomen), down the descending colon (on the left side of the abdomen) to the sigmoid colon. The sigmoid attaches to the rectum, which connects to the anus. By the time the bowel contents arrive at the large intestine, most if not all of the nutrients have been absorbed within the small intestine; however, there remains a substantial amount of



Fig. 6.21 A photograph showing the fan-shaped fatty mesentery, which attaches to the small intestine

water content. It is within the large intestine that most of the residual water is reabsorbed into the bloodstream. What remains are soft, formed stools which contain abundant bacteria intermixed with other waste.

Bacteria and other microbial organisms that are normally present on or reside within the human body, without causing illness, are referred to as normal microbial “flora.” Many of these are “commensal” organisms, where both the host (human) and micro-organism receive a benefit from the mutual existence. For example, certain GI bacteria are involved in the production of vitamins that are subsequently absorbed into the blood stream.

Each specific portion of the GI tract has a distinct gross and microscopic appearance (Disc Images 6.19, 6.20, 6.21, 6.22, 6.23). The oral cavity, tongue, portions of the larynx, and the esophagus are lined by stratified squamous epithelium, similar in appearance to epidermis, but without keratinization. The stomach, small intestine, and large intestine are lined by columnar (tall) epithelium and have various other specialized cells. For instance, the stomach also has parietal cells, which are responsible for producing hydrochloric acid. The walls of each also contain smooth muscle cells which allow for peristalsis to propel the food substance along its way. As with all organ systems, blood vessels flow to and from each part of the GI system, and lymphatic vessels and lymph nodes provide for drainage and filtering of excess extracellular fluid. The major arteries supplying blood to the GI tract include the celiac artery, the superior mesenteric artery, and the inferior mesenteric artery. Each arises from the abdominal aorta.

The liver and pancreas play a very important role in the digestive process. With regard to the liver, bile salts are produced which aid in the digestion of fats (please refer to the hepatobiliary section for more information about bile). The pancreas has two functional components which are designated as the exocrine pancreas and the endocrine pancreas. The endocrine pancreas is described further in the endocrine system below. The exocrine pancreas, which makes up the bulk of the pancreatic tissue, involves the production and excretion of digestive enzymes that aid in food digestion. Some of these enzymes include amylase (involved in carbohydrate digestion), lipase (involved in fat digestion), and various proteases (involved in the digestion of proteins). After nutrients are absorbed across the small intestine walls and into the blood stream, the blood from the intestines empties into the portal vein, which drains directly into the liver.

Hepatobiliary System

The hepatobiliary system essentially encompasses the liver, the gallbladder, and the ducts that drain the liver and gallbladder (the biliary ducts). The liver is a large organ that resides primarily within the upper right side of the peritoneal cavity, just under the diaphragm (Fig. 6.22 and Disc Image 6.24). The liver can be considered the metabolic workhorse of the body because it is within the liver that many very important metabolic activities and processes occur. As we learned from the discussion of the GI system, all nutrients absorbed within the small intestine travel via the portal



Fig. 6.22 A liver removed at autopsy, as viewed from above

vein to the liver. Within the liver, many nutrients such as carbohydrates, proteins, fats, vitamins and minerals are converted into more usable molecules. The liver is responsible for the storage, breakdown and/or synthesis of many fats, proteins, and carbohydrates. Many very important proteins are produced within the liver, including many of the components which make up the fluid portion of blood – albumin and blood clotting factors are just a few of the important proteins produced by the liver. The liver also functions as the major “detoxifying” organ within the body. Whether it is a drug, a chemical, a food byproduct, or a poison, the liver is the primary site for the breakdown or detoxification of many exogenous (external) substances. As part of its many functions the liver produces bile. Bile contains bile salts which aid digestion, as well as various substances related to red blood cell breakdown and cholesterol metabolism. Some bile substances are excreted with the feces while others are reabsorbed within the intestines.

Microscopically, the liver parenchyma cells are called hepatocytes (Fig. 6.23 and Disc Image 6.25). Coursing amongst the hepatocytes are specialized vascular channels referred to as sinusoids, which allow the hepatocytes ready access to nutrients within the blood. Scattered amongst the lobules of hepatocytes are central veins and specialized structures referred to as portal triads. The central veins drain blood from the sinusoids and combine into increasing-sized veins that ultimately exit the liver as the hepatic vein, which drains into the IVC. The portal triads each contain a small branch that ultimately arose from the portal vein (containing nutrient-rich blood from the GI system), a small branch of the hepatic artery (containing oxygen-rich blood from the aorta), and a small bile ductule, which combine with one another into larger bile ducts and eventually exit the liver as the hepatic duct. The hepatic duct is connected by a side branch to the cystic duct, which ends at the gallbladder. Past the connection to the cystic duct, the hepatic duct is referred to as the common bile duct. It enters the head of the pancreas, combines with the pancreatic duct and empties into the duodenum at the ampulla of Vater.

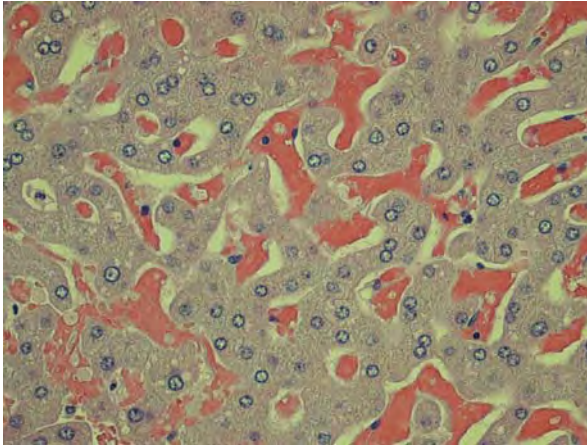


Fig. 6.23 Microscopic appearance of the liver. Liver cells are called hepatocytes. Blood courses between hepatocytes in vascular channels called sinusoids

Reticuloendothelial System (Including the Immune System)

The reticuloendothelial system is a complex system made up of a wide variety of organs and tissues, each playing a role in the make-up of blood or the surveillance of the blood and other tissues for injurious organisms or substances. The specific role relating to surveillance for injurious organisms or substances is referred to as the immune system. Since this system is relatively complex, we will attempt to address different components of the system separately.

Bone Marrow and Blood – Bone marrow is present within the medullary regions of bones. Bone marrow cells can be thought of as the cells responsible for producing blood cells. Blood has a cellular component as well as a fluid component (plasma or serum). The official name for blood-producing cells is “hematopoietic” cells and they are sometimes referred to as blood “precursor” cells. Blood cells that are produced within the bone marrow include red blood cells (rbcs) or erythrocytes, white blood cells (wbcs) or leukocytes, and platelets (sometimes called thrombocytes; produced by megakaryocytes within the bone marrow). Figure 6.24 shows a normal histologic section of bone marrow, while Disc Image 6.26 shows a normal bone marrow smear viewed under the microscope. White blood cells come in a variety of types, including neutrophils (also called granulocytes or polymorphonuclear cells (PMNs)), basophils, eosinophils, monocytes (which enter tissues to become macrophages), and lymphocytes. Lymphocytes can be further divided into T-lymphocytes and B-lymphocytes. T-lymphocytes are involved in cellular immunity and the older T-lymphocytes reside within the thymus (see below). B-lymphocytes become plasma cells, which produce antibodies.

Red blood cells are primarily responsible for carrying oxygen within hemoglobin molecules contained within their cytoplasm. Oxygen is obtained when the blood

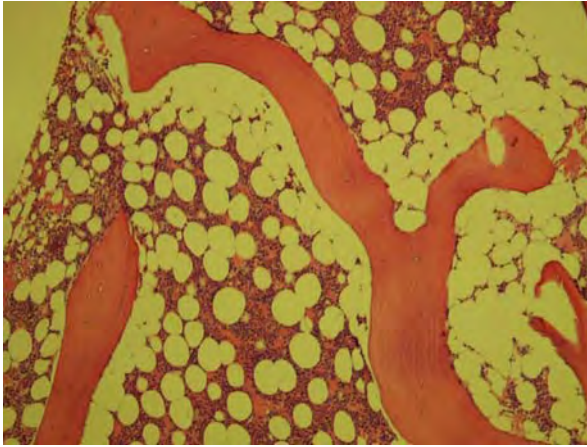


Fig. 6.24 Histology section of bone marrow. Note the bony trabeculae (large scaffolding-like structures) surrounded by bone marrow cells and fat (clear cells)

travels through the lungs, and it is released to other tissues when the blood travels through the capillaries of those tissues. A unique feature of rbc's is that they contain no nucleus, although during development within the bone marrow, the rbc precursor cells *do* have nuclei. Red blood cells only live for approximately 120 days before they become “worn out” so new rbc production is a constant requirement. Old rbc's are removed by the spleen and other mechanisms. Hemoglobin from the destroyed rbc's is broken-down in the liver and incorporated into bile. Hemoglobin can be evaluated, even in postmortem samples, via a process known as “hemoglobin electrophoresis.”

White blood cells are important in the processes of inflammation and immune reactions. The wbc's circulate within the blood and are actually able to leave the blood and enter other tissues where inflammation or an immune reaction has been initiated. Depending on the underlying reason for the inflammation, different types of wbc's may accumulate. In general, neutrophils (PMNs) tend to accumulate in response to bacterial infections or tissue necrosis (cell death in an otherwise living tissue). Neutrophilic inflammation is sometimes called suppurative inflammation. Grossly, a collection of suppurative inflammation may have the appearance of pus and can also be called purulent inflammation. A localized collection of neutrophilic inflammation in association with tissue necrosis is called an abscess. Lymphocytes accumulate in response to viral infections or chronic (longterm) inflammation. Macrophages (which begin as blood monocytes) accumulate in response to a variety of chronic infections or inflammatory processes, including the presence of foreign substances. A localized collection of macrophages associated with lymphocytes is referred to as a granuloma. Granulomatous inflammation is characteristic of several disease processes, including tuberculosis. Eosinophils accumulate in a variety of situations, including allergic reactions and parasite infections. Figure 1.2 shows the microscopic appearance of several types of blood cells.

Platelets are small cellular fragments, without nuclei, produced by bone marrow megakaryocytes and released for circulation within the blood. They play an important role in the blood clotting process (thrombosis), along with various clotting factors that are part of the coagulation cascade of proteins. A thrombus can be considered a blood clot and a thromboembolus is a thrombus that breaks free from its source and travels within the blood vessels to a distant location.

Spleen – The spleen is an abdominal organ that resides within the upper back left portion of the peritoneal (abdominal) cavity, immediately underneath the left side of the diaphragm (Fig. 6.25). The hilum of the spleen is near the tail of the pancreas. Its blood supply is via the splenic artery, and its venous drainage is via the splenic vein, which drains into the portal vein. Although it is more complex than this, one may think of the spleen as a blood filter that specifically filters blood cells, rather than the fluid component of blood. Old rbc's are removed within the spleen, as are old wbc's, and various immune reactions also occur in the spleen. While people can survive without a spleen, such individuals are at increased risk of a variety of infections. Grossly, the spleen has a capsule, as well as red pulp and white pulp. Microscopically, the spleen contains abundant white and red blood cells, as well as blood vessels and connective tissue support structures called trabeculae (Disc Image 6.27).



Fig. 6.25 Gross appearance of the spleen

Thymus – The thymus is an organ that resides within the anterior (front) portion of an area of the chest called the mediastinum (Fig. 6.26). The mediastinum occurs between the lungs. Other anatomic structures that reside within the mediastinum include the great vessels of the heart (aorta and pulmonary arteries), the lower trachea and mainstem bronchi, a portion of the esophagus, and numerous lymph nodes. The thymus is most prominent during the early childhood years, when it appears as

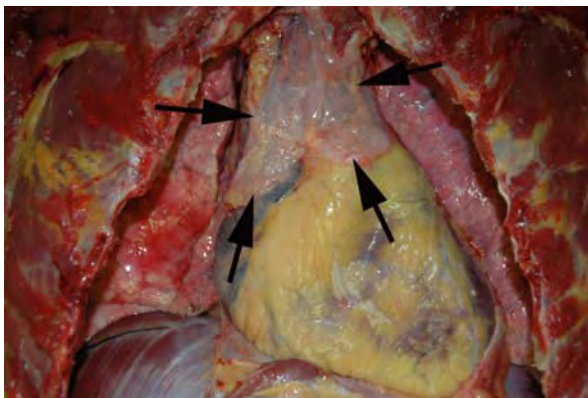


Fig. 6.26 Gross appearance of the thymus in situ (in its normal location within the body)

a relatively large, fatty structure with a lobe that frequently extends upward into the lower neck. Its primary function involves cellular immunity, a component of the immune system that is very active during the early childhood years, as children are exposed to numerous pathogens. Microscopically, the thymus contains lymphocytes as well as peculiar epithelial structures called Hassal's corpuscles (Disc Image 6.28).

Lymphatics and Lymph Nodes – The lymphatic system is a system of vessels that begin within the interstitial spaces (areas outside of cells and outside of blood vessels) within many parts of the body, including certain organs. The main function of the lymphatic system is to drain excess fluid from the interstitial spaces. Fluid moves out of the tissues into the lymphatic vessels and empties into lymph nodes in various parts of the body. Lymph nodes are relatively small, spherical to ovoid, tan structures located throughout the body (Fig. 6.27) and can be considered immune surveillance checkpoints along the lymphatic vascular system. They are composed of lymphocytes and specialized macrophages called “histiocytes” (Disc Image 6.29). The lymph fluid that enters the lymphatic vessels and is filtered by lymph nodes continues to travel through other lymphatic vessels and eventually is dumped back into the venous blood.

Endocrine System

The endocrine system encompasses numerous endocrine glands located in various places throughout the body. Endocrine glands produce hormones that are important in various metabolic processes; the glands' parenchymal cells produce the hormones and release them directly into the blood. The hormones circulate within the blood and manifest their effects in locations distant from the gland that produced them. In some instances, a specific hormone has a very specific function on only a limited number of target cells while in other instances, a hormone can have multiple effects

Fig. 6.27 Gross appearance of normal lymph nodes (*arrows*) within the hilum (central portion) of the lung. Typically, normal lymph nodes are relatively small and inconspicuous



on a wide variety of target cells. In the following paragraphs, a brief description will be provided regarding each of the major endocrine glands.

Pituitary Gland – The pituitary gland (also called the hypophysis) is considered the master gland, since it produces many hormones that act on several other glands. It is located immediately under and attached to the hypothalamus of the brain and resides within a depression within the basilar skull called the sella turcica. The pituitary has two distinct parts, the neurohypophysis, which actually represents an extension of the hypothalamus of the brain, and the anterior aspect of the gland, composed of several cells types. Vasopressin and antidiuretic hormone are released from the neurohypophysis, while the anterior pituitary produces the following hormones: adrenocorticotrophic hormone (ACTH), which acts on the adrenal gland; prolactin, which acts on breast tissue among other tissues; thyroid stimulating hormone (TSH), which acts on the thyroid gland; and follicle stimulating hormone (FSH) and luteinizing hormone (LH), which act on sex glands (ovaries and testes).

Pineal Gland – The pineal gland is something of a mystery gland. It resides deep within the brain and its cells have similarities to cells found within the eyes. Most experts believe that the pineal gland plays a role in daily cycles related to light and dark.

Thyroid Gland – The thyroid gland is located in the anterior aspect of the neck, near the thyroid cartilage. It has two lobes (right and left) connected by an isthmus that crosses in front of the thyroid cartilage (Fig. 6.28). The thyroid gland has two major parenchymal cell types, the most abundant of which are follicular cells that have a very distinctive histologic architectural appearance, wherein follicular cells surround a space containing a substance referred to as colloid. Disc Image 3.1 shows the normal histologic appearance of the thyroid gland. The follicular cells respond to TSH (made in the pituitary) and produce various types of thyroid hormone. Thyroid



Fig. 6.28 Gross appearance of the thyroid gland

hormone effects many other tissues and organs and is responsible for maintaining normal metabolism. If someone has too much thyroid hormone, they have a hyper-functioning metabolism but if they have too little thyroid hormone, they have a depressed metabolism. The other type of parenchymal cell within the thyroid gland are the parafollicular C cells and these are scattered amongst the thyroid follicles. The parafollicular C cells produce calcitonin, a hormone that plays an important role in the metabolism of calcium and phosphorus. As such, its actions occur primarily within the bone and kidneys.

Parathyroid Gland – There are usually at least four parathyroid glands, but sometimes more. They are small, pea-sized glands that reside adjacent to the thyroid gland within the anterior neck and produce parathyroid hormone, which also plays an important role in calcium and phosphorus metabolism. Some of the hormone's effects are via the hormone's ability to activate vitamin D within the kidneys.

Adrenal Glands – The adrenal glands are small, triangular-shaped glands that are present above the kidneys and have a distinct outer golden yellow cortex, as well as an inner, brown-gray medulla (Fig. 6.29). The cortex is composed of three zones of cortical cells (Disc Image 6.30): from outside to inside known as the zona glomerulosa, the zona fasciculata, and the zona reticularis, which produce mineralcorticoids (especially aldosterone), glucocorticoids (especially cortisol), and sex steroids (estrogens and androgens), respectively. Aldosterone is important in sodium and potassium metabolism. Cortisol can be thought of as the body's major steroid – it has many important physiologic roles, including immune function, glucose regulation, blood vessel tone, and bone metabolism. The adrenal medulla is the source of epinephrine (adrenaline) and norepinephrine and is also considered a part of the sympathetic autonomic nervous system.

Fig. 6.29 Gross appearance of an adrenal gland that has been sectioned



Pancreas – The majority of the pancreas is composed of exocrine pancreas cells that produce various digestive enzymes (Fig. 6.30). Scattered amongst the exocrine pancreas cells are islands of endocrine cells called the islets of Langerhans (Disc Image 6.31). There are several cell types within the islets which produce hormones such as insulin, glucagon, and somatostatin. Insulin is a very important metabolic regulatory hormone that is required by many (most) cell types to take up, or internalize, glucose (blood “sugar”) from the blood. Glucagon is a hormone that is also important in glucose metabolism, with many effects that are opposite to those of insulin.

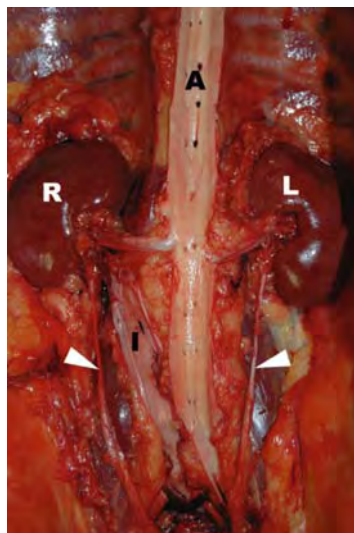


Fig. 6.30 Gross appearance of the sectioned pancreas

Genitourinary System

The genitourinary system includes the kidneys, ureters, bladder, and urethra, as well as the sex organs (ovaries and associated structures; testes and associated structures). The kidneys are best thought of as filters of the fluid component of blood (as opposed to the spleen, which can be considered a filter of the cellular component of blood) (Fig. 6.31 and Disc Image 6.32). The kidney has an outer cortex, as well as an inner medulla, which drains into a collecting system composed of several calyces and a renal pelvis (Disc Image 6.33). The collecting system is where the waste water (urine) collects and drains into the urinary bladder via the ureters. From the bladder, urine exits the body via the urethra. Microscopically, the kidney cortex contains specialized structures called glomeruli (singular: glomerulus), as well as tubules (Fig. 6.32). Blood enters the capillaries of the glomeruli and waste products diffuse into Bowman's space, which surround each glomerulus. The waste fluid then travels through a lengthy network of tubules, including the proximal and distal tubules and the loops of Henle, where various exchanges of electrolytes and fluids may occur. Finally, the fluid exits the tubules and enters the collecting system as urine, emptying into the urinary bladder.

Fig. 6.31 Gross appearance of the kidneys in situ. Note that the right (R) and left (L) kidneys are attached to the aorta (A) by renal arteries. The arrowheads indicate the ureters. The black "I" indicates the location of the inferior vena cava



In females, two ovaries and two fallopian tubes are present adjacent to a single uterus, one each on the right and one each on the left (Fig. 6.33). The uterus is an organ composed of a smooth muscular wall with an open cavity lined by endometrium. The lumen (openings) of the fallopian tubes connects to the endometrial cavity. At its lower end the endometrial cavity connects to the vagina through an opening in the cervix called the cervical os. The vagina, which is essentially a hollow tube, opens via an orifice (opening) within the perineum, the area of the

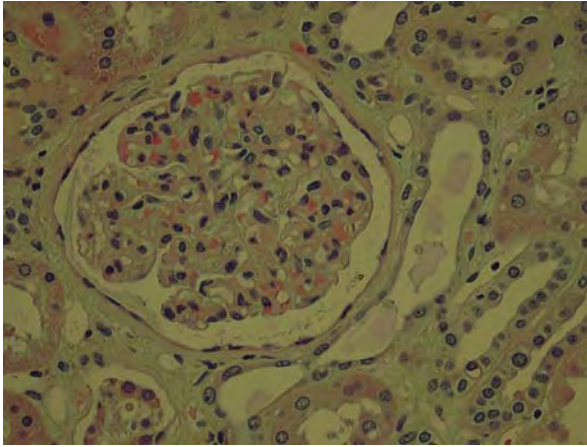


Fig. 6.32 Microscopic appearance of the kidney. Note the rounded glomerulus, as well as numerous surrounding tubules

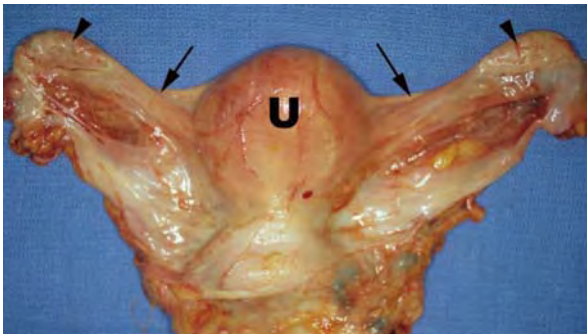


Fig. 6.33 Gross appearance of a uterus (U), with attached fallopian tubes (*arrows*) and ovaries (*arrowheads*)

groin between the legs and bounded by the anus posteriorly and the urethral orifice anteriorly. The ovaries contain a lifetime's worth of eggs (ova). In postmenopausal women, ova are not readily seen (Disc Image 6.34). During reproductive years, a woman ovulates approximately once a month, releasing an ova which typically enters the fallopian tube and travels down the tube into the endometrial cavity. The cells that surround the eggs within the ovary produce various sex hormones, including estrogen. If a sperm fertilizes the egg, the new cell created by the union of sperm and egg is referred to as a zygote. The endometrial lining goes through a monthly cycle as it prepares for a zygote to implant. If implantation occurs, the zygote grows into an embryo and placenta and bag of waters (amniotic sac), while the place where ovulation occurred in the ovary transforms into a structure called a corpus luteum of pregnancy; this structure helps to maintain the pregnancy early in its course. If fertilization and implantation does not occur, then the endometrial lining sloughs

off in the form of a monthly period, and the cycle repeats. The ovulation cycle and the endometrial cycle are under the control of follicle stimulating hormone and luteinizing hormone, produced by the pituitary gland.

In males, each of two testes (or testicles) resides within the scrotum and is closely associated with an epididymis (Fig. 6.34). The testicles contain seminiferous tubules, from which spermatozoa (sperm) are produced, and interstitial cells of Leydig, which produce testosterone (Disc Image 6.35). Sperm leave the testes and travel through the epididymis and into the vas deferens, which go upward into the groin region and then dive back downward, past the prostate gland and into the urethra. The prostate gland (Disc Image 6.36), which resides behind the urethra, below the urinary bladder (Fig. 6.35), and in front of the rectum and anus, produces seminal fluid which provides nourishment and a protective fluid medium for sperm transport.

Fig. 6.34 Gross appearance of a sectioned testicle



Special Sensory Structures

Special sensory structures include the eyes, ears, and nose. It is beyond the scope of this text to provide details regarding these structures, except to say that, on occasion, these structures may be examined at autopsy. Most common are the eyes, particularly in cases of suspected child abuse. Each eye has an optic nerve which arises from the brain and enters the back of the eyeball (the “globe”). The back of the inside of the eye is the retina, composed of numerous layers of specialized cells (Fig. 6.36) that enable vision. Vitreous humor (a clear gel-like fluid) exists in front of the retina. The lens is present near the front of the eye. The iris is the

Fig. 6.35 Gross appearance of an opened urinary bladder (B) and prostate gland (P)

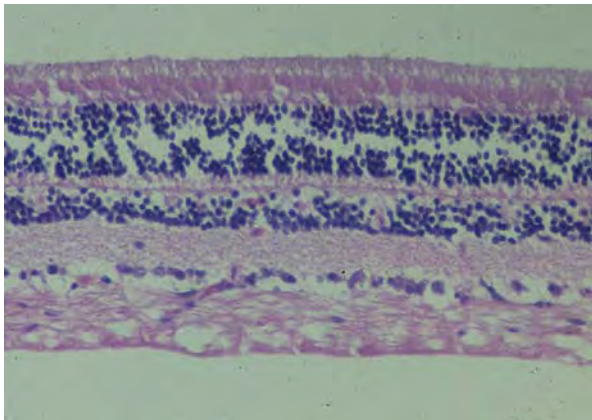
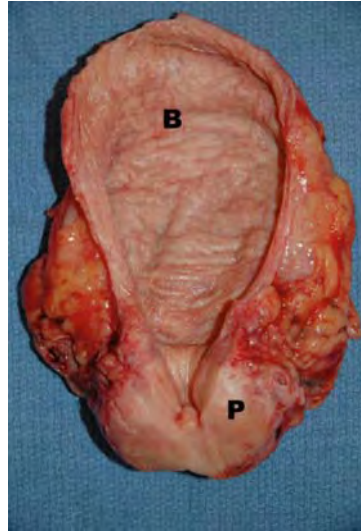


Fig. 6.36 Microscopic appearance of a normal retina

colored part of the front of the eye, visible externally, and surrounding the pupil, which can constrict (become small) or dilate (become large) in order to decrease or increase the amount of light entering the eye, respectively. The white part of the eye is called the sclera. The surface of the eye covering the iris and pupil is called the cornea. The epithelial lining covering the sclera and inner eyelids is referred to as the conjunctiva.

Disc Image Legends

- Disc Image 6.1 The upper trunk as viewed at autopsy, after the opening incisions and removal of the anterior (front) chest plate. Each lung (L) is visible. The pericardial (heart) sac (P) has not yet been opened. The diaphragm (D) separates the chest from the abdomen. Just beneath the diaphragm, a portion of liver (arrow) is evident, as is a portion of stomach (S).
- Disc Image 6.2 An autopsy photograph showing the four body cavities of the trunk, after organ removal. The pericardium (heart sac) and diaphragm (arrows) have been left in place. There are two pleural (chest) cavities (PL), a single pericardial (heart) cavity (P), and a single abdominal (peritoneal) cavity (ABD).
- Disc Image 6.3 The end of a long bone (tibia) from a child. Note that the epiphyseal plate is readily visible (arrow).
- Disc Image 6.4 A low-power microscopic section of bone, including the dense cortex and the bony trabeculae. Also evident is the bone marrow with blood-producing cells and fat cells, which are clear.
- Disc Image 6.5 Microscopic appearance of cartilage (high power).
- Disc Image 6.6 A medium-power microscopic section of the cerebrum showing the outer gray matter containing neurons.
- Disc Image 6.7 A cross-section of the cerebellum.
- Disc Image 6.8 A cross-section of the midbrain (the uppermost part of the brainstem).
- Disc Image 6.9 A cross-section of the pons (the middle part of the brainstem).
- Disc Image 6.10 The circle of Willis at the base of the brain. Portions of the cerebellum (C), brainstem (BS), and temporal (T) and frontal (F) lobes of the cerebrum are visible. The vertebral arteries (yellow arrows) and basilar artery (black arrow) are immediately adjacent to the front of the brainstem. The carotid arteries (black arrowheads) have been cut during brain removal. The circle of Willis is composed of the anterior cerebral arteries (white arrowheads), the anterior communicating artery (yellow arrowhead), the middle cerebral arteries (blue arrowheads), the posterior communicating arteries (pink arrowheads), and the posterior cerebral arteries (white arrows).
- Disc Image 6.11 A portion of the spinal cord seen within the vertebral column after the anterior (front) of the vertebral column has been removed at autopsy. The cervical (neck) (white arrow) and thoracic (chest) (black arrow) portions of the spinal cord are visible.
- Disc Image 6.12 Right atrium (RA), tricuspid valve (TV), and right ventricle (RV) of heart.
- Disc Image 6.13 Right ventricle (RV) of heart, pulmonary valve (PV), and pulmonary artery (PA).
- Disc Image 6.14 Left atrium (LA), mitral valve (MV), and left ventricle (LV) of heart.
- Disc Image 6.15 Left ventricle (LV) of heart, aortic valve (AV), and aorta (A). Note the coronary artery ostia (the openings where the coronary arteries arise) (arrows).

- Disc Image 6.16 Normal gross appearance of the larynx with upper trachea, viewed from behind.
- Disc Image 6.17 A normal formalin-fixed lung that has been sectioned at autopsy.
- Disc Image 6.18 Normal respiratory epithelium (medium power).
- Disc Image 6.19 Normal microscopic appearance of the esophagus (medium power). Like the epidermis of the skin, it is lined by stratified squamous epithelium; however, the esophagus epithelium does not form a keratin layer on its surface.
- Disc Image 6.20 Gross appearance of the normal stomach (gastric) lining (mucosa).
- Disc Image 6.21 Medium-high-power microscopic appearance of the gastric (stomach) mucosa (lining).
- Disc Image 6.22 Histologic appearance of the small intestine (medium-high power).
- Disc Image 6.23 Normal colon microscopic appearance (medium-high power).
- Disc Image 6.24 Photograph of a cross-section through a normal liver at autopsy.
- Disc Image 6.25 A medium-power microscopic section of the liver. Note the central vein (CV) and the portal triad (P).
- Disc Image 6.26 Normal microscopic appearance of a bone marrow smear. Note the fat cells (clear cells), the numerous blue cells (blood precursor cells), red blood cells (arrows), and the single large cell (a megakaryocyte (M), which produces platelets).
- Disc Image 6.27 Microscopic appearance of the spleen. Most of the cells seen are white and red blood cells. The red blood cells appear pink/red, while the white blood cells are actually stained a blue color. The pink “scaffolding”-like supportive structures are the trabeculae (T).
- Disc Image 6.28 Microscopic appearance of the thymus (medium power). Most of the cells are lymphocytes (staining blue), with occasional pink, swirled structures referred to as Hassal’s corpuscles (arrows). The clear cells are fat cells.
- Disc Image 6.29 Microscopic appearance of a lymph node (medium power), primarily composed of lymphocytes and histiocytes. The rounded structures are called “lymphoid follicles” or “germinal centers.”
- Disc Image 6.30 A medium-power microscopic section of the adrenal gland, showing three zones of cortical cells (zona glomerulosa, zona fasciculata, and zona reticularis), and a small portion of medulla (upper left corner).
- Disc Image 6.31 Microscopic section of the pancreas (medium-high power). A majority of the cells seen are glandular cells of the exocrine pancreas (producing digestive enzymes). The rounded area of other cells is referred to as an islet of Langerhans (arrows) and contains cells of the endocrine pancreas. An exocrine duct is indicated by the arrowhead.
- Disc Image 6.32 Gross appearance of a normal kidney removed at autopsy.
- Disc Image 6.33 A longitudinal section of a kidney, showing the outer cortex, the inner medulla, and the collecting system.
- Disc Image 6.34 Microscopic appearance of an ovary (medium-high power) of a postmenopausal woman.
- Disc Image 6.35 Microscopic appearance of a testis (medium-high power), with numerous sperm visible within the seminiferous tubules.
- Disc Image 6.36 Microscopic appearance of the prostate gland (medium power).

Selected References

- Alberts B, Johnson A, Lewis J, Raff M, Roberts K, Walter P. *Molecular Biology of the Cell*, 5th ed. New York, NY: Garland Science; 2008.
- Boron WF, Boulpaep EL. *Medical Physiology – A Cellular and Molecular Approach*. Philadelphia, PA: Saunders; 2003.
- Dorland's Illustrated Medical Dictionary*, 27th ed. Philadelphia, PA: W.B. Saunders Company; 1988.
- Mills SE, editor. *Histology for Pathologists*, 3rd ed. Philadelphia, PA: Lipincott Williams & Wilkins; 2007.
- Moore KL, Dalley AF II. *Clinically Oriented Anatomy*, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
- Zigmond MJ, Bloom FE, Landis SC, Roberts JL, Squire LR. *Fundamental Neuroscience*. San Diego, CA: Academic Press; 1999.

Part II
General Topics in Forensic Pathology

Chapter 7

The Postmortem Forensic Examination/Autopsy

Show me, O Lord, my life's end and the number of my days; let me know how fleeting is my life. You have made my days a mere handbreadth; the span of my years is as nothing before you. Each man's life is but a breath.

Psalm 39:4–5

Abstract Chapter 7 begins by providing a synopsis of the differences between a hospital (medical) autopsy and a forensic (medicolegal) autopsy. Most of the chapter deals with a relatively detailed discussion of the various aspects of a forensic autopsy, including investigation, external examination, internal examination, and ancillary procedures, and the chapter concludes by discussing cases in which autopsies are not performed, the role of the autopsy assistant, and office accreditation and forensic autopsy standards.

Keywords Autopsy · Forensic autopsy · External examination · Autopsy assistant · Accreditation · Forensic autopsy standards

Introduction

The reader is referred to Chapter 3 (Introduction to Forensic Pathology) as prerequisite reading for this chapter, which presents a relatively detailed explanation of the forensic autopsy and includes a section that explains the important differences between hospital (medical) autopsies and forensic (medicolegal) autopsies. Most of the chapter deals with the basic components of a forensic autopsy (Table 7.1), namely the investigation, the external examination, the internal examination, various ancillary procedures, and the autopsy report. There are also sections that specifically deal with forensic examinations without autopsies (so-called “external examinations,” where an autopsy is not believed to be necessary, but body examination still occurs) and cases that require medicolegal investigation but have no body available for examination for one reason or another.

Table 7.1 Components of a forensic autopsy

1 – Investigation
2 – External examination
3 – Internal examination
– Routine examination:
• Trunk
• Head
• Neck
– Special dissections:
• Eye removal
• Middle/inner ear removal
• Vertebral artery evaluation
• Cervical vertebral column examination
• Spinal cord removal
• Anterior neck dissection
• Posterior neck dissection
• Facial peel down
• Skin incisions
• Fracture evaluation
• Examination of deep veins of the legs
• Soft tissue dissection
4 – Ancillary procedures
– Radiology
– Toxicology
– Chemistry
– Serology
– Microbiology
– Histology
– Other tests (molecular, biochemical, etc.)
– Consultation (neuro- and cardiac pathology, other forensic disciplines)
5 – Autopsy report

Forensic Versus Hospital Autopsies

An autopsy represents a postmortem surgical examination of a human body, but not all autopsies are equivalent. There are two basic types of autopsies: forensic or medicolegal autopsies and hospital or medical autopsies. There are several very important differences between the two. The first is that forensic autopsies fall under the jurisdiction of the local governmental death investigation office (coroner or medical examiner), whereas hospital autopsies do not. As such, forensic autopsies do not require consent from the legal next-of-kin and the coroner or medical examiner may, by law, order an autopsy. Hospital autopsies require a properly executed and witnessed consent signed by the decedent's (dead person's) legal next-of-kin before the autopsy can be performed.

Making certain that the person being autopsied is, in fact, the person he/she is supposed to be is of utmost importance, no matter what type of autopsy is being performed. In virtually every hospital autopsy performed, the decedent's identity is already known and well-documented via identification bracelets and within the

medical records that accompany the body. In contrast, a very important aspect of every forensic autopsy involves the positive identification of each decedent. This issue is so important that an entire chapter of this book addresses it (Chapter 9). Depending on the structure of the death investigation system, this very important task may be the official responsibility of different entities. In certain jurisdictions, the legal responsibility rests with non-pathologist death investigators (coroners or non-pathologist medical examiners), while in other offices, the responsibility rests with those performing the autopsies. In either case, all persons and offices involved should work together to ensure positive identity in every case.

Frequently in hospital autopsies, the cause of death is already known, and the autopsy is performed to attempt to answer specific questions that family members or physicians have regarding the case, such as disease extent, effectiveness of therapy, etc. For these reasons, hospital autopsies are frequently limited to the examination of certain body parts. In addition, the complete medical record is usually available to the pathologist prior to hospital autopsy performance (Fig. 7.1). As a result of this the “background information” being readily available prior to autopsy, a pathologist can perform the autopsy with specific focus. In contrast, while the cause of death may be obvious in certain forensic cases and a certain amount of background information may be available prior to examination, forensic autopsies are usually performed with the intent of documenting all findings, rather than being focused on a single issue or two. Frequently in forensic work, all of the important background information, as well as all of the questions that will eventually be asked regarding a specific case, are not known when the autopsy is being performed. In general, if resources allow, the most thorough postmortem forensic examination involves a complete autopsy (head, neck, and trunk), although different office and practitioner policies and protocols may allow for limited dissections in specific case types.

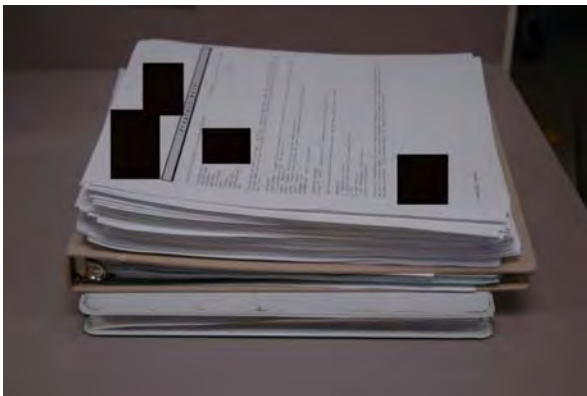


Fig. 7.1 Hospital records are typically available for review prior to hospital autopsies

Another major difference that exists between hospital and forensic autopsies is the fact that, in many hospital autopsies, the external examination is relatively



Fig. 7.2 Photography is an integral part of forensic autopsies

unimportant, whereas in forensic cases, the external examination can frequently be one of the most important aspects of the case. As such, photography is a key component of the forensic autopsy (Fig. 7.2), whereas it is usually considered optional in the hospital autopsy.

Certain other procedures and tests are common in forensic cases, but quite rare in hospital cases. The two most common are toxicology testing and radiography (X-rays). A significant percentage of forensic cases will have positive toxicology tests (drugs are detected), which will, in many cases, be an important factor in determining the cause of death. Likewise, a significant percentage of forensic cases also require radiography (Fig. 7.3). Various specialized dissections can occur in



Fig. 7.3 Radiography is commonly used in many types of forensic cases

either type of autopsy, but are probably more common in forensic cases. These include anterior (front) neck dissections, posterior (back) neck dissections, spinal cord examination, eye removal and examination, dissection of the deep veins of the legs, etc.

“Fixing” organs or tissues in formalin (a preservative solution containing formaldehyde) *prior to* dissecting them is a procedure that can occur with either type of autopsy, although it is probably more common in hospital cases, where many pathologists were trained to “fix” the organs prior to detailed dissection. In forensic cases, however, it is not unusual in certain cases to choose to fix a particular organ prior to sectioning, with the brain and heart being the most common organs in which this is done. It is important to note here that formalin fixation will change the appearance of tissues, usually causing a darker, less shiny appearance (this is obvious when comparing photographs of “fresh” organs to those of “fixed” organs), and will also cause the tissues to become more firm, thus making dissection a bit easier.

A final difference between hospital and forensic autopsies involves those who actually perform, or assist with, the autopsy. In many hospital-based pathology practices, pathology assistants (PAs) are employed to assist pathologists with many functions. In some practices, they are responsible for performing the “gross” part of the autopsy, with pathologists only performing histologic (microscopic) examinations. However, this is not acceptable practice in forensic autopsies. This topic will be addressed further at the end of this chapter.

Investigation

One of the most important aspects of a forensic death examination is the investigation surrounding the death, and much of this important information is obtained by the work of the death investigators. The title of the individuals responsible for performing the initial (and follow-up) death investigation in a particular jurisdiction varies from place to place. The role may be filled by a coroner, a deputy coroner, a non-pathologist medical examiner, a medical examiner investigator, a field agent, or a variety of other titles applied to the person responsible for receiving notification of death and then performing the initial death investigation. Depending on the jurisdiction, the forensic pathologist may or may not have the ultimate responsibility and associated authority to ensure that the death investigation is as complete as it needs to be. In settings where the pathologist does not have this authority, it is essential that there be a good working relationship between the pathologist and the death investigator. Chapter 4 deals specifically with death investigation and the important issues that relate to this essential aspect of the forensic autopsy, and the reader is referred to that chapter to review important issues related to the investigation. Pathologists cannot and should not “operate in a black box” and it is vitally important that they be provided with as much information as possible regarding a given death.

External Examination

As mentioned previously, the external examination is frequently a very important aspect of the forensic autopsy, and in certain cases, is quite lengthy. There are important forensic issues that relate to the performance of a thorough, detailed external examination. It is important for identification issues, postmortem interval estimates, injury documentation, evidence collection, and often cause and manner of death determination.

The observation and documentation of various external characteristics of the decedent is the essence of the external examination. Height, weight, skin pigmentation, postmortem changes (livor mortis, rigor mortis, etc.), eye color, general body development and nutritional status, hair color and length, presence or absence of facial hair, presence and condition of dentition (teeth), presence and description of permanent body surface markings (scars, tattoos, piercings), presence and description of clothing, jewelry, and property, and the presence and type of medical therapy are all considered parts of the general external examination (Fig. 7.4). Documentation of these features should occur in every case although different pathologists may document them in different ways – some choose to use a body diagram (Disc Image 7.1). These findings may aid in issues related to identification, cause of death, and/or manner of death determination.



Fig. 7.4 The external examination is of extreme importance in forensic autopsies. Pathologists must document clothing, medical treatment, identifying features, and injuries

Depending on the case, various items of evidence must be collected during the autopsy. This can include clothing, blood and other body fluids, projectiles, knives, and various items of trace evidence. In a hit-and-run pedestrian death, clothing examination can be of paramount importance, because paint chips or glass fragments or tire tracks from the vehicle may be present on the clothing. In these types of cases, as well as several other case types (including virtually all homicides), the clothing should be examined (or retained for subsequent examination). The clothing, as well as the skin surface, may, in certain cases, contain other important trace

evidence (hairs, fibers, gunshot residue, etc.) (Disc Image 7.2). Fingernails may contain debris or even blood or tissue that may link the decedent to a specific location or suspect and the documentation and collection of such evidence may provide very important forensic evidence in a particular case. Likewise, the collection of sexual activity evidence (using a “sexual activity kit”) can be important in certain cases. In all cases where trace and other evidence on the exterior surface of the body is considered important or even potentially important, it is essential to collect the evidence prior to disrupting the body by cleaning it. A common procedure used in many jurisdictions involves placing paper bags over the hands of the decedent, prior to transfer to the morgue, in order to preserve any trace evidence that might be present on the hands/fingers (Fig. 7.5). Identification and collection of such evidence may be very difficult at the death scene and in many instances, the morgue provides a more controlled environment for such collection – better lighting allows for better identification of such evidence. Plastic bags are not appropriate as condensation can lead to moisture accumulation and destruction, or washing away, of important evidence.



Fig. 7.5 In some cases, paper (not plastic) bags are placed over the hands at the death scene, in order to preserve any trace evidence that might be on the hands and/or fingers

Another crucial aspect of the external examination in forensic cases involves the documentation of injuries (or even the lack of injuries). As such, it is important for pathologists to identify and describe any injuries that are observed during external examination. In general, forensic pathologists usually attempt to characterize the category of injuries seen, such that all blunt force injuries are described as such, while sharp force injuries, gunshot injuries, asphyxial injuries, etc. are described separately. Detailed descriptions of these and other injury types are provided in subsequent chapters within this textbook. With certain major injuries on the skin (and elsewhere), such as gunshot wounds, sharp force injuries, and other injury types, pathologists should describe the size, shape, and character of the wound, along with the exact physical location on the body, with a description of the general location (for example: lower right abdomen), as well as measurements from the top of the

head (or bottom of the feet), the midline, and if practical, a fixed anatomic landmark (for example: the umbilicus, the left nipple, or the upper attachment of the right ear). With minor injuries, a more general description is appropriate, although providing detailed descriptions is also acceptable. Major injuries are not necessarily large, so part of the complete external examination should include careful evaluation of the conjunctival surfaces (inner eyelids and eyes) for petechial hemorrhages (pinpoint areas of bleeding), examination the neck and scalp for subtle injuries, intraoral (mouth) examination for inner cheek, lip, and gum injuries, and careful examination of the hands (including nails) and external genitalia for subtle injuries.

Photographic documentation of major injuries is considered standard practice. Identifying markers bearing the unique autopsy number, with a measurement scale, should be included in a sufficient number of photographs to ensure that the photos correspond to the specific case. In order to best document a particular injury, it is necessary to wash extraneous blood away from the skin surrounding the wound, and it may also be necessary to shave the hair from around the injury (Figs. 7.6 and 7.7). These procedures are entirely appropriate, but should only occur after collection of trace evidence (see below) or after it has been determined that such collection is not necessary. A final note regarding injuries is the fact that certain defects on the skin surface may mimic true antemortem (before death) injuries. Medical intervention, various artifacts, and even decompositional changes can occasionally result in skin changes that simulate true injuries.



Fig. 7.6 An example of how blood can obscure the true nature of an injury. In this photograph of a man with a gunshot wound of the head, it is impossible to identify the wound

A final aspect of the overall autopsy that frequently occurs during the external examination is the collection of toxicology samples. As will be discussed later, toxicology tests are best performed on peripheral blood samples, such as blood collected from the upper thigh (femoral) region. As such, attempting to collect blood from this site during the external examination is common (Fig. 7.8). Other samples, such as vitreous (eye) fluid (Disc Image 7.3) and hair samples can also be collected during



Fig. 7.7 In this photograph of the same man shown in Fig. 7.6, the body has been cleaned and the hair has been shaved from around the gunshot wound. Cleaning and shaving allows for proper documentation of injuries



Fig. 7.8 Collection of blood from the femoral region (groin) is the preferred site for postmortem toxicology testing

the external examination, although in some cases (especially certain child deaths), vitreous fluid should not be collected until the pathologist has ruled out head trauma on internal examination.

Internal Examination

The usual internal examination involves the surgical cutting open of the body with removal of the internal organs, beginning with the trunk region (chest, abdomen, and pelvis), followed by the head (brain), and finally, the neck. The process is referred to as “evisceration,” since it involves the removal of the internal organs (the viscera). The neck is often saved until last, so that as much blood drains out of the neck

as possible (both inferiorly, toward the trunk, and superiorly, toward the brain) as this makes examination easier. In certain types of injuries, very subtle internal neck injuries may be present.

Opening the skin of the trunk often involves the traditional Y-shaped incision, extending from the shoulders bilaterally (both sides), to the lower chest midline, and then running inferiorly down the midline of the abdomen to the pubic bone (Fig. 7.9). The skin and underlying soft tissues (muscles and fat) are reflected laterally (to the sides), exposing the anterior chest wall superiorly and the peritoneal cavity inferiorly (Fig. 7.10). The peritoneal cavity is the space that contains the abdominal and pelvic organs. At this point, the anterior (front) of the chest wall can be removed – some prefer to use manual rib-cutters while others use a bone-saw (Fig. 7.11). In younger individuals, simply using a scalpel and cutting through the cartilaginous portions of the anterior ribs is possible (Disc Image 7.4). When the



Fig. 7.9 The classic “Y-shaped” incision used during the performance of an autopsy



Fig. 7.10 The skin and underlying subcutaneous tissues are reflected, thus exposing the anterior (front) chest wall, as well as the opened abdominal (peritoneal) cavity. The liver, stomach, and intestines are visible



Fig. 7.11 A bone saw is used to cut the front of the chest plate off of the chest wall



Fig. 7.12 The anterior chest plate is removed, exposing the pleural cavities (containing the lungs) and the front of the pericardial sac

anterior chest wall has been removed, the pleural cavities (where the lungs reside) are visible, as is the anterior aspect of the pericardial sac (the lining around the pericardial cavity, where the heart resides) (Fig. 7.12). The anterior sac can be opened to visualize the pericardial cavity (Disc Image 7.5). At this point, all four of the trunk's cavities are visualized. The diaphragm separates the peritoneal cavity below from the pleural and pericardial cavities above. The pathologist will take note of any abnormalities within the body cavities. Usually these cavities contain a small amount of clear, yellow fluid; however, abnormalities may include excessive clear fluid, purulent fluid (pus), blood, and adhesions (scarring). If trunk injuries are present, the pathologist will probably spend some time at this point during the autopsy attempting to determine the injuries that exist.

At this stage in most forensic autopsies, the pathologist will also ensure the collection of appropriate fluids for toxicology testing (blood, urine, bile) (Disc Images 7.6, 7.7, 7.8). The next step involves removal of a majority of the intestines, usually from distal duodenum to rectum (Figs. 7.13 and 7.14 and Disc Image 7.9). Depending on the case or office policy, the intestines may or may not be “run,” or opened. From this point on, there are a variety of evisceration techniques that may be employed. A common technique is referred to as the Virchow method (named after the “father of pathology,” Rudolph Virchow) and involves the removal of organs



Fig. 7.13 The small intestines may be removed in one large block by cutting the base of the mesentery at the posterior (back) of the abdominal cavity



Fig. 7.14 Alternatively, the small intestines may be removed by cutting the mesentery attachment immediately adjacent to the entire small intestine length. Removal in this fashion allows the intestine to be more easily opened and examined internally

individually. In this technique, each individual lung is removed (Fig. 7.15), as is the heart (Fig. 7.16). The liver (Fig. 7.17) and spleen (Fig. 7.18) are removed, followed by a “block” of tissue including the duodenum, stomach, pancreas and lower esophagus (Disc Images 7.10 and 7.11). Each adrenal gland and kidney is removed individually (Disc Images 7.12, 7.13, 7.14), and then the pelvic block (bladder and prostate in males (Disc Image 7.15), bladder, upper vagina, uterus, fallopian tubes, and ovaries in females) is removed. Finally, the middle esophagus, lower trachea and mainstem bronchi, and aorta are removed (Disc Image 7.16). Each individual organ or block of organs is then systematically dissected as the pathologist looks for disease and injury. Prior to dissection of individual organs, the organs are typically weighed (most often using the metric system: grams) (Disc Image 7.17).

An alternative approach to evisceration via the Virchow method involves the removal of the entire trunk organ block in one large “en bloc” conglomeration



Fig. 7.15 The lungs are pulled forward and removed by cutting the mainstem bronchi and the pulmonary arteries and veins. In this photo, the left lung is being removed



Fig. 7.16 The heart is pulled forward (upward) and removed by cutting the inferior vena cava, the pulmonary veins, the pulmonary artery and aorta, and the superior vena cava



Fig. 7.17 The liver must be cut away from the diaphragm, as well as the inferior vena cava and biliary system in order to remove it from the body

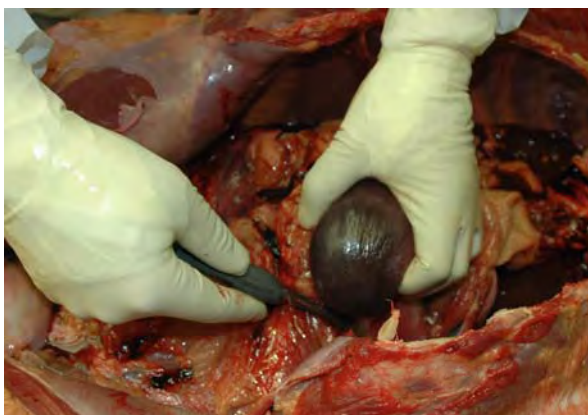


Fig. 7.18 The spleen is reflected forward and cut away from the region near the tail of the pancreas

of organs and tissues (Disc Image 7.18; also refer to Disc Image 20.39). This is commonly called the Rokitansky method, but is more accurately referred to as the Letulle (*en masse*) method. The Ghon (*en bloc*) method, where several blocks of connected organs are removed, can be considered halfway between the Virchow and Letulle methods. With either the Ghon or Letulle method, the organ blocks are dissected after removal from the body. These methods allow for better visualization of how various organs relate to one another, which can be quite important in select cases. Ultimately, the organs are individually dissected, just as they are in the Virchow method. During or after removal of organs, pathologists may note the presence of other pathology or trauma, including bone injuries and/or collections of blood within the soft tissues of the mediastinum (behind the heart and between the lungs), the retroperitoneum (behind the abdominal cavity), or the body cavity walls. Although the Letulle method is commonly referred to as the Rokitansky method, the

Rokitansky method actually represents a rarely performed in situ method, in which organs are examined and partially dissected while remaining in the body.

It is beyond the scope of this chapter to provide all of the details necessary regarding the examination and dissection of each organ; however, some basic points will be discussed. With each organ, the pathologist will perform a dissection specific to the organ in question. With hollow structures, such as blood vessels and portions of the GI tract like the esophagus and stomach, the structure is cut open in order to reveal the inside. With many solid organs, the pathologist will make many parallel cuts, in a fashion similar to slicing a loaf of bread. For this reason, some pathologists refer to the process of serially sectioning an organ as “bread-loading” the organ. No matter which dissection technique is used, pathologists will usually save at least a single small portion of each organ, preserving it in formalin (a solution containing formaldehyde) within a “save” or “stock” container (Disc Image 7.19), and this “formalin-fixed tissue” will be maintained for a relatively long time, sometimes many years. In some instances, the pathologist will submit smaller tissue samples for histology (microscopic) examination (Disc Image 7.20).

The pathologist will take note of the heart size and weight. Hearts that weigh too much are at risk for sudden, lethal arrhythmias (irregular rhythms). The overall anatomy of the heart will be evaluated, looking for any congenital anomalies. An important part of the heart examination involves looking at the coronary arteries, which are commonly evaluated by making serial cross-sectional incisions through them and the epicardial fat within which they reside, in order to evaluate for atherosclerotic narrowing and other disease processes (Figs. 7.19 and 7.20). After the major coronary arteries are evaluated in this way, the pathologist will usually make several cross-sectional cuts all the way through the right and left ventricles, beginning from the apex and moving toward the atria in order to examine the muscular walls of the ventricles (Disc Image 7.21). Finally, a common examination



Fig. 7.19 Examination of the removed heart includes serially sectioning the major coronary arteries which are embedded in the epicardial fat. This photo shows the anterior (front) of the heart, after these serial cuts have been made

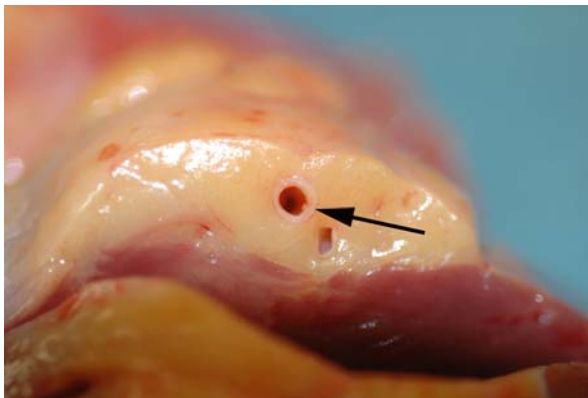


Fig. 7.20 A photo showing a section through the epicardial fat with a coronary artery

technique of the heart ends with the pathologist “following the flow of blood through the heart,” as he/she opens the remainder of the heart and evaluates the atria and heart valves. First, the right atrium is opened, followed by the tricuspid valve, and then the pulmonic valve. Next, the left atrium is opened, followed by the mitral valve and the aortic valve. Special sections can be taken at this point to evaluate the conduction (electrical) system of the heart.

The lung weights may provide useful information, as the weight increases with various situations and disease processes. For example, in situations of heart failure, the lungs are frequently heavy because of the backup of fluid into the lungs; however, the lungs could also be heavy because of an infectious process (pneumonia). Pathologists will evaluate the large airways of the lungs (bronchi), the larger blood vessels of the lungs, as well as the parenchyma (the lung tissue itself). The typical method of evaluating the parenchyma involves serially sectioning the organ (Fig. 7.21).



Fig. 7.21 Examination of the lung includes serially-sectioning the organ

The liver, spleen, kidney, adrenal glands and pancreas are mostly solid organs that require serial sectioning and examination at autopsy (Figs. 7.22 and 7.23 and Disc Images 7.22 and 7.23). The liver also has the attached gallbladder, while the kidneys actually have a non-solid component, where the urine collects and drains into the ureters. Each of these organs is individually examined by the pathologist. If a recognizable thymus is evident at autopsy, examination with sectioning is also performed on this thoracic organ. Each of the remaining “hollow organs” is opened to evaluate the mucosal surfaces (the inner lining) – this includes the esophagus, the stomach, the duodenum, and the bladder, and depending on office policy and case type, the remainder of the intestines may also be opened. In many cases, urine will be collected from the bladder for toxicology testing and, in some instances, gastric (stomach) contents may be retained as well. The aorta and its major branches, as well as the trachea, will also be opened and evaluated. Finally, the female and



Fig. 7.22 Examination of the liver involves serially-sectioning the organ



Fig. 7.23 Examination of the kidney involves cutting the organ in half longitudinally

male organs will be dissected and evaluated; testicles may or may not be evaluated, depending on office/pathologist protocol and case type. Once the organs of the trunk have been removed, the trunk represents a single, hollowed-out, empty “shell” (Fig. 7.24).



Fig. 7.24 After the trunk organs and diaphragm have been removed, the trunk appears to be one large cavity

In order to examine the brain at autopsy, the scalp and skull must first be removed. The typical scalp incision involves a cut that is made from behind one ear, over the crown, to behind the opposite ear (Fig. 7.25). The scalp is then peeled forward and backward, off of the skull (Fig. 7.26); when the scalp is peeled far enough forward, the inverted scalp actually covers the decedent’s face (Disc Image 7.24). Reflecting



Fig. 7.25 The initial step required for brain removal includes making an incision from behind one ear, across the top of the scalp, to behind the opposite ear

Fig. 7.26 The scalp is then reflected forward, exposing the underlying skull



the scalp in this fashion allows the pathologist to evaluate for subscalpular (subgaleal) trauma. Note that the location of the incision also allows for an open-casket funeral. Next, a portion of the skull is removed, using a bone saw (Fig. 7.27). Depending on the case or office protocol, the temporal muscles which attach to the sides of the skull may be removed prior to cutting the skull (Disc Image 7.25). After the cuts are made in the skull, it is carefully pulled away from the underlying brain. In some cases, the dura mater peels away from the bone as the bone is removed, such that the dura remains covering the brain (Fig. 7.28). In other cases, the dura remains



Fig. 7.27 A bone saw is used to cut-out a piece of skull, thus allowing access to the underlying brain



Fig. 7.28 In some instances, the bone can be removed without disturbing the underlying dura. In such cases, the skull cap actually peels off of the dura as it is being pulled off of the head

Fig. 7.29 In other cases, the dura remains adherent to the inner aspect of the skull cap so that when the skull is removed, the dura is also removed, thus exposing the underlying brain



adherent to the inner aspect of the skull cap as it is removed, thus exposing the underlying brain (Fig. 7.29 and Disc Image 7.26). At this point, evaluation is made for epidural, subdural, and subarachnoid hemorrhage or other abnormalities in these locations. Next, the brain is removed from the cranial cavity (Disc Image 7.27), after which the remaining dura mater must be removed from the skull, including the basilar skull, so that the skull can be thoroughly examined (Disc Image 7.28). The dura should be removed from the skull cap, as well as the basilar skull. At this point, the pituitary gland can be removed. In many forensic autopsies, the brain is examined in the fresh state; however, in select cases, the pathologist chooses to allow the brain to “fix” in formalin for several weeks prior to further evaluation (Fig. 6.11 shows a

formalin-fixed brain). Whether examination occurs in the fresh state or after formalin fixation, the typical brain examination involves serially sectioning of all parts, including cerebrum, cerebellum, and brainstem (Figs. 7.30 & 7.31).

Following removal of the trunk organs and brain, internal neck dissection can proceed. At a minimum, the upper trachea and esophagus, the larynx, the thyroid gland, and the hyoid bone should be examined. The most thorough examination of these structures involves their removal with subsequent dissection (Fig. 7.32). An even more thorough examination involves the additional removal and examination of the tongue. A layer-by-layer dissection of the anterior neck is also possible (see below). After neck examination, the pathologist should evaluate the integrity of the cervical vertebral column by palpating (feeling) and moving the neck with one hand in the cranial cavity and one over the internal neck region.



Fig. 7.30 The brain is examined and is serially-sectioned in the “fresh” (non-fixed) state, although in some cases, the brain is fixed in formalin prior to sectioning

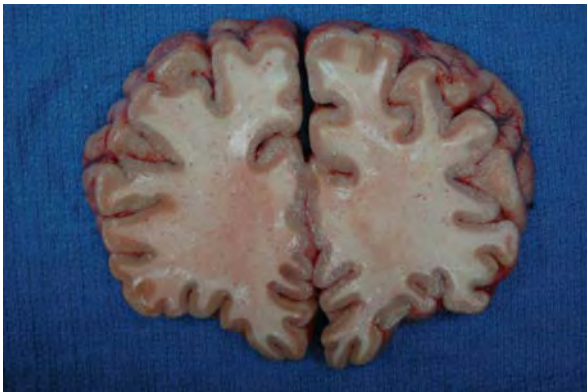


Fig. 7.31 A section of “fresh” (non-fixed) brain at autopsy



Fig. 7.32 Examination of the anterior (front) of the neck typically includes removal of the trachea, the larynx, and the thyroid gland

What has been described to this point is what most would consider the minimum procedures expected in a “complete” forensic autopsy. Depending on office or pathologist policy or practice, slight variations might exist. For example, some might not remove the tongue in every case while others always remove the tongue. In some situations, however, only a limited autopsy might be performed. Aside from the minor variations which may exist between different offices or pathologists regarding what encompasses a complete autopsy, there are a variety of other specialized dissections that may be performed, depending on the case type or the circumstances of the case. A brief description of several of these special dissections follows.

Special dissections involving the head and face include the “facial peel-down” (Disc Images 7.29 and 7.30), which allows for visualization of the bones and subcutaneous tissues of the face. This procedure is relatively disfiguring, and if a pathologist chooses to perform it, he/she should advise the funeral home that it is occurring. Eye removal is frequently performed in suspected child abuse cases; they are removed from above, through the base of the skull that normally covers them (refer to Disc Image 20.41). Subsequent examination typically occurs after formalin fixation when the eyes are usually cut open to evaluate for the presence or absence of retinal hemorrhages. Middle/inner ear removal via the base of the skull is occasionally performed, in order to look for disease processes such as infection (Disc Image 7.31). Because these structures are deeply embedded within the bones of the base of the skull, decalcification is typically necessary when this examination is performed.

Special dissections involving the neck and spine come in a variety of types. A relatively frequent dissection amongst forensic pathologists is a layer-by-layer anterior neck strap muscle dissection, where each muscle, as well as the thyroid gland and other neck structures, is meticulously dissected, in order to identify injuries (Fig. 7.33 and Disc Images 7.32, 7.33, 7.34, 7.35). When this examination



Fig. 7.33 Layer-by-layer anterior neck dissection

is performed, the hyoid bone and tongue should always be a part of the examination. A posterior neck dissection allows the pathologist to look for muscle (and skeletal) trauma involving the posterior neck. These dissections, however, may be limited (Disc Image 7.36), simply looking for gross hemorrhage, or they may be more detailed and relatively extensive. Occasionally, it becomes necessary to examine the vertebral arteries and there are a variety of methods available for such examination, none of which is easy. Some choose to approach the arteries posteriorly, some attempt an anterolateral approach, and some choose to cut a large square in the base of the skull and dissect out much of the entire cervical vertebral column for subsequent dissection. Each approach can also be used to more thoroughly evaluate the cervical vertebral column itself. If the cervical spinal cord (or other parts of the spinal cord) requires examination, the cord can be exposed and removed from an anterior approach or a posterior approach, using a bone saw.



Fig. 7.34 Spinal cord removal via an anterior (front) approach. An alternative method involves removing the spinal cord from a posterior (back) approach



Fig. 7.35 A body following complete autopsy. Note the sutured Y-shaped incision. Also note that, after the scalp is reflected back into its original position, and the incision is stitched, it is impossible to detect from the front that the head has been autopsied. This allows for adequate viewing of bodies at funerals



Fig. 7.36 Bodies are typically stored in coolers prior to and after autopsy. Coolers can contain individual “drawers” or “shelves,” as shown, or be composed of large walk-in/roll-in units

Occasionally, pathologists will make skin incisions in order to identify or rule out the presence of subcutaneous hemorrhage. This can involve large parts of the skin, such as the back or the extremities (refer to Fig. 20.19), or relatively small areas, such as the wrists or soles of the feet (refer to Figs. 21.16 and 21.17), depending on the circumstances of the particular case.

Other dissections that are frequent in certain forensic autopsies include examination of the deep veins of the legs for the presence of deep venous thromboses (blood clots) in cases of pulmonary thromboembolism (Disc Image 7.37), removal and examination of the spinal cord (Fig. 7.34), dissection of various anatomic areas in order to visualize fractured bones, and dissection of virtually any part of the body in order to identify injuries and to collect evidence, such as a bullet.

Once an autopsy is complete, assistants will typically place any residual organ and tissue parts into a plastic bag and place them back into the body cavity. They will then sew the scalp and trunk incisions and clean the body (Fig. 7.35). The body will be placed in cold storage until mortuary personnel arrive to take the body for embalming and funeral arrangements (Fig. 7.36).

Ancillary Procedures

So far, we have covered the basic components of a forensic autopsy, including the investigation, the external examination, and the internal examination. We now must address the various ancillary procedures that are sometimes utilized to complete the forensic autopsy examination. Some of these procedures are used in almost every case, while others are used in only a small fraction of cases. Each is important in its own right.

A forensic pathologist cannot properly perform his/her duties without the availability of radiology equipment. While X-rays are not required in every case, a significant number of cases do require them. From the standpoint of ensuring positive identification of a decedent, radiology may be necessary for dental or other radiological comparison. X-rays are required in virtually every gunshot wound case, even if it appears on external examination that all bullets exited the body. Many forensic pathologists perform X-rays on all sharp force injury cases as well, looking for any retained foreign objects (like a broken knife blade). In suspected child abuse cases, it is important to perform a relatively extensive X-ray examination, involving the entire skeleton. Full body X-rays are also appropriate in charred bodies, as well as certain decomposed remains. Radiographs may also be useful in a variety of other case types as they can help to identify other foreign bodies, medical therapy, fractures, collections of blood, and collections of air.

Toxicology involves the evaluation of various bodily fluids or tissues for the presence of drugs or toxins. Chapter 11 presents a more thorough review of this topic, but a quick review will be provided here. A qualitative test is a screening test that tells whether or not a particular substance is present. If a substance is present the test is said to be “positive,” but if the substance is not present, the test is said to be “negative.” In order for a test to be considered forensically significant it is essential that a second test, using a different method of detection, be used. For this reason, a quantitative test is in order whenever a qualitative test is positive. The quantitative test represents the second and confirmatory test, and it provides a specific amount (quantity) of the substance in question.

Toxicology testing is performed on most forensic autopsy cases. Exceptions involve cases where death is delayed for several days following the injuries. In these cases, it is important for death investigators to attempt to obtain hospital blood and fluid samples collected at or around the time of the initial injury. Different offices have different standard protocols for what they consider “routine toxicology testing,” and it is important to be aware of what is included in a particular

office's toxicology testing. Common sample types include urine and blood, but other samples may include cerebral spinal fluid (CSF), bile, vitreous (eye fluid), gastric contents, and various solid organs (liver, kidney, brain). In general, peripheral blood (femoral) is better than central blood (heart). It is important to label the collection site for every sample collected. Regarding blood, it is important to collect some sample in tubes containing sodium fluoride (gray-top tubes). Other specific test types may require special tube types, and consultation with the toxicology laboratory is advised so that samples are collected appropriately.

Certain postmortem chemistry tests can also be performed; some of these involve blood, but the most common type involves vitreous fluid. The reader is referred to Chapter 21 for a detailed description of the tests available. Certain serology tests, wherein specific antigens and/or antibodies are detected, and certain microbiology tests, can be performed in postmortem samples. Bacterial, fungal, and viral cultures may or may not be reliable after death and results should be interpreted in light of other autopsy findings.

Histology is the term used to describe the process by which the microscopic anatomy of a tissue is evaluated. As discussed above, there are certain cases where it becomes necessary for the forensic pathologist to look at the microscopic appearance of an organ or tissue. Protocols for which cases require histologic examination vary from one office to the next, and in some offices, every case (other than skeletal remains) has histology performed. Similarly, protocols regarding which tissues need to be examined vary from office to office and from pathologist to pathologist; however, there are certain cases that always require histologic examination in order to determine the cause of death. Routine histology involves the processing of small samples of organs and tissues such that stained, extremely thin, sections of tissues are placed on glass slides for visualization under a microscope. The routine stain that is used by many pathologists is the hematoxylin and eosin (H&E) stain. Other special stains can be used to help identify certain things, such as micro-organisms or scar tissue. Highly-specialized staining techniques known as immunohistochemistry or immunoperoxidase or in-situ hybridization (a type of molecular test) staining can be used in selected cases for identifying more complex or specialized substances or micro-organisms.

In addition to histology, the forensic pathologist may have an entire battery of additional test types or consultations available to assist in the diagnosis of various diseases or abnormalities. These include, but are not limited to, DNA testing, other molecular tests, biochemical testing, and genetic/chromosomal testing. Consultation with other medical/pathology specialists, such as cardiac pathologists and neuropathologists, is sometimes available. Consultation with other forensic scientists, such as anthropologists, toxicologists, odontologists, and entomologists, is appropriate in selected cases.

Autopsy Report

There are several parts that should be included in a forensic autopsy report. To an extent, some variation can exist from one office to the next, but all of the features

described below should be included. For legal purposes, the creation and filing and maintaining of reports must be part of the normal operating practice of the office.

Autopsy reports should contain a unique, identifying case number, as well as demographic information regarding the decedent (name, birthdate, gender). Information regarding date and time of death, positive identification, the date, time and place of autopsy, who performed the autopsy, and any pertinent information regarding death investigation and/or police agencies involved should also be included. Some reports contain information regarding autopsy assistants and who was in attendance at the autopsy. Many reports provide some of the circumstantial information regarding the case.

Many forensic autopsy reports have specific sections dealing with the external examination and the internal examination. Included in the external examination are various physical characteristics of the body (height, weight, eye color, gender, skin pigmentation, identifying marks and scars, medical therapy, etc.), as well as clothing and jewelry, and any evidence of postmortem change. Many forensic pathologists choose to not include evidence of injury within this section, and instead opt for a separate section that specifically deals with injuries.

The internal examination section provides a description of the organ systems, the body cavities, and the other tissues of the body. Usually, each system is described separately, and weights are provided for most of the organs. As with the external examination, many forensic pathologists choose not to include evidence of injury in this section, preferring to have a separate injury section.

As mentioned, many forensic pathologists include a separate “evidence of injury” section within their autopsy report. The rationale is that it seems rather disjointed to include injury descriptions interspersed amongst the descriptions of normal or diseased organs or tissues. A separate evidence of injury section allows descriptions of external injuries and internal injuries in a relatively concise fashion. For some injury types, such as blunt force head injuries, it is appropriate to start superficially (at the skin surface) and describe different depths of injury (skin, scalp, skull, dura, arachnoid, brain). For other injury types (gunshot wounds), it is more appropriate to describe the pathways of the injuries. When there is more than one specific injury of a particular type (such as gunshot wounds), it is appropriate for the pathologist to number the wounds for descriptive purposes. It is important to note that such numbering does not necessarily correspond to the sequence of injury occurrence. Many pathologists attempt to describe different injury types within different sections within the evidence of injury section. For example, a specific case may have gunshot wounds, sharp force injuries, and blunt force injuries. It is appropriate to have subsections for each injury type within the evidence of injury portion of the autopsy report. Sometimes within a certain injury type, further separation is provided based on body region. For example, with blunt force injuries, there may be separate sections for head and neck injuries, trunk injuries, and extremity injuries.

An additional section of the autopsy report should provide for listing the various ancillary procedures included in the investigation, including, for example, toxicology and histology. If specific evidence is collected in a given case, it is

appropriate to list the evidence, as well as its disposition (to whom it was given or how it was stored).

Some pathologists choose to include a summary of the major autopsy findings, either as a front page, or as a concluding page. Finally, pathologists should include opinion statements regarding the cause of death and manner of death.

External Examination Only (Without Autopsy)

In certain instances, the postmortem examination is limited to an external examination only. These cases are referred to by different names, depending on the office, and include “externals,” “sign-outs,” and “inspections.” The criteria used to determine which cases do not require an autopsy vary greatly from one jurisdiction or office to the next. Sometimes, the criteria are based on issues such as available workforce (how many pathologists are available to do the work) or budgetary constraints. Even if budget and workforce issues are not a part of the decision-making process, there remains a considerable amount of variability regarding which cases require an autopsy. Table 7.2 represents a list of case types that, in the view of the author, should always have an autopsy performed. Beyond those cases listed in the table, there can be reasonable debate as to whether or not an autopsy should be performed. A spectrum exists within death investigation offices, regarding whether or not an autopsy should be performed. At one end of the spectrum are those who tend to choose to perform an autopsy. In these places, the vast majority of suicides and accidents are autopsied (unless perhaps the person had been hospitalized for a significant amount of time prior to death, such that all injuries are adequately documented), and a significant percentage of sudden natural deaths may also be

Table 7.2 Cases that require an autopsy

All homicides and suspected homicides
Cases known or suspected to have been caused by other criminal violence
In-custody deaths and any other death associated with police action
Hit-and-run pedestrian deaths
Accidental deaths where criminal charges are expected
Suicidal deaths where investigation suggests that autopsy is necessary
Acute workplace-related deaths
Certain deaths alleged to involve medical therapy
Certain “diagnoses of exclusion,” including drug-related deaths
Deaths caused by apparent electrocution
Un-witnessed suspected drowning deaths
Infant deaths that are unexpected and unexplained
Toddler/small child deaths that are unexpected and unexplained
Charred bodies
Skeletonized bodies
Any suspicious death
Unidentified bodies in order to assist/aid in identification
Deaths that require autopsy for cause-of-death determination
Cases that require autopsy for evidence collection

autopsied. At the other end of the spectrum, an office may choose to limit autopsies to the minimum, and thus follow criteria similar to that shown in Table 7.2. Parenthetically, it should be noted that there are some offices or individuals that do not even follow these minimum criteria, such that cases that arguably should be autopsied are not.

Scene and circumstance investigation coupled with a thorough external examination can frequently substantially rule out foul play in many presumed sudden, unexpected natural deaths. Offices also vary greatly in their criteria for selecting which of these cases require an autopsy. Often, the major deciding factor is the age of the decedent. For example, if there is a sudden, unexpected, presumed natural death in a 36-year-old who has no known previous medical history, most offices would choose to perform an autopsy on this decedent. If the circumstances are identical but the decedent is 82 years old, many offices would not perform the autopsy, but would instead perform an external examination, perhaps perform routine toxicology testing (or not), and sign the death certificate with the cause of death related to atherosclerotic cardiovascular disease (heart disease) or presumed heart disease, since statistically this is the most likely underlying cause. The bottom line in this situation is that the scene investigation and external examination (with or without toxicology) have substantially ruled out non-natural causes of death, so the death is presumed to be natural. Different offices have different criteria for which cases within this category require autopsies. When only an external examination is performed, a formal report, similar to an autopsy report, should be created and maintained as a business record.

After-the-Fact and In-Absentia Cases

Another situation that remains far too common involves deaths that should have been referred to the death investigation agency (coroner or medical examiner) but, for some reason, were not until well after the decedent's remains are no longer readily available for examination. The usual circumstance involves an elderly individual who falls and fractures a hip and subsequently dies. A well-intentioned physician signs the death certificate, and lists the hip fracture from a fall as contributory to death, along with various underlying natural disease processes, but the physician fails to remember that if the hip fracture contributes to death, then the death is no longer natural and it must be considered accidental. When this error is discovered in the vital statistics bureau, the case is retroactively referred to the coroner or medical examiner for appropriate death certification. In many instances such as this, the body is no longer available for examination. As such, the death investigation agency should obtain medical records, and interview family members and health care employees, in order to determine if, in fact, the injury contributed to death. The reader is referred to Chapter 5 for further discussion regarding such a determination. These types of cases can be referred to as "after-the-fact" cases, since they are brought to the attention of the death investigation agency much later than they should have been. Properly educating physicians, nurses, other health care workers,

nursing home staffs, funeral home employees, and the general public can help to reduce the number of cases that fall into this category.

Another type of case that requires a coroner or medical examiner to certify a death without actually examining a body involves cases that, for one reason or another, require official certification by the death investigation agency but, for various reasons, do not require transportation to the morgue for examination. These case types can be called “in-absentia” cases, and they may or may not be permissible in certain jurisdictions. In some places, a physician might not be able to sign a death certificate for someone who has obviously died a natural death. Alternatively, a great distance may separate the person who must legally sign the death certificate in a case that does not require an autopsy, but still falls under the jurisdiction of the office. In all such cases, the certifying individual must place a great deal of trust in those individuals that are able to perform an external examination and those who provide the pertinent medical information concerning the case. For both after-the-fact and in-absentia cases, it is important to document the investigation and produce an official report.

Autopsy Assistants

It is unusual for a forensic pathologist to perform a forensic autopsy without any assistance. When assistance is provided by other individuals, it usually is provided by autopsy assistants, also known as “dieners.” Occasionally, assistance is provided by “pathology assistants” (PAs), but PAs are most often used by hospital-based (non-forensic) pathologists performing hospital (medical) autopsies.

The assistance provided to a forensic pathologist during an autopsy varies considerably from one office to another. This variability may be based on office policy, experience of the assistant, or preference of the pathologist. In some scenarios, assistants are responsible for preparing the body for autopsy and cleaning up after the autopsy, with the pathologist performing all or a majority of the evisceration. In some places, pathologists allow the assistant to open the bowel (if necessary) and remove the skull cap, but pathologists perform the remainder of the evisceration themselves, including brain removal. Other pathologists personally remove the heart and brain, and possibly some of the other organs, while leaving the rest of the evisceration to the assistant. Some other pathologists allow the assistant to perform the entire evisceration under their direct supervision. This typically occurs in larger offices, where dieners are very experienced, fast, and efficient. No matter which scenario, since the autopsy is considered the practice of medicine, most agree that it is absolutely essential for the pathologist to be physically present within the autopsy room to perform the external examination, to at least witness the evisceration, and to perform the dissection of organs. It should be noted that many hospital-based pathologists (and occasional forensic pathologists) disagree with this contention.

Office Accreditation and Forensic Autopsy Standards

For many years, the National Association of Medical Examiners (NAME) has operated an inspection and accreditation process by which death investigation offices may undergo an inspection process in order to become officially accredited. The inspection checklist is a relatively lengthy list of items of importance that, as a whole, have been identified as necessary in order for an office to operate at the highest possible level of quality. The process is specifically geared toward facilities and organizational structure and activities, rather than toward autopsy performance. Specific areas addressed within the inspection and accreditation checklist include: facilities, safety, personnel, notification, acceptance of, and declining of cases, investigations, body handling, postmortem examinations, identification, evidence and specimen collection, support services, reports and records, mass disaster plan, and performance improvement. Accreditation is time-limited, such that offices must be re-inspected after several years if they wish to remain accredited. Information regarding the NAME Inspection and Accreditation process can be obtained at the NAME website (www.thename.org).

Another NAME document specifically deals with forensic autopsy performance standards. The standards can be considered the minimum standards necessary when performing a forensic autopsy. The standards include the following sections: medicolegal death investigation, forensic autopsies, identification, general and specific procedures related to external examinations, internal examinations, ancillary tests and support services, and documentation and reports. This document is also available at the NAME website.

Disc Image Legends

- Disc Image 7.1 An example of a body diagram used to document injuries at autopsy.
- Disc Image 7.2 An evidence hair within the hair of a homicide victim. The collection of such trace evidence may be extremely important.
- Disc Image 7.3 Collection of vitreous fluid from the eye can provide important toxicology and chemistry information related to the cause of death.
- Disc Image 7.4 In young individuals, such as this infant, the anterior chest plate can be removed by cutting through the cartilage with a scalpel.
- Disc Image 7.5 After the anterior chest plate is removed and the pleural cavities and lungs are examined in situ (in place), the pericardial sac can be opened anteriorly.
- Disc Image 7.6 Blood samples can be collected directly from the heart.
- Disc Image 7.7 Bile can be collected from the gallbladder.
- Disc Image 7.8 Urine can be collected from the urinary bladder.
- Disc Image 7.9 After small intestine removal, the large intestine is removed.
- Disc Image 7.10 After the liver and a bulk of the intestines have been removed, the spleen (white arrowhead), esophagus (black arrowhead), stomach (arrow), duodenum, and pancreas (located behind the stomach – dashed outline) remain

- within the body. These organs may be removed in a variety of ways. Often, the spleen is removed separately, followed by removal of the other organs together.
- Disc Image 7.11 The duodenum, the pancreas, the stomach, and a portion of the esophagus may be removed in one large mass.
- Disc Image 7.12 The adrenal glands, located above and medial to the kidneys, are removed. The left adrenal gland is being removed in this photo.
- Disc Image 7.13 In order to remove a kidney, it is reflected forward, and a scalpel is used to cut through the fat surrounding the organ.
- Disc Image 7.14 After the kidney is exposed as shown in Disc Image 7.13, the capsule and surrounding fat is peeled away from the organ.
- Disc Image 7.15 The pelvic organs (urinary bladder, prostate in males [shown], uterus, fallopian tubes and ovaries in females) are then removed.
- Disc Image 7.16 The aorta, along with the bottom portion of the trachea, with attached mainstem bronchi, is removed.
- Disc Image 7.17 For most organs removed, the weight of the organ is recorded. This photo shows the heart within a scale.
- Disc Image 7.18 In this infant case, the organ bloc is freed up posteriorly, first on the right, then on the left, then from below, and finally from above (as shown).
- Disc Image 7.19 A stock container with tissue/organ samples contained within formalin, as viewed from above.
- Disc Image 7.20 Small sections of tissues can be placed into tissue cassettes, so that microscopic sections can be prepared.
- Disc Image 7.21 Following serially sectioning of the coronary arteries, the heart is sectioned, beginning from the apex (the lowermost tip) and proceeding toward the base (the part where all of the blood vessels connect to the heart).
- Disc Image 7.22 Sectioning of the adrenal gland.
- Disc Image 7.23 Sectioning of the pancreas.
- Disc Image 7.24 After the scalp has been reflected forward, the decedent's face will be temporarily covered by the "inside-out" scalp.
- Disc Image 7.25 Depending on the case or office protocol, the temporalis muscles on the side of the skull can be trimmed away from the underlying skull, in order to better visualize the bone.
- Disc Image 7.26 After skull cap and dura removal, the brain is exposed and ready to be removed from the cranial cavity.
- Disc Image 7.27 In order to remove the brain, it must be gently pulled back, in order to cut the portion of the dura which separates the cerebrum from the underlying cerebellum (the "tentorium"). This is followed by cutting of the optic nerves, the pituitary stalk, the carotid arteries, the vertebral arteries, and the upper cervical spinal cord.
- Disc Image 7.28 After the brain has been removed, it is important to remove the adherent dura from the inner aspect of the skull.
- Disc Image 7.29 Face peel-down examination.
- Disc Image 7.30 Face peel-down examination showing injury of and about the left eye.
- Disc Image 7.31 Middle/inner ear removal via the basilar skull.

- Disc Image 7.32 An anterior neck dissection in progress, at a level deeper than Fig. 7.33 Arrowheads: stenothyroid muscles.
- Disc Image 7.33 Anterior neck dissection, one level deeper than Disc Image 7.32, with the thyroid gland evident overlying the larynx. Arrowheads: reflected sternohyoid muscles.
- Disc Image 7.34 One step deeper than Disc Image 7.33 in an anterior neck dissection, revealing thyroid gland (arrowheads).
- Disc Image 7.35 The next step of a layer-by-layer anterior neck dissection Arrowhead: thyroid cartilage: Arrow: trachea.
- Disc Image 7.36 A limited posterior neck dissection.
- Disc Image 7.37 Dissection of the lower extremity in order to identify deep venous thrombosis in the setting of pulmonary thromboemboli.

Selected References

- Adams VI. Autopsy technique for neck examination. I. Anterior and lateral compartments and tongue. *Pathol Annual* 1990;25(pt 2):331–49.
- Adams VI. Autopsy technique for neck examination. II. Vertebral column and posterior compartment. *Pathol Annual* 1991;26(pt 1):211–26.
- Forensic Autopsy Performance Standards. National Association of Medical Examiners. www.thename.org.
- Inspection and Accreditation Policy and Procedure Manual, Accreditation Forms, and Checklist. National Association of Medical Examiners. www.thename.org.
- Ludwig J. *Handbook of Autopsy Practice*, 3rd ed. Totowa, NJ: Humana Press; 2002.
- Peterson GF, Clark SC. Forensic autopsy performance standards. *Am J Forensic Med Pathol* 2006;27:200–55.
- Prahlow JA. Chapter 3 – Hospital Versus Medicolegal (Forensic) Autopsies. In: Prahlow JA, editor. *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:5–14.
- Prahlow JA. Chapter 6 – Medicolegal Autopsies: An Overview. In: Prahlow JA, editor. *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:33–36.
- Sheaff MT, Hopster DJ. *Post Mortem Technique Handbook*, 2nd ed. New York, NY: Springer; 2004.

Chapter 8

Postmortem Changes and Time of Death

Jesus, once more deeply moved, came to the tomb. It was a cave with a stone laid across the entrance. 'Take away the stone,' he said. 'But Lord,' said Martha, 'by this time there is a bad odor, for he has been there four days.'

John 11:38–39

Abstract This chapter provides detailed discussion of postmortem changes, including the classic signs of death, livor mortis and rigor mortis, as well as other early postmortem changes. The chapter also discusses issues related to estimating the time of death (postmortem interval), changes encountered in the decomposition process, and postmortem injuries.

Keywords Postmortem change · Decomposition · Time of death · Postmortem interval

Introduction

The term “postmortem changes” refers to various changes that occur in a dead body after death, and although strictly speaking, the entire process represents a continuum, it can be divided into “early postmortem changes” and decomposition, both of which follow a somewhat predictable pattern. Because there are so many variables that can influence the rate and form of these changes, it is not possible to reliably estimate an accurate “postmortem interval” (time of death) based on postmortem changes alone. This chapter will first discuss early postmortem changes, followed by a description of decomposition, a brief discussion of postmortem injuries, and finally, a discussion of estimating the postmortem interval (time of death).

Early Postmortem Changes

Livor Mortis

Early postmortem changes include the classic “cardinal signs of death” (livor mortis, rigor mortis, and algor mortis), as well as several other findings that can

occur shortly after death but prior to the onset of what is usually considered decomposition. The first to be described is livor mortis, or lividity. Another name for this is “dependent hypostasis.” This finding represents the characteristic settling of blood, based on gravity, which occurs following death. During life, blood is constantly circulating within the blood vessels; however, when death occurs, this circulation stops, and the blood settles and becomes evident in the skin of the dependent parts of the body, that is, the parts that are closest to the ground. An exception is that lividity is not evident where the body is in hard contact with the floor, ground, or other object, since the pressure on these areas prevents blood from entering the skin. These areas are said to demonstrate “blanching.” Lividity typically has a dark red-purple color (Fig. 8.1), and it is usually evident within a couple of hours after death; however, it is more difficult to recognize in dark skin.



Fig. 8.1 Lividity, or livor mortis, represents the postmortem settling of blood within the dependent skin, due to gravitational forces. Note that areas of skin exposed to pressure do not develop lividity

Early on, livor mortis is easily blanchable, or unfixated, meaning that if you apply pressure to it, the blood is actually forced out of the skin in this area, leaving an area that is blanched (Disc Images 8.1 and 8.2). Usually, after removing the pressure, the blanched area “refills” with blood. After several hours (12 or so, depending on the reference and the circumstances), the livor mortis becomes “fixed,” such that blanching is not easily produced when pressure is applied. Death investigators should note the location and character of livor mortis (does it blanch or is it fixed?) when performing an initial body examination. The blanched areas that are surrounded by fixed lividity may indicate various objects that were underneath the decedent (Disc Images 8.3, 8.4, and 8.5). If lividity has fixed, and a body is subsequently moved and placed in a different position, the lividity pattern can indicate that the body has been moved after death (Fig. 8.2). It is important to note that fixation of lividity does not occur all at once. In other words, a body can have fixed lividity consistent with one position/location, be moved to another position/location, and develop fixed lividity consistent with the second body



Fig. 8.2 After several hours, lividity becomes “fixed,” such that movement of a body from one position to another may become evident because the lividity pattern is inappropriate for the current body position

position/location as well. In extremely dependent portions of the skin and other tissues, the blood that produces lividity can actually produce pinpoint, and slightly larger areas of intense, dark, subcutaneous hemorrhage known as Tardieu spots (Fig. 8.3 and Disc Image 8.6).

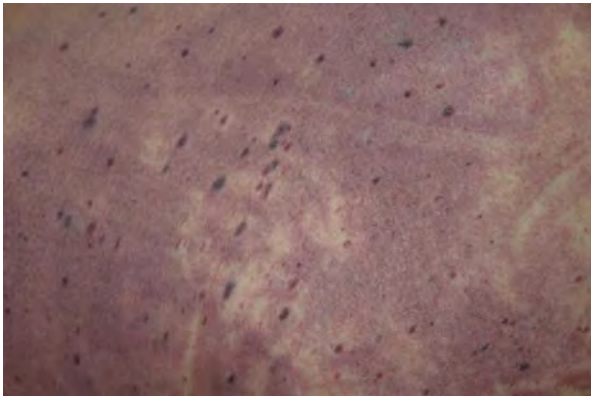


Fig. 8.3 Lividity occurs in dependent portions of the skin, where dark, pinpoint Tardieu spots may form

The character and/or color of livor mortis can sometimes aid in suggesting a particular cause of death. Persons dying of heart disease sometimes demonstrate marked lividity of the upper chest, neck and face (Disc Image 8.7). Carbon monoxide, cyanide, certain other toxins, and extreme cold can cause bright red discoloration of the lividity. Cold can have this effect even after death. Hydrogen sulfide is an extremely toxic substance that can impart a green discoloration of livor mortis and internal organs. It should also be noted that livor

mortis can take on different shades of red/purple/pink, depending on local environmental conditions (Disc Image 8.8). For example, a body that is found at a scene of death and has well-developed, fixed lividity on the posterior aspect of the body may develop some color change in certain parts of the lividity pattern after being stored in a cooler overnight.

Rigor Mortis

The second early postmortem change to be discussed is rigor mortis, or rigidity. Rigor mortis refers to postmortem stiffening of muscles. This change is a transient change, meaning that it develops and then it disappears. In life, muscle cells produce adenosine triphosphate (ATP), the molecule utilized by cells as fuel. Part of the function of ATP in skeletal muscles is that it allows the actin and myosin fibers to freely glide next to one another. Recall from Chapter 6 that the sliding action of the actin and myosin fibers within the skeletal muscle cytoplasm allows muscles to contract. When ATP is depleted in muscles, as occurs after death (since dead cells can no longer produce ATP), the actin and myosin fibers bind to one another causing the entire muscle to become stiff. The binding is different from contraction. In other words, muscles stiffen in whatever position they happen to be in at the time of death; there is no contraction that occurs after death. Even the muscles that control the hair on our skin undergo rigor mortis, and as such, certain bodies will appear to have “goosebumps” (cutis anserinus) (Disc Image 8.9). As the skeletal muscle cells continue to break down after death (early onset of decomposition), the bonds between the actin and myosin fibers break, and rigor mortis disappears.

The typical timeline is that rigor mortis begins to be noticeable within a couple of hours after death, reaching a maximum around 6–12 hours, give or take several hours, and then it slowly dissipates over the next day or so. Some attempt to grade the rigor mortis from 0 (none) to full or peak (4). It is important to note that the onset, peak, and dissipation of rigor mortis is temperature dependent. Extremely cold environmental temperatures can slow the process incredibly, such that if a body is frozen solid shortly after death, and then thawed at a later date, the body can demonstrate rigor mortis after/during the thawing process. Another point regarding rigor mortis is that the rigidity that is present can be “broken.” For example, it is not uncommon at autopsy to have a body that is in full rigor (4) or near full rigor (3). In order to remove the clothing from the body and proceed with the autopsy, the rigor present in the arms must be broken. This is accomplished by applying pressure in an attempt to move the elbow and shoulder joints. When enough pressure is applied, the rigor mortis will “break,” and the joint will be freely moveable. Once rigor is broken, it will not return.

Another caveat regarding the time of onset of rigor mortis involves situations where the decedent is extremely physically active immediately prior to death, such that his/her muscles may be lacking ATP stores. In such situations, the onset of rigor mortis can be nearly instantaneous and can sometimes be referred to as “cadaveric spasm.” The classic situation in which it occurs is drowning deaths, where a victim

can be pulled from the water within minutes of the event and still be markedly rigorized. It is not uncommon for drowning victims to have marine vegetation clenched in fully rigorized fists when their bodies are recovered (please see Chapter 16).

One final point regarding rigor mortis deserves mention. If rigor is fully developed and a body is moved to a different position, and the body is subsequently discovered, prior to the full dissipation of rigor mortis, then the “inappropriate” rigor mortis can aid investigators in determining that the body has been moved (Figs. 8.4 and 8.5). As with livor mortis, death investigators should note the presence or absence and extent of rigor mortis during initial body examination.

Fig. 8.4 If a body is in full rigor mortis, the presence of “inappropriate” rigor mortis may indicate that the body has been moved. The woman obviously did not die in this body position



Fig. 8.5 This photograph shows the original body position of the woman depicted in Fig. 8.4. The death investigator rolled her body onto her back in order to illustrate the fact that she was in full rigor mortis

Algor Mortis

The final of the three cardinal signs of death is referred to as “algor mortis.” It specifically refers to the postmortem cooling of the body that normally takes place after death, where the body temperature equilibrates with its environmental temperature. In many instances, this involves cooling, reducing the body temperature from the normal living body temperature of 98.6°F (37°C) to the cooler environmental temperature, but in certain instances (when the environmental temperature actually exceeds normal body temperature), the dead body will actually warm up, rather than cool down, after death. The rate at which body temperature change occurs following death depends on a number of variables, including environmental temperature, body temperature, the amount and type of clothing, whether or not the clothing or skin is wet, etc. There are formulas which can be employed to attempt to determine the postmortem interval based on body temperature (see below), but these are considered by many to be so unreliable as to be virtually useless.

Other Early Postmortem Changes

A variety of other changes occur within the body during the early postmortem period. The corneas (overlying the iris and pupil) of the eyes may begin to become somewhat clouded (Disc Image 8.10). The most severe corneal clouding occurs in certain fire deaths, where the corneas are virtually opaque. The sclerae (white part) of the eyes, if exposed to air because the eyes are not totally closed, will develop a dark discoloration within the part that is exposed to air. The official term for this change is “tache noire,” which means “black line” (Fig. 8.6). Other epithelial surfaces of the body can undergo a similar change, with drying and dark discoloration. The most common locations for this to occur are the lips (Disc Image 8.11), the tongue, and the scrotum, and such changes can sometimes be mistaken as injuries. Finally, it is well known that the skin of a dead body can actually darken when



Fig. 8.6 The dark discoloration of the sclera (the white part of the eye) is a common early postmortem change, known as *tache noire*

exposed to sunlight (Disc Image 8.12), this is sometimes referred to as a “post-mortem suntan.” The exact reason for the change is not known for certain, although it cannot be related to any active process requiring living cells; it probably involves ultraviolet rays acting on pre-existing pigment within the skin.

Decomposition

As mentioned previously, the early postmortem changes and the decomposition process actually represent a continuum. Decomposition specifically refers to the breakdown of the body which occurs following death. Strictly speaking, therefore, the early postmortem changes represent a part of the decomposition process, but we tend to reserve the term “decomposition” to the setting where there are additional visible changes. A corresponding feature is a definite odor that represents a hallmark of decomposition. At room temperature, decompositional changes usually become evident after about 24 hours, although variation occurs from case to case. Decomposition is classically described as having two components: autolysis and putrefaction. Autolysis refers to the situation where a body’s own enzymes are acting on itself, causing tissue and cellular destruction (“auto” means “self;” “lysis” means “breakdown”). Putrefaction refers to the situation where micro-organisms (especially bacteria and fungi) feed on and break down the tissues of a dead body. There are numerous micro-organisms that normally reside within the human body, particularly within the GI tract, and when death occurs, the normal immune defense systems are no longer functional, so these micro-organisms can grow unchecked. In a very short time period, micro-organisms can breakdown and digest a large amount of soft tissue, producing large amounts of gas and “decomposition fluid.” Although many experts within the field of forensic pathology describe the decomposition process as involving just two parts, namely autolysis and putrefaction, it is arguably more accurate to include a third part to the process, namely environmental factors. There are several environmental factors that play some very important roles in the decomposition process. The local environmental temperature is perhaps the most important; heat greatly increases the decomposition process, while cold impedes it. This is precisely the reason that dead bodies are stored in coolers. Animal activity, be it in the form of scavengers, such as rodents, predators, such as larger carnivores, or insects, such as cockroaches, ants, and flies, is another environmental factor that can significantly contribute to the decomposition process. The term “anthropophagy” specifically refers to animal feeding on a dead body. Another environmental factor that plays a role in decomposition is the physical location of the body. Bodies buried in soil typically decompose at a slower rate compared to those left open to air. Bodies in non-heated water also decompose more slowly, but usually at a faster rate than those buried in soil. An old “rule” that some forensic pathologists utilize is the following: 1 week in air = 2 weeks in water = 8 weeks in soil. As with many other issues related to the postmortem changes, it is important to recognize that wide variation exists. Different types of soil, different types of water, and even different types of air (humid versus dry, for example), can change the rate of decomposition.

Some of the earliest noticeable signs of decomposition are continuations of the changes already described with early postmortem changes. Typically, the lividity is fully fixed, rigor mortis has peaked and dissipated such that the muscles are flaccid, and the body temperature has equilibrated with the local environmental temperature. The corneas may become increasingly clouded, and various surfaces (lips, tongue, scrotum) can become dry and darkened. If the local environment is very dry, early drying/mummification can affect the nose, ears, fingers, and toes.

As decomposition proceeds, and micro-organisms continue to “feed” on the corpse, gaseous and fluid decomposition products are produced and become evident within various parts of the body. The skin begins to darken to various shades of green and brown. This is usually first seen within the right lower abdominal quadrant, where the underlying cecum is relatively close to the skin surface (Fig. 8.7). Superficial skin blood vessels can become discolored with decompositional changes. This change is sometimes referred to as “marbling,” since the appearance mimics the pattern of marble (Fig. 8.8). The superficial layers of skin can become very loose, such that blisters develop (Fig. 8.9) or they can slip off the underlying layers (Fig. 8.10), referred to as “skin slippage.” As these processes are occurring, the entire body tends to become somewhat bloated (because of the decompositional gas production), and decomposition fluid frequently is expelled, or purged, from the mouth, nose and other orifices. The presence of this red-brown fluid is referred to as “purge fluid” (Fig. 8.11) and is often mistaken as blood by those uninitiated in observing decomposed bodies. If the process continues unabated, all of the features described continue in their scourge of the corpse such that, when fully developed into a state of severe decomposition, the body will be extremely bloated (having the appearance of someone who is morbidly obese), the skin will be dark brown/green and greasy with skin slippage and blister formation, and there will be abundant purge fluid (Figs. 8.12 and 8.13 and Disc Images 8.13 and 8.14). At this stage, the eyes are frequently bulging out of their sockets, the lips are swollen

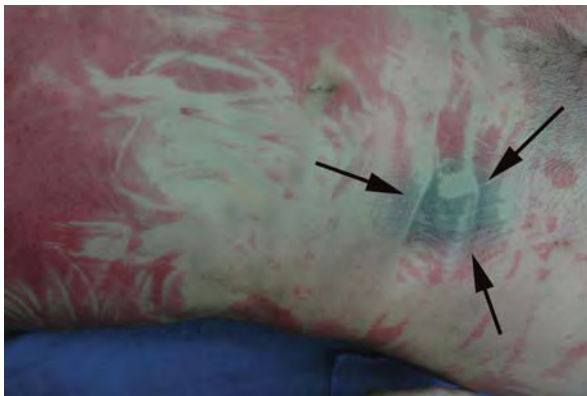


Fig. 8.7 One of the earliest signs of decomposition is green discoloration of the lower right abdominal quadrant, seen toward the right side of the photo



Fig. 8.8 As decomposition proceeds, “marbling” of the skin may become apparent

Fig. 8.9 Skin blistering is another common change early in the decomposition process



and bulging, and the tongue is often protruding from the mouth (Disc Image 8.15). Similar bulging changes can affect the anogenital area, including the scrotum.

The description in the preceding paragraph specifically deals with a dead body undergoing decomposition in a relatively “normal” environment, such that it is not extremely moist (humid) or extremely dry (arid). Depending on the presence or absence of insects and other animals, the decomposition process can continue with eventual loss of soft tissues due to micro-organism activity. The process can occur relatively quickly, especially in warm environments (Fig. 8.14), and it often occurs more quickly at sites of injury. Decomposition can be retarded, especially by cold



Fig. 8.10 Skin slippage can also occur relatively early in decomposition

Fig. 8.11 Purge fluid frequently is expelled from the mouth and nose



temperatures or embalming; however, items as simple as tight clothing may slow the process considerably compared to adjacent areas of the body (Disc Images 8.16 and 8.17). If insects or insects and other animals are present, the decomposition process is greatly accelerated. Small and large animals will actually feed on the body, and can “deflesh” a body very quickly. Insects such as cockroaches (Disc Image 8.18) and fireants (Disc Image 8.19), as well as small mammals, such as mice (Disc Image 8.20) and rats (Disc Image 8.21), frequently feed on dead bodies. This animal feeding activity, which is officially known as “anthropophagy,” is not limited to wild animals. In fact, domestic pets, left with a dead owner and no other source of food

Fig. 8.12 As decomposition continues, the body begins to bloat, and the skin becomes darkened. Note the dark discoloration of the lips



Fig. 8.13 Advanced decomposition is characterized by an extremely bloated, greasy, darkened body. This is a scene photograph of a decomposed, bloated Caucasian individual who was not very obese during life

will frequently deflesh a portion of their “best friend,” even before decomposition sets in (Fig. 8.15 and Disc Image 8.22). This defleshing often begins on the face, frequently near the mouth. More commonly, flies, particularly blowflies, will lay eggs on bodies within minutes of death (Fig. 8.16 and Disc Image 8.23), the eggs then grow into maggots, which feed on the soft tissues (Fig. 8.17 and Disc Image 8.24). Maggots frequently congregate near body orifices and at sites of injury. Some bodies become so covered with maggots, that the body can appear to be moving because of the motion of the enormous mass of maggots covering the body (Disc Image 8.25). After flies and maggots have substantially removed a significant amount of the soft tissues of a decomposing body (Disc Image 8.26), other insects, namely dermestid



Fig. 8.14 Eventually, after the initial ravages of bloating decomposition have passed, the body continues to lose more soft tissue, and the process of skeletonization begins



Fig. 8.15 Domestic pets, such as dogs, will sometimes feed on the dead body, with large portions of soft tissue removed

beetles, will arrive on the scene to continue the decomposition process. Through a combination of autolysis, putrefaction, and environmental/animal/insect factors, a dead body will ultimately be stripped of most or all of its soft tissues and be left only with bones. After careful evaluation of the partially-skeletonized body for injuries and other soft tissue findings, it is often wise to remove the remaining soft tissue so that a more thorough anthropologic examination can be performed (Fig. 8.18). If larger animals have removed larger portions of the body during the entire process, then many bones may be absent. Skeletonization can occur at a surprisingly rapid rate (Disc Image 8.27).

In extremely moist, warm environments, a different process may take precedence as the body moves into a more advanced state of decomposition. In warm, moist environments, the fats of the body can actually undergo saponification (the



Fig. 8.16 If flies are present in the local environment, they will lay eggs on the body, often in or near body orifices. This aggregate of fly eggs has an appearance that is somewhat reminiscent of grated Parmesan cheese. Also note the multiple brown spots on the skin surface. These are produced by flies and can sometimes be mistaken for blood spatter



Fig. 8.17 Fly eggs grow up into maggots, which feed on the corpse

chemical process of soap formation). This is known as “adipocere” formation and is characterized by an extremely rancid odor, and the presence of white and yellow, cheesy-appearing aggregates of saponified fats (Fig. 8.19).

In contrast, a body that is decomposing in an extremely warm but dry environment can undergo mummification, where the soft tissues become desiccated. Characteristically, the skin takes on a dry, leathery appearance (Fig. 8.20 and Disc Image 8.28). As mentioned above, early focal mummification is not uncommon involving the nose, ears, and fingers. As an aside, the old myth that suggests that fingernails continue to grow after death probably resulted from people noticing the appearance of lengthening fingernails when the skin of the fingertips became mummified and shrunk away from the nail (Disc Image 8.29).

Fig. 8.18 Autolysis, putrefaction, and animal (including insect) activity all contribute to the ultimate loss of soft tissues



Fig. 8.19 In warm, wet environments, a process known as “adipocere” formation may occur on decomposing bodies



A few other issues related to decomposition deserve mention. The first is post-mortem mold formation on the skin (Disc Image 8.30). A variety of colors are known to occur. Usually, the changes occur in damp environments, after a relatively



Fig. 8.20 In warm, dry environments, a process known as “mummification” may occur

lengthy postmortem period. Mold growth is also relatively common on bodies that are embalmed and buried for many months to years; it is evident if the body is exhumed and examined.

Internally, decomposition results in some predictable changes. Decomposition fluid accumulates within the pleural cavities. Fatty decomposition fluid may accumulate in the peritoneal cavity. The organs become markedly softened and may even “liquefy.” In some cases, gas “bubbles” actually form within the decomposing organs; the classic example of this involves the brain. If a decomposed brain such as this is fixed in formalin prior to sectioning, sections of the brain will demonstrate a “swiss cheese” appearance. The intimal surface of the aorta (and other large arteries) changes from normal yellow to dark red-brown as decomposition ensues. This is thought to be related to staining produced by red blood cell pigments after the blood cells break apart. An interesting change that sometimes affects the surface of the liver, stomach and other abdominal organs is a lace-like precipitation of minerals that is referred to as “miliaria” (Disc Image 8.31). On rare occasions, the stomach may undergo postmortem rupture secondary to marked autolysis (Disc Image 8.32), a process referred to as “gastromalacia.”

It should be noted that the decomposition process can result in a variety of changes that may be misinterpreted as true injuries. Several examples will be presented in Chapter 21 under the section entitled “artifacts and mimics.”

Postmortem Injuries

A postmortem injury is a traumatic injury to a body that occurs after death, and generally lacks a “vital tissue reaction” appearance, meaning that it does not look like it bled during life. In many instances, this is indicated by a yellow color (Fig. 8.21), rather than a red (bloody) color. Some pathologists like to add the term



Fig. 8.21 A classic postmortem injury, such as this abrasion, lacks a vital tissue reaction and appears yellow

“perimortem” to their description of apparent postmortem wounds, referring to the time immediately around the time of death, including some time immediately preceding death. Certain wounds that are inflicted during this perimortem time frame may be indistinguishable from true postmortem wounds because the injured tissue may not have been receiving much blood supply as the victim’s body was in the last stages of attempting to avoid death, with shunting of blood to the brain and other vital structures. For this reason, it is appropriate to describe postmortem wounds as “postmortem/perimortem.”

The presence of a large number of inflicted postmortem injuries (such as innumerable postmortem stab wounds) (Disc Image 8.33) is sometimes referred to as “overkill.” Their occurrence, or the presence of postmortem mutilation injuries (such as parts of the body being cut off), suggests the possibility of a murder being committed by someone well known to the victim (a so-called “crime of passion”) (Disc Image 8.34). Alternatively, their presence might indicate that the murder might represent a “hate crime” or a “bias crime,” which can be defined as a crime motivated by hatred, bias, or prejudice, based on the actual or perceived race, color, ethnicity, national origin, religion, sexual orientation, gender, gender identity, or disability of another individual or group.

One final consideration involves the fact that most readily identifiable postmortem injuries occur on non-dependent portions of the body. If a postmortem injury occurs on a dependent portion of the body, where lividity occurs, then the wound may mimic a true, antemortem wound. The occurrence of “postmortem” bruises can be explained by this phenomenon. Even after death, if a crushing injury occurs in soft tissue where lividity will occur, as lividity develops, a “bruise” can become apparent.

Time of Death Estimation

It is very difficult to accurately estimate the postmortem interval (PMI), or “time since death”. Various controlled studies have indicated that a fairly accurate estimation may be possible in certain instances if enough data regarding environmental conditions is known; however, in real world situations, reliable data is difficult, if not impossible, to obtain. Environmental conditions (temperature, humidity, etc.), size of the decedent, and the presence or absence of clothing are some of many factors that can affect the rate of decomposition. When a forensic pathologist is asked to provide an estimate, it is wise for him/her to use caution. If a definitive estimate is provided, it is best to give a wide range, since there are so many variables that can affect the onset of the postmortem changes used to make the estimate. It is also very important to evaluate the circumstances, scene and other findings (cell phone records, etc.), and incorporate such information in the estimate.

When asked to estimate the PMI, a forensic pathologist must first be provided with two very important pieces of information. The first, which, for illustrative purposes will be designated as time A, is when the decedent was last known to be alive. The second (time B) is when the decedent was found dead. When dealing with time of death estimates, the only statement that a pathologist can make with 100% certainty in most instances is that the decedent died sometime between time A and time B. Based on various other factors, including rigor mortis, livor mortis, and decompositional changes, a pathologist may be able to estimate whether or not death most likely occurred closer to time A or time B. In room temperature environments, early decompositional changes, including early skin discoloration and slippage, slight bloating, and slight purge fluid are typically evident within the first couple of days. Moderate decompositional changes tend to occur over the next couple of days, and advanced changes, characterized by marked bloating, total skin discoloration with slippage, etc. follow over the next couple of days. As can be deduced by the lack of specificity of the above statements, the timing of the onset of decomposition is so variable that it is not wise to be very specific at all when estimating time of death based on the extent of decomposition. In particularly cool environments, decomposition can be impeded greatly. Conversely, in very warm environments, especially if the body is outdoors, decomposition can be extremely rapid. Near-complete skeletonization has been known to occur in less than a week, as is indicated in Disc Image 8.14.

During life, normal cell mechanisms keep a majority of potassium molecules confined inside of cells, and sodium and chloride molecules confined to the extracellular fluid. After death, these mechanisms stop, and potassium escapes from the cells, while sodium and chloride enter the cells, until equilibrium exists. As decomposition occurs, the vitreous potassium levels increase, while the vitreous sodium and chloride decrease. A more thorough explanation of the so-called vitreous (eye fluid) electrolyte patterns is provided in Chapter 21. Some investigators have attempted to utilize the vitreous potassium concentration to estimate the postmortem interval. Two equations that use the potassium concentration are: PMI (in

hours) = $7.14 \times [K^+] - 39.1 \pm 20$ hours *or* PMI (in hours) = $5.26 \times [K^+] - 30.9 \pm 20$ hours. Unfortunately, as you can see from the error factor, the results are not very helpful in providing a specific time frame.

Postmortem changes in body temperature were briefly discussed above under the heading of algor mortis. A very rough estimate is attempted by some individuals using the following rules: body temperature decreases 1.5 to 2.0°F per hour for the first 12 hours and then proceeds at a rate of 1.0°F per hour. Some investigators have created formulas for estimating the postmortem interval based on a body's core temperature. One such formula is: PMI (in hours) = $(98.6 - \text{rectal temp.})/1.5 \pm 20$ hours. In a similar fashion to the vitreous potassium formula, it should be evident that these formulas do not provide very specific answers regarding the PMI. In fact, with an error factor of several hours, there are very few circumstances where calculating an estimated PMI based on body temperature would provide any useful information and for this reason, it is unusual that core body temperatures are taken on dead bodies. Occasionally, the police or others may request the test. If it is determined that a core body temperature PMI estimate is desired, the core temperature should be taken as close to the time that the body is discovered as possible. The most accurate core temperature requires that the temperature be taken of an internal organ. The liver is the organ that is usually chosen. A small incision must be made in the abdominal skin and in the liver, so that the thermometer can be inserted several inches into the liver. As this procedure causes some defects to the body, it is a good idea to attempt this procedure only on cases where it is absolutely considered necessary to obtain a core body temperature. Many offices refuse to perform this testing because of its lack of specificity and the fact that it requires cutting the body prior to autopsy.

Some forensic pathology references suggest that the presence or absence of gastric (stomach) contents, and the character of such contents can aid in estimating the PMI. However, the gastric emptying time varies greatly from individual to individual, and even within a single individual, such that its usefulness is highly questionable. Add to this normal variability the fact that emotional or physical stressors can theoretically slow gastric emptying considerably, and it should be clear that using this method to estimate the PMI is full of major potential errors. As such, most forensic pathologists will not even consider using this factor in estimating the time of death. Gastric content analysis may, however, be helpful in identifying the last meal that someone ingested prior to death, a fact that may or may not be important in a given death investigation.

The final topic addressed in this section of estimating the PMI is actually the one that is most scientifically sound and reproducible, involving the field of forensic entomology. Forensic entomologists are experts in evaluating insects as they relate to various forensic issues. Specifically with regard to estimating the PMI in a given death, forensic entomologists are able to evaluate the insects (and larva forms of insects) present on bodies to make a scientifically-based estimate of the time of death. The most common insect type used for this purpose are blowflies, of which there are numerous species (Fig. 8.22). As long as flies are able to land on a body, they will begin laying eggs on the corpse within minutes of death; however, many fly



Fig. 8.22 An adult fly on a decomposing body

species will only lay eggs if there is light present (daylight, indoor light). Usually, freshly laid fly eggs can be seen on a corpse within a day or so. In their smallest form, they mimic grated Parmesan cheese, as seen in Fig. 8.16. Within another day or so, the eggs “grow up” into maggots, as seen in Fig. 8.17 and as the maggots feed on the body, they grow in size. Ultimately, when the maggots reach a certain level of maturity, they will actually leave the body and pupate (they form pupa, similar to a caterpillar forming a cocoon), usually around days 6–10. After an additional several days, adult flies emerge from the pupa, and start the cycle over again. Disc Image 8.35 shows an adult fly, a pupa case, and a maggot. Each species of blowfly has a specific known life-cycle pattern. In order to estimate a time of death, entomologists must first know which species of fly is present and then they must have a representative sampling of the most mature stage of fly development associated with the corpse. In order to determine the species of fly, the adult form is easiest to evaluate. For this reason, a common method for collecting appropriate entomology samples for subsequent forensic entomology examination follows.

Pathologists (or others) should collect two samples of several of the largest maggots present on the corpse. If multiple maggot types (colors, appearances, etc.) are present, care should be taken to collect all types. At least a dozen maggots should be collected in each sample. One sample should be heat-killed by placing the maggots in a test tube filled with water and heating the tube over a Bunsen burner. The maggots will turn white and float to the top when dead. Store the maggots in a capped test tube with a 70% isopropyl alcohol solution. Any pupa cases found on or near the body should also be collected. The other sample of live maggots should be placed in an enclosed container with pin prick air holes in the lid (a clean cottage cheese-type container works well), with a food source (a piece of liver from an autopsy placed on a small square of aluminum foil), a water source (a soaked piece of paper towel), and a substance that will allow the maggots to retreat from the food source in order to pupate (vermiculite works well). The so-called “maggot motel” should be

placed on a laboratory counter for several days to weeks, and checked periodically. When it is obvious that adult flies are flying around within the container, the container should be placed in a freezer/refrigerator, to slow the flies' metabolism. Once the flies have slowed down sufficiently, they can be quickly removed and placed in a second capped test tube containing 70% isopropyl alcohol. The two test tubes (one containing samples of heat-killed maggots, the other containing adult flies) can then be sent to a forensic entomologist for evaluation. (It should be noted that collection of other insects, such as dermestid beetles, may also be beneficial.) The entomologist will determine the species of fly based on evaluation of the adult flies and will then determine the stage of development (how many days it takes for an egg to mature into the largest maggot present) for that particular species of fly, based on the largest maggots collected from the body. The entomologist will also frequently take into consideration ambient temperature variability during the time that the flies were active on the corpse. This information is easily obtained from the national weather service. Other environmental variables may also be factored into the equation. Finally, the entomologist will provide a scientifically-determined estimate regarding the PMI.

Disc Image Legends

- Disc Image 8.1 Initially lividity blanches when pressure is applied. In this photo, pressure is being applied with a fingertip.
- Disc Image 8.2 Blanching of lividity, such as seen in this photo, which corresponds to Disc Image 8.1, indicates that the lividity has not yet become "fixed."
- Disc Image 8.3 This lividity pattern developed in a decedent who died while lying on an egg-crate mattress pad.
- Disc Image 8.4 This man died face down, lying on top of his hand. Note in the next image (Disc Image 8.5) how the blanched area corresponds to his hand.
- Disc Image 8.5 An area of blanching corresponding to the decedent's hand (see Disc Image 8.4).
- Disc Image 8.6 An area of intense lividity with numerous Tardieu spots.
- Disc Image 8.7 If the lividity pattern is intense in the face, neck and upper torso, the pattern suggests a possible cardiac cause of death.
- Disc Image 8.8 A variety of factors may result in different appearances of the color of lividity. In this case, note the different colors of the lividity on the upper back. These differences are likely the result of subtle environmental changes after death, such as lying on an EMS backboard in the emergency department for several hours, and then being transferred to the morgue cooler.
- Disc Image 8.9 Rigor mortis affects even the muscles that attach to small skin hairs, giving the appearance of "goosebumps" (cutis anserinus).
- Disc Image 8.10 Slight corneal clouding noted in a decomposing body.
- Disc Image 8.11 Dark discoloration and drying of the lips is common following death.

- Disc Image 8.12 Sun exposure following death can actually lead to dark discoloration of the exposed skin surface, a so-called “postmortem suntan” or “sunburn.”
- Disc Image 8.13 An example of a body demonstrating relatively advanced decomposition, with bloating, skin discoloration, and abundant purge fluid.
- Disc Image 8.14 Another example of fairly advanced decomposition. In this case, marbling, skin discoloration, bloating, and skin slippage are prominent on the trunk region.
- Disc Image 8.15 Bulging of the eyes, lips and face, with protrusion of the tongue, all related to decomposition.
- Disc Image 8.16 An example of how tight clothing (a sock) has retarded the decomposition of the skin.
- Disc Image 8.17 Another example of how clothing can retard the decomposition process. Note that the skin of the torso, which was covered with clothing, is reasonably intact compared to the advanced decompositional changes noted on the face and neck.
- Disc Image 8.18 Cockroach bite marks. Note the absence of a “vital tissue reaction” (there is no bleeding).
- Disc Image 8.19 Fireant bite marks, with no evidence of a vital tissue reaction.
- Disc Image 8.20 Bite marks produced by the feeding activity of mice.
- Disc Image 8.21 Relatively extensive skin damage produced by postmortem rat activity. Note the dry, yellow appearance of the defects, indicating that they are postmortem in nature.
- Disc Image 8.22 Another example of domestic dog feeding activity involving the face.
- Disc Image 8.23 Another example of fly eggs on a dead body. In this example, there is virtually no appreciable decomposition; however, the decedent is the victim of multiple gunshot wounds and has quite a bit of blood on his face and embedded in his moustache. This was recognized by flies as a suitable place in which to lay eggs.
- Disc Image 8.24 Another example of maggots on a dead body.
- Disc Image 8.25 Extensive maggot activity on the face of a decomposing man.
- Disc Image 8.26 The typical appearance of a decomposing body after extensive blowfly activity.
- Disc Image 8.27 Partial skeletonization that occurred outdoors in the summertime after about 10 days.
- Disc Image 8.28 Another example of mummification.
- Disc Image 8.29 Finger mummification, wherein the soft, fleshy part of the finger has shrunk away from the overlying fingernail.
- Disc Image 8.30 Postmortem mold growth.
- Disc Image 8.31 Miliaria on a decomposing liver.
- Disc Image 8.32 Postmortem stomach rupture (gastromalacia).
- Disc Image 8.33 An example of “overkill,” with numerous postmortem stab wounds. Note that many of the wounds have a yellow color.

Disc Image 8.34 Postmortem mutilation of a corpse. (Photo courtesy of Dallas County Medical Examiners Office and Dr. J McClain).

Disc Image 8.35 An adult fly, a pupa case, and a maggot.

Selected References

- Byrd JH, Castner JL eds. *Forensic Entomology – The Utility of Arthropods in Legal Investigations*. Boca Raton, FL: CRC Press; 2001.
- Dix J, Graham M. *Time of Death, Decomposition and Identification – An Atlas*. Boca Raton, FL: CRC Press; 2000.
- Galloway A, Birkby WH, Jonew AM, Henry TE, Parks BO. Decay rates of human remains in an arid environment. *J Forensic Sci* 1989;34:607–16.
- Haglund WD, Sorg MH, eds. *Forensic Taphonomy – The Postmortem Fate of Human Remains*. Boca Raton, FL: CRC Press; 1997.
- Henssge C, Knight B, Drompecher T, Madea B, Nokes L. *The Estimation of the Time Since Death in the Early Postmortem Period*. London, England: Edward Arnold; 1995.
- Hewadikaram KA, Goff ML. Effect of carcass size on rate of decomposition and arthropod succession patterns. *Am J Forensic Med Pathol* 1991; 12:235–40.
- Komar DA. Decay rates in a cold climate region: a review of cases involving advanced decomposition from the medical examiner's office in Edmonton, Alberta. *J Forensic Sci* 1998;43:57–61.
- Mann RW, Bass WM, Meadows L. Time since death and decomposition of the human body: variables and observations in case and experimental field studies. *J Forensic Sci* 1990;35:103–11.
- Mellon PF, Lowry MA, Micozzi MS. Experimental observations on adipocere formation. *J Forensic Sci* 1993;28:91–3.
- Prahlow JA, Linch CA. A baby, a virus, and a rat. *Am J Forensic Med Pathol* 2000;21:127–33.
- Schoenly K, Griest K, Rhine S. An experimental field protocol for investigating the postmortem interval using multidisciplinary indicators. *J Forensic Sci* 1991;36:1395–415.
- Vass AA, Barshick SA, Sega G, Caton J, Skeen JT, Love JC, Synstelién JA. Decomposition chemistry of human remains: a new methodology for determining the postmortem interval. *J Forensic Sci* 2002;47:2002.

Chapter 9

Identification of Human Remains

Many casualties, piles of dead, bodies without number, people stumbling over the corpses . . .

Nahum 3:3

Abstract Chapter 9 presents the very important topic of identification. Introductory comments discuss the importance of establishing and following specific identification protocols when dealing with dead bodies. Non-scientific methods of identification (visual identification and hospital identification) are presented, as are scientific methods (fingerprint, dental, X-ray, DNA). The chapter concludes by discussing circumstantial identification and unidentified remains.

Keywords Identification · Fingerprints · Dental · X-ray · DNA · Unidentified

Introduction

One of the most important duties of coroners, medical examiners, and forensic pathologists involves the positive identification of dead bodies. In most circumstances, this process does not present much of a problem; however, a misidentification can produce dire consequences. Not only does this duty have far-reaching consequences from a societal and legal standpoint, but the impact on decedents' families and friends is substantial. It is best to have well-established policies and procedures in place in order to avoid an identification mishap. The most likely mishap involves the inadvertent misidentification of two bodies, where each is misidentified as the other. Mistakes such as these are most likely to occur when there are multiple deaths in a single incident, particularly when the gender and relative ages and sizes of individuals are the same. Misidentifications are probably more common in offices that do not have established protocols for identification and labeling of bodies (assigning unique identification numbers to bodies). Finally, mistakes are more likely to happen in a busy office, where the sheer volume of bodies entering and exiting the facility increases the chances of a mistake.

In a vast majority of deaths that are investigated by medicolegal death investigation agencies, the initial investigation will produce a “preliminary” or “tentative”

identification. Circumstantial evidence, such as a driver's license found in the decedent's possession, an automobile registration found in the car in which the person died, or knowledge of the owner of the residence in which the decedent was found dead, can be instrumental in pointing investigators toward a tentative identification. In some situations, this circumstantial information appears to be fairly "solid;" however, the death investigation agency must not simply accept this as positive identification. Steps must be taken to ensure a definitive "positive" identification is made.

A definitive positive identification can be established by a variety of means. Each office should establish guidelines/criteria for what is acceptable as a positive identification. Perhaps the most common method for positive identification involves visual identification by family members (or occasionally good friends); however, it is not considered scientific, and mistakes have been known to occur. Despite this fact, it still remains perhaps the most widely used method for ensuring positive identification within the death investigation system. Fortunately, a majority of decedents that enter the medicolegal death investigation system are suitable for visual identification.

The most reliable methods of identification are those that are considered "scientific," wherein a known standard of the individual in question can be compared to the previously-identified decedent, such that a "match" is made, to the exclusion of all others. For any case, scientific identification is the preferred method of positive identification. For certain case types, visual identification is not possible. Such case types include those that are badly burned, decomposed, or severely injured (Figs. 9.1, 9.2, 9.3 and Disc Images 9.1–9.3). Examples of scientific methods of identification include fingerprint comparison, dental comparison, X-ray comparison, and DNA typing. In all of these except for DNA typing, a known antemortem (prior to death) record/standard must exist in order for a scientific comparison to be made.



Fig. 9.1 Bodies recovered from fires may be "burnt beyond recognition"

Fig. 9.2 Badly decomposed bodies are typically not visually identifiable



Fig. 9.3 Depending on the injury type, severity, and location, persons who are victims of severe trauma may not be visually identifiable. This is a victim of a motor vehicle collision

Ultimately, if a reliable visual or scientific method of identification cannot be made, then other features may be useful in making a positive identification. For example, the location and appearance of various birthmarks, scars, implanted medical devices, or tattoos can provide sufficient information to be considered unique to a given individual. Finally, the circumstances of the death can provide some useful information in this regard as well. This would be considered the least certain method of positive identification, but sometimes, this is all that is available. For example, if a badly decomposed elderly male is found in a locked residence that is known to

be the residence of a particular elderly man, and no scientific identification is possible after a thorough search, and police investigation rules out foul play, it may be reasonable to consider the individual positively identified via circumstance.

Policies for Identification of Bodies

Each office should have established guidelines for assigning a unique identification number to each body. Frequently, this is assigned as the body enters the morgue. When multiple fatality incidents occur, it is often necessary to perform some degree of identification prior to arriving at the morgue. In all cases, investigators should attempt to determine the preliminary identification of the decedents, and then work to establish positive identification. As mentioned above, this frequently involves visual identification by relatives (see below for detailed description), but other procedures may take place as well. Many offices perform a number of basic procedures in order to aid in the identification of a body. These procedures also function as a permanent record of a particular body. Common procedures include an “identification photograph” (showing the face of the decedent), a blood sample (permanent record of DNA), and fingerprint cards. If a body is unidentified, additional procedures should occur (see “unidentified bodies”).

Common, Non-scientific Methods of Identification

Hospital Identification

In many cases that come to the coroner/medical examiner, the decedent is pronounced dead in a hospital. The length of the hospital stay varies from virtually nil (those pronounced “dead on arrival” (DOA)) to several months. Each hospital is responsible for ensuring the positive identification of its patients, and as such, has policies and procedures in place for patient identification (Fig. 9.4). In most instances, hospitals rely on self-identification (the patient provides the hospital with their name, etc.) or on visual identification by relatives or friends. As explained in the next paragraph, visual identification carries with it certain risks for error. While a hospital identification is better than no identification, it should not be considered absolutely definitive. The degree of certainty should be ascertained on a case-by-case basis.

Visual Identification

Visual identification usually depends on a relative of the decedent recognizing the decedent. Occasionally, office policy will allow someone other than a relative to make the identification. Depending on the office, a visual identification may occur

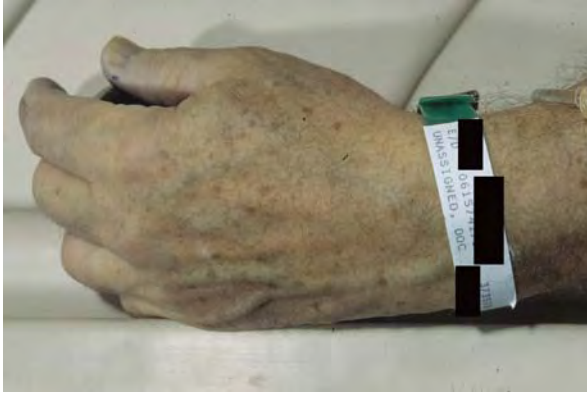


Fig. 9.4 Hospital identification bands are often considered verification of positive identity

“in person” (where the relative actually views the body while in the same room as the body), it may occur through a window from an adjacent room, remotely via video, or via still photography (Disc Image 9.4). A common procedure involves having the person making the positive identification sign a document that specifically states that they have made the identification; this document is signed by a witness and remains with the case file.

Relying on visual identification can be problematic in certain situations. If there are multiple fatalities involved in a single incident (or even if there is only one fatality, but another or several other individuals are injured severely, such that they are hospitalized and non-communicative), then extreme care must be used when assigning identity. This is particularly true if the ages and genders of the injured/dead are similar. In other cases, if there is facial trauma, this may make visual identification difficult or impossible. If there is minor facial trauma, or even no facial trauma at all, a relative may still misidentify the body as their loved one, perhaps because of the emotional nature of the event. Therefore, it is wise to remember that mistakes can be made when relying solely on visual identification.

An occasional issue that arises when visual, and sometimes other forms of identification are used, is the issue of aliases. People use aliases for a variety of reasons. The practice is particularly common amongst foreign immigrants, especially those who are in the United States illegally. When such individuals are visually identified by family members or friends, it is not infrequent to discover at a later time (usually when arrangements are being made to transport the body back to the country of origin) that the identification originally provided actually represents an alias. The death investigation agency is usually notified, because a death certificate with the correct name is necessary in order for the body to be transported out of the USA. Contacting the appropriate consulate office can be very helpful when investigating the death of an immigrant, legal or otherwise.

One final issue deserves mention when considering visual identification. It may be very tempting for investigators or pathologists to attempt to make a visual

identification themselves, by comparing the photo on a driver’s license to the decedent. This should not be considered positive identification, but only preliminary or tentative identification.

Scientific Methods of Identification

Fingerprint Identification

One of the most reliable and useful scientific identification methods is fingerprint comparison. In some offices, fingerprint cards are made (if possible) for every body coming to the morgue (Fig. 9.5 and Disc Images 9.5 and 9.6). No two fingerprints have ever been shown to be identical, and as such, each individual’s 10 fingerprints are unique, to the exclusion of all other persons. In order for a decedent to be positively identified using fingerprint comparison, two things must occur. First, a set of the individual’s fingerprints must be contained within a searchable database. The databases that are most frequently used for this purpose are police criminal databases, but other databases also exist (military, etc.). Second, the dead body must be in a condition that will allow for fingerprints to be made for comparison

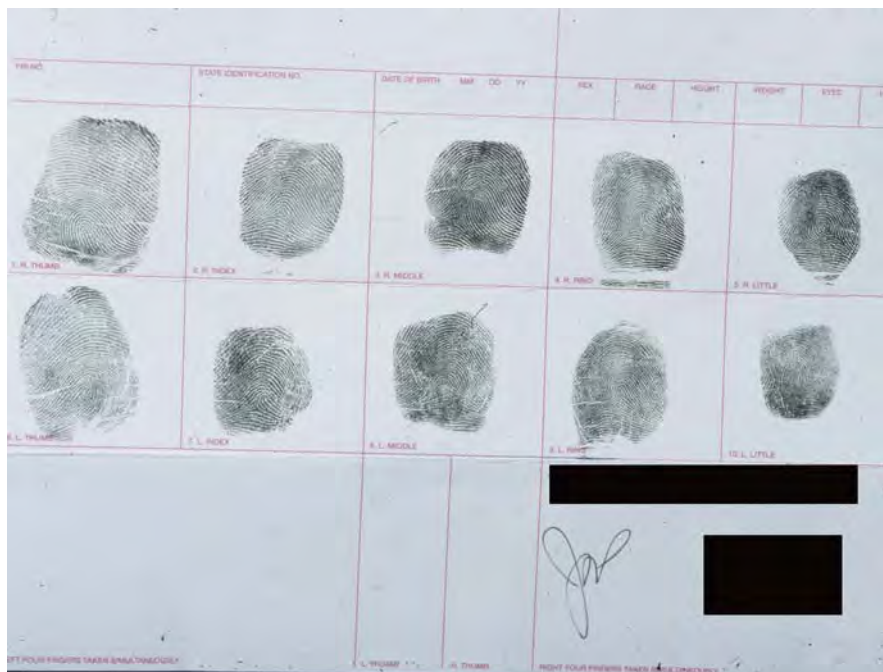


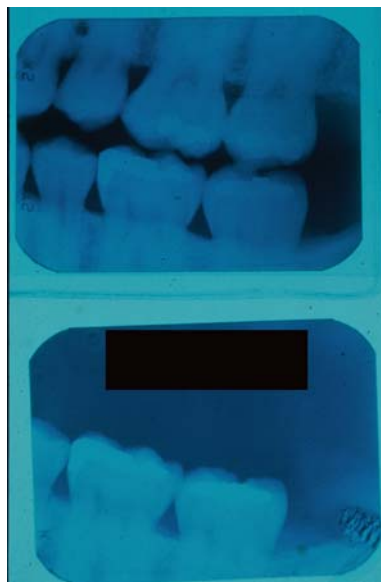
Fig. 9.5 Many offices routinely take fingerprints from all bodies. This is an example of a 10-print fingerprint card

purposes. Severe injuries, including burns, as well as severe decomposition (either severe softening or mummification) and other environmental postmortem changes (severe wrinkling from water immersion) can make postmortem fingerprint production difficult, if not impossible. Depending on the case, various techniques can be performed to try to obtain decent quality prints (Disc Images 9.7, 9.8, 9.9, and 9.10).

Dental Identification

Another very common method for identifying dead bodies is dental comparison. This method is particularly useful for decedents who are not visually identifiable and are not able to be identified by fingerprint comparison, such as occurs with severe decomposition and severely burnt bodies. Like fingerprint comparison, dental identification requires a pre-existing dental record. This is referred to as the “antemortem” (before death) record. The usual situation is that the unconfirmed identification of the decedent is known, so the person’s dentist is contacted in order to receive copies of the dental records. The records usually include the dental “chart,” which includes a description and diagram of the person’s teeth, as well as any dental X-rays. By comparing the decedent’s teeth and postmortem X-rays to the dental chart and antemortem X-rays, a positive identification can usually be made (Fig. 9.6). This process requires detailed charting of the decedent’s teeth (Disc Image 9.11), along with photography in select cases. Specific features, such as the absence of certain teeth, and the unique shapes and locations of various dental restorations (fillings, bridges, caps, root canals) are usually sufficient to make a

Fig. 9.6 Above: An antemortem (before death) dental X-ray, received from the decedent’s dentist for comparison purposes. Below: A postmortem dental X-ray performed at autopsy. Comparison of this X-ray to the antemortem X-ray allowed positive identification to be made in this case. Note the unique shapes of the fillings, which appear as radio-opaque (white) areas within the teeth



positive identification. X-rays are not absolutely essential for each dental identification, but they usually are very helpful. In many cases, the forensic pathologist is able to perform the comparison and subsequent identification (or exclusion); however, forensic odontologists can also be consulted to perform the identification, particularly when a very detailed comparison is required. This tends to occur when there are no restorations (fillings, bridges, etc.), and the odontologist must rely on detailed examination of tooth root structure, etc. to make a positive identification. When relying on dental comparison in bodies that are not suitable for an open casket funeral, it is helpful to cut away the soft tissues of the face so that adequate exposure can be attained in order to chart, photograph and X-ray the decedent's teeth (Disc Images 9.12 and 9.13). Occasionally, the mandible (jawbone) and maxilla (upper jaw), complete with teeth, are removed in order to better evaluate the teeth (Disc Image 9.14).

Radiologic Identification

Just as dental X-rays can assist in making positive identifications, a variety of other antemortem X-rays can be useful in making scientific positive identifications (Fig. 9.7). The typical situation is similar to that described with dental identification. If a tentative or possible identity has been assigned to a decedent, then death investigators can contact family members in an attempt to locate any antemortem X-rays. X-rays of many different parts of the body may prove to be valuable in making a positive identification. Particularly useful are antemortem neck or chest X-rays,



Fig. 9.7 An X-ray being performed on a badly burned body

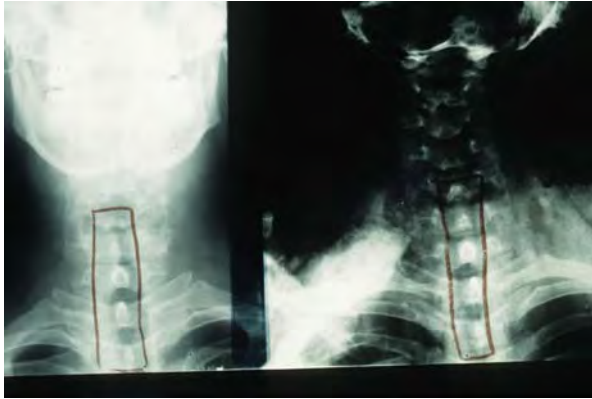


Fig. 9.8 *Left:* A close-up view of an antemortem (before death) neck X-ray, received from a hospital for comparison purposes. *Right:* A close-up view of a postmortem neck X-ray corresponding to the antemortem film. Note that the unique shapes of the spinous processes of the vertebral column in each X-ray are identical, allowing for positive identification

where the unique shapes of the spinous processes (the parts of the spinal column bones that protrude towards the skin of the back) can be matched with postmortem X-rays (Fig. 9.8 and Disc Images 9.15 and 9.16). Other useful X-rays include the head (showing the unique shapes of the frontal sinuses) (Fig. 9.9) and any that show orthopedic (bone) “hardware” (implanted devices, such as screws and plates and artificial joints) (Fig. 9.10). As explained below, if certain implanted medical devices

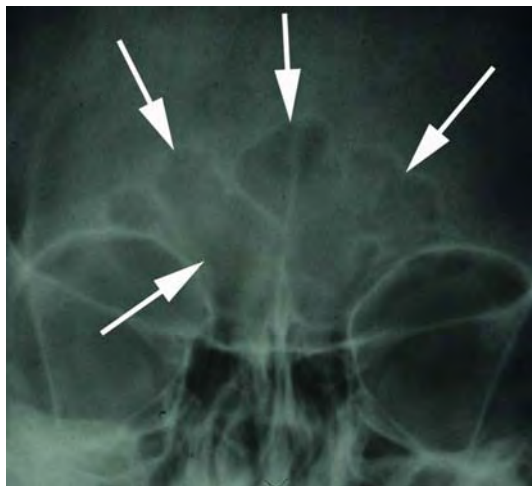


Fig. 9.9 A skull X-ray, showing the unique shape of the frontal sinuses, which represent radiolucent (*darker*) air pockets within the skull of the forehead (*arrows*)



Fig. 9.10 An X-ray showing orthopedic hardware that was useful in positively identifying a decedent

are identified on X-ray, retrieval of the devices during autopsy may allow for positive identification using unique serial numbers present on the devices.

DNA Identification

The most recent scientific method for identification involves DNA testing. The reader is referred to Chapter 2 for a brief overview of DNA testing. As with the other scientific methods used for identification, DNA testing can occur when a known antemortem DNA sample exists and can be obtained for testing. Items such as toothbrushes and hairbrushes can be useful in providing antemortem samples; however, using such items can introduce a degree of uncertainty as to whether or not the samples truly represent those of the decedent. Another option is to obtain paraffin-embedded tissue or other tissue from hospital-based pathology laboratories if the person had previously had any type of procedure that resulted in a pathology report (biopsy, surgical procedure with removal of tissue). An additional method of comparison that can be made using DNA testing is to compare the decedent's DNA to that obtained from various living relatives (parents, siblings, offspring). Every office should collect and retain a "blood spot card" or similar sample from every body coming to the morgue, in order to have a permanent sample of DNA for each decedent (Fig. 9.11). Some offices also collect pulled head hair strands (Disc Image 9.17). This is particularly useful if the decedent received multiple blood transfusions prior to death, as the blood samples in such cases will represent a mixture of the decedent's blood and donor blood.

Fig. 9.11 A blood spot card should be collected in every possible case as a permanent record of a decedent's DNA



Identification Based on Other Unique Features

Other unique features of a decedent can provide useful information, including some that are scientifically indisputable. The first of these has been mentioned previously: implanted medical devices. With some devices, such as certain types of orthopedic hardware, like an artificial hip, as well as various implanted cardiac (pacemakers, automatic defibrillators) or medication delivery devices, unique serial numbers can be traced back to the individual (Figs. 9.12 and 9.13 and Disc Image 9.18). With other implanted medical devices such as internal surgical clips and coronary artery stents, unique characterization is not necessarily possible, but their presence can certainly provide useful identifying features.

Everyone has various external (and internal) features that, in combination, make them unique, to the exclusion of all other persons. The key is identifying enough of these features to exclude the possibility that the decedent is actually someone else. Some features, although not actually part of the body, such as clothing and jewelry, can be fairly unique and as such can be useful in identification; however, basing a definitive identification solely on such items is not recommended (Fig. 9.14). Some body features, such as gender and height, are relatively basic, but are also relatively

Fig. 9.12 A postmortem hip X-ray in a badly decomposed woman shows the presence of an artificial hip



Fig. 9.13 A photograph of the artificial hip removed at autopsy. All such implanted devices have unique serial numbers that can be traced, through the manufacturer, to the individual



unchangeable (without surgical intervention). Other features that are fairly common and relatively easy to identify at autopsy include circumcision, various body piercings (Disc Image 9.19), absence of the appendix and/or gallbladder, and fallopian tube ligation. Some additional internal “devices” may also provide useful information toward ensuring positive identification. Examples include dentures (Disc Image

Fig. 9.14 Jewelry, particularly if it is unique, or personalized (inscribed with the individual's name), can be quite valuable in making a positive identification; however, basing a positive identification solely on such jewelry is not considered scientific



9.20), which can be inscribed with the person's name, and intrauterine devices (IUDs) (Disc Image 9.21). Other body features are also rather basic, but are more easily changed, such as hair color, facial hair, and body weight. Certain external features of a person can be considered unique, particularly if there is a combination of multiple features such as birthmarks, scars, amputations (Disc Image 9.22), and tattoos (Fig. 9.15 and Disc Image 9.23). In some cases, a combination of multiple, sufficiently unique, external and internal features allows a positive identification to



Fig. 9.15 A tattoo may represent one of several features of a body that allows for positive identification

be rendered. In decomposed remains, the visualization of tattoos can be improved by applying hydrogen peroxide to the skin surface.

Circumstantial Identification

In some cases, none of the above methods of identification produces a positive identification. The usual situation is a badly decomposed body found in a secure residence, where no scene findings or autopsy findings suggest foul play. The death is presumably due to natural causes, and the suitability of samples for and the expense of DNA typing is such that this method of identification is not deemed necessary or possible. Visual identification is not possible, no antemortem fingerprint, dental records, or other X-rays exist, and there are insufficient unique body features to make a definite positive identification. Nothing suggests that the body is anyone other than the person who resides at that residence. In such a situation, the identification can be deemed “positive based on circumstance.” Depending on the availability and affordability of DNA testing in the future, and the resources of the particular death investigation agency, it may be possible and practical to perform DNA testing in such cases, so long as adequate DNA samples can be obtained. It should be noted, however, that there will be similar cases where no adequate antemortem DNA sample or living relatives are available, so a circumstantial identification will still be warranted. If a death represents a homicide, every effort, including DNA testing, should be made to ensure a positive identity.

Unidentified Remains

In a small subset of cases examined by coroners/medical examiners, a positive identification cannot be made. This situation is more common in urban areas, particularly large metropolitan areas, as large cities tend to attract people who, for whatever reason, become or choose to become anonymous. For this reason, some of these unidentified persons actually *would* be visually recognizable, but for the fact that no one comes forward to identify them. Many others are badly decomposed or skeletonized. In rural areas, bodies that remain unidentified tend to be the badly decomposed or skeletonized remains. When there are absolutely no clues as to the identity of a body, forensic pathologists will usually perform all necessary procedures so that, should a possible identity be suggested, all available information regarding the decedent will then be readily available in a file. As such, office protocol should state that all unidentified bodies will have all of the following procedures performed (if possible): total body photographs, fingerprints, total body X-rays, dental charting and X-rays, DNA sample collection (blood and hair, bone, teeth). In select cases, forensic anthropologic assessment can provide useful estimates as to age, race, gender, and stature. Occasionally, a forensic artist can attempt to create a clay facial reconstruction over an unidentified skull, with the resultant reconstruction photographed and shown to the public, in the hopes that someone will recognize

Fig. 9.16 Occasionally, skeletonized remains are examined at a medicolegal death investigation office, but there is absolutely no indication of the identity of the decedent, either by body examination or scene/circumstance. After forensic anthropologic examination to characterize the race/ethnicity, gender, and approximate age of the individual, the forensic pathologist may choose to send the skull to a forensic artist, who then attempts to reconstruct the facial features of the decedent (see Fig. 9.17)



Fig. 9.17 A forensic artist's facial reconstruction over the skull shown in Fig. 9.16. A photo such as this can be published, with the hope that someone might recognize the person. If someone believes that the decedent might be a particular person, then scientific methods for identity can be attempted



the person (Figs. 9.16 and 9.17). Once all of the above data has been collected, the unidentified body will be stored (or buried in a pauper's grave), until such time that positive identification can be made. Subsequent identification in these cases is the exception rather than the rule; however, recently, nationwide efforts are being made to identify these individuals.

In 2007, the NamUS program was initiated by the National Institute for Justice. This federal program, developed in conjunction with the forensic pathology/medical examiner community, is a computerized database that allows death investigation agencies from around the country to enter data about each unidentified decedent. Information includes all of the identification parameters as delineated in this chapter, including photos, dental records, X-rays, unique external and internal features, as well as items related to date of death, etc. For many years, there have been national databases of missing persons but the hope now is that by matching cases within the NamUS and the missing persons databases, some answers and closure will be afforded to families who have not been able to locate their missing loved ones.

Disc Image Legends

Disc Image 9.1 Even if a fire victim is not totally charred, they may be burnt beyond recognition. A scientific method of identification should be sought in such cases.

Disc Image 9.2 For obvious reasons, skeletal remains cannot usually be identified visually.

Disc Image 9.3 Another example of severe trauma resulting in facial distortion such that visual identification is impossible.

Disc Image 9.4 An “identification photograph” may be shown to a family member to positively establish the identity of a decedent.

Disc Image 9.5 Ink being applied to a decedent’s finger at autopsy.

Disc Image 9.6 A fingerprint being taken from a decedent at autopsy.

Disc Image 9.7 In certain decomposition cases, all or a majority of the skin of the hands and fingers may slip off of the body. This image shows a hand without a majority of the skin, which has “slipped off.”

Disc Image 9.8 The “degloved” portion of skin that previously slipped off of the hand shown in Disc Image 9.7.

Disc Image 9.9 Using a gloved hand, the pathologist or assistant can place his/her own finger inside the decedent’s finger skin and produce a fingerprint that can be used for identification purposes.

Disc Image 9.10 A fingerprint being produced via a degloved portion of hand/fingers.

Disc Image 9.11 Besides dental X-rays, dental charts can be used to make positive dental identification. This is an example of a dental diagram created at autopsy, for comparison to antemortem dental records.

Disc Image 9.12 When the body is not viewable for funeral purposes, dental charting and radiography can be made much easier by first cutting away the skin and soft tissues of the face, thus exposing the underlying teeth. In the example shown, a dental X-ray is being performed.

Disc Image 9.13 Another example of a case in which the soft tissues of the face have been removed in order to better visualize the teeth.

Disc Image 9.14 A maxilla (upper jaw) that has been cut away from the rest of the skull in order to better visualize and X-ray the teeth more easily.

- Disc Image 9.15 An antemortem X-ray, showing the unique shapes of the spinous processes from the last cervical (C7) and first 3 thoracic (T1–3) vertebral bodies.
- Disc Image 9.16 A postmortem X-ray showing a similar region as depicted in Disc Image 9.15. Note that the unique shapes of the spinous processes from one X-ray to the next match one another, allowing for a positive identification.
- Disc Image 9.17 Some offices routinely collect pulled hair samples as another permanent record of a decedent's DNA.
- Disc Image 9.18 Implanted medical devices, such as this cardiac defibrillator within the upper left chest, can allow for positive identification to occur at autopsy, via unique serial numbers contained on the device and within the medical records.
- Disc Image 9.19 Piercing sites can be considered further evidence for a positive identification, but like jewelry they should not be the sole criteria utilized.
- Disc Image 9.20 Dentures, such as those shown in the photo, can be useful in ensuring positive identification. Caution should be exercised, since it is not unheard of for elderly individuals, particularly those in extended care facilities and those who are demented, to use other person's dentures.
- Disc Image 9.21 An intrauterine device seen within the uterus at autopsy.
- Disc Image 9.22 The history of a recent toe amputation in this decedent was one of several factors which allowed positive identification to be made.
- Disc Image 9.23 Tattoos can be fairly unique. In this decomposed case, enough of the skin of the back was intact to visualize this tattoo and provide sufficient unique findings to consider the case as positively identified.

Selected References

- Blau S, Hill A, Briggs CA, Cordner SM. Missing persons – missing data: the need to collect antemortem dental records of missing persons. *J Forensic Sci* 2006;51:386–9.
- Fields R, Molina DK. A novel approach for fingerprinting mummified hands. *J Forensic Sci* 2008;53:952–5.
- Haglund WD, Sperry K. The use of hydrogen peroxide to visualize tattoos obscured by decomposition and mummification. *J Forensic Sci* 1993;38:147–50.
- Hanzlick R, Clark S. The unidentified decedent reporting system – a model national website registry for the unidentified deceased. *Am J Forensic Med Pathol* 2008;29:106–13.
- Hanzlick R, Smith GP. Identification of the unidentified deceased: turnaround times, methods, and demographics in Fulton County, Georgia. *Am J Forensic Med Pathol* 2006;27:79–84.
- Murphy WA, Spruill FG, Gantner GE. Radiologic identification of unknown human remains. *J Forensic Sci* 1980;25:727–35.
- Paulozzi LJ, Williams DD, Nolte KB. John and Jane Doe: the epidemiology of unidentified decedents. *J Forensic Sci* 2008;53:922–7.
- Simpson EK, James RA, Eitzen DA, Byard RW. Role of orthopedic implants and bone morphology in the identification of human remains. *J Forensic Sci* 2007;442–8.

Part III
Major Causes/Mechanisms of Death

Chapter 10

Natural Deaths

One man dies in full vigor, completely secure and at ease, his body well nourished, his bones rich with marrow. Another man dies in bitterness of soul, never having enjoyed anything good. Side by side they lie in the dust, and worms cover them both.

Job 21:23–26

Abstract A large percentage of deaths investigated by forensic pathologists result from underlying natural disease processes. Chapter 10 provides a relatively detailed description of the common and many uncommon natural disease processes that are known to result in death, primarily in adults (Chapter 20 includes a discussion of childhood diseases). Sections of the chapter are grouped according to physiologic mechanism or organ system.

Keywords Natural death · Sudden unexpected death · Disease · Natural disease

Introduction

A “natural disease” should be considered an inherent pathologic process resulting from the body’s natural, usually long-term, response to a combination of genetic (internal) and environmental (external) factors. The definition specifically excludes processes that are traumatic (as detailed in many of the other chapters within this text) and those that involve the acute (quick) manifestations of an external factor. Various “lifestyle factors,” such as diet, being sedentary, and chronic substance abuse, are considered factors that are acceptable external influences within the realm of “natural disease.” Likewise, contracting an infectious disease via what would be considered a “natural” or “normal” route of transmission (breathing in influenza virus, being infected with a virus when bitten by a mosquito, developing a community-acquired bacterial pneumonia), and subsequently dying from the infection is typically considered a natural death. In many other cases, the presence of various external factors are what define the case as being something *other than* a natural death. For example, a death resulting from atherosclerotic plaques within the coronary arteries (described below), which, in turn, result from a combination

of genetic factors and poor eating habits with a diet (external factor) rich in high-fat foods, is considered a natural death. In contrast, a death resulting from physical trauma sustained in an automobile collision cannot be considered a natural death. This is true regardless of the timing of the death (which may occur decades after the crash, from long-standing complications). When we consider drugs and toxins, the rationale changes slightly from how cases of physical trauma are treated. A death due to the acute intoxicating effects of a drug or alcohol is not classified as a natural death; however, by convention, if a death results from chronic alcohol or drug abuse, the case *is* considered a natural death.

The human body is made up of cells, tissues, and organs. These structures have a limited number of ways in which to respond to stress, whether the stress is totally internal (genetic), external (environmental), or a combination (“multifactorial”). It is useful to consider some of the ways in which the body responds to stress when describing natural diseases. It is beyond the scope of this text to describe every possible response; however, some of the more common responses will be presented. A very common response involves the process referred to as “inflammation.” The inflammatory process can be thought of as a very complex process by which the body attempts to eliminate the cause of a stress, or at least minimize its effects. The stress itself can include physical trauma of a variety of types, toxins or poisons, infectious organisms, foreign substances, immune reactions, the lack of nutrients, lack of oxygen, genetic mutations, etc. . . . In living people, a variety of signs, symptoms, and laboratory tests can indicate that inflammation exists. At autopsy, pathologists are frequently able to detect areas of inflammation, both by gross (naked eye) examination, as well as microscopically. When the mechanisms that result in inflammation are extremely active, and abundant amounts of the various substances involved in the inflammatory process are circulating throughout the body, a condition known as the “systemic inflammatory response syndrome” (SIRS) can occur. This is particularly common in association with widespread (systemic) infection, but it can also occur with extensive burn injuries, extensive physical trauma, and other conditions. Whatever its cause, SIRS is associated with a significant risk of death.

Some other basic types of responses to stress include “hypertrophy” (increased cell and organ size), “hyperplasia” (increased number of cells), “atrophy” (cell/organ shrinkage), and a process referred to as “metaplasia,” in which the cells lining a surface actually change from one type to another. A variety of stressors can lead to these changes. The term “neoplasia” literally means “new growth,” and the new growths that develop are referred to as “neoplasms,” or tumors. Typically, neoplasia results from numerous mutations occurring within a cell’s genes, such that the cell transforms into something other than what it should be. A benign neoplasm does not invade adjacent tissues or “metastasize” (metastasis is the ability to break away from the primary, or original, neoplasm, travel to a distant site, and begin growing at that new site). In contrast, a “malignant” neoplasm typically has the capacity to invade as well as metastasize.

The term “ischemia” refers to a reduction in blood flow to a particular organ or part of the body. Part of ischemia is a lack of tissue oxygenation, which is referred

to as “hypoxia.” An extreme form of hypoxia is referred to as “anoxia,” where essentially no oxygen is delivered to the tissue. Hypoxia/anoxia can be transient or sometimes relatively permanent. Depending on the tissue affected, the extent of hypoxia, and the duration of the hypoxia, the tissue may actually die while the remainder of the body’s organs/tissues/cells continue to live and function normally. When cells/tissues die in this manner, they are said to have experienced “necrosis,” and the dead area is said to be “necrotic.” The name applied to an area of necrotic tissue caused by hypoxia is “infarct.” Therefore, an “infarction” is a localized area of necrotic (dead) tissue/organ within a living person. It takes many hours for necrosis to be visible grossly as well as microscopically, following the actual physiologic event. As such, if someone dies immediately or shortly after experiencing an infarct, the infarct will not be evident.

One method of categorizing diseases is to describe diseases based on the organ system involved, such as the cardiovascular system or the respiratory system. This is especially useful for diseases that primarily affect one system. Some disorders, however, affect multiple systems. Another categorization scheme involves basic physiologic mechanisms of disease. For example, some diseases primarily involve blood vessel function; some involve infection; some are primarily immune disorders; some are congenital anomalies (birth defects); some are molecular, related to genetic mutations; some involve increased workload or other stressors; some are primarily electrical in nature, such as cardiac conduction system abnormalities and brain seizures; and some are primarily metabolic in nature. The electrical and metabolic disorders can be particularly difficult or impossible to identify at autopsy. In the remainder of this chapter, an attempt will be made to present natural diseases that can lead to death, based on the physiologic mechanism or the organ system involved, followed by several miscellaneous categories that do not fit nicely into a specific organ system. Several disorders that may affect children are presented in this chapter, but some are described in further detail elsewhere. The reader is referred to Chapter 20 (Deaths in Infancy and Childhood) for additional descriptions of natural death in children.

Infectious Disease

As with many of the other topics discussed in this chapter, it is far beyond the scope of this text to provide sufficient details regarding every possible infectious disease which exists. In general terms, infections involve tissue damage and pathologic effects as a result of the growth of a micro-organism on or within the body. As such, when micro-organisms reside on or within the body without causing pathologic changes, it is not considered an infection. These organisms are called “commensal organisms” or “normal flora.” Occasionally, one of these can overgrow and cause an infection, but certain special circumstances are typically required.

There are several different categories of infectious organisms, including bacteria, mycobacteria, fungi, viruses, parasites, and prions (see Central Nervous System section). Oftentimes, bacteria produce an inflammatory process characterized by

the presence of numerous neutrophils (a type of white blood cell). Pus (or “purulence” or “suppurative inflammation”) is sometimes evident grossly. A Gram stain can be used to actually visualize bacteria in microscopic tissue sections. If a bacterial infection is suspected at autopsy, cultures should be collected and sent to the microbiology laboratory. Mycobacteria (tuberculosis) and fungal infections typically result in “granulomatous inflammation,” characterized by “granulomas” composed of lymphocytes and macrophages (two other types of white blood cells). In each type of infection (tubercular and fungal), special cultures can be collected, and visualization of the micro-organisms within microscopic tissue sections is possible using special stains. Viral infections classically cause a lymphocytic inflammatory reaction. Depending on the virus, there may be specific cellular features (“viral inclusions”) identified within infected cells, but actual visualization of the viruses using a light microscope is not possible. Special “immunostains” can help to identify the infection, along with blood “serology” tests and expensive viral cultures. Parasite infections, which often involve some type of arthropod or other animal “vector,” may or may not be readily identified grossly, depending on the organism involved. Microscopic identification, with or without additional serologic testing, is used to make the diagnosis.

Infections of specific organ systems will be presented throughout this chapter. Occasional mention is made of the term “sepsis.” This term should imply to the reader that an infection (usually bacterial) is widespread throughout the body. In other words, it represents a “systemic” infection (meaning throughout the entire body system). The term “septicemia” is similar and implies infection within the bloodstream. Sepsis and septicemia are very serious conditions which frequently lead to death. Sepsis can be considered an infectious cause of the “systemic inflammatory response syndrome,” described above.

By convention, in many deaths due to infectious processes, the manner of death is considered natural, even though the micro-organism is, strictly speaking, an external environmental factor. For example, if a child contracts bacterial meningitis and dies, the death is considered natural. In a similar way, an elderly person who develops a pneumonia and dies is a natural death. This even holds true for many cases where a specific external vector is involved, such as West Nile viral encephalitis transmitted by a mosquito bite or Rocky Mountain spotted fever (caused by a bacteria) transmitted by a tick bite. Exceptions to this MOD rule regarding infectious diseases occur when an infection develops at the site of an injury. For example, if a person sustains a gunshot wound of the abdomen and initially survives, only to die of a bacterial infection of the abdominal cavity, the death is no longer considered natural, since the infection was initiated by the gunshot injury.

Cardiovascular System

Strictly speaking, the cardiovascular system is composed of the heart (cardio-) and the blood vessels (vascular), and therefore this section will deal with diseases of both the heart and the blood vessels known to result in death. The mechanism of death in many heart diseases is related to a lethal arrhythmia. The word “arrhythmia”

refers to an irregular heartbeat and is sometimes referred to as a “dysrhythmia.” Not all arrhythmias are lethal, but some are. It is impossible for pathologists to diagnose an arrhythmia at autopsy (since there is no longer a heartbeat); however, there are a variety of diseases that are known to be associated with an increased risk of arrhythmias and sudden death. It is also important to note that an enlarged heart (cardiomegaly), from whatever reason, is at risk for arrhythmias (Fig. 10.1). Common causes of cardiomegaly include hypertension (high blood pressure), various heart valve disorders, ischemic (insufficient blood) heart disease, various cardiomyopathies (see below), and morbid obesity. Finally, the term “cardiomyopathy” is frequently used in this section: the word means “disease of the heart muscle.”



Fig. 10.1 Cardiomegaly related to a case of morbid obesity, where the size of the heart approximates that of the brain

Congenital Heart Disease

In general, congenital heart disease refers to abnormal development of the heart or the major blood vessels attached to it. Many different forms of congenital heart disease are known to occur. Some are essentially incompatible with extrauterine (outside of the womb) life. Others allow for survival, but require relatively immediate surgical intervention. Others are less severe but can become problematic during childhood. Some may be missed altogether during childhood, only to manifest sometime in adult life. It is beyond the scope of this text to provide details regarding each type of congenital heart disease.

In some disorders, oxygenated blood from the left side of the heart (having already traveled through the lungs) is shunted to the right side of the heart. Examples include atrial septal defect (ASD), ventricular septal defect (VSD), and patent ductus arteriosus (PDA). In other disorders, unoxygenated blood from the right side of the heart is shunted to the left side, thus resulting in systemic “cyanosis” (a blue discoloration related to low oxygen content within blood). Examples include tetralogy of Fallot, transposition of the great arteries, truncus arteriosus, tricuspid atresia,

and total anomalous pulmonary venous connection. In other disorders, the major problem is related to a structural obstruction that exists. Examples include coarctation (constriction) of the aorta, pulmonary stenosis (narrowing) or atresia (complete closure) associated with a hypoplastic (small) right ventricle, and aortic stenosis or atresia.

Anomalous (or “aberrant”) coronary artery anatomy is occasionally a cause of sudden, unexpected death, especially in childhood or young adulthood. Several variations exist. In some, instead of two coronary arteries (which supply blood to the heart itself), there is only a single artery. In others, one of the coronary arteries arises in an abnormal location (Fig. 10.2), such that the coronary artery is compressed between the aorta and pulmonary artery, with resultant myocardial hypoxia.

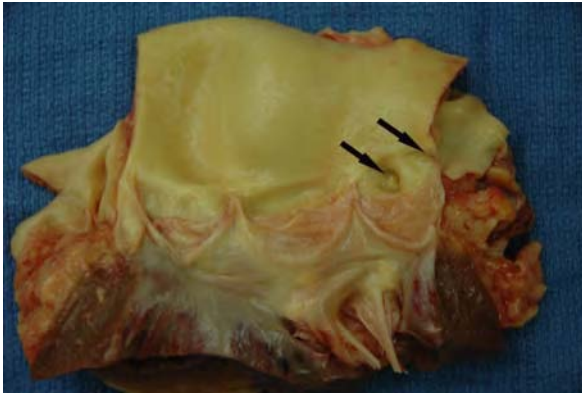


Fig. 10.2 A case of aberrant origin of a coronary artery. Note that both coronary artery ostia (openings indicated by arrows) arise from above the same aortic valve

A final example of a congenital disorder of the heart is a congenitally bicuspid aortic valve. Normally, the aortic valve has three cusps. In this disorder, there are only two cusps (Disc Image 10.1). A very rare congenital disorder is a unicuspid (one cusp) aortic valve. These disorders are associated with an increased risk of degenerative calcification, with associated aortic valve stenosis (see Valve Disorders below).

Congestive Heart Failure (Heart Failure)

Heart failure occurs when the “pump” (the heart) can no longer pump efficiently enough to supply the remainder of the body with adequate perfusion of blood. It can occur very quickly, but often occurs in settings where underlying stressors cause the heart to enlarge over many years, attempting to compensate, until the heart finally begins to fail. Heart failure can result from a variety of underlying heart disorders, including many included in this section. In addition, it may result from problems

related to other organs, such as the lung, or systemic disorders, such as hypertension. Sometimes physicians speak of left-sided heart failure and right-sided heart failure. Left-sided failure is far more common, and can ultimately lead to right-sided failure as well. Right-sided heart failure typically results from severe underlying lung disease, such that blood vessel pressure in the pulmonary artery becomes markedly elevated, resulting in the right ventricle having to work extra hard.

Coronary Artery Atherosclerosis

One of the most common cardiac causes of sudden death is coronary artery atherosclerosis (or arteriosclerosis). Sometimes, this is simply referred to as “coronary artery disease.” Atherosclerosis is a more scientific name for “hardening of the arteries.” It is a disease that affects large, medium, and small arteries, including the aorta and its branches, the coronary arteries and their branches, and the cerebral arteries, and is characterized by the build-up of fatty, cholesterol-containing “plaques” within the walls of arteries (Disc Image 10.2). The plaques can become so large in medium and small arteries that the opening on the inside of the artery (referred to as a “lumen”) can become markedly narrowed (Fig. 10.3). This is bad, as it prevents adequate blood flow (it causes ischemia). Additionally, the presence of a plaque is a potential location for thrombus (blood clot) formation. If a thrombus forms overlying an otherwise non-occlusive plaque, total blockage of the artery can result (Fig. 10.4). Pathologists categorize the extent of coronary artery atherosclerosis based on the approximate percentage narrowing, or “stenosis,” caused by the plaque. Anything less than 50% is considered mild, while 50–75% is considered moderate, and >75% is severe.

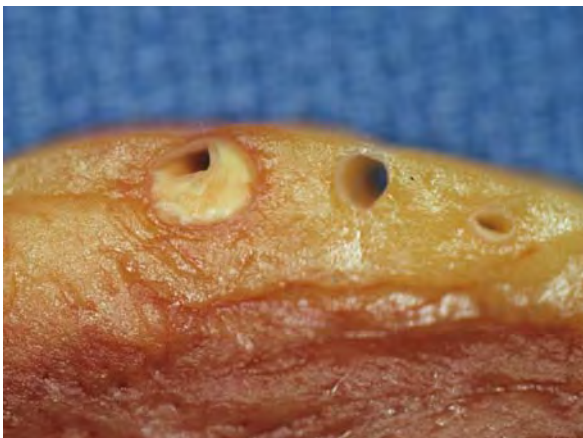


Fig. 10.3 Coronary artery atherosclerotic plaque (*left*), causing stenosis (*narrowing*) of the coronary artery lumen (*opening*). For comparison, a normal coronary artery (with no atherosclerotic plaque) is located just to the right of the disease artery



Fig. 10.4 Coronary artery thrombus (clot), causing total occlusion (blockage) of the artery

Persons with at least one severe coronary artery atherosclerotic lesion are at risk of sudden death. Therefore, if a single severe plaque is identified at autopsy, death can be attributed to coronary artery disease, so long as there is no other more logical explanation for death (like a gunshot wound). There may or may not be evidence of acute (recent) or remote (old, healed) myocardial infarcts (“heart attacks;” characterized by necrotic heart cells or scarred areas). In many cases, there is no gross or microscopic evidence of an infarct. This does not mean that a myocardial infarct did not occur: recall that it takes several hours for an infarct to become visible. Alternatively, the atherosclerosis-induced hypoxia might have caused a fatal arrhythmia. In a minority of cases, there is evidence of a definite acute myocardial infarct (Fig. 10.5 and Disc Images 10.3, 10.4, and 10.5). Sometimes, the necrotic heart muscle has actually ruptured, causing an acute loss of blood from within the

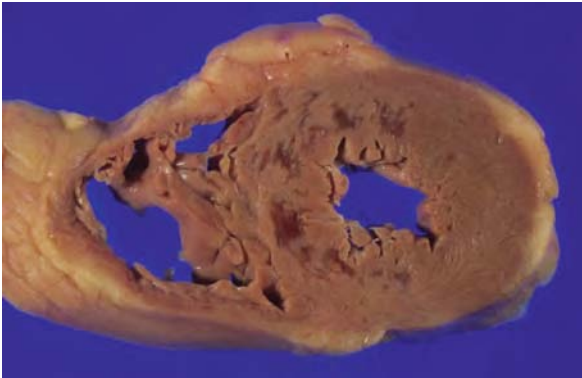


Fig. 10.5 A cross-section of the heart showing an acute myocardial infarct, characterized by patchy, dark discoloration within the myocardium, primarily within the interventricular septum

heart into and filling the pericardial cavity (“heart sac”) (Fig. 10.6 and Disc Image 10.6 and 10.7). This finding is referred to as a “hemopericardium,” and it results in a clinical entity called “cardiac tamponade,” in which the blood filling the sac constricts the heart so much that it can no longer pump. Occasionally, pathologists will discover an occlusive thrombus within a coronary artery. As described above, there may or may not be evidence of an associated myocardial infarct. Invasive therapy for coronary artery atherosclerosis includes angioplasty (compression of the plaque with a tiny balloon), followed by metallic stent placement (Disc Image 10.8), and bypass surgery (see below).

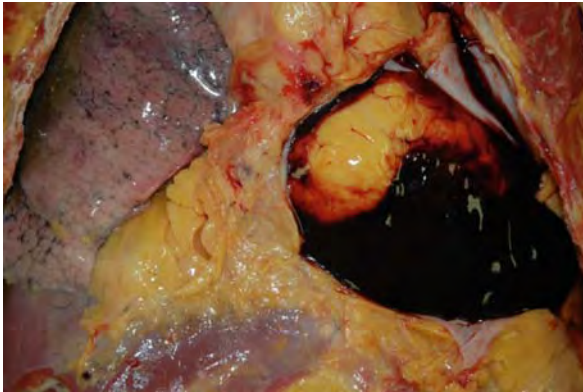


Fig. 10.6 Blood filling the opened pericardial sac at autopsy. The blood came from a ruptured myocardial infarct (such as that depicted in Disc Images 10.6 and 10.7)

In persons who have a long history of previously non-lethal coronary artery disease, there may be evidence of all sorts of heart damage. Extensive, calcified coronary artery atherosclerotic plaques are common, as are numerous remote

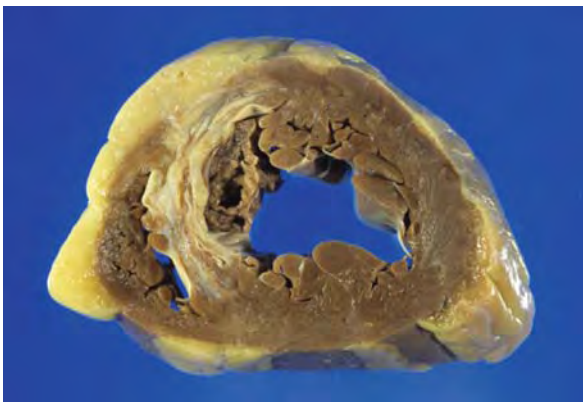


Fig. 10.7 A cross-section of the heart showing a remote (old, healed, scarred) myocardial infarct of the interventricular septum. Note that this area is thin and white/grey

(scarred) myocardial infarcts (Fig. 10.7). Aneurysmal dilation (thinned wall with associated out-pouching) of the heart may be present, with or without thrombus formation along the inside of the aneurysm. Remote (old) infarcts may also have thrombus formation within the ventricular cavity with no associated aneurysmal dilatation (Disc Image 10.9). These hearts tend to be enlarged, and a general term used to describe them is “ischemic cardiomyopathy.” In some of these hearts, there is evidence of previous therapy, including metallic stents within the coronary arteries, or the presence of coronary artery bypass graft (CABG) surgery (Disc Images 10.10 and 10.11).

Coronary Artery Dissection

A coronary artery disorder that is much less common than atherosclerosis is coronary artery dissection. In this disorder, the wall of the coronary artery becomes disrupted, such that blood is able to “dissect” through and within the wall of the artery (Disc Image 10.12). Occasionally, a coronary artery dissection is induced via “angioplasty,” wherein a cardiologist inserts a catheter into the artery and inflates a tiny balloon in order to compress a plaque and thus open up a stenotic artery. In other instances, a spontaneous coronary artery dissection occurs. This phenomenon is known to occur as a complication of pregnancy or as a condition totally unrelated to pregnancy. When it is discovered at autopsy, pathologists should consider the possibility of an underlying connective tissue disorder, such as Ehlers–Danlos syndrome (EDS). Surviving family members should be warned that such conditions often have a genetic basis.

Other Coronary Artery Disorders

Several other coronary artery disorders deserve mention. Aberrant coronary artery anatomy was presented in the section on congenital heart disease (above). “Intramycocardial tunneling” or “bridging” refers to a situation where a part of a coronary artery (usually the left anterior descending), which normally travels within the epicardial fat, actually travels within the myocardium, such that there is muscle between the coronary artery and the epicardial fat (Disc Image 10.13). Some contend that such a finding can be associated with sudden death. In the author’s experience, the finding is almost always incidental. Another coronary artery abnormality that is occasionally implicated in death is a small-caliber (diminutive) coronary artery. A disease of childhood known as Kawasaki disease can result in aneurysms (focal areas of dilation or outpouching), which can rupture later in life. Finally, it is known clinically that some coronary arteries, during life, can undergo spasm, with marked constriction and associated ischemia. This cannot be proven or disproven at autopsy.

Aortic Aneurysms (Abdominal and Thoracic)

An aneurysm represents an area of dilation (larger than normal diameter). An aneurysm may involve the heart, as described above, or a blood vessel. There are two basic types of aortic aneurysms, an abdominal aortic aneurysm (AAA) and a thoracic aortic aneurysm. Each is different from the other.

An AAA can be considered another manifestation of atherosclerosis. Severe atherosclerotic plaque within the abdominal aorta results in weakening of the aortic wall, with associated dilation. Usually, there is extensive thrombus formation within the lumen of the aorta at this location. As the aneurysm becomes larger, the risk for rupture increases (Fig. 10.8). Sudden death occurs when the aneurysm ruptures. The typical finding at autopsy is a massive hemoperitoneum (abdominal cavity filled with blood), and hemorrhage within the soft tissues behind the abdominal cavity (“retroperitoneal hemorrhage”) may also be evident.

Fig. 10.8 An opened aorta, showing severe atherosclerosis, as well as an abdominal aortic aneurysm (AAA). A rupture site is indicated with a probe



A thoracic aortic aneurysm may or may not be as readily recognizable as an AAA. Frequently, there is an overall increased diameter of the aorta as it travels upward from the heart; however, this aneurysm is not as well-defined as most AAAs. Thoracic aortic aneurysms are typically not related to atherosclerosis, although some may have coexisting atherosclerosis. The underlying abnormality in thoracic aortic aneurysms is a disorder of the connective tissue, such that the aortic wall is susceptible to dissection, in which blood breaks through the innermost part of the aorta, then travels within the wall, often lengthwise, until it ruptures through the entire wall thickness (Fig. 10.9 and Disc Images 10.14, 10.15, 10.16, 10.17, 10.18, and 10.19). This rupture can result in hemopericardium, hemothorax (in which blood fills a chest cavity, surrounding and compressing a lung)

Fig. 10.9 A thoracic aortic dissection with associated hemorrhage



(Disc Image 10.20), hemoperitoneum, or retroperitoneal hemorrhage. Under the microscope, pathologists will frequently see “cystic medial necrosis” or “myxoid (mucinous) degeneration” within the aortic wall. Sometimes, thoracic aortic aneurysms/dissections occur in the setting of a well-defined genetic disorder, such as Marfan syndrome or EDS. Most of the time, it does not occur as part of a well-defined syndrome; however, every case should be considered at least potentially familial. Therefore, surviving family members should be advised to seek medical advice from their physicians.

A subset of thoracic aneurysms is caused by *Treponema pallidum* infection, as part of the disease commonly referred to as syphilis. Syphilis has three stages, known as primary, secondary, and tertiary syphilis. In tertiary syphilis, the thoracic aorta becomes inflamed and markedly thickened. Aneurysmal dilation can accompany the thickening, as can occlusion of the coronary artery ostia (where the coronary arteries attach to the aorta). Microscopically, the aortitis (inflammation of the aorta) is characterized by “obliterative endarteritis” and perivascular plasma cell inflammation (the small blood vessels within the wall of the aorta are inflamed, with associated narrowing).

Cerebrovascular Disease

Cerebrovascular disease is the term applied to what is commonly referred to as a “stroke” or a “cerebrovascular accident” (CVA). The disease is discussed further under “Central Nervous System” below.

Vasculitis

The term “vasculitis” specifically refers to inflammation of blood vessels. There are several subtypes of vasculitis, many of which are considered autoimmune disorders. Depending on the type, a vasculitis may preferentially affect veins or arteries or very small vessels. Some forms preferentially affect certain organs, while others are more systemic in nature. Examples include “polyarteritis nodosa” and “giant cell arteritis,” but many other subtypes exist. Blood vessel inflammation resulting from an infectious disease can also occur. Certain micro-organisms preferentially infect blood vessels, including *Aspergillosis* (a fungus), *Treponema pallidum* (the spirochete bacteria that causes syphilis), and *Rickettsia rickettsii* (the bacteria responsible for Rocky Mountain spotted fever).

Fibromuscular Dysplasia

Fibromuscular dysplasia is a non-atherosclerotic arterial disease that tends to affect small arteries and arterioles (very small artery branches). It is characterized by thickening of the blood vessel walls, such that the lumen becomes stenotic (narrowed). It may preferentially affect certain arteries, such as the renal (kidney) arteries or small vessels within the heart. In the heart, the diagnosis can only be made via microscopy, which can show the disease preferentially affecting the small arteries supplying blood to various parts of the conduction system, such as the AV node or the SA node. If present in these locations, in the absence of another credible explanation for death, fibromuscular dysplasia can be considered lethal (Disc Image 10.21).

Myocarditis

The term “myocarditis” specifically refers to inflammation of the heart. The usual explanation is an underlying virus infection; however, some cases occur as a reaction to a drug, a different micro-organism infection, an autoimmune process, or, in some cases, with no apparent underlying cause. The usual case has an inflammatory process characterized by the presence of white blood cells (typically lymphocytes and macrophages) (Disc Image 10.22). The inflammation varies from focal and slight to diffuse and intense. The heart can become flabby and dilated. Presumably, cases of sudden death occur when the inflammation induces a fatal arrhythmia, or when the heart becomes so dilated that “heart failure” ensues (it no longer functions efficiently). Special studies, including submitting samples to the Centers for Disease Control (CDC), can sometimes be performed in an attempt to identify an underlying viral organism.

Hypertensive Cardiovascular Disease

Hypertensive cardiovascular disease, or simply “hypertension,” is commonly known as “high blood pressure.” This disorder is very common within the United States, where it frequently is accompanied by atherosclerosis, hence the often-used combined designation of “hypertensive and atherosclerotic cardiovascular disease.” There are many possible underlying causes of hypertension, including adrenal gland tumors, kidney diseases, renal artery fibromuscular dysplasia or other conditions, and various endocrine disorders; however, a vast majority of cases are referred to as “essential” or “idiopathic,” where an exact underlying cause cannot be determined. The major change within the cardiovascular system that is characteristic of hypertension is cardiac hypertrophy, which results in an enlarged heart (cardiomegaly). The hypertrophy involves the left ventricle, and it tends to be concentric (all walls of the left ventricle are thickened equally) (Fig. 10.10). Microscopically, there are hypertrophic (enlarged) myocytes (Disc Image 10.23).



Fig. 10.10 A cross-section of a hypertrophic heart, related to hypertension (high blood pressure)

Because of the enlarged heart that occurs with hypertension, persons with this disorder are at increased risk for sudden death due to an arrhythmia. In addition, a variety of other vascular disorders occur with increased frequency in persons with hypertension, including spontaneous brain hemorrhages, ruptured cerebral artery berry aneurysms (see Central Nervous System below), and thoracic aortic aneurysms/dissections. Another central nervous system finding that can be seen in patients with hypertension is “lacunar” infarcts, small infarcts within the basal ganglia, thalamus, and elsewhere. Characteristic kidney changes also occur in hypertension.

Valve Disorders

Recall that there are four valves within the heart. The tricuspid valve separates the right atrium and ventricle; the pulmonic valve separates the right ventricle from the pulmonary artery; the mitral valve separates the left atrium and ventricle; and the aortic valve separates the left ventricle from the aorta. Proper functioning valves are necessary for normal heart function. Normal valves are relatively thin, and very flexible. Properly functioning valves open fully to allow blood flow through the valve when the cavity “in front of” the valve contracts, and close afterwards, thus preventing “back-flow.” If a valve is firm (sometimes calcified), it cannot fully open, and it is referred to as being “stenotic” or narrowed. If a valve does not fully close, or if it is damaged so that when closed, it allows back-flow, then the valve is referred to as being “regurgitant.”

Degenerative aortic stenosis occurs as a disorder of aging and is characterized by rock-hard calcification with associated stenosis (Disc Image 10.24). For this reason, it is sometimes referred to as “senile calcific aortic stenosis.” It is best thought of as a “wear and tear” valve disorder. Patients with congenitally bicuspid (or unicuspid) valves are prone to develop this disorder much earlier in life. With severe disease, there is marked cardiomegaly, owing to the fact that the heart muscle must work extra hard to push blood through the narrow valve opening. In addition, in severe cases, the valve is not only stenotic, but also regurgitant, thus making the heart work even harder.

Mitral valve prolapse (“myxomatous degeneration of the mitral valve” or “floppy mitral valve syndrome”) is a disorder characterized by a floppy, rubbery mitral valve, with redundant (excess) tissue, such that, on gross exam, the valve leaflets appear ballooned or prolapsed (into the left atrium) (Fig. 10.11). The disorder is actually fairly common and is typically an incidental autopsy finding; however, sudden death



Fig. 10.11 A case of mitral valve prolapse (MVP) characterized by a mitral valve that is redundant and “billowing”

occasionally occurs in patients with MVP. Some individuals with MVP have an underlying connective tissue disorder, such as Marfan syndrome.

Infective endocarditis, or bacterial endocarditis, refers to a bacterial infection of the heart valves, typically associated with the growth of “vegetations” on the valves (Disc Image 10.25). The disorder may be subacute, in which the bacterial infection occurs over a period of weeks to months and usually responds to antibiotic therapy, or acute, in which there is very rapid valve destruction with associated heart failure and systemic infection. To survive the acute variety usually requires surgical removal (and replacement) of the valve (Disc Image 10.26). Infective endocarditis can occur on normal valves or on valves that are defective from other valve disorders. Intravenous drug users are at particular risk for developing infective endocarditis.

Other valve disorders include rheumatic heart disease, which starts as a myocarditis many years before the onset of valve deformities, a condition referred to as “nonbacterial thrombotic endocarditis,” and “Libman–Sacks” endocarditis, which occurs in the setting of systemic lupus erythematosus.

Hypertrophic Cardiomyopathy

A group of genetic disorders involving various components of cardiac myocytes are responsible for an entity that is known as “hypertrophic cardiomyopathy.” Other terms that have been used to describe this condition are idiopathic hypertrophic subaortic stenosis (IHSS) and hypertrophic obstructive cardiomyopathy. It is characterized by marked left ventricular hypertrophy, sometimes disproportionately involving the interventricular septum (“septal hypertrophy” or “asymmetric left ventricular hypertrophy”) (Fig. 10.12). Various blood flow problems can occur because of the hypertrophic heart muscle and associated lack of ventricular cavity space. Persons with this disorder are also at risk for lethal arrhythmias. Microscopically, hypertrophic myocytes are present. In classic cases, the myocytes are haphazardly arranged (sometimes referred to as “myofiber disarray”) (Disc Image 10.27). The underlying pathology is related to mutations of genes that are responsible for various components of cardiac myocytes. Testing for some of these is available, but is currently quite expensive. In the future, as technology advances, the availability and cost should allow many offices to have such testing performed. As these represent genetic disorders, surviving family members should be counseled to seek medical advice concerning this condition.

Dilated Cardiomyopathy

Dilated cardiomyopathy is characterized by a heart that is “floppy,” with dilated chambers (Fig. 10.13). In many cases, all of the chambers are dilated. There are numerous underlying causes of dilated cardiomyopathy, including obesity, chronic

Fig. 10.12 A longitudinal section of a heart with asymmetric left ventricular hypertrophy. Note how the interventricular septum (the central portion that separates the two cavities) is thicker than the left ventricular free wall (on the right side of photo). This condition is called “hypertrophic cardiomyopathy,” but is sometimes referred to as idiopathic hypertrophic subaortic stenosis (IHSS)

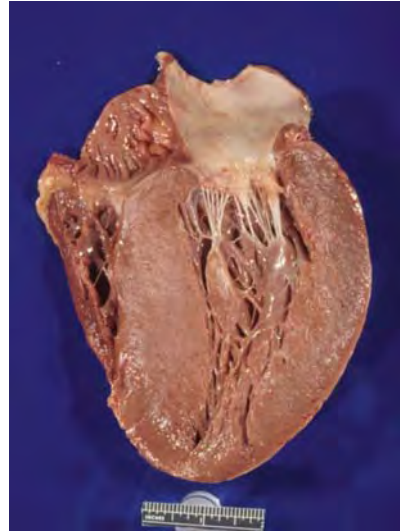


Fig. 10.13 A cross-section of a heart with dilated cardiomyopathy. Note how both the left and right ventricular cavities are markedly enlarged (dilated). In this photograph, the right ventricle is on the left, and the left ventricle is on the right

alcoholism, and viral infections. Other cases occur toward the end of or after pregnancy (peripartum cardiomyopathy). In many cases, a definite underlying cause cannot be determined, although genetic testing has revealed that many of these are caused by underlying genetic mutations (similar to hypertrophic cardiomyopathy). In all types, sudden death is possible and is frequently attributed to a presumed arrhythmia.

A specific subtype that primarily affects the right ventricle is referred to as “arrhythmogenic right ventricular cardiomyopathy” or “arrhythmogenic right ventricular dysplasia.” It is characterized by a markedly dilated right ventricular

chamber, with thinning of the right ventricular wall, which is replaced with fat tissue intermixed with fibrous tissue (Disc Image 10.28).

Restrictive Cardiomyopathy

Of the three major types of cardiomyopathy (hypertrophic, dilated, and restrictive), restrictive is the least common. In this disorder, the compliance (flexibility) of the heart is compromised, such that normal filling and function cannot occur. As with the other cardiomyopathies, there are a variety of underlying conditions that can cause a restrictive cardiomyopathy, including radiation fibrosis (scarring), amyloidosis, sarcoidosis, metastatic disease (cancer), endomyocardial fibrosis, endocardial fibroelastosis, and various inborn errors of metabolism.

Deep Venous Thrombosis

When “blood clots” develop within the deep veins of the legs, the condition is referred to as “deep venous thrombosis” (“phlebothrombosis”) (Disc Image 10.29). In some cases, there is an associated inflammatory process; such cases can be referred to as “thrombophlebitis.” The clots can break away from the veins and “embolize” to the lungs, causing sudden death from a “pulmonary thromboembolism.” The condition is further described in Chapter 21.

Conduction System Abnormalities

A variety of conditions can be considered cardiac “electric” disorders. These are characterized by an abnormality in the electrical activity of the heart. The proper term for these conditions is “cardiac conduction system abnormalities.” In life, these conditions can be diagnosed via an electrocardiogram (ECG or EKG). There is an increased risk of sudden death in many of these conditions. Unfortunately, at autopsy, there is usually no detectable anatomic/morphologic abnormality present, either grossly or microscopically. The heart is of normal size and structure. If there is a clinical history of a conduction system abnormality, the pathologist can confidently make the diagnosis, so long as no other explanation of death is found. In death investigation cases where an autopsy is totally negative (including toxicology, scene investigation, and microscopy), it is generally agreed that at least some of the cases represent presumed cardiac conduction system abnormalities. Examples of the names of some of these disorders are Wolff–Parkinson–White syndrome and the long QT syndrome. Specialized molecular tests to identify genetic mutations in these disorders may allow pathologists to readily make postmortem diagnoses sometime in the future.

Neoplastic Heart Disease

Tumors of the heart are relatively rare, but do occasionally occur. Some of these may be associated with sudden death, either via their mass effect (obstruction) within the chambers of the heart or via induction of an arrhythmia. Some primary heart tumors that have been described as causing sudden death include atrial myxomas and papillary fibroelastomas of the valves. A very small benign neoplasm, called an AV nodal tumor, “cystic tumor of the AV node” or “mesothelioma,” can result in conduction system disturbances and sudden death (Disc Image 10.30).

Central Nervous System

The central nervous system (CNS) encompasses the brain (cerebrum, brainstem, and cerebellum) as well as the spinal cord. There are numerous non-traumatic disorders of the CNS that can be responsible for death, including many that can cause sudden, unexpected death. The brain is obviously an important organ. Any disruption of the brain, especially certain portions, can result in terrible disability or death.

Congenital Anomalies

A variety of congenital anomalies can affect the CNS. Many of the lethal anomalies are commonly accompanied by many other anomalies as part of a recognized syndrome. Many of these are genetically based, for example “holoprosencephaly,” in which the brain is not divided into two halves as normal. A severe brain abnormality that is incompatible with life is “anencephaly,” in which a brain, or at least a large portion of it, fails to develop. Some disorders may be compatible with life, but can still cause significant disability, such as the “Arnold–Chiari malformation” (abnormally small posterior aspect of skull, with crowding of the cerebellum and brainstem), the “Dandy–Walker” abnormality (an enlarged posterior skull area, typically associated with a cyst), and “spina bifida” (typically affecting the spinal cord, with various degrees of complications, including paralysis).

Cerebral Palsy

Cerebral palsy (CP) is a general clinical description for a non-progressive neurologic motor (muscular) disorder characterized by muscle spasticity, lack of tone, and discoordination. It is generally believed to be due to insults suffered during intrauterine (prenatal) development and/or during the perinatal (around birth) period. It may not be readily apparent at birth, but becomes evident as a baby grows older. Persons with CP may be totally normal from a mental/intellectual standpoint; however, some have

deficiencies in these areas as well. Some patients with CP are also afflicted with a seizure disorder.

Infection

Infections of the CNS are very serious. Bacterial infections tend to occur as one of two types. “Meningitis” occurs when there is bacterial infection of the meninges (the thin membranous covering of the brain). Symptoms typically include severe headaches. At autopsy, the meninges contain purulent material (pus) (Fig. 10.14), which is sometimes hemorrhagic (Disc Image 10.31). When discovered at autopsy, pathologists should collect culture specimens (using sterile cotton-tipped swabs) and send the samples to the microbiology laboratory for immediate Gram-staining and subsequent culture. A variety of bacteria can cause this infection. In newborns, Gram-negative bacilli and group B *Streptococcus* are most prevalent. In infants and children, *Hemophilus* is most common. In teenagers and young adults, *Neisseria meningitidis* is most frequent. When *Neisseria meningitidis* (“meningococcus”), which is a Gram-negative diplococcus, is the cause, it should be considered highly contagious. All contacts, including those in the morgue, should be treated with prophylactic antibiotic therapy.

Fig. 10.14 A case of meningitis, characterized by a purulent exudate (pus) seen overlying the brain



The second type of bacterial infection in the brain is a brain abscess. Often, multiple abscesses occur. An abscess represents a focal area within the brain where bacteria are growing and causing destruction of the brain (Disc Image 10.32). Bacterial brain abscesses may occur in the setting of sepsis (widespread bacterial infection) and bacterial endocarditis (infection of the heart valves).

Viral encephalitis can be caused by a number of different viruses, including many that are transmitted by other animals (mosquitoes, ticks). Examples include Eastern equine encephalitis, Western equine encephalitis, St. Louis encephalitis, and West Nile viral encephalitis. Within the brain, areas of intense perivascular (around blood vessels) lymphocytic inflammation are characteristic (Disc Image 10.33). Blood and tissue samples can be sent to the Centers for Disease Control (CDC) for definitive diagnosis. Other viruses, such as herpes and cytomegalovirus (CMV), can infect fetuses or immunocompromised persons. Rabies is a severe viral infection that may be transmitted to humans via the bite of a rabid animal, typically a bat. It has a long incubation period and is usually considered universally fatal if, in fact, the infection fully develops within the CNS.

Other infectious micro-organisms include fungi, parasites, and prion diseases. Many of the fungal and parasitic infections are most common in the setting of a compromised immune system, such as occurs in the Acquired Immune Deficiency Syndrome (AIDS). Cryptococcus, toxoplasmosis, and aspergillosis are examples of these infections. Neurocysticercosis is a parasitic infection of the brain that can be a totally incidental finding, a cause of seizures, or a cause of death. Another parasitic infection that occasionally occurs is *Naegleria fowleri*, an amoeba that lives in stagnant water. If a person swims in such water, the amoeba can enter the sinuses, invade the brain, and cause death. Prion diseases are actually caused by the transmission of a protein. Such disorders, known as “spongiform encephalopathies,” include Creutzfeld–Jakob disease (CJD) and bovine spongiform encephalopathy. A history of rapidly progressive dementia should alert the pathologist that a spongiform encephalopathy may be present. This history should be considered a huge “red flag” within the death investigation community. The exact transmission mode of these disorders is not known, but exposure at autopsy is considered extremely high-risk. As such, it is best in these situations to refuse to perform the autopsy and instead refer the case to a medical-school-based neuropathologist.

Seizure Disorders

Seizures represent abnormal electrical activity within the CNS. A variety of seizure types occur, the most recognizable being the grand mal seizure, which manifests as generalized body convulsions. Many specific CNS disorders can induce seizures, including trauma, tumors, strokes, infections, etc. A majority of cases of “epilepsy” have no specific underlying CNS disorder, and can be referred to as “idiopathic” seizure disorders. Persons with a chronic seizure disorder are at risk for sudden, unexpected death. This is true regardless of the use of anti-seizure medication, although presumably anti-seizure medication reduces the risk by reducing the frequency of seizure activity. The presumed mechanism of death in these cases is a seizure-induced cardiac dysrhythmia. Most sudden deaths due to seizures are natural deaths; however, if the seizure disorder was initially caused by trauma, then the manner of death should be determined by the circumstances of the trauma.

Occasionally, a seizure is the cause of an accident, which then results in a traumatic death. Examples include someone experiencing a seizure while driving, running off the road and dying from blunt force injuries, and someone experiencing a seizure in a bathtub, with resultant drowning. The cause and manner of death in such cases might be written as follows: COD part I: drowning; part II: Seizure disorder; MOD: accident. It is unusual for pathologists to discover a definitive underlying microscopic cause for seizures, and hence autopsies on persons who die from seizures are typically negative. A diagnosis of death due to a seizure disorder, therefore, is a so-called “diagnosis of exclusion.” So long as there is no other explanation for death in a person with a history of seizures, then the cause of death can be ruled “seizure disorder.”

Dementia

Dementia of a variety of types may be a cause or contributory cause of death. Subtypes include Alzheimer’s Disease, vascular dementia, and senile dementia, to name a few. Many forms demonstrate brain atrophy. Alzheimer’s Disease has characteristic microscopic findings, including “senile plaques” and “neurofibrillary tangles”.

Cerebrovascular Disease

Cerebrovascular disease is responsible for what is commonly referred to as a “stroke.” The term “cerebrovascular accident” (CVA) is sometimes used. The disease involves the development of a cerebral (brain) infarct, due to lack of oxygen. Many strokes are related to atherosclerotic plaque build-up within the carotid arteries or cerebral arteries. Some are related to thromboemboli that form elsewhere (for example in the heart, or on a heart valve) and travel to the brain. Cerebral infarcts may be relatively bloodless or hemorrhagic (Disc Image 10.34). Persons who experience a stroke do not always die from the stroke; a substantial amount of brain can be involved with continued survival. “Remote” (old) strokes are evident at autopsy as areas of brain absence (Disc Image 10.35). So-called “spontaneous intraparenchymal hemorrhages” can occur within the cerebrum, the brainstem, or the cerebellum (see below). In cases of sudden death due to such a spontaneous hemorrhage, the hemorrhage is typically quite massive, and there is frequently a history of or autopsy findings suggestive of hypertensive cardiovascular disease.

Hypertensive Disease

Hypertensive disease was discussed above in the “Cardiovascular Disease” section. A variety of CNS disorders can be associated with underlying hypertension. Included here are strokes, spontaneous intraparenchymal hemorrhages, ruptured berry aneurysms, and hemorrhagic arteriovenous malformations.

Spontaneous Intraparenchymal Hemorrhage

As mentioned earlier, spontaneous intraparenchymal hemorrhage tends to occur in the deep cerebrum (basal ganglia or thalamus) (Fig. 10.15), the cerebellum or the pons. Oftentimes, it is associated with hypertensive cardiovascular disease. As such, it is appropriate to rule these deaths as follows: COD: part I – Spontaneous intracerebral (or pontine, or intracerebellar) hemorrhage; part II – Hypertensive cardiovascular disease; MOD: Natural.

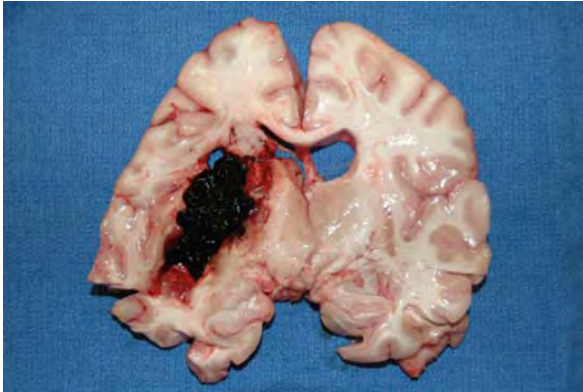


Fig. 10.15 An example of a spontaneous hemorrhage within the cerebrum (brain)

Ruptured Berry Aneurysm

A berry aneurysm is a localized out-pouching of the wall of a cerebral artery that is at risk for rupture. It is generally accepted that the propensity to develop berry aneurysms represents a congenital disorder. The aneurysms tend to be relatively small, but occasionally measure more than a couple of centimeters, and tend to be more prevalent at branch points along arteries. Occasionally, multiple aneurysms are found. Rupture of a cerebral artery berry aneurysm is characteristically accompanied by “the worst headache” the person has ever experienced. Underlying hypertension may be present. At autopsy, there is typically extensive basilar subarachnoid hemorrhage (SAH) (Fig. 10.16). In fact, a ruptured berry aneurysm is the most common non-traumatic cause of a SAH. When dense basilar SAH is identified at autopsy, pathologists should take the time to carefully wash away the blood in an attempt to find the ruptured aneurysm (Figs. 10.17 & 10.18).

Ruptured Arteriovenous Malformation (AVM)

An arteriovenous malformation (AVM) is a localized tangle of blood vessels, including arteries and veins, that are interconnected with one another. They can occur anywhere in the brain (or elsewhere in the body). Like berry aneurysms, AVMs

Fig. 10.16 The base of a brain showing basilar subarachnoid hemorrhage. When this is discovered at autopsy, the pathologist must carefully search for a ruptured berry aneurysm. This involves attempting to wash away much of the hemorrhage while maintaining the integrity of the blood vessels

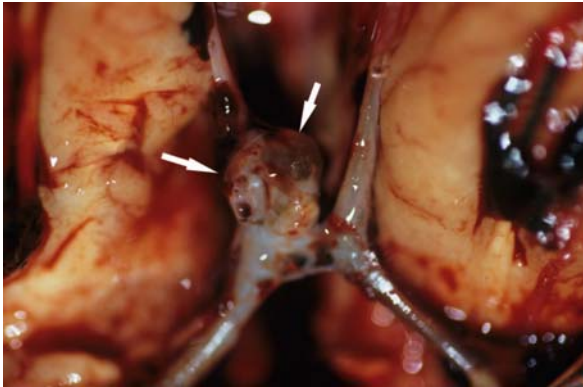


Fig. 10.17 A close-up view of the base of the brain in Fig. 10.16, after washing away much of the subarachnoid hemorrhage. Note the presence of a berry aneurysm (*arrows*)

can spontaneously rupture. Underlying hypertension may or may not be present. Usually, the AVMs will be evident grossly (Disc Image 10.36), but some examples are quite small. Microscopic identification of multiple vessels allows a diagnosis to be given (Disc Image 10.37).

Dural Sinus Thrombosis

Thrombus (blood clot) formation within the veins of the dura mater (the “dural sinuses”) is a relatively rare condition that can cause central nervous system symptoms (such as headaches), “strokes,” and even sudden death. The condition is more

Fig. 10.18 The cerebral arteries have been carefully removed from the base of the brain shown in Figs. 10.16 and 10.17. The berry aneurysm is more readily evident (*arrow*)



common in women and most commonly occurs in the third decade. Risk factors include conditions that are considered risk factors for thrombus formation elsewhere (such as the deep veins of the legs) (refer to Chapter 21).

Neoplasia

Brain tumors come in a variety of types, including benign and malignant forms. Since the cranial cavity is an enclosed space, and since the brain is such an important organ whose integrity is necessary for life, even benign tumors can result in death, usually as a result of their “mass effect” or because they obstruct the normal flow of cerebral spinal fluid (Fig. 10.19). Malignant tumors can kill by similar mechanisms or by invasion of vital structures, hemorrhage, or metastasis. The most common rapidly lethal malignant brain tumor is known as a glioblastoma multiforme (GBM) (Disc Image 10.38). Occasionally, the first manifestation of a brain tumor (benign or malignant) is sudden death.

Respiratory System

Congenital Anomalies

As with other organ systems, congenital anomalies of the respiratory system may be sufficient to cause death. In some of the more severe forms (when the trachea and bronchi are not “connected” appropriately, or if they are narrowed or not formed at all), problems may be evident at birth, or even prior to birth. Sometimes, portions

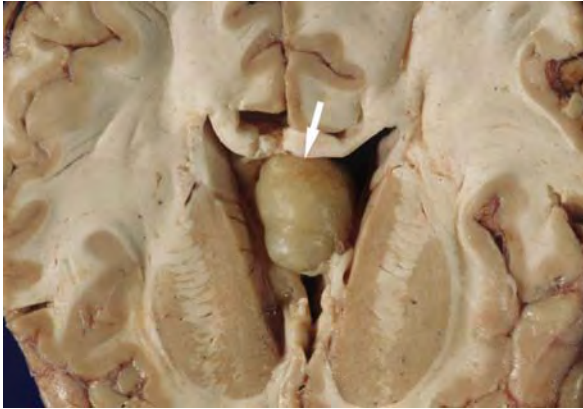


Fig. 10.19 A colloid cyst of the third ventricle of the brain (*arrow*). This biologically benign tumor caused sudden death due to the obstruction of the flow of cerebral spinal fluid

of a lung are not properly connected to the remainder of the lung, with infection or other problems occurring. Minor anomalies, such as a bronchus arising from higher up within the trachea (a so-called “tracheal bronchus”), can also cause death (usually via infection), and may not be recognized clinically.

Neonatal Conditions

Meconium Aspiration: During the birth process, infants can aspirate amniotic fluid. If the fetus has been distressed during the labor and birthing process, meconium (the first bowel movement) may be contained within the amniotic fluid. Aspirated meconium can be a serious insult to the newborn’s lungs, sometimes resulting in death.

Hyaline Membrane Disease: The lungs of very premature infants do not contain sufficient amounts of “pulmonary surfactant.” In the absence of this important substance, the surface tension within the alveoli (air sacs) is not optimal, and inflammatory substances fill and line the alveolar walls, causing “respiratory distress of the newborn.” Death can result. Oxygen therapy can save some, but problems related to oxygen toxicity can be long-term complications.

Upper Airway Conditions

The epiglottis is at the upper end of the larynx and functions to cover the airway opening during swallowing, so that food does not enter the upper airway. Severe swelling (occurring in an allergic reaction) or inflammation from infection (“epiglottitis”) can cause lethal narrowing of the airway, resulting in an asphyxial death (Disc Image 10.39).

Pulmonary Thromboembolism

A mechanism of death that is quite common and results in a classic “sudden death,” wherein the victim simply “drops dead,” is a pulmonary thromboembolism (Disc Image 10.40). As this process typically occurs in association with an underlying predisposing condition or susceptibility, some of which are not “natural,” it is discussed along with other types of emboli in Chapter 21 (Miscellaneous Topics).

Bacterial Pneumonia

When a lung becomes infected with a bacteria, the process is usually referred to as “pneumonia.” There are two basic types: lobar (in which an entire lung lobe is affected), and bronchopneumonia (in which there are patchy areas of pneumonia throughout the lungs, typically centered around bronchi). Each is capable of causing death. Particularly with bronchopneumonia, an underlying predisposing condition (disease or situation or injury) is typically identified. For example, someone can develop a bronchopneumonia following abdominal surgery. Someone else may develop bronchopneumonia because their lungs are “filling with fluid” from their congestive heart failure. Still another individual might develop a bronchopneumonia while on a ventilator in the intensive care unit following severe head injuries sustained in a motor vehicle collision. Grossly at autopsy, pneumonia causes the lung tissue to become firm, and often there is discoloration of the tissue as well. When seen at autopsy, cultures can be collected in order to identify the causative bacteria. Whether lobar or bronchopneumonia, the findings under the microscope are identical: the alveoli are filled with neutrophils (a type of white blood cell) (Fig. 10.20).

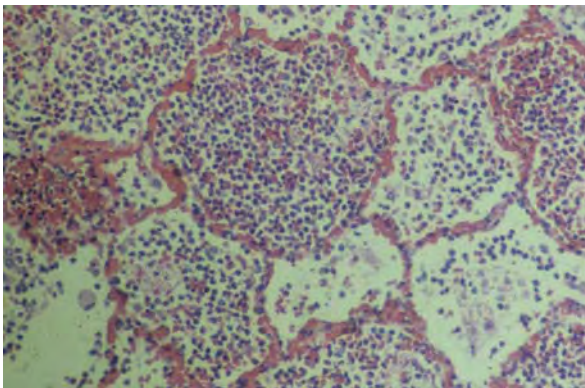


Fig. 10.20 The microscopic appearance of pneumonia, characterized by alveolar spaces (air sacs) filled with polymorphonuclear white blood cells (neutrophils)

Aspiration Pneumonia

“Aspiration pneumonia” specifically refers to a situation where gastric contents are inadvertently inhaled, and inflammation and gastric acid damage ensues. It occurs in a variety of settings, especially in persons with underlying neuromuscular disorders, as well as chronically debilitated persons. Strictly speaking, then, the underlying cause may be a natural disease (cerebral palsy) or something that is not natural (chronic brain injury following trauma). In aspiration, pathologists are usually able to identify various substances under the microscope that are consistent with food particles, as well as acid damage. When aspiration is identified, an underlying reason for the aspiration should be sought out (neurologic compromise, intoxication, extremes of age, etc.). It should be noted here that the identification of aspiration pneumonia implies that the person did not immediately die from the aspiration event, since it takes time after the event to develop the inflammation seen under the microscope.

Viral Pneumonia

Lungs that are infected with a virus typically have inflammation that is confined to the walls of the alveoli (air spaces). Pathologists refer to this pattern as “interstitial” inflammation. The alveoli are not filled with inflammatory cells (as seen in bacterial pneumonia). Grossly, the lungs may or may not have obvious changes. In some cases, they are heavy and somewhat firm. Microscopically, interstitial inflammation is present. Depending on the causative virus, certain cellular changes may be identified. Some viruses that have fairly specific cellular changes are cytomegalovirus (CMV), herpes simplex virus (HSV), and respiratory syncytial virus (RSV). Viruses can be cultured at autopsy, but such tests are usually quite expensive. Alternatively, serum (blood) can be tested for the presence of antibodies directed against certain viruses. For some viruses (RSV) and Influenza, nasal swabs can be tested by molecular techniques (like PCR). In some viral pneumonias (such as influenza), an overlying bacterial pneumonia can be superimposed on the viral pneumonia; this is referred to as a “superinfection.”

Other Infections (Tuberculosis, Fungi, Parasites)

Bacteria and viruses are not the only types of micro-organisms capable of infecting the lungs. Tuberculosis (TB, which is a “mycobacterium”), various fungal organisms (histoplasmosis, aspergillus, etc.), and certain parasites (pneumocystis) can infect the lungs and lead to death. With certain of these (fungi and TB), gross and microscopic “granulomas” (localized areas of chronic inflammation) are readily evident. For many organisms, cultures can be collected at autopsy; however, the tests usually require many weeks to complete. Under the microscope, pathologists frequently rely on the use of various special stains to help identify the organisms. The

identification of pneumocystis should prompt the pathologist to test the decedent's blood for HIV (Disc Image 10.41).

Asthma

Bronchial asthma is a relatively common respiratory disease characterized by a hypersensitive respiratory system that is capable of causing sudden death. In many patients who die of an acute exacerbation of asthma, the diagnosis is previously known; however, occasional decedents die from asthma without prior knowledge of their disease. The typical case is one of chronic asthmatic bronchitis, in which a patient knows about and has dealt with acute exacerbations of the disease. Coughing and wheezing with "air hunger" are classic clinical signs and symptoms. At autopsy, a death caused by acute asthmatic bronchitis may have several findings, both grossly and microscopically. On gross examination, the lungs may appear markedly inflated (hyperinflated), such that they protrude out of the chest cavity after the chest plate has been removed (Fig. 10.21). On sectioning, the major bronchi may contain grossly visible "mucous plugs" occluding their lumens (Disc Image 10.42). In addition, the bronchial walls may be noticeably thickened. Microscopically, the thick bronchial walls are explained by smooth muscle hypertrophy and submucosal gland hyperplasia. The "basement membrane" underlying the mucosal lining is frequently very thick, and there is typically a mixed inflammatory cell infiltrate with numerous "eosinophils," which are a specialized type of white blood cell (Disc Image 10.43). Deaths from acute asthma attacks can occur at any age, including children.

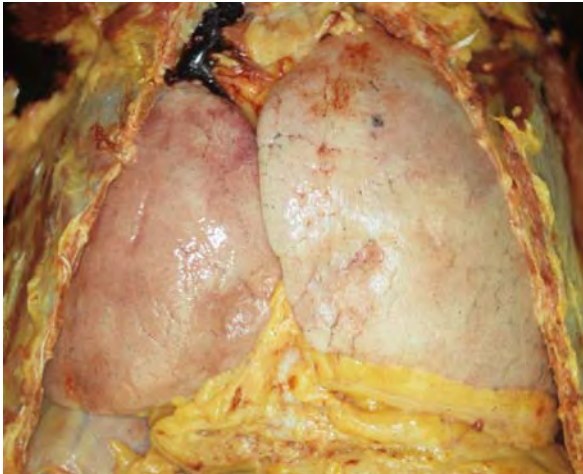


Fig. 10.21 A case of acute asthmatic bronchitis. Note that the lungs appear hyperinflated, such that the pericardial sac and underlying heart are not visible

Chronic Obstructive Pulmonary Disease (COPD)

Strictly speaking, asthma can be classified as one of several types of “chronic obstructive pulmonary diseases,” but since the other disorders classified as COPD tend to primarily affect older individuals, asthma was described separately. Besides asthma, COPD encompasses three other conditions, two of which are very common: emphysema, chronic bronchitis, and a condition called “bronchiectasis,” which involves marked dilation of the bronchi. Like asthma, each is characterized by a primarily obstructive problem within the lungs. The two common entities, emphysema and chronic bronchitis, are characterized by loss of elastic tissue/alveolar walls with enlarged airspaces (Fig. 10.22 and Disc Image 10.44) and inflammation of bronchi, respectively. Each has a strong association with tobacco use. If emphysema is identified in a non-smoker who is relatively young, the pathologist should consider the possibility of alpha-1-antitrypsin deficiency, a genetic disorder characterized by the lack of a substance which normally counteracts “elastase,” an enzyme that breaks down elastic tissue. Severe COPD can cause death in a number of ways, including chronic hypoxia, right-sided heart failure, superimposed infection, rupture of an air pocket (in emphysema) with pneumothorax, or rupture of a blood vessel (with associated “hemoptysis,” or coughing up blood). Depending on the pathologist, COPD may be a frequent disorder listed in part II of the death certificate, as persons with COPD frequently have accompanying heart disease, which is typically listed in part I.



Fig. 10.22 A case of severe emphysema. Note the grossly-visible, markedly dilated air spaces (referred to as “blebs” or “bullae”)

Chronic Lung Disease

A variety of conditions can be considered “chronic lung diseases.” Such disorders sometimes are referred to by other names, such as “interstitial lung diseases” or “pneumoconiosis.” It is far beyond the scope of this text to address these disorders. Suffice to say that there are a variety of substances that can be chronically inhaled which lead to lung injury and compromised respiratory function, for example coal dust, silica, and asbestos. Many of these may be considered “occupational lung diseases,” as they are related to occupational exposure to various substances. Some, like asbestos, are associated with an increased risk of malignancy (cancer). Although such disorders are related to an external causative factor, they are frequently considered “natural,” since they represent the body’s long-term response to that environmental insult (similar to the reasoning behind considering deaths due to chronic alcoholism or chronic drug abuse as natural deaths). Others disagree with this certification. Certainly, the designation of “natural” in such cases does not preclude surviving family members from pursuing civil action.

Pulmonary Hypertension

Pulmonary hypertension refers to a situation where the blood pressure within the pulmonary arteries (and branches) is chronically elevated. Many chronic lung diseases, including asthma, COPD, chronic interstitial diseases, etc., can cause pulmonary hypertension. In addition, a condition referred to as “primary pulmonary hypertension” also exists, and is more common in young women. When pulmonary hypertension exists, it results in increased work for the right side of the heart, which is manifest as right ventricular hypertrophy and potential right-sided heart failure. Deaths can result from cardiac problems (arrhythmias, heart failure) or the underlying lung pathology, or a combination of both.

Neoplasia

Benign lung tumors occur, but are relatively rare. Most lung tumors are malignant (cancer) (Disc Image 10.45). Many represent primary malignancies (arising in the lung), but others represent metastases (malignant growths that have spread to the lungs from a primary cancer elsewhere in the body). In a vast majority of cases, lung cancer is diagnosed before death occurs; however, occasional cases are diagnosed at autopsy. These are the cases that occur within the setting of a medicolegal death investigation system.

Sarcoidosis

Please refer to the “Multisystem Disorders” section below.

Gastrointestinal and Hepatobiliary System

Congenital Anomalies

As with other organ systems, a variety of congenital anomalies may involve the gastrointestinal (GI) or hepatobiliary (liver) systems, ranging from incidental anomalies to serious, potentially lethal defects. During intrauterine development, a portion of the GI system actually protrudes from within the abdominal cavity into the surrounding cavity, with various rotations occurring prior to and during a subsequent return to the proper location. Certain anomalies involve malrotation of the bowel or incomplete formation of the anterior wall of the abdomen. Internal anomalies may include abnormal organ location, areas of weakness within the abdominal/groin wall (predisposing to hernias; see below), defects (holes) within the mesentery (the fatty “fan-like” attachment of the intestines), and residual developmental structures that normally disappear (Meckel’s diverticulum, which may become ulcerated, inflamed, and rupture). Lack of appropriate nerve distribution within the distal (far) end of the colon/rectum results in a condition referred to as Hirschprung disease, wherein normal peristaltic (muscular movement) action cannot occur, so the colon proximal (in front of) the defect becomes massively dilated, with possible rupture.

Gastritis

Gastritis means inflammation of the stomach lining, including the innermost layer (the mucosa), as well as deeper parts of the gastric wall. Since many factors can cause gastritis, such as alcohol consumption, various drugs, and stress related to trauma or other systemic insults, it is a relatively frequent finding at autopsy. It is characterized by the presence of superficial (shallow) mucosal erosions that are typically pinpoint to up to several millimeters in size. Gastritis can result in some degree of bleeding, but usually not to the extent that it contributes significantly to death. Some special subtypes bear the name “ulcer,” although, strictly speaking, many are really erosions (involving only the superficial aspects of the mucosa) rather than ulcers (full thickness of mucosa). Examples include “Curling ulcers” (seen in burn patients), “Cushing ulcers” (seen in persons with increased intracranial pressure), and “Wischnewsky ulcers” (seen in cases of hypothermia).

Peptic Ulcer Disease (PUD)

Peptic ulcer disease (PUD) results from an imbalance between several destructive forces (including *Helicobacter* bacteria, stress, stomach acid, alcohol, certain drugs, etc.) and several protective factors (such as mucus, normal blood flow, bicarbonate, etc.) within and beyond the stomach. An ulcer is a localized area where the mucosa (lining) of an organ erodes away, leaving a crater-like lesion (Disc

Image 10.46). Ulcers in PUD can affect the stomach or the duodenum. Ulcers can lead to chronic blood loss (anemia) with associated medical problems, and can also lead to sudden death, usually by one of two mechanisms. If the ulcer erodes through the entire wall of the stomach or duodenum, gastric/intestinal contents can enter the peritoneal (abdominal) cavity and cause “peritonitis” (inflammation of the peritoneal cavity/lining), which can lead to sepsis (systemic infection) and death. Alternatively, an ulcer can erode into a blood vessel, with subsequent bleeding to death.

Cirrhosis/Esophageal Varices

Cirrhosis of the liver is a process in which liver cells are damaged and die, and new (regenerating) liver cells grow, accompanied by fibrosis (scar tissue). It can be caused by a variety of conditions, including chronic alcoholism, other toxins, hepatitis viruses, and alpha-1-antitrypsin deficiency. Cirrhosis involves the entire liver, so that it becomes noticeably nodular (Fig. 10.23). As the process continues, the normal flow of blood through the liver becomes disrupted. This results in the inability of blood coming from the GI tract via the portal vein to easily pass into and through the liver. The result is what is referred to as “portal hypertension” (elevated blood pressure within the portal vein). When portal hypertension exists, the blood within the portal vein and feeder veins “backs-up” into various parts of the GI tract, where it can actually bypass the liver on its way back to the heart. There are two main areas where the blood can do this: in the veins of the lower esophagus/upper stomach, and in the lower rectum/anus area. In each area, varicose veins (widely dilated veins) can develop and are prone to hemorrhage. The esophageal varices are particularly apt to bleed and are associated with a significant degree of mortality (Fig. 10.24 and Disc Image 10.47). Patients dying of bleeding esophageal varices tend to present with massive bleeding, frequently with marked “hematemesis” (vomiting blood).



Fig. 10.23 A cross-section of a liver demonstrating cirrhosis. Note the extreme nodularity

Fig. 10.24 Bleeding esophageal varices (dilated blue-appearing blood vessels underneath the mucosa), related to underlying cirrhosis and portal vein hypertension



Another frequent finding with cirrhosis and portal hypertension is the presence of “ascites” fluid, which is abundant, clear, yellow fluid within the peritoneal (abdominal) cavity. In and of itself, this fluid does not cause death. Occasionally, “spontaneous bacterial peritonitis” becomes superimposed, resulting in death. The lining of the peritoneal cavity is inflamed and may have adherent pus. Widespread (systemic) infection can rapidly ensue, with subsequent death. In cases of spontaneous bacterial peritonitis, no GI perforation can be identified.

Mallory–Weiss Tears

The junction between the esophagus and the stomach is at risk of tearing (lacerating) as a result of repeated retching, as occurs occasionally following a drinking binge. As such, this finding may be evident in alcoholics or other binge-drinkers. If underlying esophageal varices are present, death can occur rapidly.

Other GI Abnormalities

The acute inflammation of a portion of the GI tract, with subsequent perforation, rupture, bleeding, or peritonitis, can lead to death, as previously described with PUD. Other sites where this occurs include the appendix (acute appendicitis), a Meckel’s diverticulum (as described under “Congenital Anomalies”), strangulated hernias, infarcted bowel segments, telescoped (intussusception) or twisted (volvulus) bowel segments, and areas containing a tumor. A “hernia” occurs when

something protrudes into or through something that it should not. Examples of herniation sites involving segments of bowel (usually small intestine) include the inguinal regions (groin), the anterior abdominal wall, a defect within the mesentery, and intra-abdominal adhesions (which develop following surgery). A segment of herniated bowel can become entrapped (“incarcerated”) (Fig. 10.25). It can subsequently twist on itself, or otherwise swell to such an extent that its blood supply is cut off. When this occurs, the hernia is said to have “strangulated.” A strangulated hernia will eventually die (infarct), and perforation, inflammation, hemorrhage, or sepsis, with possible death, can occur. Segments of bowel can undergo ischemic infarction, with similar results. In some persons, the bowel actually telescopes on itself, which can lead to compression of blood vessels with infarction, perforation, etc. This process is referred to as “intussusception.” When a segment of bowel twists on itself, or its mesentery, with eventual infarction, the condition is referred to as a “volvulus.” Intussusception and volvulus may occur in association with a tumor, which acts as a mass that predisposes to the conditions. Alternatively, each of these conditions can occur in the absence of a tumor in children.

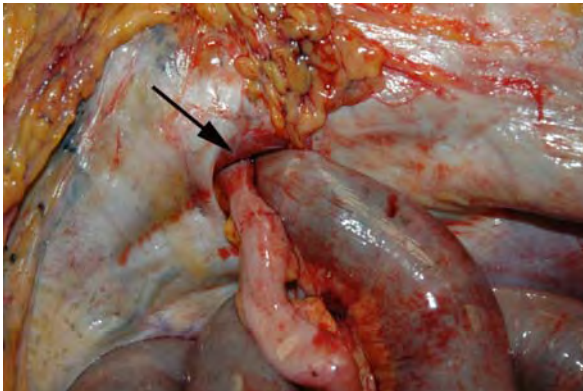


Fig. 10.25 A strangulated hernia case. Note that a portion of small intestine has herniated through a small defect in the abdominal wall and that it cannot be removed. The arrow indicates where the intestine enters the defect in the abdominal wall

Hemochromatosis

Hemochromatosis refers to excess levels of iron in the body. It can result from a genetic abnormality (primary or hereditary hemochromatosis) or from various iron-overload conditions, including multiple blood transfusions, various blood/bone marrow disorders, increased oral intake of iron, and chronic liver diseases. Humans are at risk for the disorder because they have no normal way in which to get rid of excess iron. The genetic condition is an autosomal recessive disorder (see below), caused by a mutation of the HFE gene, which normally regulates intestinal absorption of iron. In primary hemochromatosis, too much iron is absorbed. The

major manifestations of hemochromatosis include cirrhosis, diabetes mellitus, and skin pigmentation, each resulting from excess iron deposited in tissues. Under the microscope, iron pigment is readily identified using special iron stains.

Pancreatitis

Pancreatitis refers to inflammation of the pancreas. A variety of conditions can induce pancreatitis, including chronic alcoholism, various toxins, severe bodily trauma, and elevated blood fat levels. The most commonly encountered situation involves alcohol. Chronic pancreatitis is a relatively frequent finding in chronic alcoholics and is characterized by fibrosis (scar tissue). Acute pancreatitis is a life-threatening condition. In deaths resulting from acute pancreatitis, pathologists will be able to identify microscopic evidence of acute inflammation (neutrophils) within and around the pancreas, often in association with “fat necrosis,” as well as hemorrhage (Disc Image 10.48). Grossly, the pancreas may be hemorrhagic as well (Fig. 10.26). Postmortem blood levels of “amylase” and “lipase” may be elevated.



Fig. 10.26 The gross appearance of hemorrhagic pancreatitis at autopsy

Reticuloendothelial and Immune Systems

Recall that the reticuloendothelial system encompasses the bone marrow, the spleen, the lymph nodes, and the thymus, among other structures. As such, it is involved with the immune system, as well as in blood production (bone marrow). The immune system is primarily involved in the recognition and elimination of potentially harmful influences, including micro-organisms, foreign substances, and even

malignant cells. A variety of immune disorders, such as hypersensitivity reactions, autoimmune diseases or immunodeficiencies, may play a role in death; however, many of them have nonspecific morphologic findings within the organs of the reticuloendothelial system. They may or may not have more specific findings within other organ systems. A hypersensitive immune system is thought to play a role in a variety of diseases, including autoimmune disorders (see below). A specialized subtype of hypersensitivity is referred to as “anaphylaxis” or an “allergic reaction.” This is described more completely in Chapter 21 (Miscellaneous Topics), since it frequently involves an exogenous factor.

Autoimmune Disorders

There are a number of autoimmune disorders that may cause or contribute to death. Some of the systemic disorders are also referred to as “collagen vascular diseases” or “connective tissue diseases.” Autoimmune disorders are characterized by tissue damage resulting from the body’s immune system reacting against the body’s own tissues. In some cases, the injury is very isolated (autoimmune thyroid disease). In other cases, many organs and tissues are affected (systemic lupus erythematosus). In most cases, laboratory tests can be performed that help to make the diagnosis. Many of these involve the detection of specific “autoantibodies,” antibodies that are able to bind to specific structures. Such testing can be reliable with postmortem samples. Examples of systemic autoimmune disorders include systemic lupus erythematosus, Sjögren syndrome, scleroderma, rheumatoid arthritis, and several types of vasculitis, including polyarteritis nodosa. Examples of localized autoimmune disorders include Hashimoto thyroiditis, autoimmune hemolytic anemia, pernicious anemia, multiple sclerosis, Goodpasture syndrome, myasthenia gravis, Graves disease, and insulin-dependent diabetes mellitus.

Leukemia/Lymphoma

Leukemia represents a malignancy of the bone marrow cells that normally produce blood cells (red blood cells, white blood cells, platelets). Leukemias are typically characterized by markedly elevated white blood cell counts in the blood, because the malignant bone marrow cells “spill out” into the blood. The cells that account for the elevated WBC count are actually the malignant cells. Since leukemias tend to affect the blood, they are sometimes considered “fluid” malignancies. Problems arise when the malignant cells crowd out normal cells, such that anemia and infection ensue. Acute leukemias, which frequently occur in young persons, tend to have a very rapid, stormy, clinical presentation, whereas chronic leukemias, which occur mostly in adults, tend to have a more prolonged course, although chronic leukemias can convert into acute leukemias.

Lymphomas can be considered “solid” malignancies of white blood cells. They tend to involve lymph nodes, but can occur in other organs of the reticuloendothelial

system (spleen, thymus) and virtually anywhere else. There are two basic categories: Hodgkin disease and non-Hodgkin lymphoma, each with several subtypes. With each type, immune dysfunction can occur.

Many persons die from leukemia or lymphoma. Most die from the secondary effects of the disease (compromised immune system, infection). Although relatively uncommon, rare cases of sudden death are attributable to a previously undiagnosed leukemia or lymphoma.

Immunodeficiency

When the immune system is compromised, an individual is said to be “immunodeficient” or “immunocompromised.” If severe enough, immunodeficiency can result in death, usually as a result of overwhelming infection, although occasional deaths result from malignancy. Such compromise can occur as a primary, genetic disorder, or secondary to some other process. The “primary immunodeficiency” syndromes are typically genetically-based and include X-linked agammaglobulinemia of Bruton, common variable immunodeficiency, IgA deficiency, hyper-IgM syndrome, DiGeorge syndrome (thymic hypoplasia), severe combined immunodeficiency, Wiskott–Aldrich syndrome, and complement deficiency syndromes. Each is characterized by an increased incidence of infection, sometimes specific types or locations. Some have other specific characteristics as well. Some are very severe, resulting in early death, others are relatively minor and may not be readily recognized. Making a diagnosis of a primary immunodeficiency at autopsy is uncommon, but not impossible.

Secondary immunodeficiency results from non-genetic factors that destroy or otherwise compromise the normal function of the immune system. Three common causes include infection with the human immunodeficiency virus (HIV), malignancies of the reticuloendothelial system (leukemias and lymphomas), and administration of therapeutic drugs that have immunosuppressant effects (steroids, various anti-cancer drugs).

The acquired immunodeficiency syndrome (AIDS) develops in many individuals who have contracted HIV infection, often several years after initial infection. AIDS is characterized by profound immunosuppression, with infection, neoplasia, and neurologic symptoms. HIV is transmissible via blood and body fluids, such that a vast majority of cases are either sexually transmitted or related to exposure to a contaminated needle in the setting of intravenous drug abuse. It is beyond the scope of this text to provide sufficient details to describe AIDS in full; however, the reader should be aware of certain characteristics of the disease. As AIDS becomes manifest, the infected person’s WBC counts decline, specifically with a loss of a subtype of lymphocytes referred to as CD4⁺ T lymphocytes. These cells are normally extremely important in normal immune function. As their numbers decrease, the patient becomes susceptible to numerous “opportunistic” infections and neoplasms. Some of the more classic opportunistic conditions include pneumocystis pneumonia, toxoplasmosis CNS infection, candidiasis, cryptococcosis CNS

infection, disseminated (systemic) histoplasmosis or coccidioidomycosis, tuberculosis, cytomegalovirus (CMV), herpes simplex virus, Kaposi sarcoma, and various lymphomas.

Special Note: HIV infection represents a potential job-related hazard for anyone working with dead bodies. The virus is known to survive for some period of time following death. Universal precautions should be used whenever the possibility of blood or other bodily fluid/tissue exposure is present. This is particularly true during autopsy performance. If an exposure occurs (needle puncture, scalpel cut, splashing of fluids into the eyes), the exposed individual should immediately wash the puncture or cut with warm, soapy water, or flush the eyes liberally with water at an eye wash station, and then contact their supervisor. HIV testing can occur very quickly on samples taken from the decedent. Prophylactic anti-retrovirus medication should be implemented soon after exposure.

Endocrine System

The endocrine system consists of the pituitary gland at the base of the brain, the thyroid and parathyroid glands in the anterior neck, the adrenal glands just above the kidneys, and the endocrine portions of the pancreas (the islets of Langerhans). The gonads (testes in males, ovaries in females) can also be considered part of the endocrine system. One of the most serious and deadly disorders of the endocrine system, diabetes mellitus, has wide-reaching systemic effects, so it is presented below in the section entitled “Multisystem Disorders.” A few of the other endocrine conditions will be presented here.

The pituitary is the master gland, producing or transmitting many hormones that affect other endocrine glands. Examples of hormones produced or released include adrenocorticotropic hormone (ACTH), which stimulates cortisol production within the adrenal cortex, thyroid stimulating hormone (TSH), which stimulates thyroid hormone production, human growth hormone (hGH), which functions in normal growth, prolactin, which affects breast function, and luteinizing hormone (LH) and follicle stimulating hormone (FSH), each of which acts on the sex glands (testes, ovaries). Pituitary disorders can include hyperpituitarism and hypopituitarism, in which there is, respectively, too much of one pituitary hormone produced, usually as a result of a hormone-producing benign tumor or “adenoma,” or under-production of most or all of the hormones, again usually as the result of gland compression by a non-functioning adenoma, or as a result of inflammation. With overproduction or underproduction, symptoms occur throughout the endocrine system, since the pituitary gland is the “master gland.” Occasionally, pituitary adenomas become so large that the mass effect at the base of the brain causes sudden death in a fashion similar to a brain tumor.

Thyroid disorders include hyperthyroidism, hypothyroidism, and enlargement. An overactive thyroid (hyperthyroidism) is characterized by increased energy and metabolism. An underactive thyroid (hypothyroidism) is characterized by decreased energy and metabolism. Various forms of inflammation, some of which

are autoimmune, can lead to either hypo- or hyperfunction. Pituitary tumors can elaborate TSH, resulting in hyperthyroidism. Enlargement of the thyroid can be non-neoplastic hyperplasia (goiter) or neoplastic, with the neoplasms being benign or malignant.

Parathyroid disorders include increased function (hyperparathyroidism), decreased function (hypoparathyroidism), and enlargement, from either neoplasia (benign or malignant) or hyperplasia. Since parathyroid hormone is important in calcium and phosphate metabolism and bone metabolism, parathyroid abnormalities tend to result in abnormalities in these areas.

The adrenal glands are important in a variety of physiologic processes, including sodium and potassium metabolism, cortisol (steroid) production, sex hormone production, and the production of adrenalin (epinephrine) and similar substances. Clinical disorders involving the adrenal cortex include hyperaldosteronism, resulting in hypertension (high blood pressure) and hypokalemia (low potassium), hypercortisolism (Cushing syndrome), characterized by hypertension, weight gain, and a peculiar appearance (trunk obesity, moon face, fatty “buffalo” hump in posterior neck/upper back), and the so-called “adrenogenital” syndromes, evident as disorders of sexual differentiation. Some cases are related to pituitary tumors, which produce hormones that stimulate the production of adrenal hormones. Some are related to primary adrenal disorders, including cortical adenomas (benign tumors), cortical carcinomas (cancer), or cortical hyperplasia. Adrenal insufficiency (Addison disease) specifically refers to cortex under-function and occurs in a variety of settings, including lack of ACTH production within the pituitary gland, exogenous administration of glucocorticoids, massive hemorrhagic necrosis of the glands (“Waterhouse–Friderichsen syndrome,” often in association with a systemic *Neisseria meningitidis* infection), and other causes of inflammation, including autoimmune adrenalitis. Some autoimmune cases are associated with autoimmune destruction of other endocrine organs/glands, as occurs in “autoimmune polyendocrine syndromes.” The most significant pathologic process involving the adrenal medulla is neoplasia. Neuroblastomas in children and pheochromocytomas in adults can cause significant morbidity (disease) and mortality (death). Pheochromocytomas can produce severe hypertension.

As mentioned above, the endocrine portions of the pancreas are involved in diabetes mellitus (see below). In addition, islet cell tumors can occur and produce hormones that have major clinical manifestations. In those in which insulin is produced (insulinomas), death can result from associated severe hypoglycemia (reduced blood sugar). Unfortunately, a diagnosis of hypoglycemia cannot be made via postmortem testing (see Chapter 21).

Genitourinary System

The genitourinary system includes the kidneys, ureters, bladder, and urethra, as well as the various sexual organs of males and females. A variety of morphologic changes can occur in the kidneys in certain common systemic diseases, including

hypertension and diabetes mellitus. In hypertension, gross kidney changes include a granular surface (Disc Image 10.49), and microscopic changes are common in the blood vessels within the kidneys. Clinical renal (kidney) failure is a common occurrence in severely ill patients who are hospitalized. Kidney infections (“pyelonephritis”) can lead to sepsis and death. Kidney malignancies (Wilms tumor in children; renal cell carcinoma in adults) can cause death.

The sexual organs and structures of both men (especially the testes and prostate gland) and women (especially the uterus, including cervix, and ovaries) may develop malignant tumors that can lead to death. In addition, the prostate gland frequently becomes enlarged via hyperplasia in older men. As the gland enlarges, the veins around it can become dilated. In rare circumstances, venous thrombosis can develop, with subsequent pulmonary thromboembolism.

Pregnancy is a normal biological process that carries with it an increased risk of death, related to a variety of factors. Pregnancy-related maternal deaths are discussed in further detail in Chapter 21.

Bones, Joints, and Soft Tissues

There are numerous bone, joint, and soft tissue diseases. Many of these are diagnosed during life, with only rare cases being diagnosed after death. Some are potentially life-threatening; cancers are one of the most serious. Primary bone cancers tend to affect children and young adults. Infection of a bone, which is particularly difficult to treat, is referred to as “osteomyelitis.” Osteoporosis (loss of bone mass related to aging) is quite debilitating and frequently plays a role in deaths in the elderly, when death is preceded by an accidental fall with associated hip fracture.

Inflammatory joint disorders (arthritis) are quite common and can be terribly disabling. Examples include osteoarthritis (a wear and tear disorder), rheumatoid arthritis (an autoimmune disorder), and gout (related to uric acid crystal deposition).

Primary soft tissue cancers are referred to as “sarcomas.” A variety of genetic muscular dystrophies can occur, the most common and debilitating of which is Duchenne muscular dystrophy, in which skeletal muscles are abnormal, eventually resulting in atrophy and death.

Multisystem and Other Disorders

Many disorders that can cause or contribute to death are not isolated to a single organ system. Some have been presented previously within specific organ system sections above. Others are presented here. Others, specifically nutrition-related disorders, will be described in Chapter 21.

Amyloidosis

Amyloidosis actually represents a group of disorders, each characterized by the deposition of a protein substance referred to as “amyloid” within tissues or organs. Amyloid has a specific structural composition at the molecular level, such that it takes on very specific characteristics when visualized with a special stain called “Congo Red” and a specialized form of light called “polarized light.” This classic appearance is described as “apple green birefringence.” Several different proteins have the specific structural conformation that defines them as amyloid. A number of disorders, some of which can cause or contribute to death, are characterized by the presence of amyloid within tissues, including “multiple myeloma,” a malignant tumor of plasma cells (cells that produce antibodies), certain chronic inflammatory disorders, and senile systemic amyloidosis. Alzheimer disease is also characterized by the presence of amyloid within blood vessel walls and within other lesions within the brain.

Chronic Alcoholism

Chronic alcoholism is one of the most common chronic disorders encountered within forensic autopsy practice. Chronic alcoholism has wide-ranging effects on numerous organ systems, and several can result in death. Many of these have already been presented, but an attempt will be made here to summarize them.

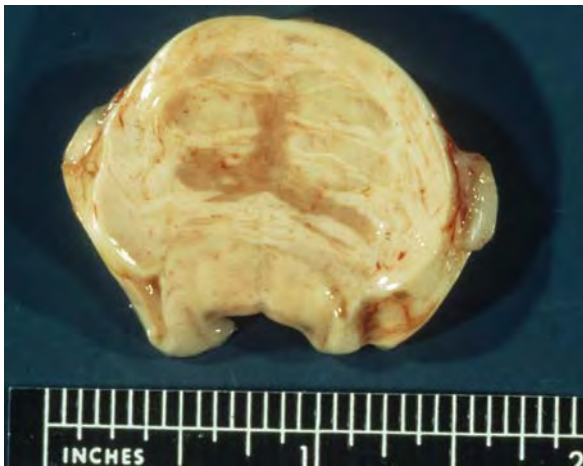


Fig. 10.27 A cross-section of the pons at autopsy, showing an area of central discoloration that represents a condition referred to as “central pontine myelinolysis” (CPM). This can occur in association with electrolyte imbalances in chronic alcoholics and represent the mechanism of death

Within the brain, chronic alcoholics can demonstrate central pontine myelinolysis (CPM) (Fig. 10.27), cerebellar vermis atrophy (Disc Image 10.50), and mamillary

body atrophy/necrosis (Wernicke–Korsakoff syndrome in association with thiamine deficiency) (Disc Image 10.51). CPM may be associated with a low-salt vitreous electrolyte pattern. It is not infrequent to identify remote (old) brain contusions in alcoholics.

Ethanol-induced dilated cardiomyopathy is a frequent finding in chronic alcoholics. The flabby heart is at risk for fatal arrhythmias. Pulmonary infections, including pneumonia, aspiration pneumonia, and abscesses, are relatively common in alcoholics. Ethanol-related liver disease includes cirrhosis, portal hypertension, and esophageal varices (described above), as well as steatosis (fatty change within the liver) (Disc Images 10.52 and 10.53) and alcoholic hepatitis. Gastritis and peptic ulcer disease are more common in alcoholics. Spontaneous bacterial peritonitis is most common in chronic alcoholics.

Diabetes Mellitus

Diabetes mellitus (DM) actually represents several distinct disorders, each characterized by systemic pathology related to abnormalities in glucose (“blood sugar”) metabolism. The two major types of DM are type I (insulin-dependent DM or juvenile DM) and type II (non-insulin dependent DM or adult-onset DM). There are also several less common forms, including “maturity-onset diabetes of the young” (MODY), caused by a variety of genetic mutations, disorders associated with insulin gene or receptor mutations, mitochondrial diabetes, and several different situations which result in diabetes secondary to islet destruction or some other disturbance of normal insulin/glucose metabolism. Included within this latter category are inflammatory conditions, neoplastic conditions, cystic fibrosis, hemochromatosis, various endocrinopathies, infections, and drug effects. Gestational DM occurs in association with pregnancy.

The major pathologic disturbances that occur in all types of DM include damage to the kidneys, eyes, nerves, and blood vessels. Since nerves and blood vessels are present throughout the body, it should be evident that the manifestations of DM tend to be systemic. The damage occurs as a result of elevated levels of blood glucose, the major energy source for cells. Insulin normally acts to allow glucose to enter cells. Only kidney, eye, nerve, and blood vessel cells do not require insulin for glucose to enter. When there is an elevated blood glucose level, the glucose readily enters kidney, eye, nerve, and blood vessel cells, resulting in cellular damage.

Type I DM, accounting for about 10% of DM cases, is characterized by beta-cell destruction within the pancreatic islets. Beta-cells are the cells responsible for producing insulin. Hence, persons with type I DM have insufficient (low) insulin levels. With a deficiency of insulin, all the cells that require insulin to receive adequate glucose are starved of glucose. The body responds by increasing the amount of glucose available within the blood, thus elevating the “blood sugar.” The glucose readily enters the kidney, eye, nerve and blood vessel cells, causing damage. Therapy for type I DM includes insulin replacement, usually via subcutaneous injection. Persons with type I DM are particularly at risk for developing “ketoacidosis,”

with severe cases possibly leading to death. Vitreous electrolyte evaluation in such cases will typically reveal a “diabetic pattern” (see Chapter 21), with glucose levels >200 mg/dL and the presence of acetone.

Type II DM, accounting for 80–90% of DM cases, is caused by cells’ resistance to insulin and an inadequate release of insulin from beta-cells. As a result, the body responds by producing more glucose, with damage to kidney, eye, nerve, and blood vessel cells. Type II DM is strongly related to obesity. Treatment includes dietary and nutritional education and manipulation, as well as medication (hypoglycemic drugs).

Classic autopsy findings in patients with long-standing DM include “diabetic nephropathy” (kidney changes), with many possible findings, such as grossly-evident granularity of the kidney surfaces and microscopic nodular glomerulosclerosis (the “Kimmelstiel–Wilson” lesion), and vascular changes, including atherosclerosis as well as arteriosclerosis (thickened walls of small arterioles). Usually, the eye and nerve changes are not evaluated in routine forensic autopsies. Diabetics are at risk for atherosclerotic cardiovascular disease-related death. In fact, from a clinical standpoint, diabetics may experience a “silent” myocardial infarct, with no recognizable symptoms. Diabetics are also more likely to experience “subendocardial” myocardial infarcts (infarcts involving the inner aspects of the myocardium) compared to the non-diabetic population, but they also can experience the more typical “transmural” (full thickness) infarcts. Diabetic patients are at risk for nasal infections (with fungi called “mucormycosis”) as well as urinary tract infections. In deaths related to ketoacidosis, the “Armanni–Ebstein” change can be seen as microscopic clearing of the cytoplasm of proximal convoluted tubule cells.

Persons on long-term insulin replacement therapy will frequently develop anti-insulin antibodies, which can be measured in postmortem blood samples. When present, much of the insulin that is present in the blood is actually bound to these antibodies, and therefore not available to function at the cellular level. Consequently, it is not unusual for these persons to have elevated “total insulin” levels. Their “free insulin” levels should be within normal limits. In endogenous insulin (that produced by the beta-cells of the pancreas), a substance called “C-peptide” is produced along with insulin. C-peptide levels can also be measured. If there is a very low C-peptide level in the presence of a normal or high insulin level, this indicates that the insulin is exogenous (injected). In deaths involving insulin-dependent DM patients, questions sometimes arise regarding whether the individual may have purposefully taken an insulin overdose, or whether a non-diabetic patient was injected with insulin. These questions are addressed in Chapter 11 (Drug and Toxin-Related Deaths).

Sickle Cell Disease

Sickle cell disease is a type of disorder referred to as a “hemoglobinopathy” (a disease characterized by the production of abnormal hemoglobin). More specifically, sickle cell disease is an autosomal recessive genetic disorder involving the gene that codes for the beta-globin chain that is part of the hemoglobin A (HbA) molecule.

Hemoglobin is the molecule contained within red blood cells (rbc) that is responsible for binding to oxygen molecules as the rbc transport oxygen from the lungs to the tissues of the body. In sickle cell disease, a single mutation in the beta-globin gene (present on both beta-globin genes) results in an abnormal beta-globin protein, such that an abnormal hemoglobin molecule results. This abnormal hemoglobin molecule is referred to as hemoglobin S (HbS). In situations of low oxygenation or dehydration, the HbS molecules can aggregate and polymerize, resulting in a characteristic sickle shape of the red blood cells. Sickle cells do not function normally, and they can actually cause blockage within small blood vessels. The cells break apart easily, resulting in anemia and an increased amount of breakdown products (including bilirubin). Clinically, patients with sickle cell disease have problems related to anemia, excess bilirubin, and occlusion of the microvasculature, characterized by pain, or “vaso-occlusive” crises. They are at increased risk for several types of infection.

Persons who have only one defective gene and one normal gene are said to have “sickle cell trait.” In other words, they are “carriers.” Although usually asymptomatic, persons with sickle cell trait can occasionally experience difficulties and even death. The usual circumstance is one in which the person, who has previously not been very physically active, is suddenly very physically active and dehydrated. In this environment, rbc can actually sickle, just as in patients with sickle cell disease.

At autopsy of patients who die from a sickle crisis, whether they have sickle cell disease or sickle cell trait, sickled rbc can usually be identified under the microscope. Although the sickling of rbc can occur as an artifactual finding related to formalin fixation, the sites of sickling in this setting are typically widespread, including tissues that are not normally congested at autopsy. In cases where survival was prolonged, ischemic tissue can be identified. In patients with full-blown sickle cell disease, evidence of previous sickle crises is common, including the presence of a small, infarcted spleen.

In persons suspected of having sickle cell disease or trait at autopsy, without previous diagnosis, pathologists can send blood samples for a hemoglobin electrophoresis test, which will allow for a postmortem diagnosis.

Inborn Errors of Metabolism

Certain genetic disorders are classified as “inborn errors of metabolism.” These disorders are typically characterized by the lack or deficiency of an enzyme that is necessary for some normal metabolic process. When the enzyme is lacking or deficient, the substance that is normally broken down by the enzyme (called the “substrate”) accumulates within the cell and causes cellular damage. Examples of such diseases (with the deficient enzyme and major organ affected in parenthesis) include Tay–Sachs disease (hexosaminidase-alpha; CNS and retina), Pompe disease (α -1,4-glucosidase; heart), Niemann–Pick disease (sphingomyelinase; CNS), phenylketonuria or PKU (phenylalanine hydroxylase;

CNS), galactosemia (galactose-1-phosphate uridyl transferase; CNS and liver), and Gaucher disease (glucocerebrosidase; CNS). Some of these have classic microscopic features.

Other Genetic Disorders

Many of the diseases presented in this chapter are genetically-based. Some are “multifactorial,” having genetic and environmental components. In this short section, several genetic disorders that have not yet been addressed will be briefly reviewed. In general, genetic disorders can be divided into chromosomal disorders, where there are losses or gains of whole chromosomes or portions of chromosomes, gene mutations, where a single gene is abnormal, and miscellaneous disorders. In most disorders involving gene mutations, a disorder is said to be “autosomal dominant” if only one copy of the defective gene is required for the disease to be present, whereas a disorder is said to be “autosomal recessive” if two copies of the defective gene are required (one on each of a pair of chromosomes). If a person only has a single copy of an autosomal recessive mutated gene, then that person is said to be a “carrier” of that disorder/gene. In many genetic disorders, a postmortem diagnosis can be made, as long as the correct samples are collected at autopsy. For some (Ehlers–Danlos syndrome, Marfan syndrome), the collection and subsequent tissue culture growth of fibroblasts may be necessary.

Chromosomal Disorders: Included here are the trisomy syndromes (where there is an extra chromosome), the deletion syndromes (where there is a portion of a chromosome missing) and the sex chromosome abnormalities (chromosomal abnormalities involving the X and/or Y chromosomes). Down syndrome (trisomy 21) patients have an extra chromosome #21, and have a characteristic facial appearance, decreased mental capabilities, and an increased risk of congenital heart defects and leukemia. They can live relatively lengthy lives. Edwards syndrome (trisomy 18) is characterized by early death, severe mental retardation, a small jaw, overlapping fingers, congenital heart defects, kidney malformations, and “rocker-bottom” feet. Patau syndrome (trisomy 13) is characterized by early death, severe mental retardation, a small head, cleft lip/palate, polydactyly (extra digits), congenital heart and kidney defects, and rocker-bottom feet. The most commonly-described deletion syndrome is cri-du-chat syndrome (where part of chromosome 5 is absent), characterized by severe mental retardation, a small head with a round face, and a peculiar cat-like cry. Sex-chromosome disorders include Klinefelter syndrome (males with extra X chromosomes) and Turner syndrome (females with a single X chromosome).

Cystic Fibrosis: Cystic fibrosis (CF) is a relatively common disorder, particularly amongst Caucasians. The underlying defective gene codes for a protein involved in chloride transportation across epithelial cells, known as “cystic fibrosis transmembrane conductance regulator” or CFTR. The severity of the disorder is quite variable, since more than 500 different mutations have been identified within the gene, each

with its own degree of severity. Clinically, patients with CF have abnormally viscous (thick) secretions. The lungs, pancreas, small intestines, and salivary glands can become obstructed, damaged, and infected, leading to death. Sweat glands are also affected, leading to the commonly-employed “sweat chloride test” (in living patients).

Ehlers–Danlos Syndrome: Ehlers–Danlos syndrome is a group of genetic disorders, each with an abnormality in genes responsible for normal collagen formation. The most serious type is type IV, in which arteries and organs (intestines, spleen, uterus) are at risk for rupture. These patients may or may not have a history of easy bruising. They may or may not have hyperflexible skin and hypermobile joints (double-jointed).

Marfan Syndrome: Marfan syndrome is a genetic disorder characterized by abnormalities in the gene for a connective tissue substance called fibrillin-1. Clinically, patients tend to be very tall and lanky, with long fingers. Abnormalities of the skeleton, eyes, and cardiovascular system are common. Most significant in terms of death investigations are a predisposition to thoracic aortic dissections and mitral valve prolapse.

Sarcoidosis

Sarcoidosis is a disorder characterized by the presence of multiple “granulomas” (inflammatory nodules containing lymphocytes and macrophages). The cause of the disease is unknown. No micro-organism or foreign substance can be identified in the granulomas. The disease can affect anyone, but women and those of African descent are at greatest risk. The granulomas can become quite large, and tend to involve the lymph nodes, particularly those within the mediastinum (central thorax area), where the enlarged nodes are sometimes referred to as “potato nodes” (Disc Image 10.54). The lungs are also frequently involved, sometimes quite extensively. Death can be related to severe respiratory compromise, but more frequently is related to the fact that the heart can also be involved. Virtually any other organ can also be involved with the disease.

Psychiatric Disease

A variety of psychiatric conditions may be associated with sudden, unexpected death, including various eating disorders (see Chapter 21), and numerous disorders characterized by psychotic behavior, such as schizophrenia or manic-depression. Some persons are at risk for various non-natural events, such as suicide or adverse drug reactions. Some individuals may experience excited delirium followed by sudden death. The excited delirium is essentially identical to that seen in drug-induced excited delirium, such as occurs with cocaine (refer to Chapter 11). A ruling on such a case might read as follows: “excited delirium due to schizophrenia.” Occasionally, the condition is referred to as “acute exhaustive mania.”

Disc Image Legends

- Disc Image 10.1 A bicuspid aortic valve, having only two cusps instead of three.
- Disc Image 10.2 The microscopic appearance of coronary artery atherosclerosis.
- Disc Image 10.3 A cross-section of a heart with an acute myocardial infarct characterized by subtle discoloration (a so-called “mottled” appearance).
- Disc Image 10.4 An acute myocardial infarct that is several days old. The dead (infarcted) muscle has a yellow color and is surrounded by dark red bloody tissue.
- Disc Image 10.5 The microscopic appearance of an acute myocardial infarct, characterized by necrosis and early inflammation with neutrophils.
- Disc Image 10.6 A heart with a ruptured myocardial infarct.
- Disc Image 10.7 A cross-section of a heart with a ruptured myocardial infarct.
- Disc Image 10.8 A coronary artery containing a metallic stent.
- Disc Image 10.9 A remote myocardial infarct (thinned wall of heart) with a mural thrombus which has a yellow, gelatinous appearance.
- Disc Image 10.10 A formalin-fixed heart that has had previous coronary artery bypass graft (CABG) surgery. The segments of vein grafts have one end attached to the aorta and the other attached to coronary arteries beyond the sites of atherosclerotic narrowing. A metal probe underlies three vein grafts.
- Disc Image 10.11 A view of the internal lining of the aorta, showing the attachment sites of two vein grafts, with visible sutures. Note at the bottom of the photograph a portion of the aortic valve. Also seen is a native coronary artery ostium (opening).
- Disc Image 10.12 A coronary artery dissection, in which blood has “dissected” its way through the artery wall.
- Disc Image 10.13 Intramyocardial “tunneling” or “bridging” of a coronary artery (white arrow). Note the presence of another coronary artery branch in the normal epicardial fat position (black arrow).
- Disc Image 10.14 A hemopericardium as seen prior to opening the heart sac (pericardium) at autopsy. Note the subtle blue discoloration of blood as seen through the pericardial lining.
- Disc Image 10.15 The hemopericardium of Disc Image 10.14 visualized after opening the pericardial sac.
- Disc Image 10.16 Evidence of aortic hemorrhage arising from an aortic dissection in the case depicted in Disc Images 10.14 and 10.15.
- Disc Image 10.17 Another thoracic aortic dissection.
- Disc Image 10.18 An aortic dissection which extends to involve the abdominal aorta.
- Disc Image 10.19 A repaired aorta with synthetic graft material in place.
- Disc Image 10.20 A right hemothorax (blood within the right pleural cavity) secondary to a ruptured thoracic aortic dissection. The head of the body is to the left, while the feet are to the right. The right lung is being held forward/upward, in order to view the hemothorax, as well as the defect (arrow) within the medial pleural lining, where the blood ruptured through pleura, into the chest cavity.

- Disc Image 10.21 The microscopic appearance of fibromuscular dysplasia, characterized by abnormal thickening of the walls of a small artery near the AV node of the heart.
- Disc Image 10.22 The microscopic appearance of myocarditis, characterized by heart inflammation with lymphocytes (the small blue cells scattered amongst the heart cells).
- Disc Image 10.23 The microscopic appearance of hypertrophic (enlarged) myocytes (heart cells) in a case of hypertension (high blood pressure).
- Disc Image 10.24 The appearance of degenerative calcific aortic stenosis. The valve is extremely rigid and “non-pliable.”
- Disc Image 10.25 Bacterial endocarditis of the aortic valve in a chronic intravenous drug abuser. Note the incidental presence of the aberrant origin of a coronary artery (two coronary artery openings adjacent to one another).
- Disc Image 10.26 A mechanical prosthetic heart valve as seen at autopsy.
- Disc Image 10.27 The microscopic appearance of enlarged myocytes that are haphazardly arranged within fibrous (scar) tissue in a case of hypertrophic cardiomyopathy.
- Disc Image 10.28 A case of “arrhythmogenic right ventricular cardiomyopathy” or “arrhythmogenic right ventricular dysplasia” (ARVD), characterized by a dilated right ventricular cavity and fat (and fibrous tissue) replacement of the wall of the right ventricle (arrows).
- Disc Image 10.29 A case with thrombosis discovered within the deep veins of the legs (arrows).
- Disc Image 10.30 Said to be the smallest tumor that can cause death, the AV nodal tumor is composed of individual tumor cells and nests of tumor cells within the heart muscle. When present within key components of the heart’s conduction system, as in the case shown, the tumor can cause a lethal arrhythmia (abnormal heart rhythm).
- Disc Image 10.31 A case of hemorrhagic meningitis.
- Disc Image 10.32 A cross-section of the brain showing a brain abscess.
- Disc Image 10.33 The microscopic appearance of viral encephalitis, characterized by a lymphocytic inflammatory infiltrate within the brain, especially surrounding blood vessels.
- Disc Image 10.34 A hemorrhagic brain infarct (a stroke).
- Disc Image 10.35 A remote infarct of the brain, characterized by loss of brain tissue.
- Disc Image 10.36 A cross-section of a brain demonstrating an arteriovenous malformation (AVM).
- Disc Image 10.37 The microscopic appearance of an AVM, composed of a mass of tangled arteries and veins.
- Disc Image 10.38 A brain with a large, hemorrhagic glioblastoma multiforme (GBM) (arrows), a malignant brain tumor, first discovered at autopsy.
- Disc Image 10.39 A case of lethal epiglottitis in which the swollen, infected structure (arrow) caused airway obstruction.

- Disc Image 10.40 Pulmonary thromboembolism. Note the occlusion (blockage) of the pulmonary artery by numerous, wormlike thrombi (bloodclots), indicated by the arrow.
- Disc Image 10.41 The microscopic appearance of pneumocystis pneumonia, in an AIDS patient.
- Disc Image 10.42 A cross-section of a lung at autopsy, showing “mucous plugging” within bronchi.
- Disc Image 10.43 The microscopic appearance of acute asthmatic bronchitis, including an intense mixed inflammatory cell infiltrate with numerous eosinophils, which are too small to readily appreciate in this relatively low-power magnification.
- Disc Image 10.44 The microscopic appearance of emphysema, characterized by enlarged alveolar spaces.
- Disc Image 10.45 A cross-section of a lung with cancer.
- Disc Image 10.46 The gross appearance of a gastric ulcer.
- Disc Image 10.47 The microscopic appearance of esophageal varices (dilated veins).
- Disc Image 10.48 The microscopic appearance of pancreatitis, characterized by acute inflammatory cells (neutrophils), mostly along the upper edge of the photo.
- Disc Image 10.49 The gross appearance of a hypertensive kidney, characterized by a granular (rough) surface.
- Disc Image 10.50 A midline section through the cerebellum of a chronic alcoholic, showing atrophy of the “vermis” (upper right portion of specimen).
- Disc Image 10.51 A cross-section of the brain showing small and darkened “mamillary bodies” (arrows) in a case of Wernicke–Korsakoff syndrome.
- Disc Image 10.52 A fatty liver at autopsy.
- Disc Image 10.53 The microscopic appearance of fatty change (the clear appearance of cells), also called “steatosis,” within the liver, occurring as a result of ethanol consumption.
- Disc Image 10.54 The gross appearance of enlarged lymph nodes (arrows) occurring in a case of sarcoidosis. Note that the lungs and heart have been previously removed from the body.

Selected References

- Batalis NI, Galup L, Zaki SR, Prahlow JA. West Nile virus encephalitis. *Am J Forensic Med Pathol* 2005;26:192–6.
- Byard RW, Cohle SD. *Sudden Death in Infancy, Childhood, and Adolescence*. Cambridge, England: Cambridge University Press; 1994.
- Byard RW, Manton N, Tsokos M. Sarcoidosis and mechanisms of unexpected death. *J Forensic Sci* 2008;53:460–4.
- Celbis O, Aydin NE, Mizrak B, Ozdemir B. Arrhythmogenic right ventricular dysplasia cases in forensic autopsies. *Am J Forensic Med Pathol* 2007;28:235–7.
- Christiansen LR, Collins KA. Natural death in the forensic setting – a study and approach to the autopsy. *Am J Forensic Med Pathol* 2007;28:20–3.

- Fineschi V, Baroldi G, Silver MD. *Pathology of the Heart and Sudden Death in Forensic Medicine*. Boca Raton, FL: Taylor & Francis; 2006.
- Gonsoulin M, Barnard JJ, Prahlow JA. Death resulting from ruptured cerebral artery aneurysm – 219 cases. *Am J Forensic Med Pathol* 2002;23:5–14.
- Gruszecki AC. Dural sinus thrombosis. *American Society for Clinical Pathology Forensic Pathology Check Sample* 2009;51:31–39.
- Gulino SP. Examination of the cardiac conduction system: forensic application in cases of sudden cardiac death. *Am J Forensic Med Pathol* 2003;24:227–38.
- Iion M, Kimura T, Abiru H, Kaszynski RH, Yuan QH, Tsuruyama T, Tamaki K. Unexpected sudden death resulting from anomalous origin of the right coronary artery from the left sinus of Valsalva: a case report involving identical twins. *Legal Med* 2007;9:25–9.
- Jing HL, Hu BJ. Sudden death caused by stricture of the sinus node artery. *Am J Forensic Med Pathol* 1997;18:360–2.
- Matschke J, Lockemann U, Schulz F. Intracranial arteriovenous malformations presenting as sudden unexpected death – a report of 3 cases and review of the literature. *Am J Forensic Med Pathol* 2007;28:173–6.
- Oeberst JL, Barnard JJ, Bigio EH, Prahlow JA. Neurocysticercosis. *Am J Forensic Med Pathol* 2002;23:31–5.
- Prahlow JA, Barnard JJ, Milewicz DM. Familial thoracic aortic aneurysms and dissections. *J Forensic Sci* 1998;43:1244–9.
- Prahlow JA, Wagner SA. Death due to Ehlers-Danlos Syndrome type IV. *Am J Forensic Med Pathol* 2005;78–82.
- Tsokos M, Braun C. Acute pancreatitis presenting as sudden, unexpected death – an autopsy-based study of 27 cases. *Am J Forensic Med Pathol* 2007;28:267–70.
- Wei JP, Kay D, Fishbein MC. Spontaneous dissection of the distal obtuse marginal coronary artery: a rare cause of sudden death. *Am J Forensic Med Pathol* 2008;29:199–201.
- Wirthwein DP, Spotswood SD, Barnard JJ, Prahlow JA. Death due to microvascular occlusion in sickle-cell trait following physical exertion. *J Forensic Sci* 2001;46:399–401.

Chapter 11

Drug-Related and Toxin-Related Deaths

Then the Lord sent venomous snakes among them; they bit the people and many Israelites died.

Numbers 21:6

Abstract Deaths related to drugs or toxins represent a sizeable percentage of cases investigated by forensic pathologists. Chapter 11 begins by discussing the investigation, autopsy findings, general toxicology issues, and death certification issues specifically related to drug/toxin deaths. The remainder of the chapter presents many common and some uncommon drugs and toxins which may result in death.

Keywords Drugs · Toxins · Toxicology · Abuse · Lethal drug levels

Introduction

Drug- and toxin-related deaths represent a sizeable percentage of cases investigated by death investigation offices. Frequently in these cases, there is no obvious visible indication of drug or toxin exposure, either on external autopsy examination or internal examination (grossly or microscopically). In a large number of such cases, there may also be no scene indicators that suggest drugs (this may or may not be due to the fact that family members or friends might have “cleaned-up” the scene). Since most drug-related deaths have no definitive external or internal findings, these deaths are included in the larger population of deaths that have “no anatomic cause of death.” Other case types that may be included in this population are drownings, certain electrocutions, temperature-related cases, seizure disorders, and deaths from cardiac conduction system disorders. Drug deaths may involve illegal (“street”) drugs, over-the-counter (OTC) preparations, or prescription medications. In order to appropriately identify deaths related to drugs or toxins, it is essential to perform forensic toxicology tests on postmortem blood (and/or other tissue) samples. For this reason, forensic pathologists and death investigators should be very familiar with the types of drugs that are routinely screened for by their forensic toxicology laboratory. Routine “drug screens” usually identify many drugs and toxins;

however, for many others, the toxicology laboratory must be asked to specifically attempt to identify *that* particular drug or toxin. In order to know which ones might be present, a thorough death and scene investigation must be performed. Slang or street names are very common for drugs of abuse. It is beyond the scope of this text to list each of these. The reader is referred to several websites to view such listings.

Investigation of Drug-Related Deaths

Death investigators must be able to recognize drugs and drug paraphernalia while at death scenes. Packaging materials, containers, syringes, homemade smoking devices, scales, prescription medications, and other items should arouse suspicion of a drug-related death (Figs. 11.1, 11.2, 11.3, and 11.4 and Disc Images 11.1 and 11.2). Over-the-counter (OTC) and prescription medications should not be ignored. Collecting and documenting all OTC and prescription medicines, and counting all pills present in each container, is an essential part of a death investigator's job. Interviewing survivors (family, friends) can often reveal a history of drug use by the decedent. It should be noted that, in certain circumstances, family members and friends may attempt to clean up the scene or hide drugs and drug paraphernalia prior to police, emergency medical personnel, or death investigator arrival at the scene.

Another scenario involves the “dumping” of a drug death body at a site distant from the place of death. If a drug abuser dies at a location that is being used by other drug users, it is not uncommon for some of the living members of the group to transport the body to a separate, isolated location. This is presumably done so as to not disrupt the drug-use environment. Sometimes, the helpful body transporters



Fig. 11.1 Drug-related death scene. In many cases, such as the one shown, it is obvious that drugs were involved in the death. Note the IV (intravenous) tubing and drug paraphernalia



Fig. 11.2 When investigating any death scene, it is important to note the presence or absence of drug paraphernalia



Fig. 11.3 Identification of ethanol containers provides a relatively obvious clue that ethanol intoxication may have played a factor in death

leave obvious signs for those who discover the body, such as a syringe and needle, or other drug paraphernalia (Figs. 11.5 and 11.6).

If a victim is transported to the emergency department prior to being pronounced dead, and if there is survival in the hospital prior to death, it is important for death investigators to obtain from the hospital laboratory any or all blood (and other tissue) samples collected from the decedent. This collection must occur immediately upon notification of death, since many laboratories discard samples after only a few days.



Fig. 11.4 Death investigators should inventory all medicines present at the death scene



Fig. 11.5 A “dumped body,” found in an abandoned warehouse



Fig. 11.6 A closer view of the dumped body shown in Fig. 11.5. Note the spoon next to the arm and the syringe in the hand

Autopsy Findings in Drug-Related Deaths

Despite the fact that there are usually no overt external (or internal) autopsy findings in drug/toxin deaths, there are some findings that may represent clues that a drug death has occurred. Occasionally, there are obvious findings, some of which will be described here. Others will be highlighted when specific drugs and toxins are described later in this chapter.

Findings on external examination that suggest the possibility of a drug or toxin-related death include drug paraphernalia on the body or within the clothing (Disc Image 11.3), fresh needle puncture marks (Fig. 11.7 and Disc Image 11.4), needle tracks (scars from intravenous drug administration) (Fig. 11.8 and Disc Image 11.5), skin-popping scars (from injecting drugs subcutaneously) (Disc Image 11.6), a perforated nasal septum (from snorting drugs, such as cocaine), stained fingers (from



Fig. 11.7 Two fresh needle puncture marks



Fig. 11.8 Needle tracks on an arm

smoke or paint), evidence of previous suicide attempts (recent shallow cuts or scars on arms/wrists) (Disc Image 11.7), evidence of pill residue or dyes in vomit within or around the mouth (Disc Image 11.8) or on clothing (Disc Image 11.9), peculiar discoloration of livor mortis (bright red: carbon monoxide or fluoroacetate; green: hydrogen sulfide), and extensive white or pink foamy, frothy pulmonary edema fluid protruding from the mouth and/or nose (Fig. 11.9). This latter finding, which is sometimes called a “foam cone,” is not specific for drug deaths, as it can occur in a variety of non-drug cases, but it can be seen in deaths related to opiates or inhalants. Drugs may be hidden in clothing or even on or within the body itself (Disc Images 11.10 and 11.11). Occasionally, a decedent will have a single fingernail that is very long compared to the other nails (Disc Image 11.12). This finding suggests a history of drug abuse, as certain individuals presumably use the long nail to insufflate (snort) various drugs.



Fig. 11.9 Pulmonary edema fluid exuding from the mouth producing a so-called “foam cone”

Internal findings that suggest the possibility of a drug or toxin death include extensive liver damage, unusual gastrointestinal contents (peculiar odor or color) (Disc Images 11.13 and 11.14), numerous pills or pill “sludge” or “residue” within the stomach (Fig. 11.10), a dusky discoloration of the brain or widespread brain swelling (Disc Image 11.15), and abundant foamy pulmonary edema fluid. The term “body stuffing syndrome” refers to a situation whereby an individual, usually in imminent threat of being apprehended or searched by police, quickly intentionally ingests (or hides in other body orifices) illegal or illegally-obtained drugs, in hopes of avoiding arrest. Subsequent massive drug overdose can ensue (Figs. 11.11 and 11.12). The term “body packing syndrome” refers to a situation whereby an individual purposefully ingests (or hides in other body orifices) illegal or illegally-obtained drugs, usually packaged in some type of carrying container (balloons, condoms, plastic bag or wrap), in order to transport the drugs across international borders, through airports, or after stealing them, with the intention of recovering the drugs at a later time and place. As with the body stuffing syndrome, subsequent massive overdose can occur.



Fig. 11.10 An opened stomach at autopsy, showing residual gastric contents containing pill residue, along with two specimen containers that contain the majority of the gastric contents. Note that a large amount of pill residue has settled toward the bottom of each container

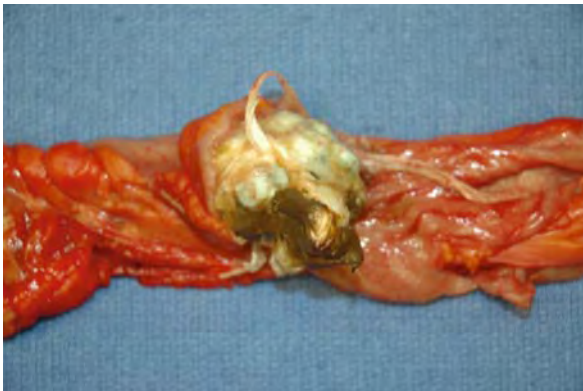


Fig. 11.11 A portion of the sigmoid colon opened at autopsy, revealing a foreign body composed of plastic cellophane material wrapped around numerous pills. The decedent presumably ingested the material several days earlier in an attempt to rob a “friend” of the drugs

Toxicology Issues

With any substance (drug, poison) that is present in a person’s body, there are a variety of terms that are used to describe processes involving that substance. The “route” of exposure or administration describes how the substance gets into a person’s blood stream. Common routes are ingestion (through the GI tract), injection (via a needle intravenously (into a vein), intramuscularly (into a muscle), or subcutaneously (under the skin)), inhalation (breathing it in, either directly or via burning or smoking it), and insufflation (snorting). The term “metabolism” describes how the human body breaks down the substance. Two common locations for drug/toxin metabolism are the liver and the bloodstream itself, where various enzymes can



Fig. 11.12 The unwrapped plastic cellophane from Fig. 11.11, with numerous oxycodone pills that were contained within the plastic. The decedent died of a massive oxycodone overdose

break down certain substances. A substance that is formed by the breakdown of a “parent” substance is referred to as a “metabolite,” or a “breakdown product.” With certain drugs, a metabolite might be “active,” meaning it has pharmacological properties, often similar to the parent drug/toxin. Many other metabolites are “inactive,” meaning that they do not have pharmacological properties. How the body actually gets rid of a drug, usually via metabolism and excretion (often via the kidneys), is referred to as “elimination.” The “half-life” (designated as $t_{1/2}$) of a drug/toxin is the length of time it takes for the body to eliminate half of the substance that was originally present. There are certain drugs that have very short half-lives. With some of these, postmortem (after death) metabolism can continue to occur, using blood enzymes that remain partially active after death. As such, pathologists often rely on toxicology tests identifying a metabolite in order to establish that a death was related to a particular drug’s toxicity.

As mentioned in the introductory paragraph, one of the most important factors in identifying drug- or toxin-related deaths is the forensic toxicology laboratory. Knowing the correct samples to obtain, the proper packaging and storage protocols, and maintaining the proper chain of custody are all necessary to obtain the most optimum toxicology results. Forensic pathologists and death investigators should be aware of the drugs and toxins that are normally screened for in a particular toxicology laboratory’s screening tests. A “qualitative” test signifies whether a substance is present (positive) or not (negative). For a test to have forensic significance, a second methodology must be used with positive results. In most laboratories, the second, confirmatory test is also “quantitative,” in that a specific level of the drug/toxin is determined. A common confirmatory method is gas chromatography–mass spectroscopy (GCMS) (Fig. 11.13). For certain substances, samples must be referred to a larger, reference laboratory. Several tubes of blood and urine should be collected (if possible) in every forensic case.

Urine is frequently used for screening tests. Although such tests can be useful in directing a toxicology laboratory toward a particular drug/toxin, it is important



Fig. 11.13 A toxicologist and a GC/MS

to remember that, in order to determine whether or not a given drug or toxin has contributed to death, the blood levels are what is most important. The best source of blood is peripheral blood, which is most easily obtained from the femoral (groin) blood vessels (Fig. 11.14). “Blind” puncture of the heart, via the chest skin, should be avoided if at all possible, since the needle actually may enter the stomach. Subclavian puncture (near the collarbone) should not be attempted if an autopsy will eventually be performed, since it can introduce tremendous artifacts that may create difficulties during internal neck examination (Disc Image 11.16). Samples of blood should be collected in tubes with sodium fluoride preservative (gray-top tubes), tubes with no preservative (red-top tubes), and tubes with appropriate preservatives for carboxyhemoglobin (carbon monoxide) testing, which vary from laboratory to laboratory. Urine samples (Disc Image 11.17) should be collected in containers



Fig. 11.14 Femoral blood collection at autopsy

with no preservative. Other body fluids and tissues can also be collected, including vitreous fluid (which can be used for toxicology testing or vitreous chemistry testing — see Chapter 21), cerebrospinal fluid (CSF) (Disc Image 11.18), bile (from the gallbladder) (Disc Image 11.19), gastric (stomach) contents, and various solid organs (liver, kidney, brain, muscle). Pathologists should work with their toxicology laboratory to determine which samples are best to retain. In cases where there has been a period of survival following injury, samples can be collected from hematomas (collections of blood outside of blood vessels; for example, a subdural hematoma) in order to be evaluated for drugs that may no longer be present in the circulating blood. When blood and/or other fluids are not available, such as frequently occurs in decomposed remains, solid tissue samples should be collected.

All samples should be labeled with the decedent's name, as well as the sample site (for example: "femoral blood") (Fig. 11.15). Occasionally, testing of hair is required. This can be performed at special reference laboratories, and sometimes can provide a timeline for drug/toxin exposure. A relatively significant amount of hair (a group of hair strands at least 1/4 to 3/8 inch in diameter) should be collected if such testing is to be performed. At autopsy, the hair can simply be pulled out, but in living patients it should be cut, with the cut end marked in some fashion. In cases where heavy metal poisoning is of concern, a fingernail or toenail can be retained for potential testing.

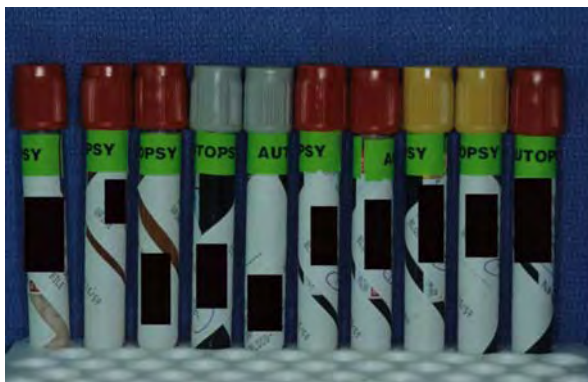


Fig. 11.15 Test-tube rack of toxicology samples from an autopsy

The interpretation of postmortem toxicology tests has some inherent difficulties. Unfortunately, even after death, the postmortem metabolism (breakdown) of certain drugs can continue to occur. This can occur in the liver and/or blood, via enzymes that continue to function even after death. Sodium fluoride preservative acts to prevent such continued metabolism. Refrigeration also decreases such postmortem metabolism. Another issue of concern is a process referred to as "postmortem redistribution," whereby drug levels in postmortem samples actually increase secondary to previously-bound drug being released from tissues or drugs and entering the

blood via diffusion from adjacent structures (stomach, liver). It is generally agreed that the best blood sample to collect in order to minimize the effects of postmortem redistribution is femoral blood. Examples of drugs that are known to exhibit marked postmortem redistribution include tricyclic antidepressants and propoxyphene. In cases where postmortem redistribution has occurred, heart blood samples have drug levels that are much higher than femoral blood samples.

Another issue of concern is the concept of “pharmacogenomics.” Different people have genetic variations in the enzymes used to metabolize certain drugs. As such, a given dose of a particular drug may cause certain, predictable results in many individuals, but have serious or even lethal effects in another individual. For example, many persons of Chinese, Vietnamese, and Japanese descent have a reduced activity of one of the enzymes involved in the metabolism of ethanol (drinking alcohol). When these individuals drink alcohol, they have a severe reaction with facial flushing.

For some drugs, a generally accepted level for lethality is known. In other words, a death will not be considered due to the toxic effects of that drug unless the blood level is at or above the generally accepted lethal level. For other drugs, such a level does not exist, or the levels in cases reportedly due to the drug are so widely spread that a generally accepted lethal level is not agreed upon. Many factors contribute to this somewhat confusing situation regarding these drugs, including postmortem redistribution, postmortem metabolism, pharmacogenomics, short half-lives, tolerance, and the fact that with many such drugs a possible mechanism of death involves the initiation of a lethal cardiac arrhythmia or perhaps a seizure. With drugs that can induce a lethal arrhythmia or seizure, it can be argued that, in a given individual, an absolutely safe level of the drug may not exist. It is beyond the scope of this text to provide detailed information regarding lethal levels of the drugs and toxins discussed. The reader is referred to an excellent reference book, *Disposition of Toxic Drugs and Chemicals in Man*, eighth edition, by Randall C. Baselt (copyright 2008 by Biomedical Publications, Foster City, CA), as an extremely useful resource regarding lethal drug levels.

Death Certification

If a death is caused exclusively by the acute toxic effects of a drug, the cause of death ruling can be written in several different ways. Examples include “toxic effects of cocaine,” “cocaine intoxication,” or “acute cocaine intoxication.” If more than one drug is involved, several options exist, for example, “mixed drug intoxication,” “combined toxic effects of multiple drugs,” or “combined intoxication with cocaine and heroin.” Occasionally, an underlying natural disease process or finding may be considered contributory to death. It is appropriate to list such a finding in part II of the death certificate. For example, part I – toxic effects of cocaine; part II – cardiomegaly. In other instances, a natural disease process may be considered the primary cause of death, but the presence of a drug intoxication is believed to have

played a contributory role in death. For example, part I – spontaneous intracerebral hemorrhage; part II – toxic effects of cocaine. If the drug-related death occurs as a result of long-term (chronic) drug use, without an acute intoxication, the cause of death ruling can be written as follows: “chronic alcohol abuse,” or “complications of chronic intravenous drug abuse,” or “sequelae of chronic drug use.” When the death is felt to be related to both the acute and chronic effects of substance abuse, it is appropriate to rule the death as follows: “acute and chronic alcoholism” or “acute and chronic effects of ethanol.”

The manner of death (MOD) in drug/toxin-related deaths can occasionally be the source of great confusion. In most jurisdictions, a death that is related to the acute toxic effects of a drug in the setting of recreational or therapeutic drug use is appropriately ruled “accidental.” This may seem strange, especially with recreational drug deaths, since the person should have known they *could have* died, therefore the death does not really represent an *unforeseen event*; however, “accident” is the best choice regarding MOD, given the other choices of natural, homicide, suicide, and undetermined. In a few jurisdictions, the MOD ruling “unclassified” is used for recreational drug abuse deaths, but this option is not available in most jurisdictions. Therefore, in most places, a recreational drug-related death due to the acute toxic effects of the drug is ruled as an accident. Some of the most difficult MOD decisions occur when a known drug abuser is found to have died of a drug overdose, and there is some evidence, but not overwhelming evidence, of a possible suicide. Other examples of accidental death rulings related to drugs/toxins include therapeutic drug overdose (sometimes in the setting of “polypharmacy,” or many drugs; sometimes related to pharmacogenomic issues) and childhood intoxication deaths and poisonings, wherein a young child accidentally ingests a drug or poison.

A suicide ruling requires evidence that indicates that the individual purposefully ended his/her life. Such evidence may be a suicide note, investigative information that confirms that the individual was speaking of harming or killing himself/herself, the presence of drugs/toxins that are not typically used for recreational abuse purposes, markedly elevated levels of drugs, or the presence of massive amounts of drug/toxin within the stomach.

Homicidal poisonings are relatively rare, but do occur. Investigative information usually provides evidence that the death is homicidal rather than accidental or suicidal. If another individual knowingly and purposefully prepares a lethal drug dosage (known as a “hot shot”) for someone else to use and that person dies after administering it to themselves, it is appropriate to rule such a death a homicide. If, however, a person prepares a “typical” dose of a recreational drug for someone else, and that person dies after administering it to themselves, it is more appropriate to rule such a death as an accidental death. Likewise, it is not uncommon for drug users to assist one another in obtaining a “high.” If a person willingly allows someone else to inject them with a drug, so long as the preparation is not a “hot shot” as described above, then it is appropriate to rule such a death as an accidental death.

Cases related to therapeutic drugs can involve a variety of special considerations. Deaths can be related to suicidal or accidental self-overdose. Occasional cases are

related to medication error or dosing error. It is appropriate to rule such cases as accidents. Rare cases involve the intentional overmedication of a patient by another individual, such as a nurse intent on “ending others’ suffering” or eliminating financial burdens on the health care system or some other reasoning (the so-called “Florence Nightingale syndrome”). Such cases should be ruled homicides. In other cases, death can be attributed to the effects of a medicine, yet the deaths are routinely considered natural deaths. The following case illustrates this scenario. A woman with metastatic breast cancer is treated with several rounds of chemotherapy, only to experience a severe reaction to the drug, characterized by a profound skin rash. Steroids are given, in order to lessen the effects of the skin rash. The combination of the chemotherapy drug and steroids causes severe suppression of the woman’s immune system, such that she develops sepsis (systemic infection) and dies. So long as the appropriate drugs were given at the appropriate doses, these deaths are typically ruled as natural deaths. The cause of death in the example given could be written in several ways. An example of a relatively detailed way in which to rule the death is: sepsis due to immunosuppression due to steroid treatment of an adverse reaction to chemotherapy for metastatic breast cancer.

Specific Drugs and Toxins

The remainder of this chapter will deal with specific drugs and toxins. Please note that carbon monoxide, cyanide, and hydrogen sulfide (the “chemical asphyxiants”) are presented in Chapter 15 (Asphyxial Deaths).

Ethanol and Related Substances

Ethanol

Ethanol (ethyl alcohol or EtOH) is what is commonly known as drinking alcohol. There are numerous alcoholic beverages, ranging from “near-beers,” which have such low levels (<0.5%) that they are not legally considered alcoholic beverages, to beer (3–8% alcohol by weight), to wine (10–20%), to various distilled liquors, such as vodka, rum, gin, scotch, brandy, whiskey, and bourbon, which may contain 40–50% alcohol by weight. The term “proof” is used to describe the quality of alcohol in wines and distilled liquors and is equal to twice the percentage of ethanol by volume. Various elixirs, cough syrups, and mouthwashes may contain substantial amounts of alcohol (up to 18%). Ethanol is also used in a variety of industries as a solvent.

The “legal limit” for driving under the influence of ethanol varies from state to state within the United States. The terminology typically used for these purposes is

the “blood alcohol concentration” (BAC). The units are expressed in weight/volume of whole blood, so the BAC can actually be written in many forms, including percent (%), grams per 100 mL whole blood (g/100 mL), grams per deciliter (g/dL), milligrams per deciliter (mg/dL), milligrams percent (mg%), or grams per liter (g/L). The appropriate equivalent values for each of these is as follows: $0.10\% = 0.1\text{ g}/100\text{ mL} = 0.10\text{ g}/\text{dL} = 100\text{ mg}/\text{dL} = 100\text{ mg}\% = 1.0\text{ g}/\text{L}$. The usually legal limit within the US is 0.08–0.10%, depending on the jurisdiction. The ratio between blood and breath ethanol percentage is constant, at 1 to 2100. This number allows the “breathalyzer” analysis to convert to an equivalent BAC. Of note regarding autopsy specimens is the fact that, in many laboratories, a postmortem blood alcohol level is actually performed on serum (the liquid part of blood, without the blood cells), rather than whole blood. The serum ethanol level is typically 12–18% higher than that of whole blood.

The usual route of administration of EtOH is via GI absorption after oral ingestion. The EtOH is absorbed predominantly in the small intestines. Food may slow absorption. A vast majority of EtOH is metabolized within the liver by enzymes that convert EtOH to acetaldehyde and then acetic acid. A small percentage of EtOH within the blood is excreted in the urine, sweat, and breath. The average non-alcoholic person’s ability to eliminate EtOH is about two thirds to one drink per hour.

Although the legal limit is established by statute, it is well documented that in many individuals physiologic impairment occurs at levels less than the legal limit. As the BAC increases, a person becomes more and more intoxicated, displaying various signs and symptoms, including slurring of speech, incoordination, and slowed responses, among others. In persons who are chronic abusers of EtOH, it is common for “tolerance” to occur, such that the individual may actually appear sober with BACs that would make most people obviously drunk. In non-tolerant individuals, BAC levels at or above 4.0 g/dL can be considered lethal; however, some argue that levels as low as 3.0 g/dL can be lethal. In living chronic alcoholics, it is not unusual to see levels higher than 4.0 g/dL.

The toxic effects of EtOH encompass a spectrum of changes within numerous organ systems. Within the CNS, EtOH has a depressant effect. Wernicke–Korsakoff encephalopathy, cerebellar vermis atrophy, central pontine myelinolysis (CPM), and seizures can occur. The liver changes include steatosis (fatty change), hepatitis (inflammation), and cirrhosis (scarring) (Fig. 11.16). GI system effects include portal hypertension, esophageal varices, peptic ulcer disease, gastritis, Mallory–Weiss syndrome (lacerations of esophagus from vomiting), and pancreatitis (inflammation of the pancreas). EtOH is considered a cardiac irritant, with intoxicated persons being at increased risk of an arrhythmia. In addition, chronic use can result in a dilated cardiomyopathy. Chronic alcoholics tend to be malnourished, have vitamin deficiencies (especially thiamine), and are prone to electrolyte disturbances, such as occurs in the low-salt vitreous electrolyte pattern. Deaths related to chronic alcoholism, without accompanying acute intoxication, should be ruled as natural deaths. This includes deaths related to alcohol withdrawal (delirium tremens).

Fig. 11.16 A case of liver cirrhosis seen at autopsy



The best specimen for postmortem EtOH determination is peripheral blood (femoral) collected in a gray-top tube (containing sodium fluoride as a preservative). Heart blood samples should be avoided, since EtOH can diffuse across the gastric wall and diaphragm. “Blind” attempts at cardiac puncture in cases where an autopsy is not going to be performed should also be avoided, since the stomach can be inadvertently punctured; EtOH contained within the stomach in such instances will create an artifactually elevated blood ethanol level. It is important to remember that a *serum* EtOH level is approximately 12–18% higher than the corresponding *whole blood* BAC. In cases with a significant amount of survival following injury and before death (such as several days), evaluation of blood contained in hematomas (for example, a subdural hematoma) may show ethanol levels consistent with circulating blood levels at the time of injury, even though circulating blood levels immediately prior to death (and in samples collected at autopsy) are negative.

Decomposition can result in EtOH production via bacteria, but such postmortem production does not occur in every decomposed body. The typical blood EtOH level in cases of postmortem decompositional production is <1.0 g/dL; however, some cases have been reported in which the level has approached 2.0 g/dL. The identification in blood samples of n-propanol and other lower order alcohols provides evidence that at least some of the blood EtOH is due to postmortem bacterial production. Vitreous and CSF samples are less likely to demonstrate postmortem EtOH production, but it is important to note that each of these fluids has a higher ethanol concentration than blood (1.2:1 ratio), due to differing water content.

It is not uncommon in cases involving acutely intoxicated individuals to smell a peculiar “alcohol odor” at autopsy. Strictly speaking, from a chemistry standpoint, EtOH is odorless. Therefore, the smell that accompanies alcoholic beverages (whether in the container, at a bar, or in a dead body) is really the other substances, or “congeners,” contained within the beverage.

Certain drugs can “potentiate” (increase) the effects of EtOH, including barbiturates, narcotics, tranquilizers, antihistamines, and hypnotic agents. If EtOH is used in combination with cocaine, another substance, called “cocaethylene,” can be produced within the body, as the EtOH and cocaine are metabolized.

Cocaethylene has pharmacologic properties similar to, but more powerful than cocaine.

Ethylene Glycol

Ethylene glycol is the major ingredient within many antifreeze preparations (Disc Image 11.20). It should be considered poisonous and is occasionally encountered in forensic casework. The typical situation is a chronic alcoholic who obtains some antifreeze, likes the sweet taste, and drinks it because of the unavailability of EtOH. Another situation is an accidental ingestion, typically by a child, who also enjoys the sweet taste. Occasional suicidal ingestions are encountered, as are homicidal poisonings. A relatively small amount (100 mL) is considered a lethal dose, and the lethal blood concentration is around 2.4 g/L. Once absorbed, ethylene glycol is metabolized to many compounds, including oxalate. The toxicity is related to CNS depression, seizures, acidosis, and renal failure. Calcium oxalate crystals can be easily identified within the kidney on microscopic examination; polarization helps to identify the crystals (Fig. 11.17). Urine samples can also contain the crystals, and sometimes the urine will fluoresce when exposed to ultraviolet light (a Wood's lamp) (Disc Image 11.21). Of importance is the fact that many routine toxicology screening methods do *not* identify ethylene glycol. As such, if it is suspected, a specific test for ethylene glycol should be requested. Alternatively, microscopic sections of kidney can be screened for the presence of calcium oxalate crystals.

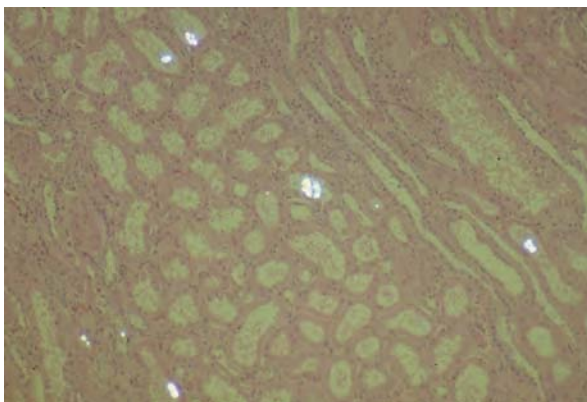


Fig. 11.17 Calcium oxalate crystals within the kidney in a case of ethylene glycol poisoning

Methanol

Methanol (methyl alcohol) is also referred to as “wood alcohol.” It is used as a solvent, a fuel, and in certain antifreezes. A small amount is produced in humans. Routes of exposure include ingestion, inhalation, and absorption through the skin.

It is metabolized to formaldehyde and formic acid. These substances are all toxic, resulting in CNS depression, acidosis, and blindness if the person survives. The minimal lethal dose is relatively small, similar to that of ethylene glycol.

Isopropanol

Isopropanol is commonly known as “rubbing alcohol.” It is used for first aid and as a method of cleaning the skin prior to needle puncture. It is considered a CNS depressant, but in reality, isopropanol is not toxic in itself, but is metabolized to acetone, which has toxic effects. Small amounts of isopropanol in postmortem blood samples are probably due to one of several possibilities, including conversion from acetone in the setting of diabetic ketoacidosis or starvation, or contamination from “alcohol wipes” used during medical or autopsy procedures. A dose of 250 mL can be lethal.

Amphetamines and Similar Substances

Amphetamines are stimulant drugs. There are numerous drugs classified as amphetamines, many with specific street names (see below). Several amphetamines have legitimate medicinal uses but most are used solely for recreational abuse. Some of these are referred to as “designer drugs” – they may be ingested, injected, smoked or snorted. The primary effects are on the CNS and cardiovascular systems, including possible hallucinations, psychosis, and elevation of blood pressure. Deaths related to the toxic effects of amphetamines may be related to CNS seizure activity or cardiac arrhythmias. Tolerance may occur with amphetamines. Unlike certain other drugs, where a specific level must be present in order to consider the death as an “overdose,” the mere presence of amphetamines within the decedent, in the absence of another explanation for death, is considered enough evidence to rule the cause of death as being due to the toxic effects of amphetamines.

Examples of amphetamines include: amphetamine, methamphetamine, dextroamphetamine (Adderal), methylphenidate (Ritalin), methathinone (CAT), methylenedioxyamphetamine (MDA), 3,4-methylenedioxymethamphetamine (MDMA or “ecstasy”), methyl-2,5-dimethoxyamphetamine (DOM), and paramethoxyamphetamine (PMA). Some of these (MDMA and PMA) are commonly used at “rave” parties. Street names are numerous and include “uppers,” “speed,” “ice,” “crank,” the “love drug,” “crystal,” “glass,” “tina,” and “pep pills.” Persons known to ingest huge quantities are sometimes referred to as “speed freaks,” and the process in which a person uses large quantities is referred to as a “speed run” or “speed binge.” Chronic methamphetamine users frequently develop “meth mouth,” a condition of poor oral hygiene, resulting in loss of teeth, probably related to a combination of drug-induced teeth-grinding (bruxism), dry mouth (xerostomia), and general lack of concern (Fig. 11.18). Methamphetamine can be produced relatively easily, although “meth labs” can be quite dangerous. Various

recipes/instructions for methamphetamine production exist and are easily found on the Internet, including “one pot” and “shake’n’bake” methods. With many ingredients or by-products may be potentially harmful.



Fig. 11.18 A typical case of poor oral hygiene associated with methamphetamine use (“meth mouth”)

MDMA (“ecstasy”) produces a massive serotonin rush and is very common within the rave scene. The drug is described as a stimulant followed by a hallucinogen “chaser.” MDMA comes in a variety of tablet forms, often with distinctive scoring (impressions) on the tablets, including various popular emblems, symbols, or characters. MDMA may be “cut” (diluted) with a variety of other substances, including methamphetamine. It is frequently concealed or hidden in various forms or packages, including otherwise “innocent” items, such as candy containers, breath mint packages, lip balm, pacifiers, ring-candy, candy necklaces, etc. Some users enjoy inhaling menthol petroleum jelly products along with abuse of MDMA. Teeth clenching, tongue chewing, and post-MDMA depression are undesirable side-effects. Slang names include “XTC,” “X,” “E,” “X-box,” “biscuits,” “adam,” “essence,” “Roloids,” “molecule,” and “Molly.” Other terms related to ecstasy use include “parachuting,” in which the drug is wrapped in tissue prior to ingestion, in order to avoid the bitter taste, and “plugging,” in which the tablet is inserted rectally.

Other Stimulants

Khat (pronounced cot) is a plant, *Catha edulis*, which is native to eastern Africa and southern Arabia. Fresh leaves contain a powerful stimulant (cathinone). As the leaves dry, a less potent stimulant (cathine) forms. The leaves are chewed, and the juice is swallowed. A form used for smoking is referred to as graba.

Mescaline (see below) is a naturally-derived drug of abuse that has certain properties that are similar to amphetamines.

Barbiturates

Barbiturates are a group of depressant drugs, commonly referred to as “downers.” Other street names include “redbirds,” “blue heavens,” “goofballs,” and “yellow jackets.” There are three basic groups, depending on their timing of onset and duration of action (short-acting, intermediate-acting, and long-acting). The drugs are typically ingested or injected and have a sedative-hypnotic effect. Overdose results in coma and death. Medicinally, they are frequently used in anesthesia or as anticonvulsants. Examples include phenobarbital, secobarbital, amobarbital, and pentobarbital.

Although barbiturate abuse has fallen out of favor, occasional overdoses are still encountered. Classic descriptions of some of the lesions associated with barbiturate abuse include the presence of “barbiturate blisters” on the skin, necrosis (death) of a particular structure in the brain referred to as the “globus pallidus,” and the identification of polarizable crystals seen under the microscope when looking at the stomach lining (if the drug was ingested). The latter finding is not specific to barbiturates, as it can be seen with numerous ingested drugs. The term “drug automatism” refers specifically to the prolonged uncontrollable intake of barbiturates.

Cocaine

Cocaine is produced from a plant called *Erythroxylon coca*. It has legitimate medicinal use as a local anesthetic agent, usually in facial-type surgery, but is far more commonly a drug of abuse. It is considered a stimulant, primarily affecting the CNS and cardiovascular systems. It acts in the nervous system by inhibiting the reuptake of the neurotransmitters dopamine and norepinephrine. It may be injected, insufflated (snorted), smoked, ingested, or applied topically. Cocaine hydrochloride is the powder form of the drug, and is sometimes referred to as “soft” cocaine. “Rock” cocaine is a very pure form of cocaine hydrochloride. “Crack”, or free-base cocaine, is smoked (Fig. 11.19), and is sometimes referred to as “hard” cocaine. Cocaine



Fig. 11.19 Crack cocaine discovered in the pocket of a homicide victim

is typically abused in order to experience the physiologic effects of euphoria and increased alertness. Street names for cocaine include “girl,” “connie,” and “teener.”

The half-life of cocaine is relatively short (30–60 minutes). Cocaine is metabolized to norcocaine (active), ecgonine methyl ester, benzoylecgonine (BE), and ecgonine. Metabolism within the bloodstream can continue after death. For this reason, blood samples should be collected in tubes containing sodium fluoride, a substance that inhibits the enzymes that break down cocaine. In the presence of ethanol, the substance “cocaethylene” can be produced within persons using cocaine. Cocaethylene is unique in that it is produced in the body by a combination of two other drugs (cocaine and ethanol), and it is pharmacologically active (similar to cocaine), with a half-life of over an hour and a half. BE’s half-life is around 4–5 hours. Cocaine metabolites can be detected in the urine for several days after use. In order to rule a death as being related to the toxic effects of cocaine, there should be evidence of cocaine or metabolites (such as BE) within postmortem blood samples. Even in the absence of measurable cocaine within postmortem blood samples, a death can still be ruled as being caused by cocaine toxicity, so long as metabolites are present in the blood and there is no other explanation for death. The absence of measurable cocaine in these cases is explained by cocaine’s short half-life, as well as the postmortem interval (between death and collection of sample), which allows for postmortem metabolism within the blood.

Deaths related to cocaine use typically result from cardiovascular or central nervous system effects. Within the cardiovascular system cocaine may induce arrhythmias, marked increase in blood pressure, myocardial infarcts (heart attacks), and cerebrovascular hemorrhages. Within the CNS, cocaine may induce seizures, hyperthermia, panic attacks, and intense paranoia with delirium. Any of these may be associated with a lethal outcome. “Excited delirium” is classically described in cocaine users (Fig. 11.20), characterized by hyperthermia, superhuman strength,



Fig. 11.20 A victim of cocaine-induced excited delirium. The victim experienced bizarre behavior, superhuman strength, paranoia, and hyperthermia (hence the absence of clothing) prior to collapsing and dying

agitation, paranoia, delirium, and often sudden death. Many in-custody deaths have excited delirium as a major contributing factor. The reader is referred to Chapter 21 for a more detailed discussion of such deaths.

A majority of cocaine-related deaths do not have evidence of excited delirium; the drug user is simply found dead. Drug paraphernalia may or may not be found at the scene or with the body (Fig. 11.21). In such instances, as discussed above, toxicology tests may or may not reveal the presence of cocaine within the decedent's blood; however, BE and other metabolites will typically be present. Unlike certain drugs and toxins, there is no "safe" level of cocaine. A relatively small amount can induce a fatal arrhythmia in a long-time user or a first-time user. In certain situations, there may be a massive overdose. The typical settings are the "body packing syndrome" or the "body stuffing syndrome," wherein a person attempts to transport or hide cocaine within the GI (or other) systems (Disc Image 11.22). Some long-term effects associated with chronic cocaine use include an enlarged heart, advanced coronary artery atherosclerosis, and a perforated nasal septum (in persons who snort cocaine). It is not unusual to encounter a case in which there is underlying heart disease (cardiomegaly and/or atherosclerosis) that is sufficient, by itself, to explain death, but with toxicologic evidence of recent cocaine use, i.e. significant levels of BE (but not necessarily cocaine itself). When such a situation occurs, it is appropriate to consider both the underlying disease and cocaine toxicity as contributing factors in death. As per the manner of death guidelines described in Chapter 5, the manner of death in such cases is dictated by the exogenous (external) factor (the cocaine). Therefore, most cases such as this will be ruled as accidental.



Fig. 11.21 A crack pipe along with a disposable lighter

Opiates and Related Substances

Opiates are a class of narcotic drugs that are frequently abused. The term "narcotic" describes a drug that is able to induce sleep. Opiates, therefore, are depressants. There are many opiates and related compounds that are used for legitimate

medical purposes. Morphine, hydrocodone, oxycodone, fentanyl, propoxyphene, and methadone all have useful pharmacologic properties, often as analgesics (pain relievers). Another common medical use for some of the less-potent opiates, such as codeine and dextromethorphan, is cough suppression. Opiates having no legitimate medicinal use in the United States include heroin and opium. The source of many opiates is the poppy plant (*Papaver somniferum*). Others are synthetically produced. Common routes of administration of opiates include injection and ingestion; less common routes include smoking and snorting.

The physiologic effects of opiates include CNS depression and analgesia (pain relief). At moderately high doses, opiates can cause euphoria, a sense of warmth, pinpoint pupils, nausea, urinary retention, and constipation. Higher doses may result in drowsiness, sedation, respiratory slowing, and a depressed cough reflex. Opiate overdose usually results in respiratory depression and failure. Other effects can include hypotension (low blood pressure), a decreased heart rate, and cold/clammy skin. Tolerance is a well-known phenomenon amongst opiate users, such that higher and higher doses may be required in order for a user to experience the effects that they desire. Interpreting a given postmortem toxicology result can be challenging, particularly when the decedent is a known opiate abuser; however, if there is no other reasonable explanation for death, the presence of opiates in postmortem blood is a strong indication that the death was due to opiate toxicity.

At autopsy, persons dying from opiate toxicity frequently demonstrate abundant pulmonary edema fluid, often with pink to white frothy fluid having been expelled through the mouth and nose (a “foam cone”). Depending on the route of administration, needle tracks or “skin popping” scars (from subcutaneous injection) may be evident. Microscopic examination may reveal the presence of polarizable foreign material from pills that have been ground up, dissolved, and injected into veins, and is usually seen at skin injection sites and within the lungs (Figs. 11.22 and 11.23).

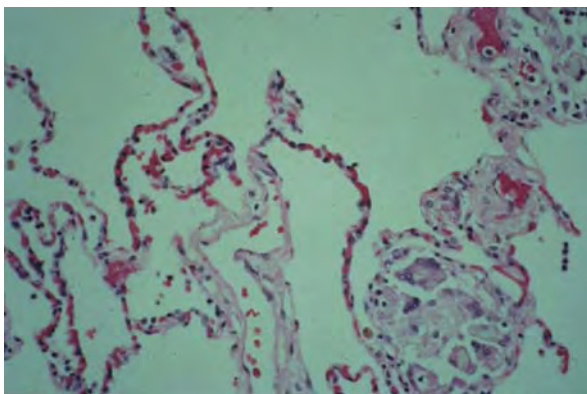


Fig. 11.22 Chronic intravenous drug abuse frequently introduces abundant foreign material into the blood stream. This foreign material can become entrapped within the lungs, causing inflammation and scarring. The material is not readily visible in routinely stained histology sections (photo courtesy of Dr. Patrick E. Lantz, MD, Wake Forest University, Winston-Salem, NC)

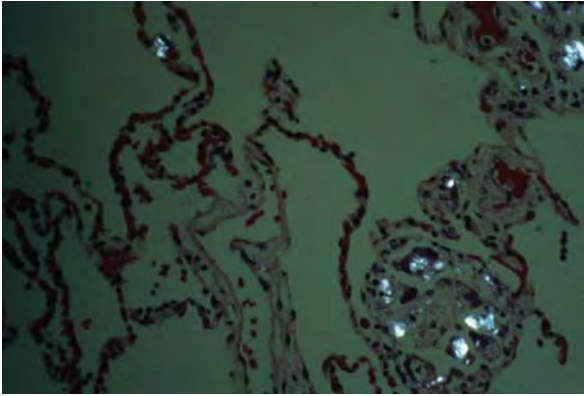


Fig. 11.23 Using polarized light, the foreign material present within the lung in Fig. 11.22 is readily apparent (photo courtesy of Dr. Patrick E. Lantz, MD, Wake Forest University, Winston-Salem, NC)

There may be gross evidence of constipation (impacted stool within the bowels) and/or urinary retention (a markedly distended bladder) (Disc Image 11.23).

The preceding paragraphs provide basic information that may be considered common to all opiates. The following paragraphs provide additional information regarding specific drugs.

Morphine

Morphine is a legal, but restricted opiate that is considered one of the best drugs for pain relief. Its use is regulated such that it may only be dispensed with a prescription. Injectable and oral preparations are common. As with other opiates, drug dependence and addiction may form in persons using this drug and, as such, abuse of morphine is relatively common. Its half-life is much longer (>1 hour) than its illegal counterpart, heroin. Since heroin is actually metabolized into morphine, morphine is frequently detected in postmortem samples of persons dying from heroin overdose.

Heroin

The chemical name of heroin is diacetylmorphine (two acetyl groups attached to morphine). Its half-life is only about 5 minutes. It is first metabolized to 6-monoacetyl morphine (6-MAM), which is subsequently metabolized to morphine. Heroin continues to break down after death, such that it is relatively uncommon to identify heroin itself in postmortem samples. The presence of 6-MAM confirms that heroin was present. The use of sodium fluoride containing tubes (gray-top tubes) for blood collection helps to prevent continued metabolism of heroin and 6-MAM. Since the metabolism of heroin is so fast, a definite postmortem “lethal level” has not

been established with certainty. If 6-MAM and morphine (and occasionally heroin itself) are present in postmortem blood, and there is no other explanation for death, then death can be attributed to the toxic effects of heroin. If morphine is present, but there is no heroin or 6-MAM, it is best to rule the death as being due to the toxic effects of opiates.

Street names for heroin include “horse,” “smack,” “tar,” “blue velvet,” “boy,” “mud,” “dogfood,” and “red devil.” “Brown tar” heroin and “black tar” heroin are mostly produced in Mexico and tend to predominate on the west coast of the US (Fig. 11.24). It is usually “cut” (diluted) with lidocaine, such that its purity is around 10% for brown and 50% for black. “China white” heroin or simply “white” heroin is produced in South America and is predominant on the east coast of the US. It is cut with quinine, and is about 90% pure. A particularly potent (high percent purity) heroin is referred to as “gunpowder heroin.” “Cheese” is the street name for a combination of heroin and Tylenol PM (acetaminophen and diphenhydramine). A “speedball” is a combination of cocaine and heroin. The term “hot shot” refers to two separate scenarios: a forced injection of heroin or a dose that is undiluted. Heroin addiction is devastating; withdrawal symptoms include insomnia, nausea, weakness, and hot and cold flashes. Heroin addicts are sometimes treated with methadone, which is a longer-acting opiate. Various forms of packaging occur with heroin, including small balloons, bindles (0.1 gram), and bundles (1 gram).

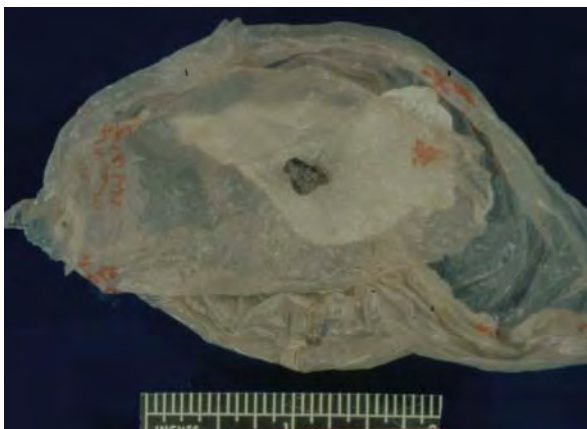


Fig. 11.24 Black (or brown) tar heroin found in the clothing of a heroin overdose decedent

Methadone

Methadone is a diphenylpropylamine derivative that has analgesic and sedative properties. It has been traditionally used as a form of chronic oral treatment for heroin addicts. By providing a heroin addict with longer-acting methadone, it is reasoned that the addict can perhaps approach a more normal lifestyle. At the very

least, they will not have to resort to crime in order to maintain their drug habit. In recent years, methadone has been increasingly advocated for use as an analgesic for numerous medical conditions. With the increase in legitimate medicinal use, the drug has become more readily available, such that its abuse has become very common. Deaths related to the toxic effects of methadone are relatively common, with many such deaths occurring in “naïve” users: those who have never used the drug, perhaps relying on a chronic user (with tolerance) to advise them on what dose to use.

Fentanyl

Fentanyl (Sublimaze; Duragesic) is a narcotic analgesic that is highly potent but has a relatively short duration of action. Routes of administration include oral, injection, and transdermal (skin patches). It is not uncommon for abusers to attempt to alter skin patches in order to receive a higher dosage of the drug. This can involve heating the patch, attempting to physically remove the drug from the patch in order to use it in a way other than transdermally, or even chewing on or ingesting the patch (Fig. 11.25). It should be noted that fentanyl is not necessarily detected in many routine toxicology screens. If it is suspected in a given case, the toxicology laboratory should be notified as specialized testing is available.

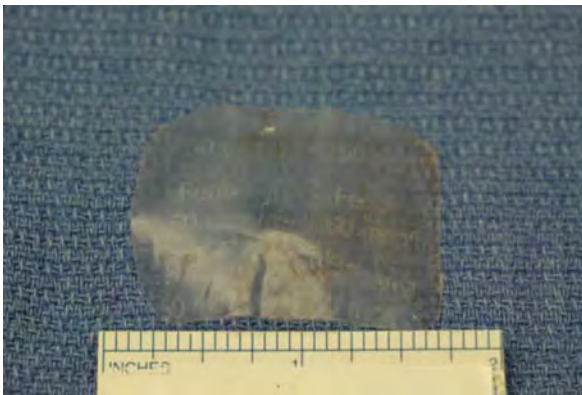


Fig. 11.25 A fentanyl patch found within the gastrointestinal system of an individual who was abusing the drug

Other Opiates

Meperidine (Demerol) is an analgesic agent with properties similar to morphine. Hydrocodone (Vicodin and others) is an opiate that is commonly used as an analgesic and antitussive (anti-cough) agent. Oxycodone (OxyContin, Percocet, and others) is an opiate that is primarily prescribed for pain relief. Sustained release

preparations, such as OxyContin, are particularly prone to abuse. Propoxyphene (Darvon) is a prescription analgesic related to methadone and as with methadone, it has a very narrow range between therapeutic and toxic/lethal levels. Kratom is a tree leaf that is chewed as an opiate substitute with stimulant properties. It has a bitter taste and is sometimes referred to as “purple sticks.”

Psychoactive Drugs of Abuse (Hallucinogens)

Hallucinogens are substances that have the ability to cause changes in a person’s perception of reality. The term “psychedelic” is applied to some of the drugs in this category. Included in this category of drugs is one of the most commonly used drugs of abuse, marijuana, as well as LSD, PCP, mescaline, and mushrooms.

Marijuana

Marijuana is another name for the plant *Cannabis sativa*, the leaf of which may be smoked or ingested (Disc Image 11.24). Other forms of varying potencies go by the names of sinsemilla, ganja, hash, hashish, and hash oil. The active ingredient is delta-9-tetrahydrocannabinol (THC). Metabolites include 11-hydroxy-THC (active), 8-hydroxy-THC (active), and 11-carboxy-THC (not active). THC metabolites can be stored in fatty tissues for weeks to months. The drug is used for its euphoric effects; however, other possible effects are lethargy, drowsiness, hunger (the “munchies”), thirst, memory loss, anxiety, paranoia, psychosis, tachycardia (increased heart rate), increased blood pressure, decreased motor coordination, and reddening of the eyes. Marijuana is not known to be lethal due to physiologic toxicity; however, it is frequently involved in lethal accidents, where marijuana intoxication may well be considered a causal factor in the accident. Blood THC levels of greater than 5–10 ng/mL indicate recent use. THC and 11-hydroxy-THC levels decline rapidly after use. Blood 11-carboxy-THC can remain elevated for several days after use and urinary carboxy-THC can be detected for several months. Various slang names are used for marijuana, specific forms of marijuana, or marijuana-containing substances. Examples include “weed,” “Mary Jane,” “herb,” “tree,” “bill,” “blunt,” “dank,” “bud,” “kush,” “seedless,” “purple haze,” and “cocoa puffs” (marijuana combined with cocaine).

Lysergic Acid Diethylamide (LSD)

LSD represents the prototypical psychoactive drug and is commonly referred to as “acid.” Other street names include “mellow yellow,” “dica,” “cid,” and “window pane.” The usual route of administration is oral, via consumption of thin squares of LSD-laced gelatin (“blotters” or “dots”). Alternatively, the drug may be snorted. LSD use results in a variety of effects, including impaired judgment, hallucinations,

rapid emotional changes, possible psychosis, and flashbacks (“post-hallucinogen perception disorder”). Hyperthermia and excited delirium may also occur. A general term used to describe the effects of LSD is “trip,” with users experiencing a “good trip” or a “bad trip.” Anxiety and paranoia tend to accompany bad trips. Death can be related to accidental injury sustained during acute intoxication (Disc Image 11.25) or in relation to excited delirium.

Phencyclidine (PCP)

PCP is approved for use as a veterinary tranquilizer. It is also considered a “dissociative” anesthetic agent. Street names include “angel dust,” “tic tac,” “boat,” and “zoom.” It may be ingested, injected, insufflated, or smoked, often in combination with marijuana, or even with some otherwise legal substance, such as oregano, parsley, or mint. Its use results in hallucinations, lethargy, feelings of strength, and decreased pain perception. Respiratory depression and increased blood pressure may also occur. Dissociation between the mind and body is also described, wherein the user is aware of the events taking place but does not feel involved, either emotionally or physically. Stupor, coma, seizures, disorientation, psychosis, excited delirium, and delusions may occur, as can hypertension (elevated blood pressure), tachycardia (increased heart rate), and sudden death. Death may also be related to injuries sustained during intoxication.

Mescaline (Peyote)

Peyote is a small, spineless cactus (*Lophophora williamsii* or *Anhalonium lewinii*) that is used by some Native Americans during religious rituals as an oral CNS stimulant/hallucinogen. It has effects that are similar to amphetamines, including the possible induction of an acute psychosis. The active ingredient in peyote is mescaline, also known as trimethoxyphenethylamine or 3,4,5-trimethoxybenzeneethanamine.

Mushrooms (Psilocybin)

Psilocybin mushrooms are another naturally-derived hallucinogenic substance used in some Native American ceremonies. They are native to the west coast, the southern US, Mexico, and South America, and are usually consumed. Street names include “shrooms,” “magic mushrooms,” and “musk.” Psilocybin and the active metabolite psilocin are responsible for the physiologic effects, which are similar to those induced by mescaline and LSD. Occasionally, users mistakenly consume similar-appearing but highly poisonous mushrooms, resulting in poisoning and sometimes death. There are several dozen varieties of mushrooms that contain known toxins, including cyclopeptides, which are considered most dangerous. The most lethal mushroom is the *Amanita phalloides*, which has a toxin (Amanita toxin) that induces

cholera-like effects via inhibition of RNA polymerase, and another (Phallin toxin) that is hemolytic (it destroys red blood cells).

Other Hallucinogens

Dextromethorphan (DXM), in amounts far in excess of doses used for its cough-suppressant effects, results in dissociative effects similar to those of PCP. It is present in a variety of over-the-counter preparations, including Robitussin and Coricidin. There are reportedly four levels or plateaus of intoxication, ranging from mild inebriation to an out-of-body experience comparable to that produced by PCP or ketamine. Abuse of excessive amounts of DXM is sometimes referred to as “robotripping” or “skittling.” It is sometimes referred to as “poor man’s PCP” or “poor man’s ecstasy.”

Dimethyltryptamine (DMT) is a naturally occurring substance in a variety of plants. It can be snorted, smoked, or injected, usually in combination with other drugs. The hallucinogenic effects typically last about an hour, hence the slang expression “businessman’s trip.”

Salvia divinorum is an herb in the mint family that is smoked, chewed, or even brewed (like tea) for its hallucinogenic/psychedelic effects, including flashbacks. The plant, which has a bitter taste, grows wild in Mexico. Its “high” is produced within 2–3 minutes and lasts for several minutes. Street names include “purple sticks” and “herbal XTC.”

Morning glory seeds contain lysergic acid amide, which is chemically similar to LSD. Consumption of 25–50 seeds can produce hallucinations which last many hours. Street names include “heavenly blue,” “pearly gates,” and “flying saucers.”

Jimson weed is a common weed found in rural areas. It can be chewed, smoked, or mixed in tea for its hallucinogenic effects. Slang names include “locoweed,” “angel’s trumpet,” “mad apple,” “stink weed,” and “green dragon.”

Ketamine is another hallucinogen that is abused. Street names include “K,” “Special K,” and “kitty valium.” “K-holing” is a term used to describe ketamine use. Flashbacks are possible, as is sudden death.

Other hallucinogens include 2C-B/2C-T-7, alpha-methyltryptamine (AMT), 5-methoxy-diisopropyltryptamine (“foxy”), GHB (see below), MDMA (ecstasy; see above), and paramethoxyamphetamine (PMA). Many of these are popular drugs at rave parties.

Over-the-Counter (OTC) Drugs

Several OTC drugs have already been mentioned. In this section, some others will be discussed. In general, OTC drugs, which are usually non-addictive, are considered relatively safe, so long as they are used according to the manufacturers’ directions.

Acetaminophen

Acetaminophen (Tylenol) is a very commonly used analgesic (pain reliever) and antipyretic (anti-fever) OTC drug. Routes of administration include oral and rectal (suppositories). A lethal dose in an adult is considered 20 g, while in a child, as little as 4 g can be lethal. A blood concentration >160 mg/L is considered toxic. If a very large amount of the drug is ingested, death can occur relatively quickly (Fig. 11.26). If lesser, but still toxic, amounts are used, the patient may initially survive, and progress through four stages of poisoning: I (24 hours) – gastrointestinal distress; II (1–2 days) – apparently better, but with ongoing liver damage; III (3 days) – jaundice, coagulation problems, continued liver damage; IV (4 days–2 weeks) – liver failure and death. Under the microscope, the classic pattern of liver destruction is described as “centrilobular necrosis.”

Fig. 11.26 The gastric contents of a man who committed suicide by ingesting three entire bottles of Tylenol. Note the abundant pill residue within the container



Aspirin (Salicylate)

Aspirin represents another very commonly used OTC drug. Besides having analgesic and antipyretic properties, aspirin also has anti-inflammatory and anti-blood clotting effects. The usual route of administration is oral. The lethal dose is said to be between 2 and 5 g. The toxic blood concentration is 500 mg/L. Persons experiencing aspirin toxicity tend to proceed through three physiologic phases: respiratory alkalosis, metabolic acidosis, and hyperthermia. Children taking aspirin during a viral respiratory illness are at increased risk of developing Reye syndrome, characterized by severe liver damage and possible death.

Ephedrine/Herbal Ecstasy

Ephedrine is a common stimulant drug that is present in a variety of OTC preparations. It is used as a nasal decongestant, an appetite suppressant, or specifically for its stimulant properties (to help stay awake). It is similar chemically to amphetamines. As a stimulant, it may affect the CNS or cardiovascular systems. It is the key ingredient of the herbal product known as “Herbal Ecstasy.” Ephedra (ephedrine alkaloids) were once common in dietary supplements, but they have since been banned by the Food and Drug Administration because of the potential for harmful CNS and cardiovascular effects.

Other Prescription Drugs

Like OTC drugs, many prescription drugs are widely available today, with many more developed and marketed every year. Each has the potential for adverse effects, including many that may cause death by overdose. As mentioned above, when investigating a death that is thought to be related to overdose, scene investigation is vital in order to provide the toxicology laboratory with an idea of what substances/drugs might be present in the decedent.

Antidepressants

Antidepressant medications come in a variety of drug classes. In past years, tricyclic antidepressants and monoamine oxidase inhibitors (MAOIs) were common. Examples of tricyclics include amitriptyline and nortriptyline. Toxic effects include dry mouth, vision changes, hyperthermia, hyperactivity, seizures, coma, arrhythmias, respiratory depression and sudden death. The interpretation of postmortem levels of tricyclic antidepressants is complicated by postmortem redistribution.

At present, selective serotonin reuptake inhibitors (SSRIs) are more commonly used as antidepressants. Examples include fluoxetine (Prozac), paroxetine (Paxil), sertraline (Zoloft), and citalopram (Celexa). These newer antidepressants are much less toxic than the tricyclics and MAOIs, but may still be involved in overdose deaths, particularly when combined with other drugs. Some research has suggested that certain SSRIs are associated with an increased risk of suicide, but this research is questioned by others because depression (the reason antidepressants are prescribed in the first place) is also associated with an increased risk of suicide. Other newer antidepressants that are similar in certain ways to SSRIs are bupropion (Wellbutrin) and venlafaxine (Effexor).

Nonbarbiturate Sedative Hypnotic Drugs

Nonbarbiturate sedative hypnotic drugs include a large group of very commonly prescribed medications. A sedative is used to provide a calming effect for someone who is anxious. A hypnotic is used to facilitate sleep.

Benzodiazepines are very commonly prescribed sedative hypnotic agents. Examples include chlordiazepoxide (Librium), clonazepam (Klonopin), cyclobenzaprine (Flexeril), and diazepam (Valium). They may be taken orally or injected. The physiologic effects include muscle relaxation, decreased anxiety, anticonvulsant effects, sedation, and drowsiness. Benzodiazepines are considered very safe; in fact, massive overdose usually does not lead to generalized anesthesia, let alone death. Overdose may, however, lead to seizure activity, hypertension or tachycardia (increased heart rate). Despite their relative safety, it is not uncommon to detect benzodiazepines in accidental or suicidal drug overdoses where multiple drugs are involved. It should be noted that flunitrazepam (Rohypnol), which is known as a “date rape” or “knock-out” drug, is a benzodiazepine that is illegal in the US. “Date rape” or “knock-out” drugs are used to cause a victim to enter a state of drowsiness with associated amnesia, such that a perpetrator is able to sexually assault them. These drugs can be secretly placed into an unsuspecting victim’s drink or food. Other such drugs include gamma-hydroxybutyrate (GHB; see below) and ketamine.

Gamma-hydroxybutyrate (GHB) is a tasteless, odorless, illegal hypnotic agent that is used as a steroid alternative for body building, as a drug to enhance sexual performance, as an intoxicating drug (at rave parties), and as a “date rape” or “knock-out” drug as described in the preceding paragraph. GHB can induce loss of consciousness, coma, seizure activity, respiratory depression, hypotension, a slowing of the heart rate (bradycardia), or sudden death. Aggression, known as “G-rage,” sometimes occurs. Terms used to describe the intoxicating effects of GHB include “carpeting out,” “throwing down,” and “scooping out.” Various GHB analogs exist, including 1,4-butanediol. GHB is a drug that is typically not detected in routine drug screens; consequently, a specific test for GHB should be ordered if it is suspected. In addition, it should be noted that GHB is produced endogenously (within the human body) as a normal breakdown product of gamma-aminobutyric acid. Another unfortunate fact is that the endogenous production of GHB can actually occur after death, such that levels can actually increase as the postmortem interval increases. As such, great care must be taken when interpreting the results of a postmortem GHB test. It is suggested that samples be collected and tested as soon as possible after death if GHB is suspected. Consultation with a forensic toxicologist is encouraged.

Antipsychotic Drugs

Antipsychotic drugs are medications prescribed to psychiatric patients for treatment of various psychiatric disorders, including schizophrenia, bipolar disorder, and mania, amongst others. Some of these drugs are also used for non-psychotic disorders, such as Tourette syndrome and Asperger syndrome. In the past, some antipsychotic drugs were referred to as “major tranquilizers.” Examples of antipsychotic drugs include haloperidol (Haldol), chlorpromazine (Thorazine), prochlorperazine (Compazine), promethazine (Phenergan), zuclopenthixol (Acuphase), clozapine (Clozaril), olanzapine (Zyprexa), risperidone (Risperdal), quetiapine (Seroquel), and aripiprazole (Abilify). Depending on the drug, various toxic effects

may be encountered, including various CNS/behavioral effects and certain cardiovascular effects, including sudden death. Several antipsychotics may be associated with a condition known as “tardive dyskinesia,” characterized by repetitive, involuntary, purposeless movements. Another condition that can develop with many antipsychotic drugs is the “neuroleptic malignant syndrome,” characterized by hyperthermia, muscle rigidity, and delirium. The condition is potentially lethal and is considered an adverse drug reaction. As mentioned above, sudden death occurring with no pre-existing symptoms has also been described in persons taking antipsychotic medications.

Anabolic Steroids

Anabolic steroids are a group of substances that function to “build up” various tissues within the body. There are a variety of legitimate medical uses for these drugs, including certain endocrine abnormalities or to treat body wasting in certain disorders. Most of the general public is aware of anabolic steroids because of the attention they have received within the world of athletics, where steroid abuse is relatively widespread. The drugs are used to increase muscle mass and to enhance athletic performance. Serious side-effects can include liver damage, the growth of various tumors, elevation of blood pressure, increased cholesterol levels, anti-masculine effects in males, pro-masculine effects in females, and abnormal growth and development in adolescents. A variety of research suggests that steroid abuse may predispose an individual toward aggressive behavior, as well as numerous psychiatric side-effects, including mood swings, manic-like symptoms, and depression.

Insulin

Recall from Chapter 10 that insulin is normally produced by beta-cells within the islets of Langerhans in the pancreas. It is a substance that is required by many cells in order to absorb glucose from the blood. In diabetes, particularly type I diabetes, insulin is not produced in sufficient quantities; therefore, patients with type I diabetes typically require insulin replacement therapy. Insulin that is essentially identical to naturally-produced or endogenous insulin, and is produced and sold by various manufacturers, may be referred to as “exogenous” insulin. When insulin is produced endogenously (in the pancreas), another substance called “C-peptide” is produced along with it. C-peptide levels can be measured in the laboratory. If the measured insulin level is high, but the C-peptide level is low, this indicates that the insulin is probably of exogenous origin. If the insulin and C-peptide levels are both elevated, then the insulin is endogenous. Diabetics who require insulin replacement therapy usually self-administer insulin via subcutaneous injection. A typical location for such an injection is the abdomen (Disc Image 11.26). In diabetics on long-term insulin replacement therapy, it is not unusual to have elevated total

insulin levels; however, measurement of “free insulin” levels should be within the normal range. This is because diabetics will frequently produce “anti-insulin antibodies” which bind to and prevent the normal function of insulin. As a result, the total insulin level (composed of bound and free insulin) can be very high, while the amount of functioning (free) insulin is normal.

In deaths where a suspected insulin overdose has occurred, the following tests should be ordered: total insulin, free insulin, C-peptide, anti-insulin antibodies. An insulin-dependent diabetic will often have elevated total insulin levels, low C-peptide levels, and a positive (elevated) level of anti-insulin antibodies. If an insulin overdose has occurred, the free insulin level will be elevated. If free insulin levels are normal, the case is likely not related to an insulin overdose. In a decedent who is not an insulin-dependent diabetic, and has not been receiving insulin for any other condition, it is unlikely that anti-insulin antibodies will be present. Normally, the total and free insulin levels are similar, and the C-peptide is appropriate for the corresponding insulin level. If the person has received exogenous insulin, the insulin (total and free) will be elevated, and the C-peptide level will be low.

Volatiles and Inhalants

Volatiles and inhalants represent a large group of substances that reach the body’s bloodstream via inhalation in the lungs. In some cases, vaporous fumes may actually enter the bloodstream via the nasal passages as well. Volatiles and inhalants are usually gaseous or particulate substances. Gaseous substances include gases and vapors. Particulates include aerosols, mists, fumes, dusts, and smoke. Strictly speaking, smoke from fires and exhaust represent inhalant deaths. As these topics are addressed elsewhere in this text (Chapters 19 and 15, respectively), they will not be described here.

Exposure to or abuse of gases can occur in a variety of ways, including directly from the source, via some type of hose or tubing, or from some type of reservoir, such as a balloon or a bag. Vapors from solvents are typically abused by using one of several methods. “Sniffing” describes a situation where vapors are inhaled directly from the source. “Bagging” describes a method by which a solvent is poured or sprayed into a bag, and then the fumes are inhaled from the bag. “Huffing” is when the drug abuser soaks a cloth with the solvent, then places the cloth over the mouth and nose in order to inhale the fumes.

As with many drug/toxin-related deaths, autopsy findings with inhalant deaths are frequently non-specific; however, some clues might suggest the possibility of inhalant abuse. The presence of pink-white, frothy pulmonary edema fluid exuding from the mouth and nose (foam cone) is a relatively common finding in deaths due to inhalant abuse (Fig. 11.27). Its presence is not specific, as it can be seen in many other conditions, including opiate deaths, drownings, and congestive heart failure. The presence of specific odors on or around the body may suggest the possibility of an inhalant death. Occasionally, residues of the specific substance used, such as paint, may be seen on the fingers, hands, clothing, or face (Fig. 11.28).



Fig. 11.27 A large “foam cone” exuding from the mouth and nose of an individual who died from inhalant abuse



Fig. 11.28 Paint on hands of an individual who died from abusing paint fumes

Many inhalants are not detected in routine toxicology testing, so specially ordered tests are necessary. With some substances, routine blood collection tubes are not sufficient to prevent volatile substances from escaping through the lids of the containers; therefore, special collection containers are in order. Consultation with a forensic toxicologist is recommended. Some pathologists and toxicology laboratories prefer to evaluate lung samples, or even aspirated air samples from within the trachea/bronchi.

Carbon monoxide: Within the death investigation community, carbon monoxide is the most commonly encountered inhalant responsible for acute poisoning fatalities. Since it is considered a “chemical asphyxiant,” carbon monoxide poisoning is described in detail in Chapter 15.

Cyanide and hydrogen sulfide: These gases are also considered “chemical asphyxiants” and are discussed in greater detail in Chapter 15.

Nitrous Oxide

Nitrous oxide (N_2O) is commonly referred to as “laughing gas.” It is a tasteless, colorless, odorless gas that is used as an analgesic/anesthetic agent (especially in dentistry), as a propellant in the food industry (whipped cream canisters) (Disc Images 11.27, 11.28, and 11.29), and as a means of supercharging race engines. It should be considered a potential drug of abuse, as persons have been known to abuse the gas. A certain amount of CNS depressant effect occurs with nitrous oxide. Accidental deaths may occur when an abuser passes out and experiences simple asphyxia (displacement or lack of oxygen) because the plastic bag used to contain the nitrous oxide prevents oxygenation (Figs. 11.29 and 11.30).

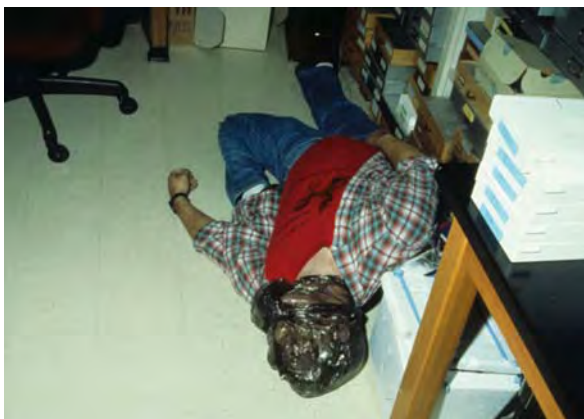


Fig. 11.29 A man found dead in a laboratory with a plastic garbage bag over his head and face. He had been abusing nitrous oxide (laughing gas)

Hydrocarbons

Hydrocarbons represent a large group of liquid and gaseous substances, primarily composed of hydrogen and carbon. Several categories exist, including aliphatic hydrocarbons (methane, propane, butane, hexane) (Fig. 11.31 and Disc Image 11.30), aromatic hydrocarbons (benzene, toluene, xylene), mixed hydrocarbons (gasoline, kerosene, starter fluid, lighter fluid) (Fig. 11.32 and Disc Image 11.31), pine wood distillates (turpentine, pine oil), freons (various fluorocarbons used as refrigerants, propellants, and solvents) (Disc Image 11.32), and various miscellaneous groups, including other chlorohydrocarbons (Disc Images 11.33 and

Fig. 11.30 A small “foam cone” in the man depicted in Fig. 11.29



Fig. 11.31 An elderly woman was found dead in her home, which was filled with natural gas. A stove burner was in the “on” position, but the pilot light was extinguished

11.34). Deaths can be accidental in relation to an accidental exposure, such as may occur in industry, accidental in relation to abuse of the substance (such as occurs in sniffing paint or gasoline), or suicidal. In general, toxicity is related to simple asphyxia (displacement of oxygen), CNS depression, or induction of a lethal arrhythmia. With some, particularly those that are liquids, aspiration of the substance may lead to aspiration pneumonia and subsequent death. As with many other drugs, intoxication may not be the immediate cause of death, but it may play a role in causing or leading to the ultimate cause of death, such as occurs when an intoxicated individual dies from blunt force injuries which might not have been sustained had the individual been sober (Disc Images 11.35).

Fig. 11.32 Mixed hydrocarbons, such as gasoline, may be abused in similar ways as other inhalants



Helium and Other Simple Asphyxiants

Helium and other “simple asphyxiant” gases, such as argon and nitrogen, which have no specific toxic properties in themselves, are occasionally encountered in forensic cases. Death results from displacement of oxygen from the environment and tends to occur in two situations: the first involves drug abusing activity, similar to that described above with nitrous oxide or paint-sniffing. Helium is occasionally used in order to “get a buzz” via the corresponding lack of oxygen (Figs. 11.33 and



Fig. 11.33 This young man was found dead in his apartment. A plastic garbage bag and a helium tank were also present at the scene. Autopsy was negative

11.34). The second situation typically occurs in a setting where an underground or otherwise enclosed space is filled with an inert (non-toxic) gas, via pipes that travel through the space or by some other means, such that oxygen is displaced from the enclosed space. Upon entering the space, the victim immediately collapses and dies due to lack of oxygen. Unfortunately, it is not unusual in these circumstances for one or several other victims to succumb to an asphyxial death as they attempt to rescue the preceding victim(s).

Fig. 11.34 The helium tank found at the scene of the individual shown in Fig. 11.33



Heavy Metals

Arsenic

Arsenic is considered one of several “heavy metals” that can be toxic or lethal. The metal is ubiquitous (existing nearly everywhere) within the environment, but can be more concentrated in certain locations, such as bodies of water, and, hence, in the fish that reside therein. It is also present in a variety of products, including ceramics and certain pesticides. Routes of exposure include ingestion and inhalation. Arsenic poisons various enzymes within the body causing acute and chronic toxicity. Symptoms include abdominal pain, nausea, vomiting, diarrhea, as well as various cardiovascular, respiratory, and CNS effects. Arsenic poisoning may result in a garlic odor of the breath. Chronic poisoning may cause thickened areas of skin, particularly on the palms and soles. A long-term complication is skin cancer. In chronic toxicity, persons may develop transverse white lines on their nails referred to as “Mee’s lines.” Since arsenic is ubiquitous, it is normally present in very low

levels within the body. Normal blood levels range from 0.002 to 0.050 mg/L. Normal hair levels are less than 1 mg/kg.

Cadmium

Cadmium is another ubiquitous metal which is used in various industries. If blood samples for cadmium testing are not collected in specialized tubes, a spurious (false) elevated level of cadmium may be measured, simply related to the fact that regular blood collection tubes may be contaminated with environmental cadmium. If an artifactually elevated level is suspected, tissue (liver) samples can be measured and usually provide a better indication as to whether or not body cadmium levels are truly elevated.

Iron

Another ubiquitous metal, present in food, the environment, and various industries, is iron. Iron is a substance that humans require for normal metabolism. Hemoglobin, as well as other vital substances within our bodies, requires iron. Iron overload is referred to as “hemochromatosis” and is discussed in further detail in Chapter 10. Toxicity is characterized by GI hemorrhage, acidosis, and hemorrhagic pneumonia.

Lead

Lead is relatively ubiquitous as well. It is present in pollution, older paints, certain forms of fuel, batteries, plastics, insecticides, ceramics, and some Chinese herbal medicines. It may be ingested or inhaled. Lead toxicity, or “plumbism,” results in a variety of physiologic effects, including anemia because of decreased hemoglobin production, abnormal calcium metabolism, GI hemorrhage, weakness, neurologic dysfunction (paralysis, encephalopathy), and kidney problems. Normal blood lead levels are less than 10 $\mu\text{g/dL}$ (0.1 mg/L). In cases of lead poisoning, pathologists may see pink inclusions within certain kidney cells under the microscope, as well as protein droplets within the CNS. Burton’s “lead lines” represent blue discoloration of the gums, at the base of the teeth. X-ray examination in children may show radio-dense accumulation of lead within the growth plates at the ends of long bones – these are also referred to as “lead lines.”

Mercury

Mercury is a metal that is a liquid at room temperature. It is present in a variety of substances, ranging from certain medicines to batteries to paint to dental fillings

to fish. It may be ingested or inhaled. The CNS is the primary target of mercury poisoning, causing long-term deficits, including dementia. Other organ systems affected include the kidneys and the GI system.

Other Poisons

Organophosphates

Organophosphates are commonly used as insecticides. They inhibit acetylcholinesterase, which normally functions at nerve endings to break down acetylcholine, which is a neurotransmitter. In the presence of organophosphates, the acetylcholine is not properly metabolized so there is continued nerve transmission, or overstimulation of the target of the nerve ending. This results in a wide variety of symptoms, including sustained muscular contractions for certain muscle groups, depressed muscular function elsewhere, increased secretions, increased sweating, increased GI tract motility, increase urination, respiratory depression, and behavioral and neurologic effects. Organophosphate exposure can be by ingestion, inhalation, or transcutaneously (skin absorption). Cholinesterase levels/activity can be accurately measured in postmortem blood samples. Decreased levels suggest that organophosphate poisoning has occurred.

Strychnine

Strychnine is a very potent poison. It is produced from the plant *Strychnos nuxvomica*, which is native to southern Asia and Australia. Its primary use today is as a pesticide (rat poison). Occasionally, the substance is reported as a contaminant in various street drugs, such as LSD, cocaine, and heroin. Numerous routes of exposure may occur, including ingestion and inhalation. The poison is an inhibitor of a neurotransmitter within the CNS. Acute toxicity is characterized by agitation, being easily startled, restlessness, painful muscle spasms, jaw tightness, and possibly difficult breathing. The urine may become dark. High doses can lead to respiratory failure, neurologic deficits, and death.

Naturally-Occurring Toxins

A variety of other poisons and toxins exist and have been known to cause death. Included here are numerous naturally-occurring toxins, including several that have already been discussed above. Animal venoms represent a type of natural toxin that can cause death. Deaths may be due to the direct toxic effects of these substances, an infection that develops as a result of the corresponding tissue destruction, or as a result of an allergic reaction to the toxin/venom. Animals that produce potentially lethal venoms include certain arthropods (bees, spiders, fire ants, scorpions,

wasps), snakes and other reptiles, and numerous aquatic animals. Numerous plants and fungi contain natural toxins. Besides those that have already been presented above, examples include hemlock, holly, jimsonweed, mistletoe, nightshade, poinsettia, and pokeweed. Ricin, from castor beans, is discussed in Chapter 21, along with other potential “terror agents”.

Disc Image Legends

- Disc Image 11.1 In certain drug-related death scenes, there may be little or no indication that drug use played a role in death.
- Disc Image 11.2 Careful evaluation of the entire death scene, including areas away from the immediate vicinity of the body, may reveal evidence of drug/toxin exposure.
- Disc Image 11.3 Drug paraphernalia found in the clothing of an overdose victim at autopsy.
- Disc Image 11.4 Needle tracks and injection sites may be masked by tattoos, as in the example shown.
- Disc Image 11.5 Another example of needle tracks.
- Disc Image 11.6 Skin-popping scars.
- Disc Image 11.7 Recent superficial wrist incisions in a suicidal overdose victim.
- Disc Image 11.8 Vomit which is discolored yellow from the pills that were intentionally ingested in this suicide victim.
- Disc Image 11.9 Pill dye (pink-red) on the clothing of an overdose victim.
- Disc Image 11.10 The belly of an obese individual who had a rather unique hiding/storage place for his crack cocaine (see Disc Image 11.11).
- Disc Image 11.11 Crack cocaine hidden/stored in the fold of skin/tissue underlying the sizeable anterior abdominal wall of the individual shown in Disc Image 11.10.
- Disc Image 11.12 A long little fingernail, presumably utilized by the decedent during life for delivering powder forms of various drugs to the nostrils.
- Disc Image 11.13 Gastric (stomach) mucosa (lining) discolored green by the pills used in an overdose.
- Disc Image 11.14 Focal green discoloration of the intestines in a case where green-colored pills were used in an overdose.
- Disc Image 11.15 Brain swelling in an overdose. This finding is “non-specific,” meaning that it can occur in a variety of other situations, not just in overdoses.
- Disc Image 11.16 Postmortem bleeding in the lateral (side) aspects of the neck region, secondary to attempting to collect blood from the subclavian blood vessels. Such bleeding can cause difficulties in attempting to document the presence or absence of subtle neck injuries.
- Disc Image 11.17 Urine collection at autopsy.
- Disc Image 11.18 Cerebral spinal fluid (CSF) collection at autopsy, by internal approach. After the organs of the trunk have been removed, and before brain removal, CSF can be withdrawn by an anterior (front) approach, using a needle inserted into the spinal canal through an intervertebral disc.

- Disc Image 11.19 Bile collection from the gallbladder at autopsy.
- Disc Image 11.20 An example of commercially-available antifreeze, which contains ethylene glycol.
- Disc Image 11.21 A normal urine sample (left) compared to a urine sample from someone who died of the toxic effects of ethylene glycol (right), viewed with a Wood's lamp. Note the fluorescent green-yellow color of the sample on the right.
- Disc Image 11.22 An example of the "body packing syndrome," wherein the stomach contains many condoms filled with illegal drugs. The victim died of a massive overdose (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner).
- Disc Image 11.23 A markedly distended urinary bladder (arrows) in a victim of opiate toxicity. This finding is not specific for opiates. It can also be seen with other drugs, most notably ethanol.
- Disc Image 11.24 Plastic wrap containing marijuana.
- Disc Image 11.25 A decedent who was high on LSD when he jumped through a plate glass window, suffering lethal sharp force injuries (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner).
- Disc Image 11.26 Multiple subcutaneous insulin injection sites of the abdomen in a diabetic.
- Disc Image 11.27 A readily-available whipped cream container.
- Disc Image 11.28 A close-up of the ingredients of the whipped cream container shown in Disc Image 11.27. Note that the whipping gas is nitrous oxide.
- Disc Image 11.29 A commercially available nitrous oxide tank.
- Disc Image 11.30 A suicide death scene. The victim's head is totally within a black garbage bag. Note that a hose connects the inside of the bag to a wall-mounted natural gas supply.
- Disc Image 11.31 A child victim of gasoline inhalational abuse. Note the extensive pulmonary edema fluid on the face.
- Disc Image 11.32 A man found in the back of his van, with his head/face within a clear plastic bag, which he had filled with freon gas. Note the green freon gas container in the foreground of the photo.
- Disc Image 11.33 A young man found dead in his residence. The scene findings shown in the accompanying image (Disc Image 11.34) clearly indicate the likely cause of death (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner).
- Disc Image 11.34 Numerous empty containers of correction fluid (trichloroethane) and plastic bags utilized by the victim shown in Disc Image 11.33 (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner).
- Disc Image 11.35 A victim of a slow-speed train versus pedestrian collision. The engineer claimed that the victim was "acting drunk" prior to being struck by the locomotive engine. Note the spray-paint can near the body. There was spray paint on the victim's face and hands. Toxicology revealed the presence of toluene in his blood.

Selected References

- Armstrong EJ, Engelhart DA, Jenkins AJ, Balraj EK. Homicidal ethylene glycol intoxication – a report of a case. *Am J Forensic Med Pathol* 2006;27:151–5.
- Baselt RC. *Disposition of Toxic Drugs and Chemicals in Man* (seventh edition). Foster City, CA; Biomedical Publications; 2004.
- Batalis NI, Prahlow JA. Accidental insulin overdose. *J Forensic Sci.* 2004;49:1117–20.
- Denton JS, Donoghue ER, McReynolds J, Kalelkar MB. An epidemic of illicit fentanyl deaths in Cook County, Illinois: September 2005 through April 2007. *J Forensic Sci* 2008;53:452–4.
- Drummer OH. *The Forensic Pharmacology of Drugs of Abuse*. London, England: Arnold; 2001.
- Gill JR, Stajic M. Ketamine in non-hospital and hospital deaths in New York City. *J Forensic Sci* 2000;45:655–8.
- Hirsch CS, Adelson L. Ethanol in sequestered hematomas. *Am J Clin Pathol* 1973;59:429–33.
- Jentzen JM, Mont EK, Revercomb C. Volatiles and inhalants (chemical asphyxia) (Chapter 26). In: Froede RC (editor). *Handbook of Forensic Pathology* (2nd edition). Northfield, IL: College of American Pathologists; 2003:237–42.
- Karch SB. Drug deaths (Chapter 25). In: Froede RC (editor). *Handbook of Forensic Pathology* (2nd edition). Northfield, IL: College of American Pathologists; 2003: 231–6.
- Kugelberg FC, Jones AW. Interpreting results of ethanol analysis in postmortem specimens: a review of the literature. *Forensic Sci Int* 2007;165:10–29.
- National Institute on Drug Abuse. www.nida.nih.gov
- O’Neal CL, Poklis A. Postmortem production of ethanol and factors that influence interpretation – a critical review. *Am J Forensic Med Pathol* 1996;17:8–20.
- Pestaner JP, Southall PE. Sudden death during arrest and phencyclidine intoxication. *Am J Forensic Med Pathol* 2003;24:119–22.
- Prahlow JA. Deaths due to animals, plants, and other natural environmental hazards (Chapter 37). In: Froede RC (editor). *Handbook of Forensic Pathology* (2nd edition). Northfield, IL: College of American Pathologists; 2003:361–74.
- Ruttenber AJ, Lawler-Heavner J, Yin M, Wetli CV, Hearn WL, Mash DC. Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. *J Forensic Sci* 1997;42:25–31.
- Sheil AT, Collins KA, Schandl CA, Harley RA. Fatal neurotoxic response to neuroleptic medications – case report and review of the literature. *Am J Forensic Med Pathol* 2007;28:116–20.
- Shields LB, Hunsaker JC, Corey TS, Ward MK, Stewart D. Methadone toxicity fatalities: a review of medical examiner cases in a large metropolitan area. *J Forensic Sci* 2007;52:1389–95.
- Smialek JE, Spitz WU, Wolfe JA. Ethanol in intracerebral clot. Report of two homicidal cases with prolonged survival after injury. *Am J Forensic Med Pathol* 1980;1:149–50.
- Westveer AE, Trestrail JH, Pinizzotto AJ. Homicidal poisoning in the United States: an analysis of the Uniform Crime Reports from 1980 through 1989. *Am J Forensic Med Pathol* 1996;17:282–8.
- Wetli CV, Mittleman RE. The ‘body packer syndrome’ – toxicity following ingestion of illicit drugs packaged for transportation. *J Forensic Sci* 1981;26:492–500.
- Wetli CV. Fatal cocaine intoxication. A review. *Am J Forensic Med Pathol* 1987;8:1–2.
- Wick R, Gilbert JD, Felgate P, Byard RW. Inhalant deaths in South Australia: a 20-year retrospective autopsy study. *Am J Forensic Med Pathol* 2007;28:319–22.
- Winston DC. Suicide via insulin overdose in nondiabetics: the New Mexico experience. *Am J Forensic Med Pathol* 2000;21:237–40.
- www.streetdrugs.org
- www.whitehousedrugpolicy.gov/streetterms
- www.drugfree.org

Chapter 12

Blunt Force Injury Deaths

If a man strikes someone with an iron object, . . . a stone, . . . a wooden object, . . . and he hits someone so that he dies, he is a murderer.

Numbers 35:16–18

Abstract Chapter 12 deals with some of the most common deaths encountered by death investigation offices, namely blunt force injuries. The chapter includes discussion of the classification of blunt force injury types (abrasions, contusions, lacerations, fractures, and avulsions), followed by a relatively detailed discussion of blunt trauma of the head and neck. The final section of the chapter deals with special topics related to blunt force injuries and includes mechanisms of death, delayed deaths, patterned injuries, clothing examination, and specific subtypes of blunt force injury.

Keywords Blunt force · Head trauma · Craniocerebral trauma · Brain injury · Patterned injuries

Introduction

One of the most common injury types that result in traumatic death is blunt force injury, defined as an injury resulting from impact with a blunt object. A blunt object can be considered a non-sharp object, such as floors, walls, furniture, hammers, baseball bats, fists, the interior surfaces within an automobile, roadways, trees, and even fluid objects, such as bodies of water. There are five basic types of blunt force injuries (described below), and each is related to the transfer of force from the blunt object onto or into the body. It is important to recognize that with many blunt force injury cases, particularly those that occur in relation to vehicular collisions and falls/jumps from heights, forces associated with sudden deceleration (or acceleration in certain motor vehicle-related cases) may significantly contribute to the overall injuries. Consequently, the term “blunt force injury” should be understood to include deceleration/acceleration forces in some instances.

Classification of Blunt Force Injuries

As mentioned above, there are five basic types of blunt force injury: abrasions, contusions, lacerations, fractures, and avulsions. A “chop wound,” which will be discussed in Chapter 13, represents a special type of injury which is best considered a combination of blunt force and sharp force injury. It should also be noted that, in many other injury types (sharp force, gunshot, asphyxial, electrical, etc), certain cases may have associated blunt force injuries, including some associated directly with the non-blunt force mechanism of injury.

Abrasions

Abrasions are also known as scrapes or scratches. Actual tissue disruption occurs on the surface of the epidermis, frequently with injuries extending into the underlying dermis. Abrasions can be linear, rounded, irregular (Figs. 12.1 and 12.2 and Disc Images 12.1 and 12.2), or of various specialized types (see below). Very superficial abrasions (those that do not extend into the dermis) may not bleed. Abrasions only occur at the site of impact with a blunt object; although they do not occur at *every* blunt impact site. Occasionally, the directionality of the applied force can be determined based on the observation of “peeled-off” superficial layers of epidermis adherent to the skin surface of the abrasion, and located away from the origin of the force (Disc Image 12.3). Friction abrasions (for example, rug burns) are another type of abrasion. “Road rash” is a term used to describe extensive friction abrasions that occur when a victim’s body hits a roadway (pedestrian or cycle rider or person ejected from a vehicle) (Fig. 12.3). Certain abrasions may “take on” the shape of the blunt object; these are referred to as “patterned injuries” (see below).



Fig. 12.1 An example of many different types of abrasions, including linear and rounded



Fig. 12.2 Another example with abrasions of various sizes and shapes



Fig. 12.3 An example of extensive abrasions on the trunk of a pedestrian, sometimes referred to as "road rash."

Fresh abrasions appear red and moist, although postmortem drying frequently causes them to have a dark, dry appearance. Healing, with scab formation, indicates subacute or remote (healing) abrasions. Some pathologists refer to scabbed abrasions as "crusted" abrasions.

Microscopically, the presence of healing may reveal that the injury did not immediately precede death. The proliferation of collagen-producing cells (fibroblasts), excess collagen fibers, inflammatory cells, and a breakdown product of red blood cell hemoglobin referred to as "hemosiderin" are all indicators of healing.

Contusions

Contusions are also known as bruises. Various other terms are sometimes used to describe contusions, including “ecchymosis.” However, strictly speaking, the term “ecchymosis” refers to any situation where blood escapes into soft tissues from blood vessels (including various natural disease processes); whereas the term “contusion” implies a traumatic cause. Unlike abrasions, which only occur at the site of blunt force impact, contusions can occur at sites of impact (Fig. 12.4 and Disc Image 12.4), as well as at sites distant from the impact site. In those that occur at the site of blunt impact, the contusion may or may not have associated epidermal abrasions and/or lacerations. Occasionally, a skin contusion may be subtle. Incising the contusion can “prove” that the bruise is real, as the underlying hemorrhage within the subcutaneous fat will be readily apparent (Disc Image 12.5). Elderly individuals frequently have numerous bruises involving their fragile forearm skin (Disc Image 12.6). Classic examples of contusions occurring away from the site of impact include so-called “raccoon’s eyes” (bilateral periorbital ecchymosis, or two “black eyes”), occurring as a result of basilar skull fractures (Fig. 12.5), so-called Battle’s sign (bruising over the “mastoid process,” the skull protrusion evident behind the ears), also occurring as a result of basilar skull fractures (Disc Image 12.7), Grey Turner’s sign (flank or side of abdomen ecchymosis), occurring with extensive retroperitoneal (behind abdominal cavity) bleeding (Disc Image 12.8), and Cullen’s sign (periumbilical or around the belly button ecchymosis), occurring when there is extensive internal abdominal hemorrhage. Occasionally, contusions are patterned (see below).



Fig. 12.4 An impact contusion of the chest

The appearance of a contusion is not always immediately visible. The color changes that occur in contusions and are frequently described in various textbooks are not reliable in attempting to determine the age of a bruise. Various factors



Fig. 12.5 Bilateral periorbital ecchymoses (raccoon's eyes), resulting from basilar skull fractures that were sustained in a fall

play roles in the color of a bruise, including extent of hemorrhage, depth of hemorrhage, tissue location of hemorrhage, etc. Most forensic pathologists can recall examples of bruises less than 24 hours old that have a yellow appearance, which, according to some, suggests older injuries. Consequently, it is not advisable to estimate the age of a contusion based solely on its color (Disc Image 12.9). Microscopically, changes of healing may be seen; however, absolute determinations regarding the age of such injuries is generally not possible. Contusions may also occur in internal organs (Disc Image 12.10).

Lacerations

A laceration is a splitting apart of tissues, usually resulting from blunt force (or deceleration/acceleration) injuries. On the skin surface, lacerations may or may not be associated with abrasions and/or contusions (Fig. 12.6 and Disc Images 12.11 and 12.12). Lacerations may be linear, jagged, irregularly-shaped, or occasionally patterned (see below). Linear lacerations may occasionally be confused with sharp force injuries. A feature that favors a diagnosis of a laceration versus a sharp force injury is the presence of "tissue bridging," which describes the presence of intact nerves, blood vessels, and other strands of tissue that "bridge the gap" between the two sides of the laceration, deep to the skin surface (Fig. 12.7 and Disc Image 12.13). Tissue bridging tends not to occur with sharp force injuries, as these structures would likely be severed along with the skin and underlying soft tissues. Other features that tend to occur with lacerations include abrasions and contusions, although these may also occur with sharp force injuries. If the direction of force that causes a laceration is angled rather than directly perpendicular to the skin surface, there will frequently be an abrasion on the side of the laceration from which the

Fig. 12.6 A laceration of the scalp. Note the somewhat jagged appearance, as well as the marginal abrasions



Fig. 12.7 Another scalp laceration, with minimal marginal abrasions. Note the presence of tissue bridging toward the left side of the laceration

force is applied. The opposite side may demonstrate “undermining,” the presence of a cavity underlying the skin (Disc Images 12.14 A, B).

Because of the fact that it is sometimes difficult to differentiate a laceration from a sharp force injury, many persons within healthcare professions misuse the term “laceration,” referring to true sharp force injuries as lacerations. The term should be reserved for blunt force injuries.

Lacerations frequently occur at sites of blunt force impact; however, they can also be found away from the site of impact. The classic example occurs in pedestrians struck from behind by a motor vehicle, where decedent’s frequently have



Fig. 12.8 Numerous stretch lacerations of the inguinal (groin) region in a pedestrian struck from behind. These lacerations are yellow, indicating that the blood pressure immediately dropped on impact (due to severe internal injuries)

stretch-type lacerations in their inguinal (groin) regions (Fig. 12.8), related to hyperextension that results from excessive force applied from behind.

Lacerations from blunt force (and acceleration/deceleration) injuries are also common within internal organs and tissues. They frequently occur in the aorta (Fig. 12.9 and Disc Image 12.15), the lungs (Disc Image 12.16), the liver (Fig. 12.10 and Disc Image 12.17), and the spleen (Disc Image 12.18), but can occur in virtually any organ or tissue (Disc Image 12.19). Lacerations frequently involve the surface of organs; however, internal lacerations may occur without overlying surface injuries.

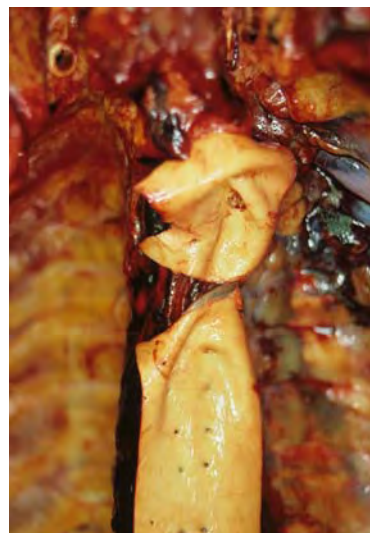


Fig. 12.9 An aortic laceration sustained in a sudden deceleration/blunt impact incident (motor vehicle collision)



Fig. 12.10 Multiple liver lacerations

Fractures

The breaking of a bone (or cartilage) is referred to as a fracture. There are a variety of fracture types and several methods of categorizing them. One classification scheme is based on skin involvement. If the skin overlying a fracture is intact, the fracture is referred to as a “closed fracture” (Fig. 12.11). In contrast, if the skin overlying the fracture is lacerated, the fracture is referred to as an “open fracture” or a “compound fracture” (Fig. 12.12). Open fractures are more susceptible to infection.



Fig. 12.11 A closed humerus (upper arm bone) fracture

Another system that can be used to classify fractures describes the characteristics of the fracture itself. A “simple” or “complete” fracture describes a linear (straight line) fracture that is relatively perpendicular to the long axis of the bone shaft, and involves both sides (both cortices) of the bone. An “incomplete” fracture does not involve both sides of the bone shaft. “Greenstick” fractures are incomplete fractures where the bone, usually in a child, is essentially “bent.” A “buckle” fracture



Fig. 12.12 An open (compound) fracture of the tibia (shin bone)

is an example of an incomplete fracture where one cortex is essentially collapsed or buckled. A “spiral” fracture has a helical shape, twisting along the long axis of the bone shaft. Depending on the circumstances, spiral fractures may suggest the possibility of child abuse. A “comminuted” fracture can be thought of as a fracture with multiple fragments of bone. Another fracture type occurring in children that suggests the possibility of child abuse is the epiphyseal plate fracture. Depending on the X-ray view of this type of fracture, epiphyseal fractures are variably referred to as “bucket-handle” or “corner” fractures (refer to Chapter 20).

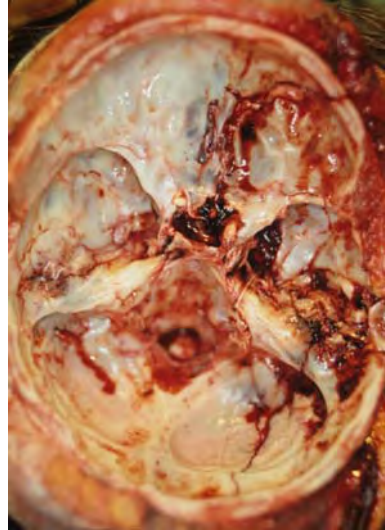
Rib fractures are common in blunt force injury deaths (Disc Image 12.20), as are skull fractures. Skull fractures can be “linear” (Fig. 12.13), “depressed” (indented



Fig. 12.13 Multiple linear and curvilinear skull fractures

inward), or comminuted (Disc Image 12.21). “Basilar” skull fractures involve the base of the skull (Fig. 12.14) and have a variety of subtypes (described below). “Diastatic” fractures represent a splitting apart of a child’s skull suture lines (where the skull plates join each other).

Fig. 12.14 Multiple basilar skull fractures, predominantly on the right side



Fractured bones that are in the process of healing can be differentiated from fractures that occurred immediately prior to (or coinciding with) death. Evidence of healing can be visualized by X-ray as well as grossly and microscopically (Disc Image 12.22).

Avulsions

An avulsion injury represents a blunt force injury in which a portion of a body part (or tissue/organ) substantially separates from or totally separates from the body (or tissue/organ) (Fig. 12.15). Amputation injuries are considered a type of avulsion injury in which an entire extremity or portion thereof is severed from the body (Disc Image 12.23). The most extreme example of an avulsion injury is a decapitation injury, in which the head separates from the body (Fig. 12.16). Not all such injuries are the result of blunt force (some are due to sharp force injuries); however, a significant percentage of these types of injuries are related to severe blunt force/deceleration force. Total body transection may also occur (Disc Image 12.24 and 12.25). Internal organ avulsion injuries may also occur, particularly when severe blunt force/deceleration occurs. Examples include avulsion injuries of portions of organs, such as the liver, or entire organs, such as the heart and lungs.



Fig. 12.15 An avulsion injury of the skin of the face, sustained in a pedestrian versus motor vehicle collision

Fig. 12.16 A decapitation injury sustained in a motorcycle versus fixed object collision



Blunt Force Head and Neck Trauma

A significant percentage of deaths due to blunt force trauma are related to head and brain injuries. As such, it is appropriate to discuss some special issues related to blunt force head trauma. It is useful to consider head injuries in a relatively organized fashion, beginning from the external skin surface and working inward to the brain itself. It should be noted that several other terms can be applied to certain head injuries. The term “craniocerebral trauma” can be used to describe the presence of skull (“cranio”) and brain (“cerebral”) injury. If there are no skull fractures, brain

injuries are sometimes referred to as “closed head injuries.” When neck and upper spinal cord injury play a role in death, a term describing these injuries can also be included in the overall description of injuries (“craniocerebral and neck trauma”).

Skin and Mucosal Injuries

Skin injuries, including abrasions, contusions, and lacerations, can occur on the scalp as well as the face. Their location, size, and shape should be noted. Hair (on the head and/or face) can sometimes hamper the visualization of these injuries. In certain cases, the hair should be shaved, in order to adequately evaluate and document the skin injuries (Fig. 12.17). Not every blunt force impact of the head/face (or anywhere else, for that matter) will result in skin surface injuries, and it is possible to sustain blunt force head injuries without any external evidence of injury. Mucous membrane injuries, including eye and intraoral (inside the mouth) trauma, can also result from blunt force.



Fig. 12.17 The hair overlying this blunt force head injury has been shaved in order to better visualize the wound. Note the presence of abrasion, contusion, and multiple lacerations

Subcutaneous Injuries

Subscalpular subcutaneous tissues: Upon scalp reflection during autopsy performance, pathologists are able to visualize the subscalpular (or “subgaleal”) soft tissues, which are primarily composed of fat (adipose tissue) and muscle. The most common blunt force injury in this location is the contusion (bruise), which is typically described as “subscalpular (subgaleal) hemorrhage” (Fig. 12.18). Another type of injury that can involve the subscalpular soft tissues is a laceration; most of these also involve the overlying skin surface. It is not infrequent for pathologists to identify definite subscalpular hemorrhage without associated overlying skin

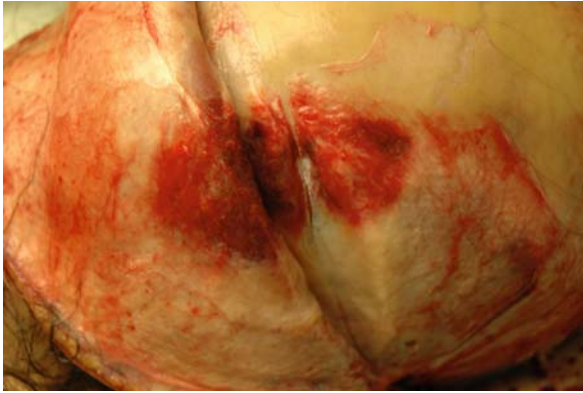


Fig. 12.18 A focal subscalpular (subgaleal) hemorrhage indicating a site of scalp impact

injuries. Most areas of subscalpular hemorrhage associated with blunt trauma should be considered an indication that an impact has occurred in that location. An exception is when there is hemorrhage that arises from a fractured skull, with seepage of blood into the layers of subscalpular soft tissues that immediately overlie the fracture site.

Facial subcutaneous tissues: Occasionally, pathologists may choose to examine the subcutaneous soft tissues of the face, looking for trauma. In order to do this, the pathologist must perform a “facial peel-down” examination, wherein the skin and superficial subcutaneous tissues are dissected downward over the face. Such an examination tends to be very destructive from the funeral home standpoint, and the performance of a facial peel-down should only be performed when absolutely necessary.

Skull and Facial Bone Injuries

During the performance of a routine forensic autopsy examination, excellent visualization of the skull is of paramount importance. Prior to cutting the skull cap off, the skull should be visually inspected and palpated for the presence of fractures. If need be, the muscle and periosteum (soft tissue adherent to the bone) of the skull can be removed in order to better visualize the outer cortical surface of the bone. Once the skull cap and brain have been removed, the dura mater should be stripped away from the bone, so that the inner cortical surface of the skull can be seen. Skull fractures may be linear, comminuted, nondisplaced, displaced, and/or depressed. Diastatic skull fractures occur in young children when intracranial pressure is so great that the unfused skull suture lines separate from one another. It is important to note that the presence of a skull fracture does not necessarily mean that there is underlying brain injury. Alternatively, it is important to note that lethal brain injury can, and frequently does, occur without associated skull fractures.

Skull fractures may involve the crown, the sides, the front, the back, or the base. Those occurring in the base are referred to as “basilar skull fractures” and can occur anywhere within the base of the skull, including the front third (anterior cranial fossa), middle third (middle cranial fossa), or back third (posterior cranial fossa). The basilar skull within the anterior cranial fossa, overlying the eyes, tends to be very thin and, as such, this area can be more prone to fracture (Disc Image 12.26). Fractures at this site will frequently lead to “periorbital ecchymosis,” as described above. If a basilar skull fracture extends all the way from one side to the other, across the middle cranial fosse area, such a fracture is frequently referred to as a “hinge fracture” (Fig. 12.19); this frequently occurs with side impacts. In persons who sustain significant force on the crown (top) of their head, or those who fall or jump from a height, landing on their feet or buttocks, with significant amounts of force being driven upward, along the spinal column onto the base of the skull, a “ring fracture” can be present (Disc Image 12.27). In this basilar skull fracture, there is a circular or oval fracture of the base of the skull, surrounding the foramen magnum (the “hole” in the base of the skull where the spinal cord attaches to the brain). Although ring fractures classically occur when forces are applied from above or below, they may also occur with chin impacts, or with virtually any other direction of impact.

Fig. 12.19 A “hinge fracture” of the base of the skull resulting from a side impact



In routine autopsies, facial fractures are identified by palpation (feeling them). In rare cases, a facial peel-down may be performed in order to actually visualize the skeletal trauma. Jawbone (mandible) fractures are usually easy to palpate, as are cheekbone (zygomatic) fractures.

Epidural, Subdural, and Subarachnoid Hemorrhage

After the skull cap has been removed, the presence of hemorrhage inside the skull but outside of the brain should become evident. There are three types of hemorrhage that can result from blunt force head trauma: epidural, subdural, and subarachnoid. The dura mater represents a relatively tough membrane that adheres to the inside of the skull. Bleeding can occur between the skull and the dura, the so-called epidural area, or it can occur between the dura and the brain, the “subdural space.” The brain itself is covered by the very thin membrane called the meninges or arachnoid membrane. Cerebral spinal fluid is present underlying this membrane, but outside of the brain. When a brain is removed at autopsy, the arachnoid membrane remains covering the brain. If hemorrhage occurs underneath the arachnoid membrane, but outside of the brain, it is referred to as a “subarachnoid” hemorrhage.

Epidural hemorrhage: An epidural hemorrhage occurs between the skull and the dura. Normally, the dura is attached fairly well to the skull, and there is no real “space” between the two structures. If a skull fracture occurs, blood can get in between the skull and dura, creating an epidural hemorrhage. The usual circumstance is a temporal bone (side of head) skull fracture, in which the “middle meningeal artery” is injured. The high pressure within the injured artery is of sufficient strength to peel the dura away from the bone, creating a pocket of epidural blood (epidural hematoma) (Fig. 12.20). These can become quite large and life-threatening, secondary to the accompanying brain compression. The classic scenario is that a person sustains a blow to the side of the head and then has a “lucid” interval, where they remain conscious and alert, only to be followed by loss of consciousness some time later, as the blood eventually works its way between the skull and dura. The epidural is the least common of the three types hemorrhages that

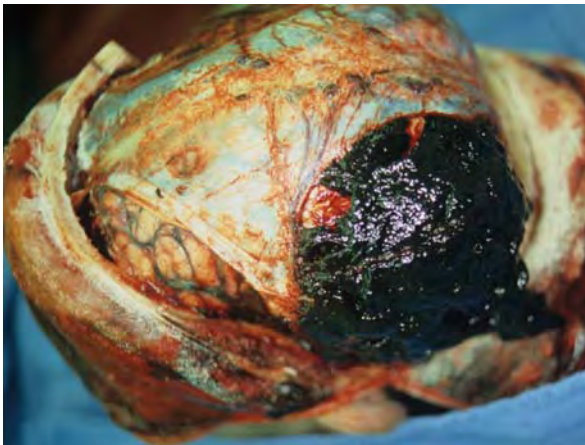


Fig. 12.20 An epidural hemorrhage. Note that the collection of blood is above the level of the dura (a portion of the dura on the left side of the image is reflected, exposing the underlying brain)

can occur inside the skull but outside the brain. Of note is the fact that artifactual epidural hematomas can occur in fire victims, related to heat-induced postmortem skull fractures.

Subdural hemorrhage: A subdural hemorrhage occurs when the “bridging veins” that normally connect the underlying arachnoid membrane to the overlying dura are torn as a result of blunt force trauma or sudden acceleration/deceleration (Disc Image 12.28). The ensuing hemorrhage accumulates within the “subdural space” (Fig. 12.21 and Disc Images 12.29, 12.30, and 12.31). If the hemorrhage is large enough, it can cause brain compression and possibly death. Most subdural hemorrhages are not large enough to cause such brain compression; however, the presence of any amount of subdural hemorrhage is usually interpreted by forensic pathologists as an indicator that the amount of force sustained by the individual was likely sufficient to cause lethal brain injuries. Having said this, it is entirely possible for individuals to survive a subdural hemorrhage. The location of a subdural hemorrhage does not necessarily correlate to the location of the blunt force impact site; in many instances, there is absolutely no correlation.

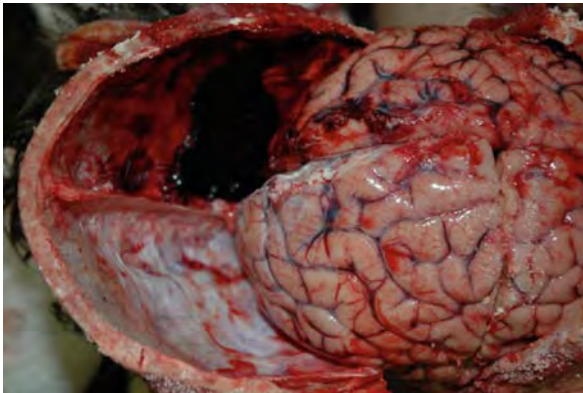


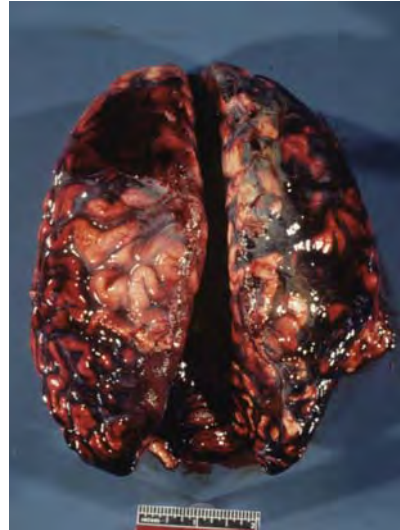
Fig. 12.21 An acute subdural hemorrhage. The dura in this case has remained adherent to the inner aspect of the skull

“Healing” subdural hemorrhages can be seen at autopsy. These “subacute” or “remote” subdurals demonstrate various changes in their appearance, both grossly and microscopically, such that a very rough estimate of the age of the subdural hemorrhage can be ascertained. The gross appearance of healing involves the development of what is referred to as a “neomembrane” (Disc Images 12.32 and 12.33). This is frequently accompanied by yellow discoloration, resulting from the breakdown of hemoglobin within the red blood cells contained within the subdural. Microscopically, as the subdural heals, a process referred to as “organization” occurs. The presence of collagen-producing fibroblasts, collagen fibers, and the red blood cell breakdown product “hemosiderin” are features that indicate that the subdural hemorrhage is organizing.

If a death is delayed for several days or longer following injury, collecting subdural blood at autopsy for subsequent toxicology testing may allow for identification of ethanol and/or other drugs that were present at the time of the injury.

Subarachnoid hemorrhage: The final type of hemorrhage that can occur outside of the brain but inside the skull is the subarachnoid hemorrhage, where bleeding occurs underneath the thin arachnoid membrane (Fig. 12.22 and Disc Images 12.34 and 12.35). Such hemorrhages will not wash away when the brain is rinsed with water. Like subdural hemorrhages, when a pathologist sees a subarachnoid hemorrhage at autopsy in cases of blunt force trauma, it is usually considered an indicator that lethal brain injury may have occurred. Subarachnoid bleeding can be minimal, severe, or anywhere in between. It may be localized, multifocal, or diffuse. Like the subdural, the location does not necessarily correlate with the site of impact in blunt force trauma. Of the three hemorrhage types described, the subarachnoid is the most likely to have a non-traumatic cause. Various natural disease processes, most notably a ruptured berry aneurysm, can cause significant subarachnoid hemorrhage.

Fig. 12.22 Extensive subarachnoid hemorrhage surrounding the brain



Gross Brain Injuries

If a death from blunt force injuries results from head trauma, the mechanism of death in most cases is related to traumatic brain injury. There are a variety of blunt force injury types that may affect the brain, including lacerations, contusions, avulsion injuries, and a specific injury type referred to as “traumatic axonal injury.” These and other mechanisms of injury are discussed below. Whether or not such injury can actually be visualized at autopsy depends on several variables, including how

quickly the death occurred. This last point is especially important. In numerous cases of death due to blunt force head trauma, the only intracranial injuries that are evident at autopsy include subdural and subarachnoid hemorrhage. None of the other injury types described below are evident. In these cases, it is presumed that some of these brain injuries actually occurred, but because death occurred so quickly, they are not evident by gross or microscopic examination. In this regard, subdural and subarachnoid hemorrhage may be considered “markers” or indicators that lethal brain trauma may have occurred.

As with other organs and tissues, the brain can be lacerated. As with lacerations elsewhere, brain lacerations are evident as splitting apart of the brain substance. These injuries can be difficult to identify at autopsy, often because there are numerous other injuries, such as contusions. Extensive lacerations may ultimately result in avulsion injuries. The usual situation where brain avulsion occurs involves severe, open skull fractures. In infants, lacerations from blunt force injuries may be more common than contusions, whereas in older individuals, contusions are much more common.

Brain contusions represent areas of hemorrhage within the brain resulting from trauma. There are several different types of brain contusions, based on location and/or mechanism of injury. Many contusions occur right at the surface of the cerebral cortex (the “crests” of the gyri), in areas closest to the overlying skull. Grossly, they appear as multiple, relatively small (several millimeters long), linear areas of hemorrhage with the long axes perpendicular to the surface of the brain (Fig. 12.23). Frequently, there is associated overlying subarachnoid hemorrhage. Cerebral contusions commonly occur immediately underlying a skull fracture site, and are referred to as “fracture” contusions. If a cerebral contusion occurs directly under a scalp impact site (without an associated fracture), the cerebral contusion is referred to as a “coup” contusion but if it occurs in a location opposite from the side of impact, the injury is referred to as a “contrecoup” contusion. Both coup and contrecoup



Fig. 12.23 Multiple cerebral cortical contusions in a formalin-fixed brain

contusions may occur with a single impact. The coup contusion tends to be more severe than the corresponding contrecoup contusion if a relatively stationary head is struck by a moving object (such as a 2×4). In contrast, the contrecoup contusion tends to be more severe than the corresponding coup contusion if a moving head (such as occurs in a fall) strikes a stationary surface (such as a concrete floor).

There are three additional types of cerebral contusions. “Intermediary” contusions represent contusions within the deep structures of the brain, along a line starting at the point of scalp impact and running in the direction of the force of impact (Disc Image 12.36). One of the most common scenarios is when an impact occurs on the top of the head.

“Gliding” contusions are considered by some to represent hemorrhage related to shearing forces. They tend to occur in the white matter and adjacent gray matter of the cerebral cortex (Disc Image 12.37), but they can occur elsewhere within the white matter. Grossly-evident white matter contusions associated with traumatic axonal injury (see below) are considered by some to represent gliding contusions. In contrast to intermediary contusions, gliding contusions do not occur along the long axis of a line drawn to coincide with the direction of force but originate at the site of impact.

The final contusion type is the “herniation” contusion. When severe brain injuries are sustained and death does not occur immediately, the brain responds in many ways, including the development of severe swelling (fluid accumulation, or “edema”) at the location of injury. This swelling can cause the brain to become extremely compressed within the skull. Another cause for traumatic brain compression is related to brain-shifting occurring as a result of a large hemorrhage, such as can occur with certain subdural or epidural hemorrhages. When brain compression occurs, the brain substance will move toward areas of least resistance. There are basically four places where the swollen brain may “herniate” (protrude). One is underneath the falx cerebri, the part of the dura that separates the two cerebral hemispheres. The specific brain gyrus that herniates under the falx cerebri is the “cingulate” gyrus, so this type of herniation is called a “cingulate gyrus herniation” or a “subfalcine” herniation (Disc Image 12.38). The second type involves the “uncus” of the lower central cerebrum herniating over the tentorium (the part of the dura that separates the overlying cerebrum from the underlying cerebellum). This type of herniation is called an “uncal” or “transtentorial herniation” (Disc Image 12.39 and 12.40). The third type of herniation involves the lower, central, midline parts of the cerebellum herniating downward into the foramen magnum. This type of herniation is called “tonsillar herniation” (Disc Image 12.41). The final type of herniation occurs when brain tissue herniates through a defect in the skull. This type of herniation is called a “transcalvarial herniation” (Disc Image 12.42). With each type of herniation, brain tissue can be compressed to such an extent that tissue damage and hemorrhage occurs; these areas of hemorrhage are referred to as “herniation” contusions.

“Duret hemorrhages” represent multifocal areas of hemorrhage within the brainstem (pons and midbrain) (Fig. 12.24) that occur as areas of secondary hemorrhage, following severe brain injury with subsequent marked brain swelling, or related

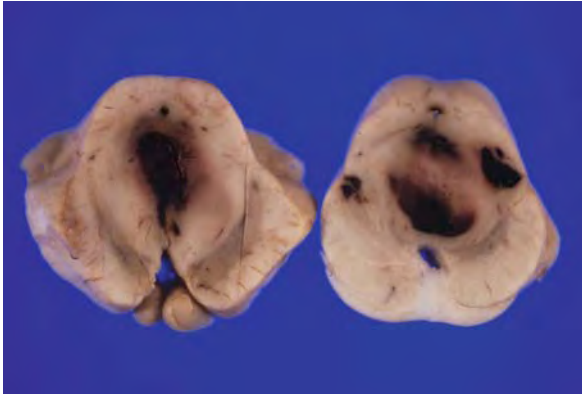


Fig. 12.24 Duret hemorrhages within the midbrain of a person who sustained severe head trauma, but survived for several hours prior to death

to brain-shifting resulting from an epidural or subdural hemorrhage. Similar to the development of herniation contusions, the development of Duret hemorrhages requires some amount of survival following the initial injury.

In rare cases, the only intracranial injury identified at autopsy is a markedly swollen brain (Fig. 12.25). The swelling, or edema, may be diffuse or it may be localized to a single side or portion of the brain, with an associated “midline shift.” The condition is referred to as “malignant cerebral edema.”



Fig. 12.25 Widespread, severe, cerebral edema (brain swelling)

Microscopic Brain Injuries

As alluded to above, when certain lethal brain injuries occur, there are no immediately recognizable gross or microscopic injuries within the brain itself (but usually, there *is* evidence of subdural and/or subarachnoid hemorrhage). If survival is long

enough for cerebral contusions to be evident on gross examination, then microscopic examination will be characterized by blood extravasation (blood cells located outside of blood vessels), with or without associated brain tissue disruption.

The term “diffuse traumatic brain injury” has been used to describe another type of injury that is associated with trauma. From a clinical standpoint diffuse traumatic brain injury represents a spectrum of severity, ranging from concussions, where there is altered or temporary loss of consciousness, to transient unconsciousness with residual permanent brain injury, to severe but non-lethal brain injury in association with coma or persistent vegetative state, to unconsciousness with a period of survival followed by eventual death, to such severe initial injury that death occurs at or very shortly after the injury occurs. With each of these situations, it is believed that traumatic injury of the axons within the brain occurs. The term used to describe this type of injury is “diffuse axonal injury” (DAI). Some advocate the use of the term “traumatic diffuse axonal injury” in order to differentiate the traumatic injury from axonal “injury” that occurs as a result of ischemia (lack of blood flow), which can accompany trauma or be caused by other disorders. The latter is referred to as “ischemic” or “vascular” axonal injury.

Axons can be thought of as extensions that arise from neurons (brain cells) and connect to other neurons. The white matter of the brain is primarily composed of many axons. When traumatic DAI occurs, shearing forces result in the disruption of axons, with associated central nervous system symptoms. The specific mechanism involved in the disruption of the axons is thought to be related to rotational and acceleration/deceleration forces, each of which may occur in association with blunt force head injuries. With concussions, the axonal damage is considered largely reversible; however, at the other end of the spectrum, when sufficient vital axons are injured to a severe enough degree then death can occur very quickly. Unfortunately, it is not possible to visualize axonal injury under the microscope if death occurs at or around the time of injury. In fact, many hours (>12 hours or longer) of survival may be necessary before the injuries can be recognized under the microscope using routine staining methods. However, a specialized immunohistochemistry stain known as beta-amyloid precursor protein (β -APP) can identify DAI at approximately 2 hours following injury (Disc Image 12.43). DAI and vascular (non-traumatic) axonal injury both stain positively with β -APP. The patterns of staining for each entity can overlap, and in certain cases of traumatic brain injury, both types of axonal injury are frequently present.

Neck, Spinal Cord, and Vertebral Artery Injuries

The neck is a vital and, to an extent, vulnerable part of the human body. A variety of injury types can affect the neck. In this chapter, cervical spinal cord and vertebral artery injury related to blunt force injury will be addressed. The reader is referred to Chapter 15 for a discussion of asphyxial neck injuries.

The integrity of the cervical (neck region) spinal cord is vital for continued survival because nervous system control centers for breathing reside within this area.

A variety of blunt force injuries can result in disruption of the cervical spinal cord and its junction with the medulla (the lowest part of the brainstem). A fractured (broken) neck is one type of blunt force injury that can cause spinal cord contusion, laceration, or transection. Disruption of the atlanto-occipital junction (where the occiput, or base of the skull, “connects to” the first cervical vertebral body, which is also known as the “atlas”) can result in similar injuries. Palpation of the atlanto-occipital joint at autopsy, after brain removal, allows pathologists to check the integrity of this important structure.

A blow to the side of the head or face, with a resultant abrupt twisting or sideways flexion motion of the neck, can result in a laceration of the vertebral artery. The vertebral arteries travel through the sides of the cervical vertebral bodies and enter the base of the cranium, in front of the brainstem, where they join to form the basilar artery. Laceration of a vertebral artery typically results in severe basilar subarachnoid hemorrhage, with subsequent loss of consciousness and death. At autopsy, the pathologist must attempt to identify the laceration site; this can be particularly difficult if the laceration is in a part of the artery that is contained within the vertebral bodies. Special dissection techniques are required to visualize the vertebral arteries in this location.

Special Topics Related to Blunt Force Injuries

Mechanisms of Death in Blunt Force Trauma

A number of mechanisms of death can be involved in blunt force injury deaths. The following discussion will be divided into acute (quick) deaths and delayed deaths.

Regarding acute deaths related to blunt force injuries, a general description of blood loss will be followed by specific comments about certain organ systems. One of the most common mechanisms of death involves the acute loss of blood. Exsanguination (blood loss) can occur externally or internally (Disc Image 12.44). For blood loss to occur, vascular (blood vessel) trauma must be present. When there is an insufficient amount of blood within the circulatory system (heart and blood vessels), oxygen is not able to be delivered to the tissues of the body. This lack of “tissue perfusion” (lack of blood delivery to tissues) is referred to as “shock,” and eventually becomes irreversible, and death occurs. Deaths related to traumatic blood loss can involve virtually any organ or tissue. Particularly common sites of injuries where blood loss causes death include the aorta, heart, lungs, liver and spleen. In order for death to be attributed to acute blood loss, approximately 33% of the blood volume must be lost.

It is important to note that, in certain situations, death can also occur from bleeding even when there is a relatively small amount of blood involved. If blood accumulates within the pericardial cavity (the “heart sac;” where the heart resides), the condition is referred to as a “hemopericardium.” When this occurs, the accumulating blood can compress the heart to such an extent that it is no longer able to beat.

This condition is referred to as “cardiac tamponade.” In a similar way, if there is a traumatic hemorrhage within or around the brain, the compressive effect (or “mass effect”) of the blood can cause death, even though the total amount of blood is not sufficient to cause death from blood loss alone.

Physical disruption of the central nervous system is another very common mechanism of death involved in blunt force injury deaths. Transection or injury of a vital CNS structure, such as the brainstem or upper cervical spinal cord, represent examples of grossly-evident CNS disruption. Diffuse axonal injury, as described above, represents a type of physical disruption that typically cannot be seen grossly, but may be evident microscopically, so long as an individual survives long enough for it to become evident. The term “*commotio cerebri*” refers to a death that occurs following a sudden blow to the head wherein autopsy fails to reveal any lethal trauma or markers/indicators of trauma (subdural and/or subarachnoid hemorrhage). Such cases probably represent deaths related to severe traumatic diffuse axonal injury. Another rare occurrence, “malignant cerebral edema,” is similar to *commotio cerebri* in that there is no intracranial hemorrhage, but there is severe diffuse cerebral edema (swelling). Again, traumatic diffuse axonal injury may be a contributory underlying mechanism.

Injuries around and/or involving the lungs can result in the inability of air exchange to occur. This leads to the lack of blood oxygenation, which ultimately leads to lack of tissue oxygenation and death. Injuries of the lungs themselves can result in the air spaces (alveoli) filling with blood, thus preventing air from getting into the air spaces. If the surface of the lung is injured, or if the chest wall is injured (a “sucking chest wound”), air can enter the pleural cavity (chest cavity) surrounding the lung, resulting in a “pneumothorax.” If air can enter the pleural cavity but cannot get out, then a “tension pneumothorax” can result, wherein the accumulating air compresses the lung (and sometimes the heart and other lung), such that air can no longer enter the lungs. In a similar fashion, the lung can be compressed by accumulating blood (“hemothorax”) (Disc Image 12.45). In each of these situations, the lack of air exchange within the lung acts as a mechanism of death. If a pneumothorax is suspected, the pathologist can create a “pocket” between the reflected chest wall and the side of the rib cage prior to removing the anterior chest plate. This pocket can be filled with water, and then an incision can be made between the ribs, so that the incision connects the pleural cavity with the water. If a pneumothorax is present, air bubbles will escape into the water. The bubbles can be collected in an inverted, water-filled graduated cylinder in order to measure the amount of air (Disc Image 12.46). A final lung-related finding that can occasionally be encountered in blunt force, as well as other types of injuries, is aspiration (breathing in) of blood. Aspirated blood has a characteristic gross appearance at autopsy (Disc Image 12.47).

A relatively rare cardiac mechanism of death that occurs in certain blunt force injury cases is “*commotio cordis*.” A lethal arrhythmia can be induced by a blow to the chest that occurs at a very specific time within the electrical cycle (electrocardiogram tracing) of the heart. There may be evidence of anterior chest wall injury, but not always. By strict definition, there should be no trauma of the heart itself,

although some have argued that this definition is too limiting, and cases with cardiac injury that do not lead to exsanguination or cardiac tamponade should be included in the definition of *commotio cordis*.

Traumatic disruption of a pre-existing natural disease process (or even a pre-existing injury that was previously not severe enough to be lethal) with subsequent hemorrhage and death is also known to occur. An example is a person who has a cerebral artery berry aneurysm that ruptures when someone punches the person in the face. In such a case, the blunt force trauma and underlying natural disease process should be included in the cause of death statement. The manner of death should be determined based on the circumstances of the blunt trauma. In the example case provided, homicide is an appropriate ruling.

Delayed Deaths Related to Blunt Force Injury

Death does not have to occur immediately when blunt trauma is the underlying cause of death. Such delayed deaths may occur several hours, days, weeks, months, or even years after the initial traumatic event. As long as an uninterrupted chain of events can link the underlying trauma to the eventual death, it is appropriate to rule the underlying trauma as the underlying cause of death.

A frequent example of a delayed death following blunt trauma is a situation where the victim initially survives the trauma as a result of valiant efforts by emergency medical services and hospital personnel. Despite these efforts, the massive stress associated with severe traumatic injuries can be too much for the victim to overcome. Various complications related to shock, trauma, inflammation, and stress can ultimately lead to death despite all efforts to avoid such complications.

A variety of other situations can cause death in persons who have survived the initial effects of blunt trauma, the most frequent being pulmonary embolism. Persons who are bedridden for whatever reason have an increased risk of developing thrombi (blood clots) within the deep veins of their legs and the risk is increased even more if there are also injuries of the legs. Many trauma victims are in such a situation. If the clots within the leg veins break free and travel upward into the inferior vena cava and ultimately through the right side of heart into the pulmonary arteries, they can cause a massive “pulmonary embolism” (Fig. 12.26) which results in an abrupt stopping of all blood flow to the lungs and sudden death. This is discussed further in Chapter 21.

Another complication that can occur following any type of injury is infection. As with any infection, a localized infection can spread to involve the entire body (referred to as “sepsis”) which is a life-threatening event and can rapidly lead to death.

The fat embolism syndrome is a relatively rare complication, and usually occurs several days after severe trauma, typically including skeletal trauma. Clinically, it is characterized by the sudden onset of respiratory distress, with or without neurological symptoms. At autopsy, gross examination of the brain can reveal numerous small



Fig. 12.26 Numerous pulmonary thromboemboli evident near the opened pulmonary artery of the lung. The “worm-like” thrombi arose within the deep veins of the legs, following a motor vehicle collision. They broke loose and embolized to the lungs, where they blocked the pulmonary arteries

hemorrhagic areas within the white matter (Disc Image 12.48). Microscopically, fat and bone marrow emboli can be visualized within blood vessels of the brain, as well as blood vessels within the lungs (Disc Image 12.49) and evaluation of frozen brain and lung tissue can aid pathologists in their identification. It should also be noted that the microscopic identification of fat and bone marrow emboli within lung tissue is frequently seen in cases of blunt trauma, in absence of the clinical scenario associated with the fat embolism syndrome as described above.

A final type of delayed death that deserves mention involves seizure disorders that are initiated by blunt force brain injuries. Sudden death as a result of a seizure disorder is a well-known occurrence (see Chapter 10). If the seizure disorder resulted from brain injury sustained in relation to blunt force head trauma, then a seizure-related death is ultimately a result of the blunt force injury, even if it occurs decades after the initial trauma.

Depending on the timing of death following blunt force head injury, brain examination at autopsy may reveal cavitory lesions (absence of brain substance where a previous contusion existed), or a discolored (yellow-brown), depressed old (or “remote”) contusion (Disc Image 12.50).

Patterned Injuries

A “patterned injury” is an injury that has a specific and characteristic shape, or pattern. The typical scenario involves various types of blunt force injury, where the injury takes on the shape of the blunt object; although various other injury types (besides blunt force injuries) can also be “patterned.” Regarding patterned blunt force injuries, abrasions, lacerations, contusions, and even fractures can demonstrate specific patterns that take on the shape or characteristics of the inflicting object

(Fig. 12.27 and Disc Images 12.51, 12.52, 12.53, 12.54, 12.55, 12.56, and 12.57). One pattern that is occasionally encountered is a “tram track” injury, composed of two parallel linear contusions or abraded contusions produced when a cylindrical object strikes the skin surface (Fig. 12.28). The central zone of pallor correlates to where the object strikes the skin. Other examples include tire tread abrasions and contusions, hammer-induced scalp abrasions, lacerations, and contusions, as well as skull fractures, and stomping abrasions/contusions caused by the soles of shoes.

Fig. 12.27 A patterned abrasion produced when the decedent sustained a blunt impact. Note that the pattern on the skin mimics that of the weave pattern of the clothing



Fig. 12.28 A patterned injury with a “tram track” appearance



Clothing Examination

As with many other injury types, the examination of a decedent's clothing in cases of blunt force injury death can provide important information. Clothing defects or other markings may correlate with injuries on the skin surface. Transfer of trace evidence from the blunt object that caused trauma can sometimes be identified and collected for subsequent examination in the crime laboratory.

Specific Subtypes of Blunt Force Injury

Certain types of blunt force injury death are common or specific enough to deserve special discussion. The most frequent of these are motor vehicle collision deaths and pedestrian fatalities. Less common case types that will be described are deaths from aircraft crashes and falls/jumps from heights.

Motor vehicle collisions – Over the past several decades, numerous safety features have been implemented within automobiles. Safety belts, airbags, collapsible steering columns, softened interior dashboards, antilock brakes, and numerous other safety features have contributed to the saving of countless lives. Despite such improvements, a substantial number of deaths continue to occur each year as a result of motor vehicle collisions. Some injuries can even be associated with some of these safety features (air-bag injuries, especially problematic in children; safety belt-induced injuries). Accident reconstruction experts can be tremendously helpful in deaths involving motor vehicles. The following describes some unique issues related to injuries and deaths associated with motor vehicle collisions.

Patterned injuries can occur when impact occurs with various objects within the interior of the automobile. One of the most common patterned injuries occurs from seatbelts, including lap and shoulder straps (Fig. 12.29). With the sudden deceleration that occurs in many motor vehicle collisions, patterned abrasions and contusions (and occasionally lacerations) frequently occur horizontally on the lower abdomen and diagonally across the chest to the shoulder (right or left, depending on location of victim). Particularly in older model automobiles, patterned abrasions from impact with the steering wheel can occur on the chest (Fig. 12.30).

Seatbelt injuries are common in motor vehicle collision fatalities (Disc Image 12.58). A patterned injury produced on the chest/shoulder by a seatbelt shoulder strap can assist the pathologist in determining if a particular occupant was the driver or a passenger. Another type of injury that can assist in this regard are “dicing injuries” on a particular side of the body, usually the face, neck, and upper extremity. When the side windows of a car shatter, they break into numerous small cube-shaped pieces of glass. These cubes actually have relatively sharp edges, so the resulting injuries, referred to as “dicing injuries,” actually represent a patterned sharp force injury (Fig. 12.31 and Disc Images 12.59 and 12.60). If they occur on the right side of the body in a traditional left-sided steering column vehicle, then it is likely that the person was sitting on the right side of the vehicle. If they occur on the left side of the body, then it is possible that the individual was the driver.

Fig. 12.29 A patterned injury produced by the driver-side seatbelt shoulder strap

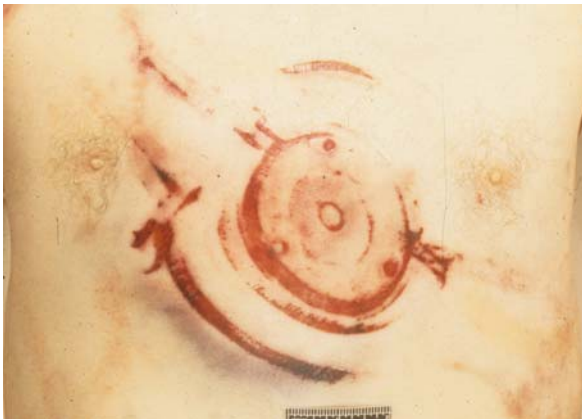


Fig. 12.30 A patterned injury produced by impact with a steering wheel

Examination of soles of the shoes worn by the driver involved in a motor vehicle collision can sometimes reveal the presence of imprint marks from the control pedal that the driver was depressing at the time of impact (Disc Image 12.61). Note that the patterns on the gas and brake pedals are purposefully different from one another.

Internally, a variety of severe injuries can occur in motor vehicle collision deaths, presumably due to sudden deceleration. One of the most classic types is the lacerated descending thoracic aorta. Others that can occur include atlanto-occipital dislocations, and lacerations of numerous internal organs.



Fig. 12.31 Dicing injuries on the left cheek of a driver involved in a motor vehicle collision

In rare cases, exhaust system problems may be implicated in contributing to the crash (carbon monoxide-induced sleepiness). For this reason, it may be prudent for pathologists to test the blood for carbon monoxide. Care must be taken in the interpretation these results, particularly if a fire occurred in association with the crash.

Pedestrians – Pedestrians struck by motor vehicles can demonstrate numerous characteristic features. These become particularly important if the death represents a “hit and run” incident, wherein the vehicle leaves the scene, and police are searching for the vehicle. Frequently, when a pedestrian is struck by a motor vehicle, the initial impact actually throws the pedestrian upward onto the hood of the vehicle. Secondary impacts with the vehicle will frequently result in additional injuries, as will subsequent injuries related to impact with the pavement and possibly other vehicles. Extensive “road rash” abrasions are common. Differentiating injuries caused by initial impact, secondary impact, roadway impact, and other vehicle impact is frequently not possible; however, reconstruction experts can assist if these issues become very important. Occasionally, pedestrians are actually “run over.” This will tend to occur if the pedestrian’s center of gravity is lower than the impact site, as occurs with shorter pedestrians and larger vehicles. As mentioned above, accident reconstruction experts can be extremely valuable in attempting to determine exactly how a particular incident occurred.

Patterned marks or injuries that correlate to specific parts of the vehicles can be present (Fig. 12.32). The clothing can demonstrate similar marks or, more frequently, tire imprints. Paint or chrome chips or, occasionally, pieces of light fixtures or other car parts, can be transferred to the clothing or skin (or deeper tissues) of the pedestrian. Likewise, hair, blood, and other tissue can be transferred from the pedestrian to the vehicle. For this reason, pathologists should collect standard hair and blood samples for potential comparison. As mentioned above, stretch-type lacerations are frequent in the inguinal (groin) regions, particularly when the pedestrian



Fig. 12.32 Tire track marks on a nude murder victim

has been struck from behind. Lower extremity (leg) fractures tend to correlate with the bumper height of the offending vehicle. Consequently, pathologists will usually measure the distance from the bottom of the foot to an external impact site or the fracture site (Disc Image 12.62 and 12.63). The fracture itself will sometimes be of such a shape that allows the pathologist to determine the direction of force that caused the fracture.

Internal injuries tend to be severe. Crushing injuries, including multiple bilateral rib fractures (“flailed chest”) may indicate that the pedestrian was actually run over. Descending thoracic aortic lacerations are probably related to sudden acceleration occurring upon impact.

It should be noted that, because of differences which exist in various forensic training programs around the country, some forensic pathologists will rule the manner of death in “hit and run” pedestrian fatalities as “homicides,” while many others will rule these as “accidents.” Others prefer an “undetermined” ruling.

Aircraft fatalities – Some of the most severe blunt force/deceleration injuries that forensic pathologists see occur in airplane crashes. Amputation injuries, decapitation injuries, and sometimes total body fragmentation may occur. Because of the severe trauma, identification by visual means is not always possible and, as such, appropriate identification methods should be employed in such cases. It should be noted that it is not uncommon for airplane-induced blunt force/deceleration injuries to be associated with other injury types, such as fire-related trauma. For this reason, pathologists will frequently test airplane crash victims for carbon monoxide. Elevated levels of carbon monoxide in crash victims who have blunt force injuries that are immediately lethal suggest that an on-board fire preceded the crash. So-called “control injuries” (fractures of the hands/wrists, as well as the feet/ankles) may indicate that a pilot was attempting to control the plane when impact occurred. Aircraft fatalities are discussed further in Chapter 21.

Falls/jumps from heights – Depending on the length of the descent and body positioning upon impact, injury type and severity in falls/jumps from heights can vary from one case to the next. In some cases, the skin can be surprisingly intact, even when significant heights are involved. In contrast, internal examination frequently reveals a tremendous amount of trauma. If impact occurs on the soles of the feet, forces transferred upward can result in significant pelvic trauma, as well as a “ring fracture” of the basilar skull, as forces “drive” the spinal column upward into the cranial cavity. Ring fractures may also occur when impact occurs on the top of the head. The long bones of the legs can be forced through the soles of the feet (Disc Image 12.64). One of the most crucial considerations in these types of cases involves the manner of death ruling. While suicide should immediately be considered, pathologists and investigators should consider all possible explanations.

Defensive injuries – Although commonly associated with sharp force injuries, “defensive” injuries, typically occurring on the hands and forearms, may occur in victims of blunt force injury (Disc Image 12.65).

Massive body trauma – There are a variety of situations that may result in massive body trauma. Examples include high-speed vehicle crashes, pedestrians run over by many vehicles (Fig. 12.33), falls/jumps from extreme heights, and industrial/machinery-related deaths. In many such instances, blunt force injuries represent a major component of the injury types which exist. Certainly, deceleration injuries also play a role in many such deaths. In some cases, other mechanisms of death are also instrumental in causing death. Examples include asphyxial injuries, sharp force injuries, and fire/thermal injuries. On occasion, a pathologist may not be able to, or not choose to, identify which injury type plays the most important role in death. In such cases, it is appropriate to be more general in the COD statement. Examples include “multiple severe injuries” or “multiple traumatic injuries” or some similar description. Often in these cases, identification of the decedent becomes of primary importance.



Fig. 12.33 Massive body trauma resulting from a multiple motor vehicle collision in which the decedent was ejected from the vehicle and then run over by numerous other vehicles, including multiple semi tractor trailer trucks

Disc Image Legends

- Disc Image 12.1 Abrasions resulting from striking an automobile windshield.
- Disc Image 12.2 Multiple linear and confluent abrasions on a murder victim's abdomen, produced when her body was moved (dragged). Note the yellow discoloration, indicating peri- or post-mortem occurrence.
- Disc Image 12.3 An abrasion with peeled up superficial layers of the epidermis. Their location toward the left of the abrasions shows that the direction of force was from right to left in the photograph.
- Disc Image 12.4 A relatively small impact contusion of the arm.
- Disc Image 12.5 A scalpel incision of a contusion at autopsy, allowing visualization of the hemorrhage within the subcutaneous fat.
- Disc Image 12.6 An example of senile ecchymosis.
- Disc Image 12.7 An example of Battle's sign, related to basilar skull fractures.
- Disc Image 12.8 Grey Turner's sign, indicating extensive internal (retroperitoneal) hemorrhage.
- Disc Image 12.9 A multicolored bruise known to have occurred 7 days prior to death.
- Disc Image 12.10 A contusion of a heart. Note that this heart has an extraordinary amount of epicardial fat.
- Disc Image 12.11 A laceration with extensive associated abrasions.
- Disc Image 12.12 Extensive lacerations of the scalp, with minimal associated abrasions.
- Disc Image 12.13 Another example of a laceration with tissue bridging.
- Disc Images 12.14 **A:** A scalp laceration. **B:** The same laceration, showing prominent undermining.
- Disc Image 12.15 Another example of an aortic laceration.
- Disc Image 12.16 Multiple lung lacerations.
- Disc Image 12.17 Extensive liver lacerations.
- Disc Image 12.18 Multiple spleen lacerations.
- Disc Image 12.19 Extensive heart lacerations sustained in a high-speed motor vehicle collision.
- Disc Image 12.20 Multiple acute rib fractures.
- Disc Image 12.21 Multiple comminuted skull fractures.
- Disc Image 12.22 Multiple healing rib fractures (arrows).
- Disc Image 12.23 An amputation injury sustained in a farming accident.
- Disc Image 12.24 Body transection occurring in a motorcycle accident.
- Disc Image 12.25 Another example of body transection involving a motorcycle rider.
- Disc Image 12.26 Orbital plate (anterior cranial basilar skull) fractures.
- Disc Image 12.27 A "ring fracture" involving the base of the skull.
- Disc Image 12.28 Visualization of the bridging vessels that connect the dura (attached to the under surface of the skull) to the meninges overlying the brain.
- Disc Image 12.29 A subdural hemorrhage visualized through the relatively translucent dura membrane, which remains over the brain in this photo.

- Disc Image 12.30 A relatively dense subdural hemorrhage. A flap of dura is being reflected upwards in order to visualize the underlying subdural blood.
- Disc Image 12.31 Basilar subdural hemorrhage, after brain removal.
- Disc Image 12.32 A healing (or “organizing”) subdural hemorrhage.
- Disc Image 12.33 Another example of an organizing subdural hematoma.
- Disc Image 12.34 The base of a brain with minimal subarachnoid hemorrhage.
- Disc Image 12.35 Focal subarachnoid hemorrhage overlying areas with cerebral contusions.
- Disc Image 12.36 Deep contusions within the brain. Note the “midline shift” of central structures, from right to left, due to the injury and associated swelling.
- Disc Image 12.37 A gliding contusion of the cerebral cortex.
- Disc Image 12.38 A brain with a cortical contusion (lower left), associated brain swelling with midline shift, and a cingulate gyrus, subfalcine herniation (arrow).
- Disc Image 12.39 Bilateral (both sides) temporal lobe uncal herniation contusions.
- Disc Image 12.40 An example of uncal “notching,” when the uncal regions are compressed but not yet hemorrhagic (arrows).
- Disc Image 12.41 Cerebellar tonsil herniation contusion.
- Disc Image 12.42 A transcalvarial herniation occurring in a head trauma victim who underwent craniotomy (removal of a portion of the skull) surgery to relieve pressure. Note the relatively large area of swollen brain tissue protruding outward from the remainder of the brain, as seen in the upper aspect of the photograph (arrows).
- Disc Image 12.43 An immunohistochemistry stain showing brown staining areas indicative of axonal injury.
- Disc Image 12.44 A case where extensive internal hemorrhage occurred into the peritoneal cavity (hemoperitoneum).
- Disc Image 12.45 An example of a hemothorax, where blood has filled the right thoracic (pleural, chest) cavity. The heart sac is indicated by the asterisk. The diaphragm is indicated by the double asterisk. The compressed and injured right lung is indicated by the arrow.
- Disc Image 12.46 A pathologist checking for the presence of a pneumothorax. The inverted cylinder (filled with water) is used to collect air bubbles escaping from the chest cavity. (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)
- Disc Image 12.47 Aspirated blood within a lung.
- Disc Image 12.48 The gross appearance of a brain from an individual who experienced fat embolism syndrome. Note the numerous areas of prominent pinpoint blood “spots” within the white matter.
- Disc Image 12.49 The microscopic appearance of fat emboli (clear round to oval-shaped areas) within a lung blood vessel.
- Disc Image 12.50 A remote (old) contusion of the brain. Note the depressed, brown-yellow appearance.
- Disc Image 12.51 A patterned injury produced by contact with a steel cable.
- Disc Image 12.52 Numerous patterned injuries produced by an electrical cord “loop” that was used as a “whip.” Notice that some of the marks have a “tram

track” appearance. (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)

Disc Image 12.53 Multiple patterned injuries produced by blows from the claw end of a hammer. These injuries are most appropriately referred to as “chop” wounds (see Chapter 14).

Disc Image 12.54 A different case with multiple hammer strikes to the head.

Disc Image 12.55 A third case with multiple hammer strikes. The two injuries shown were produced by the round end of a hammer.

Disc Image 12.56 Patterned depressed skull fractures produced by hammer blows.

Disc Image 12.57 A patterned abrasion produced by the zipper of a jacket. The decedent was forcibly struck in the chest by a tool that was flying through the air as a result of an industrial site explosion.

Disc Image 12.58 A lap strap seatbelt injury.

Disc Image 12.59 Another example of dicing injuries sustained by a driver in a motor vehicle collision.

Disc Image 12.60 An example of the cubes of glass produced when the side windows (tempered glass) of an automobile shatter. The sharp edges of the cubes are responsible for producing dicing injuries.

Disc Image 12.61 Pedal imprint marks on the sole of the shoe of a driver involved in a motor vehicle collision. (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)

Disc Image 12.62 A yardstick adjacent to a lower extremity of a pedestrian, showing the height of an impact abrasion above the bottom of the foot.

Disc Image 12.63 Incision of the leg, with visualization of a tibia fracture.

Disc Image 12.64 Lacerations and fractures involving the feet of an individual who jumped from a multiple story building, landing on his feet.

Disc Image 12.65 “Defense”-type injuries on the hands of an individual who was attacked by an individual wielding a hammer.

Selected References

- Burke MP. *Forensic Medical Investigation of Motor Vehicle Incidents*. Boca Raton, FL: CRC Press; 2007.
- Dix J, Graham M, Hanzlick R. *Investigation of Road Traffic Fatalities – An Atlas*. Boca Raton, FL: CRC Press; 2000.
- Ehrlich E, Maxeiner H. External injury marks (wounds) on the head in different types of blunt trauma in an autopsy series. *Medicine Law* 2002;21:773–82.
- Langlois NEI. The science behind the quest to determine the age of bruises – a review of the English language literature. *Forensic Sci Med Pathol* 2007;3:241–51.
- Marshall DT, Gilbert JD, Byard RW. The spectrum of findings in cases of sudden death due to blunt cardiac trauma – “commotio cordis.” *Am J Forensic Med Pathol* 2008;29(1):1–4.
- Murphy GK. “Beaten to death.” An autopsy series of homicidal blunt force injuries. *Am J Forensic Med Pathol* 1991;12:98–101.
- Pearl GS. Traumatic neuropathology. *Clinics Lab Med* 1998;18:39–64.
- Teresinski F, Madro R. Evidential value of injuries useful for reconstruction of the pedestrian-vehicle location at the moment of collision. *Forensic Sci Int* 2002;128:127–35.

- Toro K, Szlavik N, Meszaros A, Dunay G, Soos M, Keller E. Jumping and falling death in children, adolescents, and young adults. *J Clin Forensic Med* 2006;13:129–34.
- Turk EE, Tsokos M. Pathologic features of fatal falls from height. *Am J Forensic Med Pathol* 2004;25:194–9.
- Zivot U, DiMaio VJ. Motor vehicle-pedestrian accidents in adults. Relationship between impact speed, injuries, and distance thrown. *Am J Forensic Med Pathol* 1993;14:185–6.
- Zugibe FT, Costello JT. Identification of the murder weapon by intricate patterned injury measurements. *J Forensic Sci* 1986;31:773–7.

Chapter 13

Gunshot Wound Deaths

Do not let them out of your sight.
Proverbs 4:21

Abstract Chapter 13 begins by providing a general presentation of weapon and ammunition types. The chapter then discusses various characteristic features of low-velocity (typically handgun) gunshot wounds, including entrance wounds, range of fire, exit wounds, graze wounds, caliber, and miscellaneous features. After presenting sections that deal with high-velocity wounds and shotgun wounds, the chapter concludes with a discussion of various miscellaneous topics, including internal examination, mechanism of injury, documentation, X-rays, clothing examination, gunshot residue, manner of death, special ammunition, weapon types, and circumstances.

Keywords Gunshot wounds · Handguns · Rifles · Shotguns · Ammunition

Introduction

Deaths related to gunshot wounds are common in the United States. Injuries caused by projectiles (missiles) fired from guns, or firearms, have features that are somewhat characteristic. Some authors prefer to describe such firearm-related injuries as penetrating or perforating injuries. The term “penetrating” indicates that the projectile/missile enters the body, but does not exit, whereas the term “perforating” indicates that the projectile/missile traveled all the way through the body.

Types of Weapons and Ammunition

Before considering the various features of gunshot wounds, it is appropriate to provide a general review of weapon types, as well as ammunition types. This section is not meant to be an all-inclusive description of these topics, but rather an overview. There are several other excellent resources available that provide greater detail. There are three basic types of firearms that will be described: handguns, rifles, and

shotguns. Prior to describing these types of weapons, some basic information will be provided. The reader is also referred to the section on firearms and toolmarks examination in Chapter 2 of this book.

The basic configuration of a firearm includes the grip (the part that is held with the hand that has the trigger finger), the barrel (the part from which the bullet exits the weapon), the trigger (an external part near the grip that enables the person holding the weapon to discharge the gun), the internal mechanisms that transfer the trigger pull into the firing of the bullet (usually including a “firing pin”), the firing chamber (containing the cartridge that contains the bullet or projectiles to be fired), and frequently, a mechanism which allows for a mechanized means of quickly supplying another round (bullet) to be fired. In long guns (rifles and shotguns), there is frequently a stock that extends rearward from the grip and is placed against the shoulder during discharge. Also, in long guns, the barrels are relatively long (by US law, minimum length of 16 inches for rifles, and 18 inches for shotguns), while in handguns, they are relatively short. Figure 13.1 shows a general comparison of two handguns and a rifle (Fig. 13.1).



Fig. 13.1 Two handguns (semi-automatic and revolver) and a rifle

The barrels of most handguns and rifles are “rifled,” meaning that the inside surface of the barrel has spiraling grooves traveling the length of the barrel. The official names for these are “lands” (the raised parts) and “grooves” (the depressed parts in-between the lands). Rifling imparts a spin to the exiting bullet, giving it a more aerodynamically sound (straight) flight. Rifled barrels can differ in the direction of spin or twist of the lands and grooves with either a right twist or a left twist. They can also differ in the number and size of the lands and grooves. The “caliber” of a weapon is determined by the diameter of the barrel (which corresponds to the diameter of the bullets that are fired). The combination of a rifled weapon’s caliber, as well as the number, size, and twist of lands and grooves determine the weapon’s “class characteristics.” Disc Image 13.1 shows two bullets recovered from autopsy with rifle impression marks. The unique microscopic striations discussed in Chapter 2 represent the weapon’s “individual characteristics.”

Shotguns tend to have “smooth” bores, meaning the internal surface of the barrel is not rifled but is smooth. The diameter of the barrel is referred to as the shotgun gauge. From smallest to largest, examples of different gauge shotguns include .410, 20 gauge, 16 gauge, and 12 gauge. The “choke” of a shotgun refers to the extent of constriction that occurs at the muzzle end of the barrel. From most constricted to least constricted, the different terms used to describe choke include: full choke, modified choke, improved cylinder, and cylinder.

Fig. 13.2 An unspent (unfired) handgun cartridge. The silver-colored casing contains the unspent bullet, which has a copper jacket



A “cartridge” is what many people might consider an “unfired bullet” (Fig. 13.2) but an unfired bullet is, actually, only part of the cartridge that is used in handguns and rifles. A cartridge is composed of a “casing” that contains in its base a primer (a method for converting the trigger pull mechanism into ignition of the gunpowder), the gunpowder itself, and the bullet, which is tightly embedded into the open end of the casing (Disc Image 13.2). The two types of primers are the centerfire primer, located in the center of the base of the cartridge, and the rim primer, located in the outer rim of the base of the cartridge casing (Disc Images 13.3 and 13.4). The most well-known rimfire ammunition is .22 caliber. When the firing pin strikes the primer, the primer explodes and ignites the gunpowder and the exploding gunpowder expels the bullet from the end of the cartridge. Traveling at a great velocity down the barrel, the bullet is forced to spin along its longitudinal axis by the rifling of the inside surface of the barrel. Besides the bullet, there are other items that travel down and out of the barrel. Especially in handguns, the flames from the exploding gunpowder can be seen at the muzzle (open end) of the barrel. Also, a cloud of smoke (soot) created by the exploding gunpowder is expelled from the muzzle. Finally, particles of unburnt as well as burning gunpowder are also expelled from the end of the barrel (Fig. 13.3). The cartridge casing does not exit the weapon via the barrel. Depending on the type of weapon, the casing may be mechanically ejected from the weapon, or it may require manual removal. Usually semi-automatic weapons have a recessed indentation just above the rim base, on the side of the casing, whereas revolver



Fig. 13.3 Diagram of a discharging firearm, showing a small amount of flame at the muzzle, a cloud of smoke/soot, particles of gunpowder, and the bullet

ammunition has a rim at the base of the casing with a diameter greater than the remainder of the cartridge (Disc Image 13.5).

Low-velocity versus high-velocity: most handguns and some rifles fire what is typically referred to as “low-velocity” ammunition. The muzzle velocity of these bullets varies from around 750 ft/s to around 1400 ft/s. In contrast, high-velocity ammunition (which tends to be either military or hunting ammunition fired from high-velocity rifles) usually has a muzzle velocity in the range of 2300 ft/s to over 3000 ft/s.

Handguns: A majority of gunshot wound deaths that occur in the United States occur with the use of handguns. Handguns are weapons with short barrels that can generally be held and controlled with one hand. The usual handgun barrel is rifled. There are two basic types of handguns, revolvers and semi-automatic pistols, but there are also various miscellaneous types.

Revolvers: Revolvers contain a cylinder within which multiple bullets are placed. After one bullet is fired from a revolver, in order for another bullet to be fired, the cylinder must rotate, so that the next bullet is in proper alignment with the firing pin and the barrel. In many revolvers, a “hammer,” which is an external lever mechanically connected to the firing pin mechanism, near the back top of the weapon, can be manually pulled back. When this occurs, the cylinder rotates so that the next cartridge is properly aligned for firing, and the hammer becomes engaged in a “cocked” (ready-to-fire) position. When the trigger is pulled, the hammer drops and the firing pin strikes the primer on the base of the cartridge casing, resulting in discharge. In most “single-action” revolvers, this is the only method for firing the weapon. In many “double-action” revolvers, besides the “single-action” method just described, pulling back on the trigger will actually cause the hammer to mechanically move back, while simultaneously rotating the cylinder. As the trigger is pulled all the way back, the “hammer cocked” position is bypassed and the hammer drops, striking the firing pin and causing weapon discharge. This type of mechanism is referred

to as “double-action.” Some double-action revolvers allow either single- or double-action trigger pulls. Others are double-action only. Also of note with revolvers is the gap that exists between the cylinder and the barrel. On discharge, a certain amount of smoke is discharged from this “cylinder-barrel gap.” Figure 13.4 shows a closer view of a revolver.



Fig. 13.4 A .357 revolver

Semi-automatic (self-loading) pistols: Semi-automatic handguns are frequently mischaracterized by the media, as well as others, as “automatic” weapons. An automatic firearm is a weapon that fires multiple times with a single pull of the trigger and will continue to fire as long as the trigger is being pulled. Automatic weapons cannot be sold or purchased within the United States unless special permits are obtained. In contrast, a semi-automatic (or autoloading) firearm requires a separate trigger pull for each discharge. The “semi-automatic” description applies to the self-loading aspect of the firing mechanism. In a semi-automatic weapon, the discharge provides enough energy to mechanically eject the spent (used) cartridge casing from the weapon and insert a new, unspent cartridge into the firing chamber. In this way, after a bullet is fired, the weapon is ready to fire again. Most semi-automatic weapons have special devices (magazines) that contain multiple bullets, so that the self-loading as described above can occur until all bullets contained within the device are gone. In semi-automatic pistols, the magazines are typically contained within the grip portion of the weapon. Figure 13.5 shows a semi-automatic pistol.

Miscellaneous handguns: There are a variety of non-revolver, non-semi automatic handguns available. Many require the manual loading of a single cartridge, or sometimes two (with two separate triggers and barrels) at a time. An example is the derringer. Most of these have rifled barrels.

Handgun ammunition: A variety of ammunition types are available for handguns (Disc Image 13.6). In general, the diameter of the bullet can be used to classify a given bullet as either small, medium, or large caliber. Examples of small caliber bullets include the .22 and the .25 ACP. Examples of medium caliber bullets include



Fig. 13.5 A 9 mm semi-automatic pistol

the .32 ACP, the .38 special, the .380 ACP, and the 9-mm. Examples of large caliber bullets include the .40 S&W and the .45 ACP. .22 caliber cartridges have their primer components within the rim of the casing, hence they are commonly referred to as “rimfire” cartridges. Most others have their primers located within the center of the casing base, hence they are called “centerfire” cartridges. In general, there are two basic types of bullets, those that are unjacketed lead and those in which the lead core is covered with a metal jacket. Varieties of each exist, with variation in shape, extent of jacketing, etc. Some special types of handgun ammunition also exist (Disc Image 13.7), containing different components or materials specifically designed for particular purposes. Some of these are more specifically described later in this chapter.

Rifles: Rifles are long guns with rifled barrels. There are two general categories: low-velocity (usually rimfire .22) and high-velocity (with a variety of calibers/ammunition types). High-velocity rifles may fire hunting ammunition or military ammunition. In general, hunting ammunition is designed to break apart on impact, so that the projectile is less likely to completely perforate the target and harm something behind the target. In contrast, most military rounds are designed to completely perforate the target, although there are exceptions to this rule.

Shotguns: Shotguns are long guns having smooth (non-rifled) barrels (bores), as described above. There are generally two types of ammunition utilized in shotguns. They can fire “shot,” which are spherical metal (lead or steel) pellets that are available in a variety of sizes, ranging from quite small (.05 inch diameter) to rather large (.36 inch diameter). The smaller sized shot (.05 to .22 inch diameter) is referred to as “birdshot” (Disc Image 13.8), while the larger shot (.24 to .36 inch diameter) is called “buckshot.” The shot pellets are contained within a shotshell cartridge, where the collection of pellets overlies various forms of “wadding,” which acts to separate the pellets from the underlying gunpowder (Disc Image 13.9). The metallic base of the shell casing contains a centerfire primer and gunpowder. The cylindrical portion of the shell, above the metallic base, is typically made of plastic, and acts to

contain the pellets. Different ammunition manufacturers utilize different types of wadding: some are rather basic, consisting of cardboard or cork. Others are composed of specially-designed plastic inserts that have “petals” that extend along the side of the pellet mass. Figure 13.6 shows several different shotgun shells.



Fig. 13.6 Shotgun shells of various sizes (gauges)

A second type of ammunition used in shotguns is the shotgun “slug.” A slug is a single, large piece of metal (usually lead) that is fired from the weapon (Disc Image 13.10). The shell casing appears similar to those used for shot pellets, and there may or may not be additional components, such as plastic “sabot” pieces that assist in the slug’s travel through the barrel, but then fall away from the slug once it has exited the weapon.

Gunshot Wounds

The first question that forensic pathologists must address regarding deaths with presumed gunshot wounds is whether or not the injuries present truly represent gunshot wounds. The circumstances of the death frequently assist tremendously in this regard; however, if the circumstances are not known, then wound and body examination is the most important factor in answering this question. X-ray examination of the body is required in all gunshot wound or suspected gunshot wound cases (Fig. 13.7, Disc Image 13.11). Recognizing the characteristic features of gunshot wounds on external, internal, and X-ray examination allows forensic pathologists to determine that a particular case is related to gunshot wounds. Despite the recognition of an injury as a gunshot wound, it is not altogether uncommon for victims of firearm injury to also have other injury types (blunt force, sharp force, etc.). It should also be noted that, particularly on external examination, various non-gunshot injuries and findings can sometimes mimic gunshot wounds. Finally, living persons



Fig. 13.7 A head X-ray showing a bullet and some bullet fragments

(and hence dead persons) may have “retained bullets” within their bodies from previous gunshot injuries, so the mere presence of projectiles on X-ray exam does not mean that the death is necessarily related to firearm injury.

Entrance Wounds

Once it has been determined that a person has sustained gunshot injuries, the next important question regarding a particular gunshot wound is whether it represents an entrance wound or an exit wound. In cases of low-velocity projectiles that strike the skin directly, an entrance wound has a round or oval-shaped skin defect, surrounded by a rim of abrasion (Fig. 13.8). This rim is variably referred to as an “abrasion collar” or a “circumferential marginal abrasion” (the “margin” of the wound being the edge or rim). The abrasion occurs as the bullet stretches and then bursts through the skin, with the wound edges rubbing against the side of the bullet as it enters the body. The width of the marginal abrasion can provide an indication of the relative angle of the bullet as it enters the skin. If the marginal abrasion is of a consistent width, then the bullet entered the skin in a relatively perpendicular fashion. In contrast, if the marginal abrasion measures 1/4 inch at the 11 o’clock position (a common method for describing round wounds is to use the clockface to indicate the location of various features within or around the wound), and <1/16 inch at the 5 o’clock position, then the bullet entered the skin at an angle, from the 11 o’clock direction (Disc Image 13.12).

If a bullet goes through something else prior to striking the skin, it is said to have passed through an “intermediary” or “interposed” target. If this interposed target is relatively thin clothing, the underlying entrance wound may have an appearance identical to the usual entrance wound. If the clothing is very heavy, or if another interposed target (such as a wall, window, door, etc.) has been perforated, then the entrance wound can have an irregular central defect (because the bullet is deformed



Fig. 13.8 A gunshot entrance wound. Note the central round defect (hole) and the surrounding marginal abrasion. There is no surrounding soot or gunpowder stippling, making this a distant (indeterminate) range entrance wound. Note that the marginal abrasion is wider on the left side, indicating that the bullet was coming more from the left, as opposed to straight-on

and/or tumbling), and the marginal abrasions tend to be rather wide and of inconsistent width (Fig. 13.9). These wounds are referred to as “atypical entrance wounds.” It is not uncommon for various body parts, such as the arms, to act as interposed



Fig. 13.9 An atypical gunshot entrance wound, characterized by a large size and relatively broad marginal abrasions. Such a wound typically occurs when the bullet has passed through an interposed target prior to striking the victim

targets, with bullets perforating the arm and then re-entering the body elsewhere (Disc Image 13.13). Surrounding skin injuries produced by the interposed target may also be evident. If a bullet fragments when striking an interposed target, multiple entrance wounds can be caused by the various bullet fragments (Disc Image 13.14). Bullets that have ricocheted off of another object also tend to produce atypical entrance wounds.

Entrance wounds on the thick skin of the palms of the hands/fingers or soles of the feet/toes are often quite different in appearance from entrance wounds elsewhere. They frequently lack a characteristic marginal abrasion, they may have associated subcutaneous contusion, and they may have a somewhat stellate (starburst) appearance (Disc Images 13.15 and 13.16). With high-velocity projectiles, a marginal abrasion may or may not be evident. Instead, there may be numerous marginal “microlacerations” or “microtears” (see below).

One final note regarding entrance wounds deserves mention. Because of the elasticity of skin, the size of an entrance wound will not necessarily correlate to the size (caliber) of the bullet. Occasionally, forensic pathologists are asked to estimate the caliber of bullet used, particularly in perforating (through and through) gunshot wounds where a projectile has not been recovered from the scene. Great caution should be used when providing such estimates, and the size of the skin entrance defect should not be the sole factor utilized in making such estimates.

Range of Fire

Contact range: Entrance wounds can vary in their overall appearance based on the “range of fire” (how far the muzzle of the weapon is from the target/skin). In “contact wounds” that occur over the skull (or any other bone that is relatively close to the skin surface), the explosive gases and smoke that discharge from the weapon can dissect between the skin and the bone in the area immediately surrounding the entrance defect. As these high-pressure gases dissect under the skin, the skin splits, thus creating numerous lacerations around the entrance defect. The overall appearance of these contact wounds is described as “starburst” or “stellate” (Fig. 13.10 and Disc Image 13.17). Depending on a variety of factors, including the strength of the round and how firmly the muzzle is placed against the skin, stellate lacerations may or may not be present or extensive (Disc Image 13.18). There may be charring of the skin, and there is usually soot deposited within the depths of the wound. If the muzzle is only loosely in contact with the skin, there is often extensive soot surrounding the entrance wound (Fig. 13.11). If the muzzle is tightly in contact with the skin, the explosive gases can force the skin back against the muzzle of the weapon, such that a “muzzle imprint abrasion” is formed (Fig. 13.12A, B). Contact wounds that occur without underlying bone will demonstrate charring of the skin, as well as soot within the depths of the wound (Fig. 13.13 and Disc Image 13.19), and may also demonstrate muzzle imprint abrasions. Occasionally, carbon monoxide and other gases that are forced into the wound cause a pink-red discoloration of the surrounding skin (Disc Image 13.20). Intraoral gunshot wounds are almost always suicidal.

Fig. 13.10 Contact entrance wound of the scalp (forehead), showing the characteristic stellate shape. Note the central round defect, as well as the soot



Fig. 13.11 Loose contact wound of the chest, with extensive soot deposited around the entrance site

Photographing these wounds can present a challenge; however, the use of a mirror can often produce a very good photograph (Disc Image 13.21). Intraoral wounds may create lacerations which extend from the sides of the mouth (Disc Image 13.22). This is particularly common when larger caliber weapons are used, when there may also be other facial lacerations.

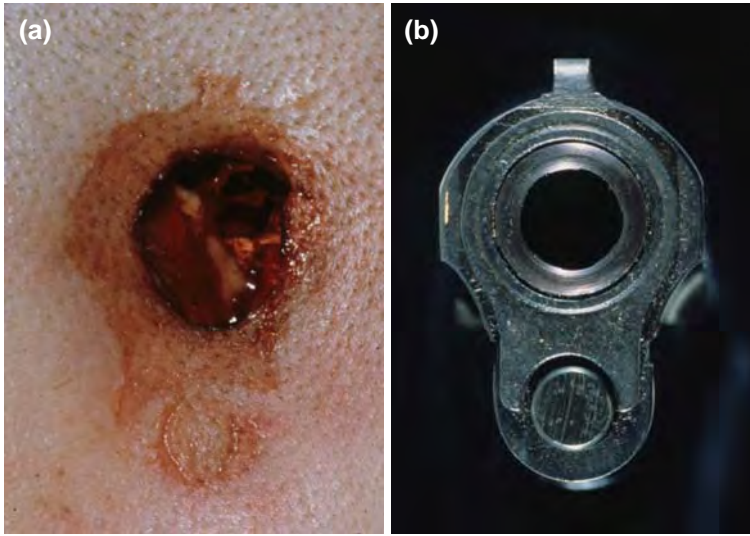


Fig. 13.12 **A.** A contact entrance wound of the scalp, with a muzzle imprint abrasion. **B.** A view of the muzzle of the weapon used in this case



Fig. 13.13 A contact gunshot wound of the neck. Note the surrounding subcutaneous bruising

Close range: If a weapon's muzzle is close to, but not in contact with the skin, then soot and gunpowder will be evident around the entrance wound (Fig. 13.14 and Disc Images 13.23 and 13.24). The soot can sometimes be washed away, but the gunpowder particles actually strike and injure (and sometimes become embedded within) the skin. The resulting marks cannot be washed away and are referred to as



Fig. 13.14 A close-range gunshot wound, characterized by soot and gunpowder deposition

“gunpowder stipple marks” or “gunpowder tattooing.” With most handguns, soot in combination with gunpowder stippling around gunshot entrance wounds can occur when the muzzle of the weapon is up to about 12 inches from the skin. Test firing a suspect weapon using paper targets and identical ammunition may provide a more specific approximation regarding range-of-fire. Entrance wounds with surrounding soot and gunpowder stippling are referred to as “close-range” gunshot wounds. It is important to remember that overlying clothing (or other interposed targets) may block some (or all) of the soot and gunpowder, and therefore clothing should always be examined (Disc Images 13.25 and 13.26).

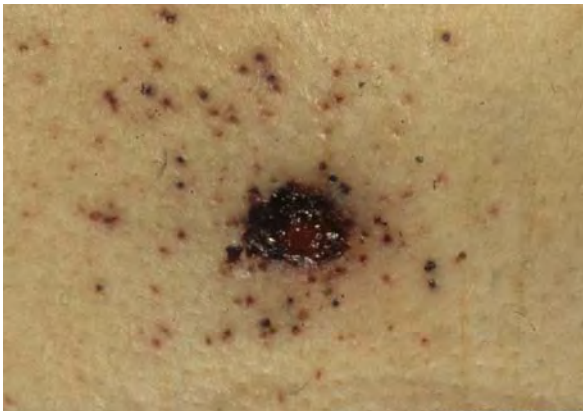


Fig. 13.15 A medium (intermediate)-range gunshot wound, characterized by gunpowder stippling, but no soot

Medium (intermediate) range: Beyond about 12 inches, the smoke/soot no longer deposits around gunshot entrance wounds. In contrast, the more dense gunpowder particles can travel further and produce stipple marks out to a distance of about 3 ft. Gunshot entrance wounds with gunpowder stippling, but no soot, are commonly referred to as “medium-range” or “intermediate range” wounds (Fig. 13.15 and Disc

Image 13.27). As described above, test firing may allow for more specific estimates regarding range-of-fire. Depending on the distance from muzzle to skin, and the angle of entrance, the stippling pattern may be more extensive or dense on various sides of the entrance wound (Fig. 13.16). Interposed targets such as clothing or hair may block the gunpowder, preventing or reducing stippling (Disc Image 13.28). It is important to note that gunpowder comes in a variety of sizes and shapes, thus influencing the appearance of gunpowder stipple marks (Disc Image 13.29).



Fig. 13.16 An angled entrance wound with more dense stippling on the skin closest to the gun (*toward the right*), and less dense stippling away from the gun (*lower left*). Note that the stippling extends a greater distance on the side opposite the gun

Distant (indeterminate) range: Once the weapon is more than about 3 ft from the skin (or clothing) surface, gunpowder particles do not usually have enough energy to actually produce stipple injuries. However, in some weapons, gunpowder particles may still be deposited on the clothing or skin surrounding the entrance wound beyond the 3 ft distance (but with no stippling). Gunshot entrance wounds with no associated soot or gunpowder stippling are referred to as “distant” wounds, meaning more than about 3 ft. A better term that is preferred by many forensic pathologists is “indeterminate,” since closer range shots where the soot and gunpowder is totally blocked by an interposed target may produce identical appearing wounds. The wounds shown in Fig. 13.8 and Disc Image 13.12 are examples of distant (indeterminate) entrance wounds. It should be noted that it is common for the margins of indeterminate gunshot wounds to darken (and appear almost black) due to post-mortem drying, and the dark discoloration can mimic soot (Disc Images 13.30 and 13.31).

Other causes of stippling: Strictly speaking, multiple small injuries of whatever source surrounding an entrance wound may be referred to as “stipple marks.” For example, injuries surrounding an entrance wound caused by shattered glass fragments from an interposed window can be referred to as “stipple marks” (Disc Image 13.32). Consequently, it is best to use the complete term “gunpowder stippling” when referring to stipple marks produced by gunpowder.

Exit Wounds

Exit wounds from low-velocity firearms tend to be relatively small, and they can have a variety of shapes, ranging from slit-like to comma-shaped to X-shaped to irregularly-shaped (Figs. 13.17 and 13.18 and Disc Images 13.33, 13.34, and 13.35). Exit wounds may or may not have central, round to oval defects, but the typical exit wound does not have marginal abrasions. In certain circumstances, however, an exit wound can mimic a classic entrance wound. This occurs when the skin surrounding the exit site is supported (“shored-up”) by firm, tight clothing or another firm object (wall, concrete floor, etc.). The resulting exit wound is referred to as a “shored” exit wound (Fig. 13.19). High-velocity exit wounds tend to be very large and destructive (see below). Frequently bullets lack enough energy to actually exit the body and may remain just underneath the skin surface. Sometimes, their presence is obvious (Fig. 13.20) and occasionally is indicated by subcutaneous hemorrhage (Disc Image 13.36).



Fig. 13.17 A stellate exit wound. Note the absence of a central round defect (a feature which is characteristic of an entrance wound)

Graze Wounds

Graze gunshot wounds (those that strike the skin surface in a tangential fashion) are not uncommon. They can range from wounds that only injure the very superficial layers of the epidermis to those that completely disrupt the epidermis and also injure the underlying dermis, and possibly the subcutaneous fatty tissues. The typical graze wound has an elongated oval shape, with an elongated marginal abrasion being seen at both ends of the elongated oval shape; however, it is more typical at the end that the bullet struck first. The direction of the travel of the bullet can be determined if the wound is deep enough to produce a series of pointed-tip “skin tags” along the

Fig. 13.18 An irregularly-shaped exit wound. Note the absence of marginal abrasions



Fig. 13.19 A shored exit wound, which mimics an entrance wound

sides of the wound. The points of these skin tags point back toward the gun, whereas the angles at the outside aspects of the lacerations are pointed in the direction that the bullet traveled (Fig. 13.21 and Disc Image 13.37).

Caliber

In general terms, smaller caliber bullets produce smaller skin entrance wounds, while larger caliber bullets produce larger skin entrance wounds. However, it is important to note that, because of the elasticity of skin, the dimensions of the

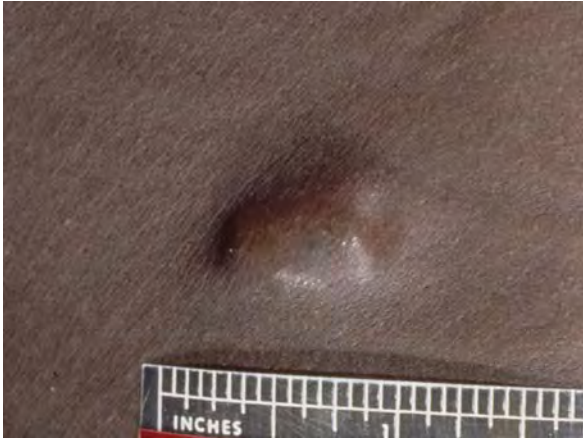


Fig. 13.20 A subcutaneous bullet



Fig. 13.21 A graze gunshot wound. The direction of bullet travel was from left to right

entrance wound as measured on the skin will not necessarily coincide with the caliber of the bullet. The wound may be smaller, larger, or the same size as the bullet. A better estimate of the bullet caliber may be provided by measuring the defect produced by the bullet within bone. This is particularly true with skull shots. Although skull defects may be larger than the bullet caliber, defects smaller than the bullet are unlikely. One final note regarding caliber involves X-ray examination: because of the magnification that can occur during the performance of an X-ray, they should not be used to estimate the caliber of bullet.

Miscellaneous Features of Handgun Wounds

When a revolver is fired, a linear, L-shaped, V-shaped or T-shaped area of soot/burning may be produced on the skin (or clothing) adjacent to the weapon. This occurs because of the small gap that exists between the cylinder and the barrel on revolvers. The exploding gases/soot/gunpowder escape through this gap and create the so-called “cylinder-barrel gap” mark (Disc Image 13.38).

In semi-automatic handguns, when the firearm discharges, the slide associated with the barrel moves backward and then forward. This motion allows for the spent cartridge casing to be ejected, and a new cartridge to be inserted into the firing chamber. If a hand is relatively large compared to the weapon, injuries may occur on shooter’s hand, between the thumb and index finger, as the slide moves backward and then forward upon discharge. Such injuries may be elsewhere on the hand/fingers, depending on how the person held the weapon (Disc Image 13.39).

With either type of handgun, soot and gunshot residue may be deposited on the shooter’s hand (Fig. 13.22). Alternatively, a victim may hold their hands up or out in a defensive posture, with subsequent soot deposition related to someone else firing the weapon.



Fig. 13.22 Soot present on the hand of a suicide victim

Characteristics of High-Velocity Wounds

Many of the features of high-velocity wounds related to range-of-fire and general features of wound types are similar to those of low-velocity wounds, with the noted exceptions described above. Many high-velocity entrance wounds do not have a significant marginal abrasion but they may, instead, frequently demonstrate multiple marginal microlacerations (Fig. 13.23). Depending on the location of the entry wound, a distant high-velocity entrance wound can have relatively large, stellate, marginal lacerations (Disc Image 13.40). High-velocity exit wounds tend to be quite

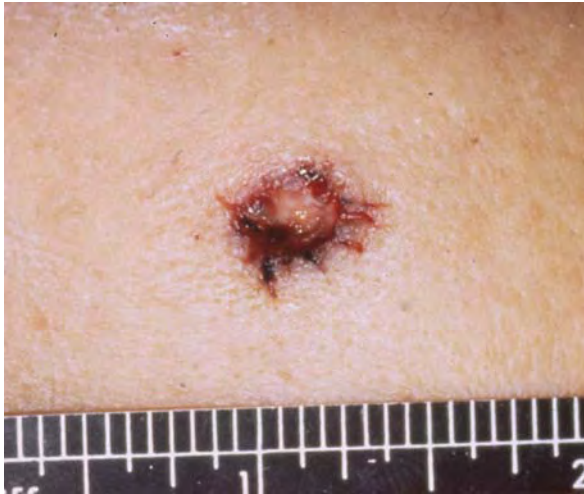


Fig. 13.23 A high-velocity entrance wound. Note the relative absence of a marginal abrasion and the presence of numerous marginal microlacerations

large and destructive, sometimes occurring as multiple exit sites (Fig. 13.24 and Disc Images 13.41, 13.42, and 13.43). Contact and non-contact shots to the head can be incredibly destructive (Fig. 13.25), but contact wounds can be markedly so, such that it may be difficult to identify the location of the entrance wound. Contact wounds of the trunk region may be similar in appearance to contact handgun wounds, with no associated stellate lacerations (Disc Image 13.44). The internal damage produced by high-velocity gunshot wounds tends to be quite extensive. X-ray examination of high-velocity wounds from bullets that fragment characteristically show numerous

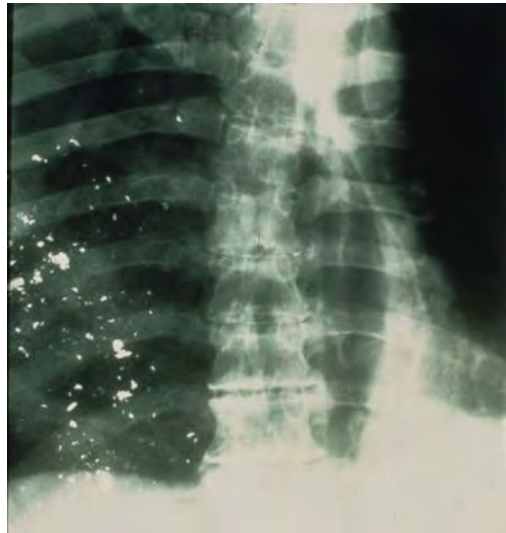


Fig. 13.24 A high-velocity exit wound, with extensive tissue damage

Fig. 13.25 A suicidal contact high-velocity rifle wound of the chin, with a massive exit wound complex of the left side of the face



Fig. 13.26 The characteristic “lead snowstorm” X-ray associated with a high-velocity projectile (typically hunting ammunition)



fragments of bullet along the pathway of the bullet, described as a “lead snowstorm” appearance (Fig. 13.26).

Shotgun Wounds

As described above, there are two basic types of projectiles that can be fired from a shotgun: shot pellets and slugs. Some of the features of shotgun wounds are

similar to those described with low-velocity gunshot wounds, but there are many additional features that deserve attention. Contact wounds, particularly of the head, are extremely devastating, whether birdshot, buckshot, or slugs are used (Fig. 13.27 and Disc Image 13.45). Reapproximation of portions of the face and head may be necessary to identify the exact location of the entrance wound. Intraoral and contact wounds of the lower chin (“submental” region) frequently demonstrate stretch-type lacerations of the cheeks and face, resulting from the explosive gases that rapidly expand and tear the soft tissues (Disc Image 13.46). Contact wounds elsewhere on the body mimic those produced by handguns, with soot deposition but no marginal lacerations (Fig. 13.28). Muzzle imprint abrasions may also occur (Disc Image 13.47). In non-contact wounds, if the muzzle is close enough to the skin, gunpowder stippling and soot may surround the entrance site. With some buckshot loads, a white filler material (which looks like little Styrofoam balls) can also cause stipple marks (Disc Image 13.48). These are produced by the filler material when the shotgun is up to about 9–10 ft from the skin, while the filler material can actually travel up to about 24 ft (without enough energy to cause stipple marks).



Fig. 13.27 A contact shotgun wound of the forehead, with massive destruction

When shot pellets are used, shotgun wounds where the muzzle is within 2–3 ft of the skin tend to have a single, large, circular entrance site where all of the charge (pellets, with associated wadding) enters the body. The closer the muzzle to the skin, the more smooth the margin of the defect appears. As the distance from muzzle to skin increases, the margin takes on a “scalloped” (“cookie-cutter”) appearance (Fig. 13.29). With additional distance, starting around 4 ft, separate individual pellet entrance sites surround the central defect (Fig. 13.30). When there are only a couple of these separate pellet entrance wounds, they are sometimes referred to as “satellite” wounds. If a plastic wad with petals is present within the shotgun shell, then there may be abrasions produced around the entrance defect by the petals that are opened up while traveling toward the target (Fig. 13.31 and Disc Image 13.49). When these are present, the wad material is probably within the body. Such wad



Fig. 13.28 A contact shotgun wound of the chest

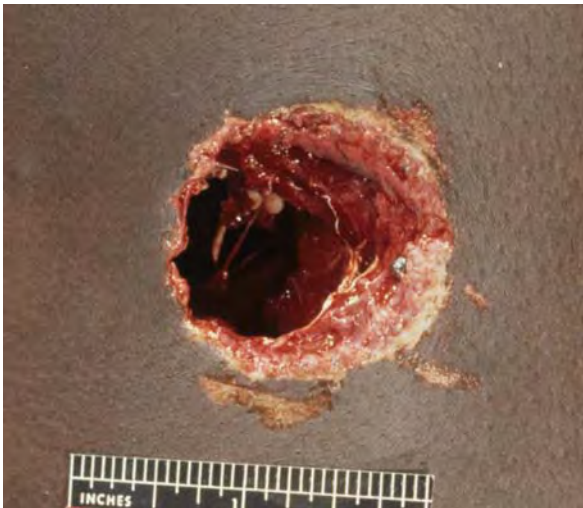


Fig. 13.29 A shotgun wound with “scalloped” margins

petal marks may occur at distances up to a couple of feet. Other “wad strike marks” may also be present and can either be near to, or some distance from the pellet entrance site (Disc Image 13.50). As the distance from muzzle to skin increases even further, the pellets spread out into larger and larger diameter areas (Fig. 13.32 and Disc Image 13.51), until there is no longer a central defect, but only separate pellet entrance injuries (Fig. 13.33). Close inspection of the separate pellet entrance defects will reveal a central circular or oval entrance defect with a circumferential marginal abrasion. Buckshot entrance defects mimic handgun bullet entrance

Fig. 13.30 A shotgun wound with a central defect, as well as occasional satellite pellet wounds



Fig. 13.31 A shotgun entrance wound with petal strike marks evident along the wound’s margins

wounds (Fig. 13.34). Measuring the diameter of the pellet spread pattern on the skin surface can assist in estimating the range-of-fire, regardless of the size of pellet used (Disc Image 13.52). It should be noted that since shotguns can have different “choke” settings, which increase or decrease the length that it takes the pellets to spread to a certain diameter, it is not as easy to estimate the range-of-fire with shotgun injuries. It is always best to perform test firing, using the suspect weapon, the same shot shell (same manufacturer, same shot size, etc.), and paper targets at varying distances.

Fig. 13.32 A shotgun entrance wound with a central defect and numerous individual pellet entrances



Fig. 13.33 A shotgun entrance wound complex, with absence of a central defect. The shot pellets have totally spread apart prior to striking the body

Birdshot pellets tend not to exit, but instead remain within the body. Buckshot pellets may or may not exit, but the exit wounds appear similar to those produced by low-velocity handguns (Disc Image 13.53). X-ray examination of shotgun pellet injuries will reveal the approximate size (bird versus buck), but are not reliable for estimating the skin spread pattern, since internally the pellets strike tissues and each other, resulting in the pellets “spreading out.” This is sometimes referred to as the “billiard ball effect” (Fig. 13.35).

Shotgun slugs are tremendously large projectiles that can produce massive injuries. Even non-contact wounds of the head with shotgun slugs can produce injuries that might otherwise appear to be contact wounds (Disc Image 13.54). Slug

Fig. 13.34 Buckshot entrance wounds

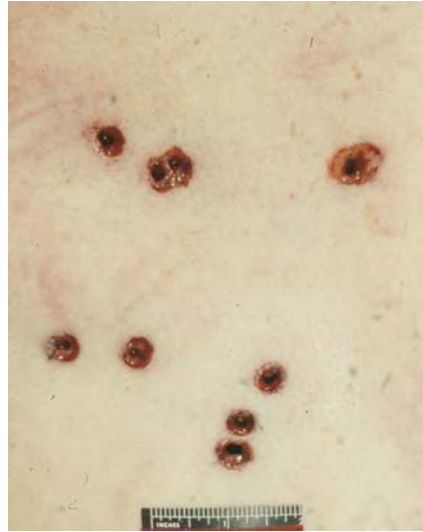


Fig. 13.35 The typical appearance of a birdshot shotgun wound X-ray



entrance wounds mimic classic entrance wounds although they tend to be larger. Slugs often break apart within the body and X-ray examination frequently shows these fragments to have a C-shape (depending on the direction of X-ray beam) (Disc Image 13.55). Pathologists should find out from investigators or police the type of slug used in a particular case. Certain slugs have portions that are non-metallic which may fall away from the slug in flight (plastic sabots) while others

may be connected to the slug (Brenneke slugs, where a cork-like base is attached to the slug by a screw).

Miscellaneous Issues

Internal Examination

No matter the projectile type, pathologists must determine the pathway of the projectile during the internal examination. If a projectile does not exit the body, it is said to be a “penetrating wound.” If a projectile exits the body, it is said to be a “perforating wound.” Occasionally, a projectile will partially exit the skin, but still remain within the skin or subcutaneous tissues (Disc Image 13.56). Depending on the projectile type, portions of a fragmented bullet may exit, while other portions remain in the body. The pathway of the projectile can be referred to as a “track”; however, some medical professionals, as well as others, inappropriately refer to it as a “tract.” In many instances, the bullet track is a relatively straight line within/through the body. Occasionally, particularly when the bullet strikes bone, a certain amount of deflection can occur; the smaller the projectile, the more likely such deflection is. For example, with birdshot pellets, deflection and ricocheting within the body is quite common. In contrast, internal deflection/ricocheting is quite rare with a large caliber bullet, such as a .45 ACP. It is not uncommon for small caliber bullets, such as .22 or .25 ACP, to ricochet within the skull.

During internal examination, pathologists will note the projectile track, keeping a record of the organs that the projectile injures (Disc Images 13.57, 13.58, 13.59, and 13.60). Also noted will be collections of blood within body cavities. In some contact wounds, soot can be readily identified within the subcutaneous tissues. Histologic examination can help to confirm the presence of soot (Fig. 13.36). Contact gunshot

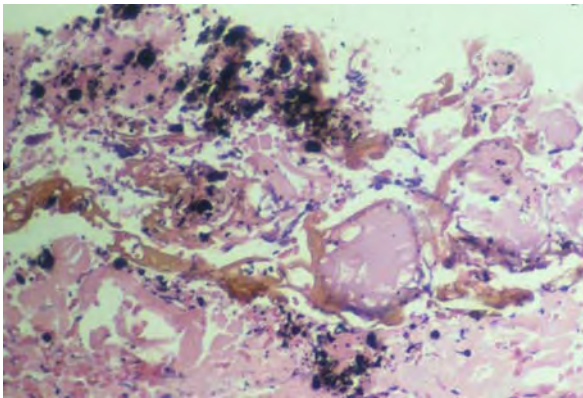


Fig. 13.36 A microscopic section of a contact gunshot entrance wound, showing extensive black particulate matter (soot) deposition

wounds of the head will frequently have soot deposited on the dura matter and/or bone that surrounds the skull entrance site (Disc Images 13.61 and 13.62). Another useful finding in head gunshot wound cases involves the presence of skull bone “beveling.” When a projectile strikes the skull, the side of the bone initially struck by the bullet will typically have a relatively well-defined hole that approximates the size and shape of the projectile. The opposite side of the skull will have a defect, typically of the same shape, but of much wider diameter. The two sides of this skull defect are connected by an angled, or beveled, surface of bone. The bone fragment that is “punched out” resembles a cone shape. Entrance wounds, by definition, result in skull bone defects that demonstrate “internal beveling” (the angled surface is evident on the inside of the skull) (Figs. 13.37, 13.38, 13.39, and 13.40), whereas exit wounds have skull defects with “external beveling.” Similar beveling can occur in other relatively flat bones, such as ribs and the sternum. Tangential gunshot wounds of the head can create a “keyhole” shaped defect, with internal beveling associated with the round end of the keyhole shape, and external beveling associated with the triangular end of the keyhole (Fig. 13.41). Gunshot wounds of the head can produce basilar skull fractures, particularly in the front parts of the skull, just over the eyes, causing hemorrhage around the eyes (“periorbital ecchymosis”) (Disc Image 13.63).

When describing the path of the gunshot wound, the pathologist should provide an overall pathway with regard to three planes (front/back, right/left, and up/over/down). For example, the bullet traveled from front to back, from left to right, and upward. Some pathologists prefer to estimate (or attempt to measure) the exact angles in relation to these planes.

If projectiles are identified on internal examination, they will be collected as evidence (Disc Images 13.64 and 13.65). In cases involving birdshot, only a representative sample needs to be collected. In buckshot cases, an attempt should be



Fig. 13.37 An outer view of a gunshot entrance wound of the skull, after scalp reflection (and after peeling away the soft tissue normally adherent to the outer surface of the skull), showing absence of beveling

Fig. 13.38 An inner view of a gunshot entrance wound of the skull, showing classic internal beveling

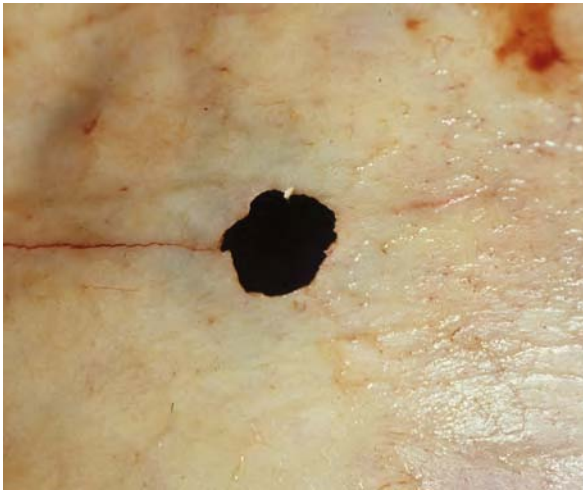


Fig. 13.39 An inner view of a gunshot exit wound of the skull, showing absence of beveling

made to collect all of the pellets. If any wadding material is present within the body, it should also be collected (Fig. 13.42); however, it should be noted that wadding is not readily apparent on X-ray examination. Another item that is difficult or impossible to see on X-ray examination is aluminum jacket material from certain bullets. In cases with markedly fragmented projectiles, the largest fragments, as well as any smaller fragments that are easily found, and any fragment that appears to be part of the jacket material, should be collected.

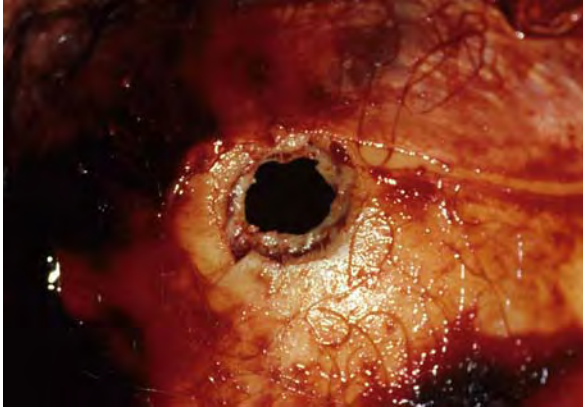


Fig. 13.40 An outer view of a gunshot exit wound of the skull, showing classic external beveling

Fig. 13.41 A “keyhole” defect in a skull, produced by a relatively tangential shot of the head, with the bullet traveling from bottom to top of the photograph



Different offices and forensic pathologists have varying protocols regarding whether or not a given projectile should be “inscribed” (marked) by the pathologist. In some places, the pathologist will inscribe his/her initials and perhaps the autopsy case number on the projectile (Disc Image 13.66), if there is a suitable location for such markings. Inscription is performed using a diamond-tipped pen. Care should be taken to avoid marking on the sides of the bullets, where rifle marks and striations exist and are used to attempt to match a bullet to a specific weapon. Other pathologists/jurisdictions prefer not to mark the bullets.



Fig. 13.42 Evidence collected from a body that was the victim of a shotgun injury. Note the presence of a representative sampling of the birdshot pellets, as well as plastic wadding material

Mechanism of Injury

The mechanism of injury in gunshot wound cases mimics those in many other traumatic causes of death. If the deep central structures of the brain and/or brainstem are disrupted by the projectile, it is generally accepted that immediate incapacitation results. Upper spinal cord injury will result in the inability to breathe. Brain injuries that are limited to more peripheral regions can be associated with longer survival, and perhaps even consciousness, depending on the location of the injury. Isolated frontal lobe injuries may not induce immediate unconsciousness or incapacitation. The mass effect associated with the injury and associated swelling can lead to compression of deeper brain structures and eventual death. In non-central nervous system sites, deaths frequently result from loss of blood. Other mechanisms, such as respiratory compromise (lung compression or lungs filling with blood) and air embolism (resulting from a neck or upper extremity venous injury with subsequent “sucking” of air into the vein) may also contribute to death. Delayed deaths can be related to infection, other medical complications (multiple organ failure, systemic inflammatory response syndrome, sepsis) and deep venous thrombosis due to pulmonary thromboembolism, to name some possibilities.

Gun enthusiasts and others occasionally become embroiled in discussions regarding which weapon/ammunition is the best for “stopping” someone. The term “stopping power” is occasionally used in this regard. Ultimately, the degree of physical injury sustained by a given tissue/organ is related to the amount of energy transferred from the projectile to that tissue/organ. Kinetic energy is calculated by the following formula: $KE = \frac{1}{2} mv^2$ (KE: kinetic energy; m : mass; v : velocity). It should be evident from this formula that the bigger the bullet (that is, the greater the mass), the more energy it contains. In fact, the amount of energy available to transfer from a moving bullet is directly proportional to the bullet’s mass. In contrast, there is an exponential increase in the amount of available energy within a moving

bullet as the bullet's velocity increases. Therefore, strictly speaking, increasing the velocity of a bullet will do more for "stopping power" than increasing the bullet's mass. In reality, one cannot totally separate one from the other. In general, the bigger the bullet, and the faster the bullet, the more potentially destructive it is. An important aspect related to tissue damage produced by projectiles is the concept known as the "temporary cavity." After a bullet has passed through a tissue, a bullet track or pathway remains which is referred to as the "permanent cavity." During the split second that a bullet is actually traveling through a tissue, a larger diameter, so-called "temporary cavity" also occurs. Tissues within this area can also sustain a significant amount of damage. The size and overall shape of the temporary cavity depend on the amount of kinetic energy transferred to the tissues from the bullet, as well as various features of the tissues themselves.

Despite all of these issues related to the energy, size and velocity of projectiles, probably the most important aspect of gunshot wound injuries is the location of those injuries. If a small caliber bullet penetrates the brain or transects the carotid artery, it is frequently more lethal than a high-velocity or large caliber bullet wound of an extremity.

A variety of other misrepresentations regarding gunshot wound injuries are propagated by the entertainment industry, or by word-of-mouth. A common misconception is alluded to elsewhere in this chapter, and suggests that, with all ammunition and weapon types, the exit wound is larger than the entrance wound. This is simply not true. Another myth is that when struck by a bullet, a person's body will respond by forcefully moving in the direction of the bullet. Still another myth is that a person struck by a bullet will be immediately incapacitated. While many shots to the central nervous system will result in immediate incapacitation or paralysis, not all of them do. For non-CNS shots, it is common for victims of gunshot injury to be able to function for many seconds to many minutes following infliction of the wound.

Documentation

It is wise to document all gunshot wound cases via written report, diagram, and photography. In certain cases, taking photographs of the victim "as is" (as they first appear, without disturbing anything) is appropriate. Subsequent washing away of blood, dirt, and other extraneous debris from around the wounds allows for better documentation. This should only be performed after any important trace evidence is documented and collected. In cases where the location of the wound is obscured by hair, it is appropriate to shave the hair from around the wound (Disc Images 13.67 and 13.68). Occasionally, it may become necessary to attempt to reapproximate wound edges, in order to better visualize an entrance wound. This is most common in contact gunshot wounds of the head, including those caused by certain handguns, but also very frequently in cases involving high-velocity rifles or shotguns. One method that can be employed involves using cyanoacrylate (super-strong glue) (Disc Images 13.69 and 13.70).

X-Rays

Even if it appears on external examination that a bullet has entered and exited the body, X-rays should be performed, as it is not uncommon in such instances to see a bullet fragment on X-ray. X-ray examination can sometimes provide clues as to the type of ammunition present, since various types of ammunition can have very characteristic radiological appearances. Examples include birdshot, buckshot, shotgun slugs, high-velocity bullets (“lead snowstorm”), and an array of special ammunition types. Particularly if a bullet has fragmented, X-ray examination can sometimes provide information regarding the general path of the bullet, as fragments of the bullet are scattered along the wound track. It is a good idea to perform X-ray examination prior to removal of clothing and medical therapy, as bullets or bullet fragments may be present within these items (Disc Images 13.71 and 13.72). A variety of radio-opaque (visible on X-ray) objects may mimic bullets. In addition, certain bullets have aluminum jackets (Disc Image 13.73) which, if separated from the lead core of the bullet, may not be identified on X-ray examination.

Clothing Examination

As mentioned several times already in this chapter, the examination of the clothing is absolutely essential in gunshot wound cases. If possible, the pathologist should be allowed to perform at least a basic clothing examination at autopsy. This is relatively easy if the decedent arrives at the morgue wearing their clothing, but if the person has received medical treatment, it is likely that the clothing has already been removed, and sending the clothing to the morgue with the body is an option. If death did not occur immediately, the clothing may have already been collected by the police. In some jurisdictions, bodies are transported many miles for a forensic autopsy and depending on the protocols of various jurisdictions, the clothing may or may not accompany the body. All police and death investigators should be aware of the fact that forensic pathologist examination of the clothing in gunshot wound victims is the preferred method of handling these cases.

Sometimes, the direction of projectile travel can be ascertained by examining the gunshot defects within the clothing. As mentioned earlier, gunshot residue (soot and gunpowder) may be deposited on the clothing. The type of clothing worn may provide clues as to why a shored exit wound is present. Projectiles or fragments of projectiles may be present within the clothing. If clothing has been moved out of the way of a contact gunshot wound of the chest, the case is most likely a suicide; however many, if not most, suicidal gunshot wounds of the chest occur through the clothing.

Gunshot Residue

The term “gunshot residue” is used by some to describe the smoke/soot and gunpowder that exits the barrel (or cylinder-barrel gap) when a firearm discharges. The term

“gunshot residue,” in another usage, refers to only the smoke and gases produced when a firearm discharges. Contained within this cloud of smoke are small quantities of substances that are contained within the primer of the cartridge; most common are the elements barium, antimony, and lead. A person’s hands can be tested for the presence of these primer components. Usually, trace amounts of material are collected from the backs and palms of the hands and then analyzed. In one type of test, the hands are swabbed with a cotton-tipped swab premoistened with hydrochloric or nitric acid, and then the swabs are tested by flameless atomic absorption spectroscopy and/or neutron activation analysis. In another method, special adhesive lifting devices are used to collect the specimen, with subsequent analysis by a scanning electron microscope.

The usefulness of testing someone’s hands for the presence of gunshot residue is debatable at best. Ideally, a positive test would indicate that a person fired a weapon recently, and a negative test would indicate that a person did not fire a weapon recently. In reality, the test can be negative in persons who have definitely fired a weapon recently, and it can be positive in persons who definitely have not fired or even handled a weapon recently. The mere handling of a weapon or simply being in the vicinity of a discharging firearm, can result in a positive test and, because of this, many offices and jurisdictions do not perform gunshot residue testing at all. Some offices, however, still perform these tests, as it can provide some useful information in selected cases.

As the components contained within primers continue to develop and transform, and as testing techniques become more specialized, future gunshot residue tests may actually do a better job in addressing the issues presented above.

Manner of Death Issues

Homicides: Many gunshot wound deaths represent homicides. While contact homicidal gunshot wounds can and do occur, most homicidal gunshot wounds are not contact range. Occasionally, “defense” type injuries are seen, wherein it is obvious on external examination that the victim was aware that someone was shooting at them, and they have injuries on their hands/forearms indicating that they had thrown their arms upward in a defensive (or surrendering) posture.

Suicides: A vast majority of suicidal gunshot wounds are contact wounds. Close range wounds are rarely present. Suicidal gunshot wounds from long guns (rifles or shotguns) are relatively common. A standard practice amongst certain pathologists in these cases is to measure the distance from the entrance wound to the fingertip, in order to determine whether or not the person could have pulled the trigger. Unfortunately, this technique does not take into consideration the fact that most individuals are able to extend their arm/hand/fingers more fully via shoulder movement. Also, it is not unusual to find that a person has used another object (coat hanger, stick) or even their toes to depress the trigger (Disc Image 13.74). Common locations for suicidal gunshot wounds include the temple, the forehead, the roof of the mouth (intraoral), the lower (submental) part of the chin, and the chest, overlying the heart. It may be important for investigators to find out if the person was right- or

left-handed. While relatively uncommon, it is certainly not unusual for a person who commits suicide to have more than one wound (Disc Image 13.75). In these cases, it is important, on internal examination, to determine that it was, in fact, possible for the person to fire all of the shots. In some offices and jurisdictions, Russian roulette cases are ruled as accidents; however, many others argue that the act is purposeful, dangerous, and knowingly potentially lethal, and as such should be ruled as suicide.

Accidents: The topic of accidental gunshot wound fatalities is the source of a great deal of debate amongst forensic pathologists. Many agree that if a very young child fires a weapon, the incident represents an accident. If the discharge involves an older child or teenager, arguments arise that suggest that the shooter should have known the inherent dangers of handling the weapon. If a shooter claims that the firearm malfunctioned and a firearm examiner inspects the weapon and discovers that it does not function properly, it can be appropriate to rule such deaths as accidents. So-called hunting accidents come in all sorts of scenarios but each situation must be evaluated on its own merits, and examination of the firearm by a firearm examiner is essential.

Special Weapons, Ammunition, and Circumstances

A variety of special weapons, ammunition types, and circumstances exist. It is well beyond the scope of this chapter to provide details regarding all of these. A few comments will be provided for some of these.

“Zip guns” are homemade weapons, made of a variety of objects, from pipes to ink pens, and can be quite dangerous. They are not very common in the United States, where cheap handguns are readily available.

Air-powered guns, such as BB guns and CO₂ cartridge guns, are relatively common. Depending on a variety of circumstances, these can, in fact, produce lethal injuries.

Nail and stud guns are industrial tools that use air or gunpowder cartridges to “shoot” nails and other fasteners into wood, concrete, steel, etc. When misused or used carelessly, they can result in serious bodily injury or death.

Captive bolt devices are specially designed weapons used to slaughter livestock. The explosion produced by a blank cartridge (containing only primer and gunpowder) causes a bolt to rapidly protrude from the end of the device, but remain attached to the device. Placing the end of the device against the animal’s head, followed by discharge, results in death of the animal.

Black powder firearms may be genuine antique weapons or newly-made weapons specially designed to use old-time (black powder) firing mechanics. The projectile in some of these weapons is a metallic ball (like a musket ball) and there is usually a great deal of black soot produced on discharge (Fig. 13.43). In order to estimate the range-of-fire with these weapons, it is best to perform test firing.

Blank cartridges are cartridges that contain a primer and gunpowder, but no projectile. Because of the explosive force created upon discharge, these cartridges are

Fig. 13.43 An example of the dense soot produced with a black powder weapon



capable of producing lethal injuries at contact range. Air guns can likewise cause serious injury or death.

Handgun shot cartridges are available in a number of calibers and styles. These cartridges contain small shot pellets and are sometimes referred to as “snakeshot” or “ratshot” (Disc Image 13.76). In some, the brass casing is simply elongated and crimped over the pellets. In others, a plastic cap covers and contains the pellets within the cartridge. Injuries mimic shotgun injuries (Fig. 13.44).



Fig. 13.44 An example of the appearance of a wound produced by a handgun shot cartridge

NYCLAD bullets are coated with nylon in order to reduce the amount of lead particles in the air of indoor shooting ranges. Unfortunately, rifle markings and striations from the barrel are not as easily seen on the nylon material, making ballistics comparison more difficult than with normal lead or jacketed bullets (Disc Image 13.77).

A relatively common type of ammunition that can also be difficult to evaluate from a ballistics standpoint is the small caliber, “gilded” or “washed” bullet. These bullets, usually of .22 or .25 caliber, have a copper-colored coating that is often substantially scraped off by the time it comes to rest within a body (Disc Image 13.78). As such, evaluation for striations can be difficult.

Glazer bullets are composed of a copper jacket containing numerous lead pellets and covered with a Teflon plug. The special design is marketed as a round that will not perforate unintentional interposed targets (such as a wall), thus reducing the chances of someone behind the interposed target from being injured (Disc Image 13.79).

A variety of bullet types are made up of compressed material that may or may not break apart into smaller fragments when a target is hit. These “frangible” bullets cannot be easily matched to a specific weapon.

A few bullets actually have jackets that are composed of steel. As such, the jacket material is subject to corrosion (rust formation). Pathologists frequently place recovered bullets in a solution of water and soap or bleach in order to wash away blood and debris. If steel-jacketed bullets remain in contact with water for even a short time, corrosion can occur, thus obscuring rifle markings.

“Sabot” ammunition uses a plastic sleeve surrounding a typical bullet; it can be used with shotgun slugs (see above), or with rifled ammunition. With rifled ammunition, the plastic sleeve will have rifle marks, but the bullet will not (Disc Image 13.80).

“Homemade” loads are most commonly associated with shotgun shells, where any number of items may be placed within the shotgun shell, including virtually any metal item small enough to fit inside the shell.

Some weapons are able to fire bullets that are not the correct caliber, resulting in various bullet deformities. In a similar fashion, weapons whose barrels have been removed will fire bullets that have unusual deformations (as well as lack of rifle marks).

“Tandem bullets” describe a situation wherein a bullet is lodged within the barrel of a weapon and a subsequent discharge results in two projectiles exiting the barrel. If the shot is a non-exiting contact wound, the victim will have a single entrance but two bullets inside their body.

Another explanation for the presence of more bullets inside a body than can be explained by the number of entrance wounds is the presence of a “retained” bullet. A retained bullet is a bullet within a body from a previous gunshot wound, sometimes many years previously. A bullet may be left in a body for various medical reasons. If a person subsequently dies, the retained bullet will still be evident on X-ray. On internal examination, the typical retained bullet is surrounded by scar tissue, with no evidence of acute injury (bleeding) surrounding the bullet (Disc Image 13.81).

Occasionally, at autopsy, a bullet cannot be found internally on initial, routine examination. The bullet track ends within the heart or aorta or other large caliber blood vessel. On further X-ray examination, it becomes apparent that the bullet (usually of small caliber) has “embolized” (traveled within the blood vessel to a site distant from its origin). The bullet may be discovered inside a branch artery of the aorta, such as within the pelvis or even the leg.

Disc Image Legends

Disc Image 13.1 Two bullets with rifle marks.

Disc Image 13.2 An intact cartridge and an opened cartridge, showing the casing, the gunpowder, and the bullet.

Disc Image 13.3 Comparison of a rimfire cartridge and a centerfire cartridge.

Disc Image 13.4 Additional comparison of rimfire versus centerfire cartridges.

Disc Image 13.5 Comparison of semi-automatic and revolver cartridges.

Disc Image 13.6 Multiple examples of handgun ammunition of various calibers. Top (L to R): .22, .38 special, .44 magnum; Bottom (L to R): .25, .32, .380, 9 mm, .40 S&W, 10 mm, .45 ACP, .50 caliber.

Disc Image 13.7 A variety of special bullet types. The lower row shows unfired bullets only, while the upper two rows show an unfired bullet (middle row) immediately below an example of a corresponding fired bullet (upper row). Lower row (L to R): full metal jacketed bullet, brass-colored full metal jacketed bullet, wad-cutter, semi-wad-cutter, Nyclad, aluminum jacketed bullet. Middle and upper rows (L to R): jacketed soft point, Black Talon, Hydrashok, Silvertip, Golden Saber, Gold Dot.

Disc Image 13.8 A variety of different sized birdshot pellets. L to R: #2, #4, #5, #6, #7 1/2, #8, and #9.

Disc Image 13.9 Various types of wadding material from within shotgun cartridges.

Disc Image 13.10 Various types of shotgun slugs.

Disc Image 13.11 Another example of an X-ray showing the presence of projectiles.

Disc Image 13.12 An example of an angled gunshot entrance wound. The bullet entered the skin at an angle, with initial contact at approximately the 11 o'clock position, traveling downward and slightly to the right as viewed in the photo.

Disc Image 13.13 A bullet has entered and exited the arm and then re-entered the left side of the thorax/back.

Disc Image 13.14 Fragmented bullet entrance wounds.

Disc Image 13.15 An entrance wound of the thick (palmar) skin on the side of the hand. Note the presence of rare gunpowder stipple marks.

Disc Image 13.16 An entrance wound of the thick skin on the sole of a foot.

Disc Image 13.17 Another example of a contact entrance wound of the head (forehead).

Disc Image 13.18 Contact .22 caliber gunshot wound of head (temple). Note the presence of soot, but the absence of significant stellate lacerations.

Disc Image 13.19 Contact gunshot wound of the chest.

- Disc Image 13.20 Contact wound of the chest, with associated pink-red discoloration surrounding the wound.
- Disc Image 13.21 Intraoral gunshot wound of the roof of the mouth, as viewed via a mirror that has been inserted into the open mouth. Note the muzzle imprint and soot deposition.
- Disc Image 13.22 Multiple lip lacerations associated with an intraoral suicidal gunshot wound.
- Disc Image 13.23 Another example of a close-range gunshot entrance wound, with soot and stippling.
- Disc Image 13.24 Another close-range gunshot entrance wound.
- Disc Image 13.25 An article of clothing demonstrating soot deposition.
- Disc Image 13.26 An article of clothing demonstrating gunpowder deposition.
- Disc Image 13.27 Another example of a medium (intermediate)-range gunshot entrance wound.
- Disc Image 13.28 A medium-range gunshot wound where much of the gunpowder stippling has been blocked by hair (which has been shaved at autopsy).
- Disc Image 13.29 Five examples of different gunpowder types from five different cartridges.
- Disc Image 13.30 Dark discoloration of the margins of an entrance wound caused by postmortem drying. This can be misinterpreted as soot.
- Disc Image 13.31 Another example of a non-contact gunshot entrance wound, with drying of the marginal abrasion.
- Disc Image 13.32 An example of a gunshot entrance wound with surrounding stipple marks produced by fragments of an interposed target. Note the atypical appearance of the entrance wound, characteristic of a wound caused by a projectile that has struck an interposed target prior to hitting the body.
- Disc Image 13.33 An example of a slit-like gunshot exit wound.
- Disc Image 13.34 An example of a comma-shaped gunshot exit wound.
- Disc Image 13.35 An angled gunshot entrance wound (left) adjacent to an unrelated gunshot exit wound (right). Note the presence of a marginal abrasion in the entrance wound and the absence in the exit wound.
- Disc Image 13.36 An area of subcutaneous hemorrhage indicating the location of a subcutaneous bullet.
- Disc Image 13.37 Another example of a graze gunshot wound. In this case, the direction of travel of the bullet was from right to left in the photograph.
- Disc Image 13.38 An L-shaped cylinder-barrel gap mark.
- Disc Image 13.39 An injury on the hand of a shooter, caused by the slide of a semi-automatic pistol.
- Disc Image 13.40 A distant range, high-velocity entrance wound of the scalp, with relatively extensive marginal lacerations.
- Disc Image 13.41 A high-velocity rifle wound injury complex of the legs. The small entrance wound is just visible on the outside edge of the right thigh (left side of photo). The large exit wound involves the inner aspect of the right thigh. Finally,

there are multiple re-entrance wounds from bullet fragments located on the inner aspect of the left thigh.

Disc Image 13.42 A closer view of the high-velocity entrance wound shown in Disc Image 13.41.

Disc Image 13.43 A closer view of the high-velocity exit wound shown in Disc Image 13.41.

Disc Image 13.44 A contact high-velocity entrance wound of the chest.

Disc Image 13.45 A contact shotgun wound of the lower chin. Note the stretch-type lacerations of the right side of the face.

Disc Image 13.46 Lacerations of the corners of the mouth and stretch-type lacerations of the cheek, related to a contact shotgun wound of the lower chin.

Disc Image 13.47 Contact shotgun wound of the chest from a double-barrel shotgun, with a muzzle imprint abrasion.

Disc Image 13.48 Extensive stipple marks related to shotgun filler material from a buckshot charge. Note the presence of the small white filler material within and around the entrance defect.

Disc Image 13.49 Another example of shotgun wadding petal marks on the skin around an entrance wound. In this case, the plastic wadding had very firm petals, thus causing very well-defined abrasions.

Disc Image 13.50 A shotgun wad strike mark, with the wadding material adjacent to the injury (photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC).

Disc Image 13.51 Another example of a shotgun wound, with a central defect surrounded by numerous satellite pellet entrance wounds.

Disc Image 13.52 A homicide victim with three shotgun wounds of the back. All were fired from the same weapon at a similar range. Two shots utilized buckshot, while the third contained birdshot (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner).

Disc Image 13.53 Three buckshot exit wounds, one just under the nose, and two on the chin.

Disc Image 13.54 A non-contact shotgun slug wound of the head, with massive destruction.

Disc Image 13.55 Shotgun slug fragments as seen on X-ray. Note the C-shape of the largest fragment.

Disc Image 13.56 A partial gunshot exit wound of the neck, where the copper-jacketed bullet protrudes through the skin (arrow).

Disc Image 13.57 A gunshot wound of the aorta.

Disc Image 13.58 A gunshot wound of the lung.

Disc Image 13.59 A gunshot wound of the heart.

Disc Image 13.60 A shotgun slug wound of the heart.

Disc Image 13.61 Soot identified on the bone underlying a contact gunshot wound of the head.

Disc Image 13.62 Soot identified on the dura underlying a contact gunshot wound of the head.

- Disc Image 13.63 Bilateral (involving both sides) periorbital (around the eyes) ecchymosis (bleeding) caused by basilar skull fractures produced by a gunshot wound.
- Disc Image 13.64 A deformed (“mushroomed”) bullet collected as evidence.
- Disc Image 13.65 Multiple bullet fragments collected from a single gunshot wound.
- Disc Image 13.66 Inscription on the base of the bullet.
- Disc Image 13.67 A photograph showing a suicidal gunshot wound of the head, prior to shaving the hair from around the entrance wound.
- Disc Image 13.68 A photograph taken after cleaning the body and shaving the hair from around the entrance wound.
- Disc Image 13.69 Contact gunshot wound of head, after cleaning and shaving.
- Disc Image 13.70 Contact gunshot wound of head, after cleaning, shaving, and reapproximating wound edges using cyanoacrylate.
- Disc Image 13.71 An X-ray taken prior to clothing removal in a toddler who was being held in his mother’s arms when she was shot multiple times.
- Disc Image 13.72 An X-ray taken after clothing removal in the case shown in Disc Image 13.71. Note that a majority of the bullets exited the body but were retained within the clothing.
- Disc Image 13.73 Aluminum-jacketed bullet, with the lead core separated from the aluminum jacket.
- Disc Image 13.74 A suicide victim who used a coat-hanger to depress the trigger of his shotgun (photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC).
- Disc Image 13.75 A suicide victim who was witnessed by numerous people to place the small caliber weapon under his chin and discharge the weapon. After each of the first three shots, he looked at the weapon, placed it back under his chin, and fired again, for a total of four shots. At autopsy, only one bullet was found to have entered the brain.
- Disc Image 13.76 Snakeshot or ratshot cartridges.
- Disc Image 13.77 NYCLAD cartridge and bullet.
- Disc Image 13.78 A gilded small caliber bullet recovered from a body at autopsy. Note that the copper-colored material has “flaked off” the sides of the bullet.
- Disc Image 13.79 Glazer cartridge, unfired bullet, and fired bullet.
- Disc Image 13.80 Sabot rifle cartridge, plastic sabot, and bullet.
- Disc Image 13.81 A retained bullet recovered at autopsy. Note that the bullet is embedded within a piece of muscle, with no surrounding hemorrhage.

Selected References

- Cina SJ, Ward ME, Hopkins MA, Nichols CA. Multifactorial analysis of firearm wounds to the head with attention to anatomic location. *Am J Forensic Med Pathol* 1999;20:109–15.
- Clark MA, Micik W. Confusing wounds of entrance and exit with an unusual weapon. *Am J Forensic Med Pathol* 1984;5:75–8.
- Cunliffe CH, Denton JS. An atypical gunshot wound from a home-made zip gun – the value of a thorough scene investigation. *J Forensic Sci* 2008;53:216–8.
- DiMaio VJM. *Gunshot Wounds*, 2nd ed. Boca Raton, FL: CRC Press; 1999.

- Dodd MJ. *Terminal Ballistics – A Text and Atlas of Gunshot Wounds*. Boca Raton, FL: Taylor & Francis; 2006.
- Jacob B, Huckenbeck W, Daldrup T, Haarhoff K, Bonte W. Suicides by starter's pistols and air guns. *Am J Forensic Med Pathol* 1990;11:285–90.
- Juvin B, Brion F, Teissiere F, Durigon M. Prolonged activity after an ultimately fatal gunshot wound to the heart: case report. *Am J Forensic Med Pathol* 1999;20:10–2.
- Karger B, Kneubuehl BP. On the physics of momentum in ballistics: can the human body be displaced or knocked down by a small arms projectile? *Int J Legal Med* 1996;109:147–9.
- Kury G, Weiner J, Duval JV. Multiple self-inflicted gunshot wounds to the head: report of a case and review of the literature. *Am J Forensic Med Pathol* 2000;21:32–5.
- Molina DK, Wood LE, DiMaio VJM. Shotgun wounds: a review of range and location as pertaining to manner of death. *Am J Forensic Med Pathol* 2007;28:99–102.
- Molina DK, Martinez M, Garcia J, DiMaio VJ. Gunshot residue testing in suicides: part I: analysis by scanning electron microscopy with energy-dispersive X-ray. *Am J Forensic Med Pathol* 2007;28:187–90.
- Molina DK, Castorena JL, DiMaio VJ. Gunshot residue testing in suicides: Part II: analysis by inductive coupled plasma-atomic emission spectrometry. *Am J Forensic Med Pathol* 2007;28:191–4.
- Shields LBE, Hunsaker JC, Stewart DM. Russian roulette and risk-taking behavior – a medical examiner study. *Am J Forensic Med Pathol* 2008;29:32–9.
- Zaki SA, Hanzlick R. Gunshot wound with asphalt related pseudosooot, pseudo-tattooing, and pseudo-scorching. *J Forensic Sci* 1987;32:1136–40.

Chapter 14

Sharp Force Injury Deaths

When Phinehas . . . saw this, he left the assembly, took a spear in his hand and followed the Israelite into the tent. He drove the spear through both of them – through the Israelite and into the woman’s body.

Numbers 25:7–8

Abstract Sharp force injuries result from contact with sharp-edged or pointed objects. This chapter reviews the three major types of sharp force injury: stab wounds, incised wounds, and chop wounds, and provides further information regarding various miscellaneous topics and issues, including internal examination, mechanism of injury, trace evidence, clothing examination, defensive wounds, and hesitation marks, among others.

Keywords Sharp force injuries · Stab wounds · Incised wounds · Chop wounds

Introduction

Sharp force injuries are caused by contact with sharp-edged or pointed objects. This is in contrast to blunt force injuries, as well as missile/projectile injuries and other injury types. There are three basic types of sharp force injury: stab wounds, incised wounds, and chop wounds. It is important to re-emphasize here what was previously detailed in Chapter 12 regarding the term “laceration.” Far too commonly, persons, including many medical personnel, refer to a sharp force injury as a “laceration.” A laceration is a splitting of tissue that results from blunt force. Usually, the circumstances as well as the presence of “tissue bridging” within the depths of the wound enable lacerations to be distinguished from sharp force trauma.

When describing sharp force injuries, one must become familiar with certain definitions. Many sharp injuries have a relatively linear (line-like) shape. The ends or tips of such an injury are commonly referred to as “angles,” and the sides or edges of the wound are typically referred to as “margins.” Because of the elastic

qualities of the skin and subcutaneous tissues, many sharp force injuries can be described as “gaping,” since the margins (edges) fall away (gape) from one another (Fig. 14.1). When examining such wounds, it is important to re-approximate the wound edges (Fig. 14.2). This allows better visualization and examination of the angles (ends) of the wound, and it provides a more accurate measurement of the wound. Pathologists will also take note of any abrasions or other injuries located along or near the margins and/or angles of the sharp force injury. It is appropriate



Fig. 14.1 A gaping stab wound. The elastic forces of the skin allow the wound’s margins to “gape” open

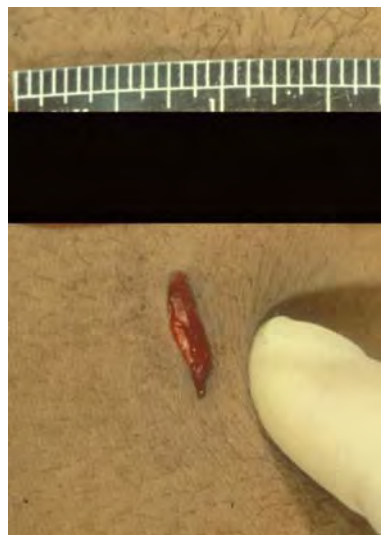


Fig. 14.2 Reapproximation of a sharp force injury’s margins, such as in the stab wound depicted in Fig. 14.1, allows for a more accurate measurement of the wound

to describe the shape, the length and the width of sharp force wounds. When linear, it is appropriate to describe the direction of the long-axis of the wound. Clock-face nomenclature can be utilized in this regard; for example, “the long axis of the wound runs between the 1 and 7 o’clock positions.” Descriptions of the angles and margins of the wound, along with any associated injuries (abrasions, contusions, etc.) should be included. Internally, the direction of the wound (front–back, right–left, up–down), as well as the maximal depth of penetration should be noted.

Stab Wounds

Stab wounds are caused by sharp, pointed objects, with the direction of force oriented more-or-less perpendicular to the skin surface. In most instances, stab wounds are deeper than they are long (Fig. 14.3 and Disc Image 14.1). Other names that are sometimes used to describe stab wounds are “puncture” wounds, “perforating” wounds, or “penetrating” injuries. The reader should be reminded that the term “perforating” should imply that the wound (whether it is produced by a bullet or a knife) is a through-and-through wound, having an entrance site and an exit wound. In contrast, the term “penetrating” implies that the wound enters the body but does not exit. If the object inflicting the injury is in motion, a stab wound can be thought of as resulting from a thrusting action of the object. Occasionally, stab wounds are produced when a moving body impacts a relatively stationary pointed object.

Fig. 14.3 A single stab wound of the neck, deeper than it is long



Most stab wounds encountered by forensic pathologists are the result of knives (Disc Image 14.2). Knives can be single-edged, double-edged, or a combination, and may have a smooth sharp edge or a serrated edge (having “teeth”). When a single-edged knife causes a stab wound, the angle corresponding to the blunt edge

of the blade will demonstrate a squared-off (blunt) appearance, while the angle corresponding to the sharp edge of the blade will come to a sharp point (Fig. 14.4). A double-edged blade will produce a stab wound with two sharp angles (Fig. 14.5). Occasionally, because of postmortem drying or other complicating factors, a particular angle may not be recognized as either blunt or sharp. It is appropriate in such situations to describe the angle as “indeterminate.” In addition, twisting of the knife (or movement of the body), as well as multiple stabs within the same general area, can result in stab wounds with various irregular shapes (Disc Image 14.3). Often there is more than one stab wound present (Disc Image 14.4). When there are numerous sharp force injuries, it is acceptable to describe multiple wounds together;



Fig. 14.4 A single-edged stab wound. Note that the left angle is blunt, while the right angle is sharp



Fig. 14.5 A double-edged stab wound. Note that both angles are sharp

describing “clusters” of stab wounds within a given region of the body is an example (Fig. 14.6).

Abrasions or other markings associated with the margins or angles of a stab wound may indicate certain characteristics of the knife. Abrasions can be caused by the handle of the knife if the knife is totally inserted into the skin. The presence of numerous superficial parallel incised wounds/abrasions can indicate that a serrated knife was used (Fig. 14.7 and Disc Image 14.5). Without the presence of such surface markings, it is usually impossible to differentiate wounds caused by a serrated-edged blade from those produced from a smooth-edged blade.

Through-and-through (perforating) stab wounds are not uncommon on the arms (Disc Image 14.6). When they are present, it may be impossible to determine with certainty which wound represents the entrance and which represents the exit. Clothing examination may or may not assist in this determination.



Fig. 14.6 A cluster of stab wounds on the neck



Fig. 14.7 A stab wound produced by a serrated knife. Note the multiple, superficial (shallow), parallel incised wounds along the upper margin of the wound

When correlating a stab wound to the corresponding knife blade, it is important to keep track of the nomenclature of the blade and the wound. The blade itself has a measurable length, a measurable width, and a measurable thickness. Likewise, the wound itself has a measurable length (produced by the width of the blade), a measurable width (produced by the thickness of the blade), and a measurable depth (produced by the length of the blade). It should be noted that a given blade can produce wound lengths equal to, longer than, or even shorter than the width of the blade. This can be explained by movement of the blade as well as elasticity of the skin. Likewise, a given blade can produce wound widths that are equal to, greater than, or even less than the thickness of the blade. Finally, a given blade can produce wound depths that are equal to, less than, or even longer than the blade length. The latter can be explained by the fact that there is a certain amount of flexibility of the human body, such that if a knife is plunged into the body all the way to the handle, the stab wound can continue to travel deeper if the skin and other tissues are compressed by continued pressure. This can happen even with chest/rib cage compression.

Many other objects/weapons can produce stab wounds. In general, the stab wound tends to take on the shape of the particular object. With some objects, such as a screwdriver (Disc Image 14.7), it is important to remember that the shaft of the tool may have a shape different from the tip of the tool. Rectangular wounds suggest a standard screwdriver tip (Fig. 14.8), while cross-shaped wounds suggest a Phillips screwdriver tip. The shafts may be square or round. Ice-picks and awls usually produce round wounds (very similar to the appearance of gunshot wounds). Scissors blade wounds mimic knife blades if the scissors was open when the wounds were inflicted. If closed, the wound mimics the overall shape of the closed scissors blades. Arrows produce stab wounds that take on the shape of the arrow itself: target arrows are round (like gunshot wound entrances), while hunting arrows have an X- or cross-shape. In general, stab wounds take on the shape of the inflicting



Fig. 14.8 Multiple stab wounds produced by a standard (rectangular-shaped) screwdriver. Note that many of the wounds have a rectangular shape

Fig. 14.9 A superficial (shallow) puncture wound of the skin produced by a Phillips-style screwdriver

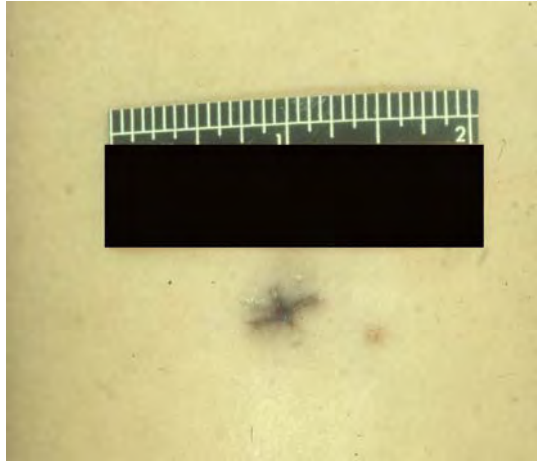
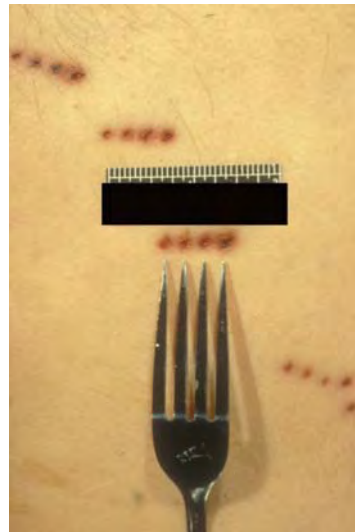


Fig. 14.10 Multiple superficial (shallow) stab wounds produced by a fork



object/weapon (Figs. 14.9 and 14.10). Firm cylindrical objects, such as rebar, fence T-posts, pipes, etc. can also produce stab or penetrating/perforating wounds.

Incised Wounds

Incised wounds can also be referred to as “cutting” injuries. They result from contact with a sharp-edged object, with the force of impact running in a direction that is roughly parallel (or tangential) to the skin surface (Fig. 14.11 and Disc Image 14.8). If the object inflicting the injury is in motion, an incised wound can be thought of as resulting from a slashing action of the object. Typically, the angles at either end



Fig. 14.11 An incised wound of the neck. Note that it is longer on the skin surface than it is deep

of an incised wound will have a sharp appearance. Determining the direction that an incised wound was inflicted – for example, from right to left across the neck, or from left to right – is not usually possible, unless small tags of skin can be seen “rolled-up” along the margin of the wound, in a fashion similar to certain abrasions where the direction of force can be determined.

Incised wounds produced by smooth-edged blades and serrated-edged blades can be indistinguishable from one another; however, serrated weapons may produce surrounding superficial skin injuries as described above. Alternatively, there may be similar marks elsewhere on the skin surface, distant from stab and incised wounds (Disc Image 14.9). As with clusters of stab wounds, incised wounds can also be clustered (Fig. 14.12).



Fig. 14.12 A cluster of incised wounds across the face and neck. (photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)

Although knives are the weapon used in a majority of incised wounds encountered in most forensic practices, any object with a sharp point or a sharp edge can result in incised, or cutting, injuries. Examples include razor blades, broken glass, scissors, barbed wire, and box cutters. Many of the injuries produced by these items are quite similar to those produced by knives. Exceptions involve a very jagged piece of glass or an object with multiple sharp points (such as barbed wire), such that the associated sharp injuries have a more irregular shape compared to the more linear wounds produced by the other instruments (Figs. 14.13 and 14.14 and Disc Image 14.10).

Fig. 14.13 Multiple sharp force injuries produced by barbed wire



Fig. 14.14 Multiple irregular incised wounds produced by a broken bottle



Chop Wounds

Chop wounds can be considered a combination of both sharp and blunt force injuries, and are typically produced by objects that have a relatively sharp edge in association with a significant degree of force. This force results from the weight of the object or the speed at which the object is moving. Examples of items that can produce chop wounds include a hatchet, an axe, a tomahawk (Disc Image 14.11), a boat propeller, and a lawnmower blade.

The typical chop wound has a sharp appearance on the skin surface, frequently with associated abrasions and contusions (Fig. 14.15 and Disc Image 14.12). The wounds often mimic the shape of the object. In addition, there is frequently significant underlying trauma. In areas with underlying bone, such as the scalp, there is frequently underlying bone trauma (fractures). Boat propellers tend to produce multiple parallel chop marks (Disc Image 14.13). Some chop wounds are extremely destructive, such as those produced by farm equipment (Disc Image 14.14).

Fig. 14.15 Multiple chop wounds of the scalp, produced by the “claw” end of a hammer. Note the associated abrasions. There were underlying skull fractures



Special Issues

Internal Examination

Internal examination of sharp force injuries should result in documentation of the pathway (track) of each injury, with notation of wound direction, maximal depth of penetration, and a listing of the tissues/organs injured (Figs. 14.16 and 14.17 and Disc Images 14.15 and 14.16). Stab wounds may not produce a significant amount of external bleeding, since the margins of the wound can sometimes easily

Fig. 14.16 A stab wound of the heart



Fig. 14.17 A stab wound of the aorta

reapproximate with one another and prevent external hemorrhage. In these cases, it is not uncommon for there to be a significant amount of blood within the body cavities. Measuring the amount of blood collected within body cavities is important. If air embolism is present (see below), it should be noted.

If a knife blade or other weapon cuts through or into bone or cartilage, careful examination of the bone or cartilage may reveal the presence of “tool marks” from the weapon. This is particularly true with cartilage (Disc Image 14.17). It is not uncommon for the cartilage of the anterior aspects of the ribs to be cut in stab wounds involving the chest. The cartilage should be removed and retained for possible tool mark examination.

Sometimes, when a stab wound on the skin surface is obscured by postmortem drying, such that the angles are indeterminate, examination of organ surfaces or body cavity lining surfaces on internal examination will allow the pathologist to determine whether the blade was single- or double-edged.

Mechanism of Injury

As with many other injury types, a frequent mechanism of death in sharp force cases is related to loss of blood, while others may include various respiratory mechanisms (as described in the blunt force and gunshot wound chapters). One mechanism that is perhaps somewhat more likely in cases of sharp force trauma is the entity known as “air embolism.” Stabbing or cutting injuries to a large-caliber vein can result in air being “sucked into” the vein, due to the negative venous pressures produced by the pumping action of the heart. When enough air is sucked into the vein, the air can move into the heart and create an obstruction that can be thought of as a sort of “vapor-lock.” Air within the right side of the heart prevents blood flow through the heart and causes relatively instantaneous collapse and subsequent death. Air embolism can be visualized on X-ray, and specialized dissection techniques at autopsy can allow the pathologist to document its presence (see Chapter 21).

Trace Evidence

Many, but not all, homicidal sharp force injury cases require close contact between the attacker and the victim. As such, it is important to remember in these cases that valuable trace evidence may be present on the victim and the victim’s clothing. Placing paper bags on the victim’s hands while at the scene can help to preserve such evidence until a better examination with evidence collection can be performed at the morgue. Careful examination at autopsy with collection of any trace evidence is of paramount importance. Depending on the circumstances, it may also be appropriate to collect sexual activity evidence.

Clothing Examination

Trace evidence can be present on clothing, as well as the skin, so careful examination of clothing should be performed. In addition, clothing examination should be performed in order to correlate body injuries to defects on the clothing (Disc Image 14.18). Depending on the clothing material type, defects through certain types of clothing may provide better approximations of the weapon type and measurements.

Defensive Wounds

So-called “defensive wounds” are injuries sustained on a homicide victim’s hands and fingers, forearms, and sometimes the upper arms, as the victim attempts to fend off the attacker (Fig. 14.18 and Disc Image 14.19). Occasionally, defensive type wounds may be encountered on the lower extremities, if the victim is on their back, attempting to fend off the attacker using their legs and feet. The presence of defensive wounds is highly indicative of homicide; however, their presence is certainly not required in order to make a ruling of homicide. Also, occasional hand wounds may be seen in individuals who themselves are attacking someone else with a knife, when their hand slips off of the blood-soaked handle onto the blade.

Fig. 14.18 Defensive wounds



Hesitation Marks

“Hesitation marks” (sometimes called “tentative” injuries) are multiple, superficial, parallel incised wounds present on the wrists (or elsewhere) of decedents who have committed suicide (Fig. 14.19). In some cases, there is an adjacent, deeper, lethal (or potentially lethal) wound. In other cases, there are lethal sharp force injuries elsewhere on the body. In still other cases, there is a completely different mechanism of death, such as a drug overdose. The presence of hesitation marks is believed by many to be absolutely indicative of suicide; however, rare homicidal sharp force injury cases may have similar appearing injuries (typically of the neck), perhaps inflicted as the perpetrator held the knife against the victim’s throat prior to inflicting lethal injuries (Disc Image 14.20).

Fig. 14.19 Hesitation wounds on the wrists of an individual who eventually stabbed herself in the heart



Self-Inflicted Wounds/Suicide

While less common than firearms, hanging, and drug toxicity, the use of sharp force injuries as a method for suicide is more common than one might think. As detailed above, the presence of hesitation marks should raise suspicion that the case represents a suicide. Occasionally, a suicide victim who has died from some other cause (such as drug overdose) will have evidence of superficial incised wounds on the wrists (Disc Image 14.21). The presence of such wounds provides a great deal of evidence that the individual was suicidal (as opposed to dying from an accidental drug overdose). Occasionally, sharp force suicide victims will thrust a knife upward, under the front of the rib cage, into the heart (Disc Image 14.22). This is sometimes mistakenly labeled as a “hara-kiri” suicide. Strictly speaking, hara-kiri involves cutting the abdomen and disemboweling oneself.

“Self-Injurious Behavior” (“Cutting”)

It is important to note that not all self-inflicted sharp force (or other) injury should be regarded as suicidal. The most common scenario involves individuals known as “cutters,” because they deliberately self-injure themselves by cutting their skin, frequently on their arms. By definition, persons who engage in “self-injurious behavior” (“self-harm” or “self-injury”) deliberately injure themselves *without suicidal intent*. Typically, persons who participate in this type of activity are young, including preteens, teenagers, and young adults. In some, there may be an associated psychiatric disorder, such as borderline personality disorder or various depressive

disorders; however, in others, there is no definite associated psychiatric illness. There is sometimes a history of low self-esteem, an eating disorder, or some form of abuse. Some contend that the activity is an attention-seeking behavior, but most authorities disagree with this. Instead, the activity is best considered a type of coping mechanism to relieve emotional stress or discomfort. While sharp force injury is a common injury type associated with this behavior, it should be recognized that other forms of injury may also be utilized. Also, while the activity per se is typically not a suicidal gesture, it should be recognized that persons who are truly suicidal frequently have some of the same stressors and/or underlying psychiatric conditions associated with this self-injurious behavior. Therefore, a history of cutting or other self-injurious behavior should not rule out the possibility of suicide.

Accidental Sharp Force Injuries

Accidental sharp force injury fatalities are rare but may occur. Cases may involve items typically associated with homicidal or suicidal sharp force injuries, such as knives, but can also involve various other sharp items, such as broken glass (Disc Image 14.23). Careful evaluation of the wounds, the scene, and the circumstances is required, as some cases may initially appear quite bizarre. Various animal attack cases involve a certain degree of sharp force trauma. Such cases are explored further in Chapter 21.

Direction of Incised Wound

As mentioned above, it is usually not possible to determine the direction that an incised wound was inflicted. For example, if there is a relatively horizontal wound of the front of the neck, it is usually not possible to determine if it was inflicted from right to left or from left to right. Even if there is an upward angle at one end of the wound, this does not provide any proof one way or the other. One possible way to determine the direction would be to identify abrasions/lacerations on the margins of the wound (or on internal exam) that might indicate the direction of force (rolled-up skin within the abrasion, skin tags associated with lacerations).

Handedness of Attacker

Some investigators claim to be able to determine that a particular set of homicidal sharp force injuries (or even a single sharp force injury) was inflicted by a right-handed or a left-handed attacker. The location of a majority of injuries on the right side of the front of a victim's body would certainly suggest that a right-handed individual may have been responsible, but this is assuming that the two individuals were standing, facing each other, and that the attacker was using a "forehand" type

of swing of the arm. The same set of injuries might just as easily have been produced by a left-handed attacker approaching the victim from behind, approaching the victim from “above” while the victim was lying on the floor, or via a “backhand” type of left arm swing from the front. Homicidal incised wounds of the front of the neck inflicted from behind will generally tend to involve more of the lateral (side) of the neck compared to those inflicted from in front. If such a wound involves the front of the neck, with extension onto the right side, near the jawline by the right ear, it is concluded by some that a left-handed individual inflicted the injury from behind. While this certainly may be the case, perhaps a right-handed attacker chose to use his stronger hand to forcibly pull the head back, and then used his non-dominant hand to inflict the incised wound. Many other scenarios could also account for the injury. In summary, caution should be exercised when making statements regarding the handedness of the attacker. Opining with unequivocal certainty, without the possibility for another scenario, is unwise.

X-Rays

As with gunshot wound cases, all sharp force injury cases should be X-rayed. Such X-rays are performed for a variety of reasons. Occasionally, a weapon or a broken-off portion of a weapon remains within the body (Fig. 14.20 and Disc Image 14.24). Identification of such an occurrence is best accomplished by X-ray examination, and it allows extra care to be taken during the autopsy, so that injuries to the pathologist and assistants may be avoided. Chest X-rays may indicate the presence of air within certain regions of the body, including the body cavities (for example, a pneumothorax) and the heart (an air embolism) (Disc Image 14.25). Air on X-ray appears as an



Fig. 14.20 An X-ray showing that a knife blade remains embedded in the body

area of “radiolucency” (darkness). Special dissection techniques employed by the pathologist can confirm the presence of a pneumothorax or an air embolism (refer to Chapter 21). As previously mentioned, an air embolism is most likely to occur when there are injuries of veins within the arms or neck, with subsequent “sucking” of air into the veins via the pumping action of the heart.

Decomposition

Sharp force injury cases may be partially or markedly obscured by the presence of decomposition (Fig. 14.21 and Disc Image 14.26). Depending on the extent of decomposition, there may or may not be sufficient skin or other soft tissues available for examination. It should also be noted that with relatively advanced decomposition, it is not infrequent for the decomposing skin to develop splits that sometimes mimic sharp force wounds. Careful examination of underlying structures is necessary in all such cases. Careful examination of the bones and cartilage, after removing adherent soft tissues, for any indication of sharp force trauma is one of the best ways to confirm the presence of such trauma (Fig. 14.22 and Disc Images 14.27 and 14.28). However, not all sharp force injury cases will have skeletal trauma, so the absence of such trauma does not rule out sharp force trauma as a potential cause of death.



Fig. 14.21 A victim of multiple sharp force injuries, with decomposition. Note that the wounds do not appear as distinct, secondary to the decomposition process

Postmortem/Perimortem Wounds

As discussed in Chapter 8, postmortem/perimortem wounds are typically characterized by a yellow color (lack of vital tissue reaction). Postmortem/perimortem sharp force injuries are frequently seen in cases where extensive homicidal injuries have

Fig. 14.22 Stab wounds of the sternum (breast-bone)



been inflicted. Such findings occur in “rage killings,” where the term “overkill” is sometimes employed (Fig. 14.23 and Disc Image 14.29). In such cases, there are many more injuries present than would be necessary to cause death. The perpetrator usually knows the victim well and is enraged for some reason, be it infidelity or some other indiscretion. If the wounds are inflicted after death, there will not be a “vital tissue reaction,” and the wounds, particularly those that are not in dependent portions of the body, will have a yellow appearance (Disc Image 14.30).



Fig. 14.23 An example of “overkill,” in which excessive stab and incised wounds have been inflicted in this murder victim

Artifacts

A final issue that requires mention with regard to sharp force injury is the presence of various artifacts that can mimic true antemortem injury. One of the most common types involves various medical interventions. Emergency medical personnel have, as their primary goal, the saving of human lives, and as such, a variety of sometimes very invasive procedures are employed during attempts at resuscitation. Common amongst these are the placement of chest tubes, the performance of an emergency “thoracotomy,” and the performance of an emergency “laparotomy.” Chest tubes are placed through a relatively small (1–2 inch) incision along the side of the chest wall (Fig. 14.24), between the ribs, in order to evacuate blood and/or air from the chest (pleural) cavities. A thoracotomy refers to the surgical opening of the chest cavity, and is performed via a skin incision which extends from the side of the chest to the front of the chest, in order for the physician to attempt to surgically repair internal chest injuries (Disc Image 14.31). A laparotomy is a surgical incision of the abdomen, again performed in order for physicians to attempt to surgically repair any internal abdominal trauma.



Fig. 14.24 A chest tube incision site, produced during emergency resuscitation efforts. As it is produced by a scalpel, it is a true sharp force “injury,” but it is therapy-related

When such medical intervention is performed in cases of sharp force injury, the medical incisions can sometimes be misinterpreted as true sharp force injuries. Also, on occasion, the medical incisions may actually incorporate a true injury into them, thus masking the true injury’s presence (Disc Image 14.32). In order to avoid such complicating factors, all emergency medical personnel are encouraged to document all findings and procedures and to avoid cutting through any pre-existing injuries. Also, it is best for medical personnel to leave all treatment on/in the body if death occurs, so that the treatment can be better recognized at autopsy.

Besides medical therapy, a variety of other injury types can sometimes mimic true sharp force injury. Perhaps the most common involves lacerations that have a very clean, linear appearance (Fig. 14.25 and Disc Image 12.13). As mentioned



Fig. 14.25 A laceration (from blunt force trauma), mimicking a sharp force injury

earlier, the circumstances and the presence of “tissue bridging” within the depths of lacerations will aid in distinguishing them from sharp force trauma. Severe blunt trauma/deceleration injuries that result in amputations and/or decapitation can also mimic sharp force trauma. Injuries sustained when a part of the body comes into contact with a tight cable or rope can likewise mimic a sharp force injury. To an extent, with some of these injuries, the trauma may actually be best thought of as representing a combined sharp/blunt injury complex.

Two other artifacts that can mimic sharp force injury include decompositional skin splitting, which was addressed previously, and artifacts related to the embalming process. An instrument known as a “trocar” is utilized for embalming purposes, and is a relatively long, hollow, rigid metal rod with a sharp end. It is inserted through a stab wound placed in the abdomen. Embalming fluid is then injected through the device, into the organs of the trunk, as the trocar is thrust repetitively into the internal organs. When an autopsy is performed on a body that has been previously embalmed using a trocar, the internal organs of the trunk have numerous trocar “stab wounds” (see Chapter 21).

Disc Image Legends

Disc Image 14.1 A single stab wound.

Disc Image 14.2 A knife used in a multiple stab wound homicide.

Disc Image 14.3 An irregularly-shaped stab wound characterized by multiple intersecting stab wounds.

Disc Image 14.4 Multiple stab wounds of the back.

Disc Image 14.5 A serrated knife blade.

Disc Image 14.6 A stab wound of the arm with an entrance and exit site. Often, it is impossible to determine which wound is the entrance and which is the exit.

Disc Image 14.7 A regular, standard screwdriver.

Disc Image 14.8 Another example of an incised wound of the neck.

- Disc Image 14.9 Superficial skin injuries produced by a serrated blade on the skin distant from deeper stab and incised wounds.
- Disc Image 14.10 Multiple superficial sharp force injuries produced by barbed wire.
- Disc Image 14.11 A tomahawk used in a homicide by multiple chop wounds.
- Disc Image 14.12 A chop wound produced by the claw end of a hammer.
- Disc Image 14.13 Chop wounds produced by a boat propeller.
- Disc Image 14.14 Severe chop injuries which occurred when the victim fell off of a tractor and was run over by a chopping implement.
- Disc Image 14.15 Stab wound of lung.
- Disc Image 14.16 Stab wound of spleen.
- Disc Image 14.17 The cut surface of the cartilage of a rib.
- Disc Image 14.18 Examination of the clothing of a sharp force injury victim includes identification of clothing defects that correlate to wounds on the body.
- Disc Image 14.19 Multiple defense wounds on the arm of a homicide victim.
- Disc Image 14.20 Homicidal hesitation marks beneath a larger incised wound of the neck.
- Disc Image 14.21 A superficial incised wound on the wrist of a suicide victim who died of a drug overdose.
- Disc Image 14.22 A suicide with the knife left in the body. This form of suicide is sometimes incorrectly referred to as “hara-kiri.”
- Disc Image 14.23 A deep incised wound of the arm caused by the sharp edge of a broken plate-glass window. The victim reportedly cut himself as he was attempting to break through the window.
- Disc Image 14.24 A knife remaining in a homicide victim.
- Disc Image 14.25 An air embolism within the heart (arrows).
- Disc Image 14.26 A decomposing larynx, showing multiple sharp force injuries of the thyroid cartilage (arrowheads) and laryngeal cartilage (arrows).
- Disc Image 14.27 A skull from a body that was completely skeletonized (no soft tissues; just bones) when discovered. Careful examination of the bones revealed the presence of some sharp force trauma (see Disc Image 14.27).
- Disc Image 14.28 A knife tip embedded in the inner aspect of the mandible (jawbone) from the case depicted in Disc Image 14.27.
- Disc Image 14.29 “Overkill” involving numerous stab wounds.
- Disc Image 14.30 Multiple postmortem incised wounds in an elderly woman who died of sharp force injuries of the head and neck.
- Disc Image 14.31 Thoracotomy incision adjacent to a stab wound.
- Disc Image 14.32 A thoracotomy incision (surgical opening of the chest) cutting through a stab wound (left) and a chest tube insertion site (right). The wounds had been stitched by medical personnel prior to autopsy.

Selected References

- Betz P, Tutsch-Bauer E, Eisenmenger W. “Tentative” injuries in a homicide. *Am J Forensic Med Pathol* 1995;16:246–8.

- Biedrzycki L, Eason EA. Penetrating and sharp edged injuries (chp 12). In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:91–6.
- Byard RW, Klitte A, Gilbert JD, James RA. Clinicopathologic features of fatal self-inflicted incised and stab wounds: a 20-year study. *Am J Forensic Med Pathol* 2002;23:15–8.
- Davis GJ. Patterns of injury: blunt and sharp. *Clin Lab Med* 1998;18:339–50.
- Di Nunno N, Costantinides F, Bernasconi P, Di Nunno C. Suicide by hara-kiri: a series of four cases. *Am J Forensic Med Pathol* 2001;22:68–72.
- Gill JR, Catanese C. Sharp force injury fatalities in New York City. *J Forensic Sci* 2002;47:554–7.
- Prahlw JA. Sharp force injuries (Chapter 16). In: Froede RC, ed. *Handbook of Forensic Pathology*, 2nd ed. Northfield, IL: College of American Pathologists; 2003:159–73.
- Prahlw JA, Ross KF, Lene WJW, Kirby DB. Accidental sharp force injury fatalities. *Am J Forensic Med Pathol* 2001;22:358–66.
- Racette S, Kremer C, Desjarlais A, Sauvageau A. Suicidal and homicidal sharp force injury: a 5-year retrospective comparative study of hesitation marks and defense wounds. *Forensic Sci Med Pathol* 2008;4:221–7.
- Taupin JM. Comparing the alleged weapon with damage to clothing – the value of multiple layers and fabrics. *J Forensic Sci* 1999;44:205–7.

Chapter 15

Asphyxial Deaths

So Judas threw the money into the temple and left. Then he went away and hanged himself.

Matthew 27:5

Abstract Chapter 15 provides an overview of asphyxial deaths, those deaths related to lack of adequate tissue oxygenation. Several different types of asphyxia are encountered. Suffocation includes several subcategories, including simple (environmental) asphyxia, smothering (external airway obstruction), choking (internal airway obstruction), mechanical (traumatic) asphyxia, and positional asphyxia. Asphyxia from neck compression (strangulation) can result from a variety of mechanisms, including hanging, manual strangulation, and ligature strangulation. Several toxins can cause chemical asphyxia, most notably carbon monoxide and cyanide. Each of the above types of asphyxia are presented, followed by a section dealing with miscellaneous topics, including autoerotic asphyxia, the choking game, and restraint asphyxia.

Keywords Asphyxia · Suffocation · Strangulation · Hanging · Choking

Introduction

The term “asphyxia” literally means “without a pulse,” but the more common and accepted definition means “without oxygen.” More precisely, it refers to a lack of tissue oxygenation (delivery of oxygen to the body’s cells). Clinically, the term “hypoxia” refers to the lack of oxygen. Anything that results in lack of tissue oxygenation could be considered an asphyxial process. If this definition is taken to the extreme, then all sorts of other mechanisms of death actually incorporate an asphyxial component. For example, if a traumatic injury results in extensive bleeding, the lack of blood, which functions to deliver oxygen to tissues, leads to the lack of tissue oxygenation with eventual death. Certain drug overdose cases result in respiratory depression with eventual absence of breathing, which leads to a lack of tissue

oxygenation with subsequent death. In this chapter, we will limit our discussion of asphyxia to the types of cases that are traditionally considered asphyxial in nature. There are three major categories, each of which has several subtypes (Table 15.1). The first is suffocation, where there is failure of oxygen to reach the blood. The second is strangulation, where there is traumatic compression of the neck, including blood vessels and the airway. Hence, with neck compression, there is lack of oxygen from airway compression and lack of oxygenated blood being delivered to the brain due to blood vessel compression. The third is chemical asphyxia, where oxygen can get to the blood, but poisoning of a vital cell process prevents tissues from receiving or utilizing the available oxygen.

Table 15.1 Categories of asphyxia

Suffocation (oxygen unable to reach blood)
Simple (environmental) asphyxia
Smothering (external airway obstruction)
Choking (internal airway obstruction)
Traumatic (mechanical) asphyxia
Positional asphyxia
Combination
Strangulation (neck compression)
Hanging
Ligature strangulation
Manual strangulation
Non-ligature, non-manual neck compression
Chemical asphyxia (oxygen in blood unable to be used)
Carbon monoxide
Cyanide
Hydrogen sulfide
Combination

It should be noted that, despite an attempt to neatly classify various types of asphyxia into categories as presented below, not every death fits into a specific category. There are cases where overlap occurs between the listed categories. Examples of such deaths will be provided throughout the chapter.

As with many other types of deaths investigated by forensic pathologists, the scene investigation is extremely important in asphyxial deaths. In fact, there is no postmortem laboratory test that forensic pathologists can order to make a diagnosis of hypoxia (lack of oxygen). With many asphyxial deaths, the autopsy is negative or has nonspecific findings that might only suggest the possibility of asphyxia. Some of the nonspecific findings that may occur include cyanosis (a blue discoloration), congestion of blood vessels, engorgement of the right heart with fluidity of blood, and “petechiae” (pinpoint hemorrhages). None of these is specific, and many can be seen in non-asphyxial deaths.

Petechiae (pronounced *pah-TEE-key-eye*), sometimes called “petechial hemorrhages,” represent pinpoint areas of bleeding. They can occur on the skin surface (Fig. 15.1), on various other membranes, such as the conjunctival surfaces of the eyes (covering the inner eyelids and the eyeball itself) and the oral cavity, as well



Fig. 15.1 Petechiae represent multiple pinpoint areas of bleeding. They are not specific for asphyxial deaths, but are frequently present in certain types of asphyxia

as on or within various internal organs. Contrary to the abundant misinformation provided by various sources (individuals who don't know, but act as if they do, and certain elements within the entertainment industry), these pinpoint areas of bleeding that can occur on the inner surfaces of the eyelids and elsewhere are *not* diagnostic of asphyxia. Petechiae can occur in any setting where there is increased venous blood pressure, such that the small blood vessels within the skin and/or mucous membranes (eye and eyelid lining, inner mouth lining, etc.) rupture (Disc Images 15.1, 15.2, and 15.3). Such an increase in venous blood pressure certainly can and does occur with some (but not all) forms of asphyxia, but it can also occur with various natural disease states (such as heart disease (Disc Image 15.4)) and has even been described in association with resuscitation (CPR). Consequently, it cannot be stressed enough that, while the presence of petechiae may be an important finding that suggests the possibility of certain types of asphyxia, their presence is not specific for asphyxia.

Suffocation

The term “suffocation” is used here to describe asphyxia cases where the sole mechanism involves the failure of oxygen to reach the bloodstream. There are several subtypes of suffocation, depending on the underlying mechanism responsible for preventing oxygen from reaching the blood.

Simple Asphyxia (Environmental Asphyxia)

“Simple asphyxia” is a term that is used by some to describe a lack of environmental oxygen. This can occur in a variety of settings. In one, a person is confined within a space that initially contains oxygen, but is such that no additional oxygen can

enter the space. With time, the oxygen is used up by the person, without subsequent environmental replacement. Some refer to this situation as “entrapment,” as might occur if a person becomes trapped inside of an older refrigerator. A second setting also typically involves a relatively enclosed space, such as a grain silo, or an underground tunnel or room. The typical scenario is that the oxygen within these environments has been used up (by micro-organism metabolism or a chemical reaction) or displaced by other gases. As such, there is insufficient oxygen within the local environment. If a person enters the environment, they will typically collapse within seconds. Note that the collapse is due to lack of oxygen, not the presence of a poison or toxin. It should be noted that first responders (and sometimes death investigators) may be at risk in such environments. Extreme care should be exercised when attempting to recover a body that is within an enclosed or otherwise secluded space.

In some deaths that initially appear to be related to carbon monoxide poisoning (see below), the presence of a very efficient catalytic converter may prevent carbon monoxide levels from elevating significantly. Instead, abundant carbon dioxide (not a lethal poison like carbon monoxide) is produced and can displace oxygen, thus resulting in death.

Another variation of the “simple asphyxia” category involves a localized enclosed space that encompasses the victim’s head/face region. The classic example involves a plastic bag over the head (Fig. 15.2). Once all the oxygen within the bag is consumed, unconsciousness ensues, followed by death. This category overlaps, to an extent, with the next category (external airway obstruction), particularly if the bag actually occludes the mouth and nose. Autopsy findings are typically absent in simple asphyxia deaths. As such, scene investigation is of particular importance in diagnosing these deaths. Petechiae are not typically observed.



Fig. 15.2 A case of “simple” asphyxia combined with external airway obstruction by a plastic bag, in a suicidal drug overdose. (photo courtesy of Dr. Patrick E. Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)

Certification of the cause of death in simple asphyxia usually requires additional descriptive terminology, rather than simply using the term “simple asphyxia.” For example, if someone is found dead in an underground utility room at an industrial complex where oxygen has been displaced by nitrogen gas, the cause of death may be written as follows: “asphyxia due to displacement of oxygen by nitrogen gas within an enclosed space.”

Note: one final category of simple asphyxia is actually a special category that has its own chapter in this textbook (Chapter 16 – drowning). Strictly speaking, drowning occurs when air is displaced by a liquid (usually water).

Smothering (External Airway Obstruction)

Smothering occurs when there is an external, mechanical obstruction of the nose and mouth. A variety of scenarios and objects/substances can be involved in smothering deaths. Scenarios can be accidental, suicidal, or homicidal. Accidental smothering deaths include young infants with external airway obstruction by large, soft bedding material (example: face-down on an adult pillow), infants/young children with plastic bags over their heads, and drug-abusing adolescents or adults, where they have used a large plastic bag to assist in drug-abusing behavior. The classic “Final Exit” suicide involves a drug overdose in combination with placing a plastic bag over the head. Homicidal smothering deaths involve someone else covering or occluding the mouth and nose; items that can be used include pillows, bedding, gags, hands, etc. Homicidal and accidental smothering deaths in infants frequently have no physical findings whatsoever. In older homicidal smothering victims, there may be evidence of face, chin, lip and intraoral (tongue, inner cheek) trauma (Fig. 15.3 and Disc Image 15.5). Facial and conjunctival petechiae may also



Fig. 15.3 Injuries of the inner aspect of the lower lip in a smothering death

be seen in homicidal smothering deaths, but typically not in the accidental or suicidal forms. The terms “smothering” or “external airway obstruction” are suitable for cause of death statements.

Occasional smothering deaths involve substances such as sawdust, grain, or sand. Frequently in such cases, these substances can be discovered within the mouth and nose, so, strictly speaking, the cases do not simply represent external mouth and nose obstruction, but are best considered a combination of smothering and internal airway obstruction (choking) mechanisms. In addition, there may also be an additional component of traumatic asphyxia (being crushed or compressed by the substance, such that chest expansion cannot occur).

Choking (Internal Airway Obstruction)

When the external mouth and nose allow air flow, but air cannot move into (or out of) the lungs because of an obstruction of the back of the mouth (oropharynx), the throat (pharynx), the voicebox area (larynx), or the trachea (windpipe) and mainstem bronchi (the air tubes that split from the trachea to supply air to each lung), then death can occur. The mechanism of these asphyxial deaths is referred to as “choking” or “internal airway obstruction (occlusion).” Most choking deaths are accidental, although homicidal, suicidal, and even natural cases can occur.

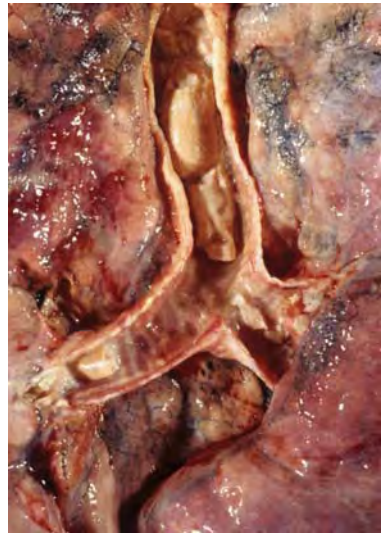
Accidental choking via a foreign body tends to occur in one of several general scenarios. The first is in infants and small toddlers, where the child places something in their mouth and subsequently chokes on it. Examples include balloons, small toys, and pieces of food (Fig. 15.4 and Disc Image 15.6). The second is in intoxicated individuals, whose gag and other reflexes are suppressed by the intoxicant. The



Fig. 15.4 Obstruction of the inner airway by food is one of the most common causes of choking deaths. In this case, food occludes the larynx

usual occluding substance in such cases is food. The third scenario involves a person with some type of underlying neurologic, psychiatric, or physical disorder that prevents normal swallowing and/or breathing. Again, the most common obstructing substance in these cases is food. Regarding food, a variety of types can be involved, although hotdogs (Fig. 15.5) and nuts are said by some to be common. Another type of accidental internal airway obstruction death involves the development of an allergic reaction, or “anaphylaxis” (to a drug, an insect sting, or radiocontrast material used during X-ray), with subsequent massive laryngeal edema (swelling) (Disc Image 15.7). It should be noted that frequently other non-asphyxial, systemic mechanisms, such as markedly reduced blood pressure, are also at play in deaths due to anaphylaxis.

Fig. 15.5 A choking death due to airway obstruction by pieces of hot dog, evident within the trachea and mainstem bronchi



An example of a homicidal choking death involves the use of a gag forcibly stuffed into a victim’s mouth and throat. In such cases, the relative inability of the person to swallow oral secretions likely plays a role in the mechanism of death. Cases of infanticide have been reported where a mother stuffs tissue or other substances into the mouth/throat of her newborn.

An example of a suicidal choking death involves a person intentionally forcing themselves to “swallow”/ “aspirate” (breath in) an obstructive object, such as a balloon or latex glove (Fig. 15.6 A, B).

Examples of natural death due to internal airway obstruction include infections of the epiglottitis (particularly in small children), with associated swelling and spasm of the epiglottitis and larynx, as well as the occlusion of the internal airway by tumors or hemorrhage (bleeding) (Disc Image 15.8).

In cases of death related to upper airway obstruction, autopsy will typically disclose the obstruction. Occasionally, emergency medical treatment will have resulted in the removal of the obstruction, with death occurring at a later time. In such cases,

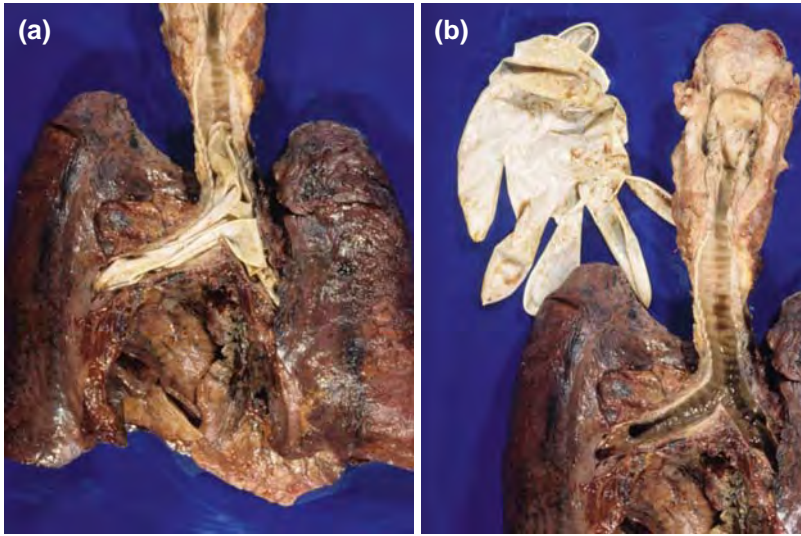


Fig. 15.6 A, B Internal airway obstruction with a latex examination glove

documentation of such an obstruction is essential in order to properly certify the death. Review of medical records is usually sufficient, although interviews with first responders may also be required. Petechial hemorrhages of the face and conjunctiva may or may not be evident in these cases.

An example of the wording that can be used in certifying a choking death follows: “asphyxia due to airway obstruction by foreign body (food).” If an underlying contributing factor, such as intoxication or a neurologic or psychiatric disorder, is thought to have played a role, the condition can be listed in part II of the cause of death section of the death certificate.

It should be noted here that it is not uncommon for persons dying from a wide variety of causes to experience what pathologists typically refer to as “terminal aspiration.” When present, there is evidence of gastric contents within the upper airways (larynx, trachea, and sometimes the mainstem bronchi) at autopsy. Typically, it is not “occlusive,” but instead a relatively small amount. In the usual case, another cause of death is readily apparent. In some cases, the significance of gastric contents within the upper airway is not so clear, for a number of reasons: abundance of gastric contents, lack of a readily apparent explanation of death (many times when this happens, the toxicology findings reveal an intoxication). Each case should be evaluated on its own merits.

Mechanical Asphyxia (Traumatic Asphyxia)

Mechanical asphyxia is also referred to as traumatic asphyxia. In this type of asphyxial death, pressure on the chest and/or abdomen prevents the victim from

being able to expand their chest to breath (Fig. 15.7). As such, air (oxygen) cannot reach the bloodstream. Such deaths are usually accidental. In many adult cases, petechiae are very extensive (face, conjunctiva, anywhere on the skin above the level of compression) (Fig. 15.8). Examples include a person crushed under a car or other structure (Disc Image 15.9), a worker crushed by the earth contained in the walls of a collapsing trench, and a person killed in an avalanche of snow. Certain types of infant death probably involve a mechanical asphyxial component. Cases of overlay,



Fig. 15.7 A case of traumatic asphyxia in which a worker became pinned in between an I-beam and an elevator when the elevator came down from above as he was leaning into the elevator shaft



Fig. 15.8 Intense conjunctival petechiae occurring in a case of traumatic compression occurring when a man became pinned underneath an overturned tractor

where a larger person's body (or body part) lays atop an infant, represent such a case. Petechiae are far less common in such instances. In adult and infant cases, for the death to be solely related to mechanical asphyxia, internal examination must not reveal the presence of other lethal traumatic injuries (blunt force, etc.). If such injuries exist, and an element of traumatic asphyxia is also present, it is perfectly acceptable to include both traumatic asphyxia and blunt force injuries as the cause of the death. Occasional traumatic asphyxia cases involving marked distortion of the neck will demonstrate extensive neck muscle hemorrhage (Disc Image 15.10).

Positional Asphyxia

The term "positional asphyxia" refers to cases where the victim's neck is kinked (bent) in such a way that breathing is compromised, *or* when the position of the entire body is such that breathing is compromised. While oxygen may initially be able to reach the lungs (and blood), the neck and/or body position either acutely or eventually leads to a reduction of lung and/or blood oxygenation. Probably the most common scenario involves an extremely intoxicated individual who somehow ends up in an upside-down position (Fig. 15.9 A, B). Another relatively common scenario involves persons who end up in an inverted position from which they cannot escape in a motor vehicle collision (Disc Images 15.11 and 15.12). Very young children, the elderly, and persons with various physical disabilities may also be at risk for such deaths. Interviewing the person who discovered the body in these cases is of utmost importance, because the autopsy findings may be essentially negative. Petechiae may or may not be present. Another example of positional asphyxia involves a rollover vehicular accident, in which a vehicle occupant is suspended upside-down for a prolonged period of time, resulting in death. Crucifixion and other ancient methods of torture can involve a component of positional asphyxia (body position resulting in compromised breathing).

Combination Forms of Suffocation

A variety of examples have already been provided wherein a combination of asphyxial types play a role in death. A few additional comments will be presented here. Overlying occurs when a larger person's body (or body part) lays atop an infant, usually when both are sharing a sleeping location. Besides involving an element of mechanical asphyxia, overlying cases may also involve elements of positional asphyxia as well as smothering, and possibly even environmental asphyxia (re-breathing air containing elevated levels of carbon dioxide). "Wedging" is a term used by some forensic pathologists to describe a situation in which an infant is trapped in-between two objects, such as a wall and a mattress or a couch cushion and the back of a couch. Like the overlying cases, these cases may include elements of smothering, environmental, mechanical, and positional asphyxia. "Burking" is a



Fig. 15.9 A, B Positional asphyxia related to acute ethanol intoxication. Note the fact that the body is essentially “upside-down” and the neck is markedly kinked

term that is not frequently encountered. It describes a situation where a victim, who is usually intoxicated, is thrown to the ground, on their back, with the assailant straddling the victim and sitting on the victim’s chest/abdomen. While doing this, the attacker holds their hands over the victim’s mouth and nose. Consequently, burking is a combination of smothering and mechanical (traumatic) asphyxia.

Neck Compression (Strangulation)

The term “strangulation” describes the mechanical compression of the neck. The neck contains two major anatomic structures whose function and integrity are necessary for proper tissue oxygenation. These structures are the internal upper airway (pharynx, larynx, and upper trachea) and the blood vessels that supply oxygenated blood to the head (carotid and vertebral arteries) and drain blood from the head (veins). With strangulation, compression of either or both of these anatomic

components may play a major role in death. A third neck component that deserves mention is the “carotid body,” which is a specialized group of cells within the wall of the carotid artery that, when stimulated, for example by pressure, can result in significant changes in heart rhythm and rate, as well as blood pressure. Classically, there are three subtypes of strangulation: hanging, ligature strangulation, and manual strangulation. These are described below, along with some variations that do not fit any of these three categories. It should be noted that certain individuals consider hanging deaths to be distinct from strangulation, whereas others consider hanging to be a subtype of strangulation death.

Hanging

The term “hanging” refers to a neck compression situation where the person’s own body weight (or part of their body weight) contributes to the compressive force on the neck. Most hanging deaths involve a “ligature,” something that either completely or partially encircles the neck. When the ligature only partially encircles the neck, the front (anterior) and front-sides (anterolateral) of the neck are typically in contact with the ligature. Ligatures are usually flexible, relatively narrow (so that pressure can be relatively focused on the neck), and fairly sturdy (strong).

For an asphyxial hanging death to occur, it is not necessary for the body to be completely suspended (with feet off the ground) (Fig. 15.10). In fact, partial suspension hangings, in which the feet (or other body parts) are touching the ground, are quite common (Fig. 15.11). In many of these, it is likely that the original intent of the individual was a full-suspension hanging, but because of stretching of the ligature, the victim’s feet are actually touching the ground when the body is discovered. There is frequently an object adjacent to the body that the victim stood upon to affix the ligature to an overhead object (Disc Image 15.13). The pressure necessary



Fig. 15.10 An example of a full-suspension hanging death, with the feet totally off the ground

Fig. 15.11 An example of a partial-suspension hanging death, with the feet touching the ground



to compress and totally occlude the jugular veins is said to be around 4–5 pounds, compared to about 9–11 pounds for the carotid arteries, around 33 pounds for the trachea, and about 66 pounds for the vertebral arteries.

As mentioned above, ligatures can be quite variable. Examples include ropes, belts, scarves, bedsheets, clothing, shoelaces, and electrical cords, although anything that can be looped around the neck could theoretically act as a ligature (Figs. 15.12



Fig. 15.12 An example of a hanging death, with a rope used as a ligature

and 15.13 and Disc Images 15.14, 15.15, and 15.16). The indented mark on the skin of the neck, produced by the ligature, is referred to as a “furrow” mark. Frequently, the furrow mark in a hanging death has a dry, yellow appearance, and it can be quite deep, especially in complete suspension hangings (Fig. 15.14 and Disc Images 15.17 and 15.18). Occasionally, subcutaneous fat liquefies under the pressure of the ligature and exudes through the skin. In most hanging deaths, the furrow mark is relatively horizontal across the front of the neck, but it then angles upward (toward the point of ligature suspension) on the sides of the neck (Disc Images 15.19 and 15.20). If a body has remained suspended for a long enough time, intense lividity



Fig. 15.13 Another hanging death. In this case, an extension cord was used as the ligature



Fig. 15.14 A “furrow” mark is evident underlying the ligature. Note that it takes on the shape of the braided rope which was used as the ligature in this case and angles sharply upward to the point of suspension

with Tardieu spots can develop in the lower extremities (Disc Image 15.21). If the case is a partial suspension hanging, blanching of lividity may be evident on the portions of the body touching the ground (Disc Image 15.22). Protrusion (with drying) of the tongue is another frequent finding in hanging deaths (Disc Image 15.23). Petechial hemorrhages of the face and eyes are comparatively rare in suicidal hanging deaths. In males, reflex ejaculation may occur during the hanging process. On internal neck examination, suicidal hanging deaths rarely demonstrate neck strap muscle hemorrhage or hyoid bone/laryngeal injury. Exceptions occur when the suicidal hanging has a significant “drop” associated with it. For example, if a person commits suicide by hanging by using a rope tied to a bridge railing, placing the noose end of the rope around their neck, and then jumping from the bridge, such that the entire body weight comes to an abrupt stop when the rope tightens, there may be evidence of internal neck injury. In fact, if the drop is of sufficient length, the mechanism of death is more likely to be related to cervical spinal cord disruption related to a fractured neck, rather than asphyxia. This is the desired mechanism of death in judicial hanging. The more typical suicidal hanging death does not involve a significant body drop; instead, the person steps off of a chair or a ladder, etc. In such cases, internal neck injury is extremely rare.

Accidental hanging deaths are known to occur. Cases involving young children who become entangled in a cord or rope are particularly sad cases (see Chapter 20). Occasional cases involve objects that would not normally be considered a “ligature.” For example, if an infant’s body slides out of one of the leg holes of an infant swing and the neck becomes impinged on the edges of the seat’s leg hole, death can result from a hanging mechanism, even though there is no rope or seatbelt or other “ligature” involved. In accidental hanging deaths occurring in the setting of autoerotic asphyxia (see below) or variations on the choking game (see below), petechial hemorrhages of the face/eyes may be present, and neck marks may be conspicuously absent, since the person may have been attempting to pad the neck to avoid recognition of the activity.

Certifying a death by hanging can occur in a variety of ways. One simple method is to simply rule the COD as “hanging.” Some prefer to say “asphyxia due to hanging.” Others attempt to be a bit more specific by using other terminology, such as “partial suspension hanging” or “full suspension hanging.”

Strangulation

When compression of the neck occurs without the person’s body weight contributing to the compressive pressure, the case is no longer considered a hanging death, but a strangulation death. There are two major types of strangulation, those utilizing a ligature (ligature strangulation), and those utilizing only the hands (manual strangulation). A majority of strangulation cases are homicidal. As such, scene investigation and the search for valuable trace evidence that may link a suspect to the crime is of utmost importance (Fig. 15.15).



Fig. 15.15 A strangulation death scene. Careful examination of the scene, the clothing, and the body may provide valuable evidence that could link a perpetrator to the crime

Ligature Strangulation

If a ligature is involved, the case is referred to as a “ligature strangulation” death. A majority of such deaths are homicidal in nature; however, suicidal and accidental ligature strangulation deaths do occur. The key in differentiating these from hanging deaths relies on the recognition that the person’s body weight (or part thereof) did not contribute to the neck compression.

In most homicidal ligature strangulation cases, there are ligature marks on the neck (Fig. 15.16 and Disc Image 15.24). The perpetrator may or may not leave the ligature around the neck (Fig. 15.17 and Disc Image 15.25). Intense congestion of the skin and tissues may be evident above the level of the ligature (Disc Image 15.26). Because there is frequently a struggle involved in such cases, the



Fig. 15.16 Ligature marks in a homicidal strangulation case



Fig. 15.17 Ligature (telephone line cord) left in place in a homicidal strangulation case

ligature marks tend not to be as distinct and “simple” in appearance as the ligature furrow marks seen with hanging deaths. There may be additional abrasions and contusions in homicidal ligature strangulation cases as well, created when the victim desperately attempts to pull the ligature away from the neck. Small, curvilinear abrasions may result from the victim’s own fingernails as they attempt to loosen the ligature. In contrast to hanging ligature marks, which generally have an upward angle toward a suspension point, such upward angling is frequently absent in ligature strangulation cases. In fact, the ligature marks are frequently more horizontal about the neck, as evident in previous images, as well as Disc Image 15.27. Conjunctival petechiae, as well as facial and sometimes intraoral and/or laryngeal petechiae, are frequently observed in homicidal ligature strangulation cases (Disc Image 15.28). Internally, focal or multifocal anterior neck strap muscle hemorrhage is the rule (Fig. 15.18 and Disc Image 15.29), and hyoid bone and/or thyroid cartilage injuries may be present (Disc Images 15.30, 15.31, and 15.32), particularly in older individuals, where the cartilage and bone tend to be more susceptible to breakage.

Manual Strangulation

Manual strangulation is a form of homicidal strangulation in which the perpetrator uses their hands to compress the neck of the victim. In such cases, ligature marks will be absent. Frequently, there will be evidence of abrasions and contusions on the anterior and/or lateral neck (Fig. 15.19 and Disc Image 15.33). These result from the hands of the perpetrator, as well as the hands of the victim, as they attempt to fend off the attack. Curvilinear abrasions representing fingernail marks can be seen as in ligature strangulation cases (Disc Image 15.34). Conjunctival and other petechiae are frequently present in manual strangulation cases, and internal

Fig. 15.18 Anterior neck “strap” muscle hemorrhage in a strangulation case

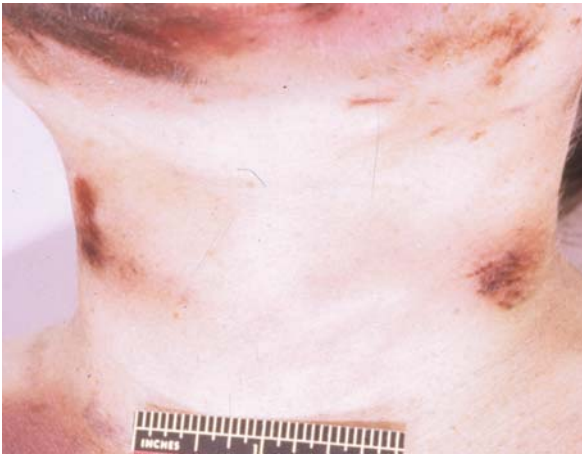
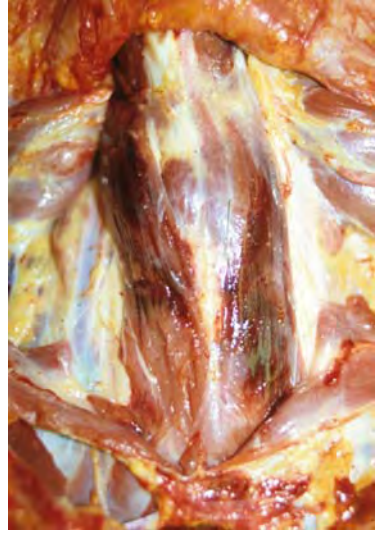


Fig. 15.19 A manual strangulation case

examination tends to reveal anterior neck injuries as described in homicidal ligature strangulation cases. Posterior neck hemorrhage can occur in both manual and ligature strangulation cases (Disc Image 15.35).

Non-ligature, Non-manual Neck Compression

Depending on how the term “ligature” is defined, there are neck compression cases that do not fit into the previously described categories (hanging, ligature strangulation, manual strangulation). For most individuals, the term “ligature” implies that

the object has a certain amount of flexibility, allowing it to conform to the neck. As such, the examples described above as certain forms of “accidental hangings” involving the boards of a crib, do not really involve a classic ligature. In a similar fashion, if an attacker uses a 2×4 board to apply pressure to the anterior neck of a victim, the case represents a form of strangulation, but it really does not seem correct to refer to the 2×4 as a ligature. To refer to such a case as a “ligature strangulation” seems inappropriate to many. While, strictly speaking, it may be true that the death resulted from “ligature strangulation by a 2×4,” it may be more appropriate to describe the death as follows: “compression of neck by 2×4.”

Various neck holds such as the “carotid sleeper hold” or the “choke hold” have similar difficulties regarding nomenclature. The carotid sleeper hold is performed by approaching the victim from behind, wrapping one’s arm around the victim’s neck, with the “bend” of the elbow (the antecubital fossa) over the middle of the anterior neck, and then compressing both sides of the neck, one side with the forearm, the other with the upper arm. The choke-hold involves placing the forearm or a weapon, such as a billy club, directly over the front of the neck and compressing the trachea between the arm (or billy club) and the vertebral column. Strictly speaking, portions of the arms other than the hands are used in the carotid sleeper hold, and a forearm or weapon (billy club) is used in the choke-hold. Therefore, the term “manual” should not be used, since that term specifically refers to the hands. The term “ligature” could conceivably be used to describe the arms or the weapon, but the same confusion occurs as with the 2×4 example above. “Compression of neck” is appropriate terminology, along with other specific descriptors, such as “by billy club.”

Chemical Asphyxia

The third major category of asphyxial deaths, besides suffocation and strangulation, is called “chemical asphyxia.” In chemical asphyxia, oxygen is able to reach the bloodstream; however, a toxin prevents either the transport of oxygen within the blood or the utilization of oxygen at the cellular level, such that death occurs. In the following paragraphs, one very common and two less common causes of chemical asphyxia will be presented.

Carbon Monoxide

Carbon monoxide (CO) is a tasteless, odorless, poisonous gas that is lighter than oxygen. It is produced via the incomplete combustion of carbon-containing fuels. As such, it is produced via the operation of internal combustion engines, as well as via the burning of organic substances. Carbon monoxide that is breathed into the lungs is able to quickly diffuse into the bloodstream, where it binds to hemoglobin, the molecule within red blood cells that normally binds to and transports oxygen to the tissues of the body. CO binds to hemoglobin with an affinity that is several hundred

times stronger than oxygen's. Consequently, even if there is some oxygen within the air that is breathed in, the CO will still bind to and "poison" the hemoglobin, thus effectively shutting down the blood's ability to transport oxygen to the cells of the body.

The two most common settings where carbon monoxide causes death are in cases of automobile exhaust inhalation (usually suicidal) and in structural fires, in the form of smoke and soot inhalation (usually accidental). Fire-related deaths will be discussed in greater detail in Chapter 19. A few more comments regarding automobile exhaust will be presented here. Suicidal CO intoxication from automobile exhaust is fairly common, and two general settings occur. In one, the suicidal person parks their running automobile within an enclosed garage (Fig. 15.20 and Disc Image 15.36). In the other, the suicidal person uses some type of tubing (such as a vacuum cleaner hose), connects one end to the exhaust pipe, and places the other inside the running automobile, thus effectively allowing the vehicle's interior to fill with exhaust (Fig. 15.21 and Disc Images 15.37 and 15.38). Typically, the person performs this act in a relatively isolated location, so that they will not be discovered until their suicide attempt has been successful. It should be noted that not all exhaust-related CO deaths are suicidal. Faulty exhaust systems, occluded exhaust systems (by heavy snow, when motorists are stranded and keep their engines running to stay warm), and other situations where victims do not think that running the car for a short time will harm them, can all result in accidental CO death.

Other settings of CO-related deaths include any other type of situation where combustion of a fuel takes place, and the exhaust/smoke is not properly expelled or the local environment is not properly ventilated. Examples include operating a



Fig. 15.20 A scene photograph showing a woman who committed suicide by carbon monoxide/exhaust inhalation in her garage



Fig. 15.21 A scene photograph in a case of suicidal carbon monoxide inhalation in which the victim used a vacuum cleaner hose to blow exhaust into the interior of a car. (photo courtesy of Dr. Patrick E. Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)

charcoal grill indoors, a furnace that is improperly vented (Disc Images 15.39 and 15.40), malfunctioning space-heaters, and running a gasoline-operated generator in an attached garage, with CO fumes entering the house. Depending on the case, scene investigation may require the assistance of the fire department, a heating/cooling specialist, or various other entities that are able to test the environment for CO levels.

Although most instances of CO production involve the combustion of organic substances, the poison can also be produced in other ways. Methylene chloride (paint thinner/stripper) is toxic if consumed. When this substance is contained in the human body, the liver actually converts some of this toxin into carbon monoxide. Various chemical reactions can also lead to the production of CO; one such involves the combination of formic acid and sulfuric acid. Each of these chemicals is quite readily available, and some advocates of suicide actually market a device that uses this method of CO production.

The most impressive autopsy finding in cases of carbon monoxide poisoning is the bright red (sometimes called “cherry red”) discoloration of tissues (Disc Image 15.41). This bright red color is typically evident in the lividity, as well as internally, involving the organs and tissues (Disc Image 15.42). It should be noted that CO is not the only source of bright red lividity and tissue discoloration: such a finding can occur with cyanide, very cold temperatures, and certain other substances. If someone initially survives CO poisoning, they may develop necrosis (death of cells) within a deep part of the brain called the “globus pallidus,” which is part of the “basal ganglia.”

In cases of CO intoxication, pathologists and their assistants may observe that the formalin within the “save” (stock) containers (where tissue samples from autopsy are saved) will take on a bright red color, compared to a normally dull yellow or brown color.

Postmortem toxicology testing is able to reliably determine the carbon monoxide level in blood. The test, sometimes referred to as the “carboxyhemoglobin” test,

shows the percentage of hemoglobin that is bound to CO. Normally, the level is less than around 3%, while smokers can have levels two to three times as much. Some labs require special collection tubes in order to perform the carboxyhemoglobin test. In the absence of blood for testing, some labs are able to utilize liver samples. In exhaust deaths, the CO levels are usually well over 50%, frequently within the 70s. In fire deaths, the CO levels are often greater than 50%, although in some cases the levels are less than 50%. In these cases, other toxic gases, the absence of oxygen, coexisting thermal injuries, or underlying severe natural diseases are probable contributing factors in death. The cause of death in CO-related fatalities can be recorded in a variety of ways, including “toxic effects of carbon monoxide” and “carbon monoxide poisoning.”

A final note concerning carbon monoxide is that some studies have shown that postmortem production of CO can occur in the setting of decomposition, presumably via the decomposition of hemoglobin, myoglobin, and other substances.

Cyanide

Cyanide, in a variety of chemical forms, is a deadly poison that functions as a cellular asphyxiant (Fig. 15.22). Cyanide binds to “cytochrome oxidase,” an enzyme molecule within our cells that is required for the normal utilization of oxygen in the very important biochemical process known as “oxidative phosphorylation.” When cyanide binds to cytochrome oxidase, the enzyme is essentially poisoned and unable

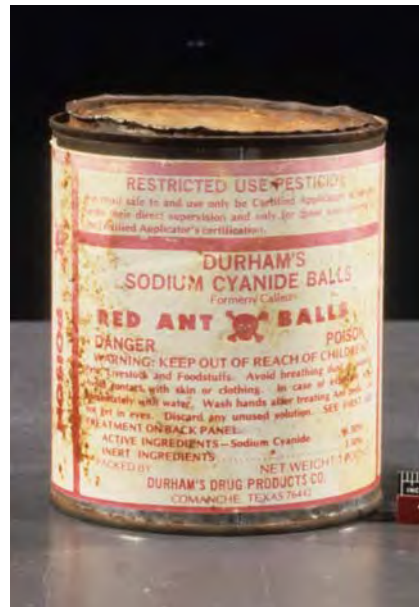


Fig. 15.22 A container of cyanide used in a suicidal ingestion case. (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)

to function properly. As such, oxygen is unable to be utilized at the cellular level, and cell death (and tissue, organ, and organism death) occurs.

Depending on its source, cyanide can be ingested, injected, or inhaled as a gas. Certain preparations have an odor that is said to be “almond”-like; however, only a certain percentage of the population is physiologically able to detect that odor. A general autopsy feature that is sometimes noted in cyanide cases is a bright red discoloration of tissues (similar to that which occurs in CO cases). If the cyanide-containing substance is ingested, a frequent autopsy finding is hemorrhage of the lining of the stomach (Disc Image 15.43).

Hydrogen Sulfide

Hydrogen sulfide is a very poisonous gas that may be present in various natural settings (volcanoes, decomposition) as well as in various industrial processes, including oil-refining. It acts in a similar way as cyanide, by poisoning the oxidative phosphorylation process, and thus blocking the use of oxygen at the cellular level.

The most significant autopsy finding in hydrogen sulfide poisoning cases is the remarkable green discoloration of the lividity and internal organs (Disc Image 15.44). Another clue that hydrogen sulfide poisoning may have occurred is a dark discoloration of metal objects (coins, belt buckles, zippers) that is known to occur in the presence of the gas.

Other Issues

Autoerotic Asphyxia

Autoerotic asphyxia is a type of intentional asphyxial activity that is typically, but not always, performed in isolation as a means of increasing sexual arousal and experience. Apparently, the relative hypoxia (decreased blood oxygen) associated with autoerotic asphyxia somehow leads to heightened sexual response. The typical participant is a relatively young adult male, although cases do occur in older men, and rare cases have been described in women. It should be stressed here that these cases represent accidental deaths, even though the initial appearance may suggest suicide. A frequent pronouncement during the initial police investigation is, “this is the strangest suicide I’ve ever seen.”

The typical scenario involves some sort of neck compression. This frequently involves a ligature, with the person’s own body weight contributing to the compressive force. As such, most of these cases are considered accidental hanging deaths. Persons participating in this activity may purposefully use padded or soft ligatures so as to not leave any ligature marks. Also, as this is distinctly *not* a suicidal activity, participants often have various “escape” devices or mechanisms in place, in an attempt to avoid unintended accidental death. Careful evaluation of the scene will frequently result in the discovery of evidence that the activity has been repeated. In

many, but not all cases, there is evidence of pornographic material and various other “sexual items” at the scene (Disc Image 15.45). Cross-dressing (wearing women’s clothing) is also a fairly frequent, but not universal, finding (Fig. 15.23 and Disc Image 15.46). Elements of bondage are not infrequent (Disc Image 15.47).

Fig. 15.23 An autoerotic asphyxia death. Note that the victim is dressed in women’s clothing. Note also that his hands are bound loosely



Although ligature asphyxiation is the most frequent finding in autoerotic asphyxia, it should be noted that other forms of asphyxia may also be employed. One such example involves the use of an inhalant gas, which can result in hypoxia by simply displacing oxygen (Fig. 15.24 A, B). In addition, certain gases may have additional intoxicating properties.

Choking Game

The “choking game” is a term applied to a variety of asphyxial “games” that are typically played by children and young adolescents. Other names for the game include Black Hole, Black Out, Funky Chicken, Space Cowboy, and Space Monkey, among many others. The apparent intent is to produce neurologic effects, such as lightheadedness, a woozy, “high” feeling, or even temporary unconsciousness (“blacking-out”). There is absolutely no indication that the game has anything whatsoever to do with autoerotic activity or suicide. There are a variety of mechanisms employed in the games, many of which involve neck compression. The usual situation involves asphyxiating to the point of blacking-out, at which time the neck compression is removed. Frequently, the game is played in groups or pairs, with one

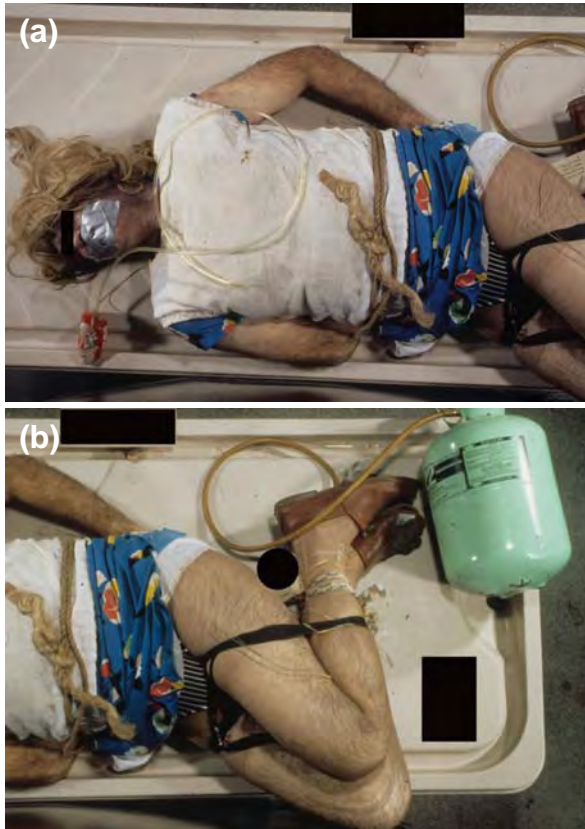


Fig. 15.24 A, B An autoerotic asphyxia death in which the cross-dressed victim used duct tape, various ligature mechanisms, and freon gas

person applying the neck compression. This results in a built-in protection mechanism, in that another individual is present to assist the asphyxiating individual should problems occur. Deaths have been reported when ligatures are used and a person chooses to play the game in isolation, resulting in accidental hangings (Fig. 15.25).

Restraint Asphyxia

The “carotid sleeper hold” and the “choke-hold” are two types of neck restraint maneuvers that may be employed while attempting to subdue someone. At one time, police agencies were routinely taught these maneuvers. The basic description of each is presented in the section above entitled Non-ligature, Non-manual Neck Compression. Variations of these two maneuvers may also be employed during restraint. In addition, there are additional maneuvers and procedures that may

Fig. 15.25 Ligature marks on the neck of a 9-year-old boy found partially suspended from a bandana looped about his neck, as a result of the “choking game.”



be employed during police or other forms of restraint (for example, the restraint of a psychiatric patient). One of these involves the placement of pressure on a person's chest, back, or abdomen when that person is on the ground (face-up or face-down). The typical situation is a suspect who has been resisting arrest and has been forced to the ground. Several officers are atop the suspect, some with their knees on the suspect's back, while attempts are made to handcuff the suspect (Fig. 15.26). Other procedures that may be employed during the restraint process include the use of “pepper spray” and/or an electrical stun gun device. Placing a restrained person in the “hog-tie” position involves placing the individual face-down on the ground, with



Fig. 15.26 Restraint asphyxia death re-enactment. Notice that the victim is face-down, with his hands cuffed behind his back, with multiple police officers applying force to his back

the arms/hands bound to one another and the feet, behind the back. Arguments have been made for and against the possibility of each of these factors contributing to the overall concept which has come to be known as “restraint asphyxia.”

The concept of restraint asphyxia has been the center of much controversy over the past several years. A complicating factor in police-involved cases is the fact that in many instances the suspect who is being restrained is probably experiencing a condition referred to as “excited delirium.” This condition can occur in persons under the influence of a variety of drugs, most notably cocaine, or occasionally as a result of an underlying psychiatric condition. The condition is characterized by agitation, hyperthermia (elevated body temperature), increased blood pressure, heart rate, and respiration, superhuman strength, paranoia, and various other psychiatric disturbances. Persons experiencing excited delirium are at risk of sudden death. If additional stressors, physical or otherwise, are added to the situation, and then death occurs, it becomes a very difficult task for the forensic pathologist to attempt to determine which factors contributed to death.

If, in the forensic pathologist’s opinion, any of the procedures/maneuvers involved in the restraint of the individual contributed to death, then the actions of another person or persons contributed to death, and an appropriate manner of death ruling is “homicide.” Others disagree and prefer to rule these as “accidents.” As for the terminology used in describing the mechanism of death in these cases, some forensic pathologists choose to use the term “restraint asphyxia,” while others prefer to use a less specific and more descriptive phrase, such as “police restraint” or “sudden death during restraint.” In cases where death or the initial collapse occurs during the actual physical restraint of the individual, it is impossible to ignore the potential contributing role of the restraint. In cases where the death or initial collapse occurs some time (several minutes or longer) after the restraint has been accomplished, and no continued restraint forces are believed to be in effect, the restraint is much less likely to be a contributing factor in death.

Combination Asphyxial Deaths

Several examples of combined mechanisms of asphyxia have already been presented, most notably in the “suffocation” section above, where various forms of suffocation can play a combined role in death. It should also be noted that there can also be combined asphyxial mechanisms involving the three major asphyxia categories (suffocation, strangulation, and/or chemical asphyxia). For example, a suicidal individual may fill a garbage bag with exhaust fumes and place the bag over their head. In this way, there would be a combined suffocation and chemical asphyxial death. Another example is a case of an accidental infant death where the baby’s body position is such that there appears to be neck compression, as well as mechanical/traumatic asphyxia. Another example is the case of a kidnapping victim who is placed in an automobile trunk, bound and gagged, and is later found dead by the kidnappers, after having transported the victim several miles. Postmortem

toxicology revealed an elevated CO level. Finally, it should be noted that there can be cases where an asphyxial mechanism of death is combined with another completely different type of death, such as in a suicide victim who places his neck in a noose and then shoots himself in the head. Figure 5.7 from Chapter 5 represents an example of such a case. Another example involves a child who was found dead in an old refrigerator. While asphyxia may have played a major role in death, some contend that hyperthermia may also have played a role.

Drowning

The reader is referred to the next chapter (Chapter 16) for a description of drowning deaths.

Disc Image Legends

- Disc Image 15.1 Conjunctival (eye/inner eyelid lining) petechial hemorrhages.
- Disc Image 15.2 Intraoral (buccal) mucosa petechiae in an asphyxial death.
- Disc Image 15.3 Laryngeal petechiae occurring in an asphyxial death.
- Disc Image 15.4 Conjunctival petechiae occurring in a death due to heart disease.
- Disc Image 15.5 Inner cheek injuries in a homicidal smothering case.
- Disc Image 15.6 A choking death in an intoxicated individual, with occlusion of the larynx and upper trachea by a large fragment of meat.
- Disc Image 15.7 An edematous (swollen) larynx and epiglottis in a death related to anaphylaxis (allergic reaction).
- Disc Image 15.8 An asphyxial death due to underlying natural disease in a decomposed body. Note the tumor occluding the laryngeal opening. The tumor is a benign tumor of fat tissue (a “lipoma”), but because of its location it was lethal.
- Disc Image 15.9 Chest injuries occurring in a man who was working under a mobile home when it fell off of its supports and crushed him. Other than florid facial petechiae, the only other injuries present at autopsy were the external chest injuries depicted here.
- Disc Image 15.10 Extensive posterior neck muscle hemorrhage identified in a motor vehicle collision-related traumatic asphyxial death.
- Disc Image 15.11 Facial petechiae in an individual who died of positional asphyxia (upside-down body position) in a rollover motor vehicle collision.
- Disc Image 15.12 Another case of extensive facial petechiae in a case of positional asphyxia.
- Disc Image 15.13 A partial-suspension hanging death. Note the presence of the milk crates, which the victim presumably used to stand on in order to attach the ligature to an overhead support.
- Disc Image 15.14 A suicidal hanging death using a chain as the ligature.
- Disc Image 15.15 An X-ray of the case depicted in Disc Image 15.14.

- Disc Image 15.16 A suicidal hanging death using a bed sheet as the ligature.
- Disc Image 15.17 A furrow mark caused by coaxial cable in a suicidal hanging death.
- Disc Image 15.18 A less obvious furrow mark produced by a bed sheet (same case as Disc Image 15.16).
- Disc Image 15.19 Note the marked upward angle of the rope along the left side of this suicide victim's neck and head.
- Disc Image 15.20 Ligature furrow mark on the posterior aspect of a suicidal hanging victim's neck, with upward angulation (inverted "V" shape).
- Disc Image 15.21 Tardieu spots on the leg of a hanging victim.
- Disc Image 15.22 Fixed lividity with blanching on the sole of a hanging victim's foot, indicating that the foot was in contact with the ground.
- Disc Image 15.23 Dry, protruding tongue in hanging case.
- Disc Image 15.24 A ligature strangulation case, viewed from the side. Note the lack of an upward angulation of the ligature mark.
- Disc Image 15.25 A homicidal ligature strangulation case in which a "zip-tie" was used as a ligature. Unlike most strangulation cases, the internal neck examination in this case revealed no soft tissue hemorrhage.
- Disc Image 15.26 Another homicidal ligature strangulation case. Note the extensive congestion of the skin above the level of the ligature mark.
- Disc Image 15.27 A subtle ligature furrow mark in a homicidal strangulation case, viewed from the back. Note that the mark is relatively horizontal, lacking the upward angulation that is typical of hanging furrow marks.
- Disc Image 15.28 Conjunctival petechiae in a homicidal strangulation case.
- Disc Image 15.29 Multiple, relatively small areas of anterior neck strap muscle hemorrhage in a homicidal strangulation case.
- Disc Image 15.30 Multiple fractures of the laryngeal cartilages (with surrounding soft tissue hemorrhage), related to a homicidal strangulation.
- Disc Image 15.31 A localized fracture of the right horn of the thyroid cartilage in a homicidal strangulation case.
- Disc Image 15.32 A hyoid bone fracture related to strangulation. Note the hemorrhagic soft tissue surrounding the site of the fracture (arrow).
- Disc Image 15.33 Another homicidal manual strangulation death.
- Disc Image 15.34 A close-up view of a fingernail scratch mark on the neck of the manual strangulation victim shown in Disc Image 15.33.
- Disc Image 15.35 Posterior neck hemorrhage in a homicidal strangulation case.
- Disc Image 15.36 Interior view of the automobile used in the carbon monoxide suicide depicted in Fig. 15.20. Note that the key is in the "on" position.
- Disc Image 15.37 Interior view of the case depicted in Fig. 15.21. Note the vacuum cleaner hose (photo courtesy of Dr. Patrick Lantz, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC).
- Disc Image 15.38 A vacuum cleaner hose connected to the exhaust pipe (photo courtesy of Dr. Patrick Lantz, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC).

- Disc Image 15.39 An elderly woman found dead in her bed. The previous day, her furnace had been professionally serviced. She had lethal levels of carbon monoxide in her blood.
- Disc Image 15.40 Examination of this furnace by an expert in the heating business revealed that the furnace was not venting properly.
- Disc Image 15.41 Comparison of lividity color in a natural death (left) versus a carbon monoxide death (right) (photo courtesy of Dr. Patrick Lantz, MD Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC).
- Disc Image 15.42 Internal cherry red tissue discoloration in a carbon monoxide case.
- Disc Image 15.43 Gastric (stomach) mucosa hemorrhage in a case of cyanide poisoning (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner).
- Disc Image 15.44 The brain of a man who died of hydrogen sulfide gas inhalation (above) compared to a brain from someone who died of natural causes (below) (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner).
- Disc Image 15.45 Pornographic and other items found at the scene of an autoerotic asphyxia death.
- Disc Image 15.46 Another photograph showing the outfit worn by the autoerotic asphyxia victim depicted in Fig. 15.23.
- Disc Image 15.47 A cable wrapped about the body of a victim who died of accidental autoerotic asphyxia.

Selected References

- Andrew TA, Fallon KK. Asphyxial games in children and adolescents. *Am J Forensic Med Pathol* 2007;28(4):303–7.
- Azmak D. Asphyxial deaths – a retrospective study and review of the literature. *Am J Forensic Med Pathol* 2006;27:134–44.
- Byard RW, Hucker SJ, Hazelwood RR. A comparison of typical death scene features in cases of fatal male and female autoerotic asphyxia with a review of the literature. *Forensic Sci Int* 1990;48(2):113–21.
- Carrick C, Collins KA, Lee CJ, Prahlow JA, Barnard JJ. Sudden death due to asphyxia by esophageal polyp: two case reports and review of asphyxial deaths. *Am J Forensic Med Pathol* 2005;26(3):275–81.
- Conroy C, Eastman AB, Stanley C, Vilke GM, Vaughan T, Hoyt DB, Pacyna S. Fatal positional asphyxia associated with rollover crashes. *Am J Forensic Med Pathol* 2007;28:330–2.
- Dix J, Graham M, Hanzlick R. *Asphyxia and Drowning – An Atlas*. Boca Raton, FL: CRC Press; 2000.
- Ely SF, Hirsch CS. Asphyxial deaths and petechiae: a review. *J Forensic Sci* 2000;45(6):1274–7.
- Focardi M, Gualco B, Norelli G. Accidental death in autoerotic maneuvers. *Am J Forensic Med Pathol* 2008;29:64–8.
- Hashimoto Y, Moriya F, Furumiya J. Forensic aspects of complications resulting from cardiopulmonary resuscitation. *Legal Med* 2007;9(2):94–9.
- Kojima T, Nishiyama Y, Yashiki M, Une I. Postmortem formation of carbon monoxide. *Forensic Sci Int* 1982;19:243–8.

- Prahlow JA, Barnard JJ. Fatal anaphylaxis due to fire ant stings. *Am J Forensic Med Pathol* 1998;19(2):137–42.
- Prahlow JA, Doyle BW. A suicide using a homemade carbon monoxide “death machine.” *Am J Forensic Med Pathol* 2005;26(2):177–80.
- Schon CA, Ketterer T. Asphyxial suicide by inhalation of helium inside a plastic bag. *Am J Forensic Med Pathol* 2007;28:364–7.
- Shields LBE, Hunsaker DM, Hunsaker JC. Autoerotic asphyxia: part I. *Am J Forensic Med Pathol* 2005;26(1):45–52.

Chapter 16

Drowning

The water flowed back and covered the chariots and horsemen – the entire army of Pharaoh that had followed the Israelites into the sea. Not one of them survived.

Exodus 14:28

Abstract Although drowning deaths may be considered a form of asphyxial death, discussion of these relatively common deaths deserves its own chapter. The chapter begins by presenting a discussion regarding the physiology and mechanisms involved in drowning. Next, the chapter emphasizes the importance of scene investigation and autopsy performance, and concludes with sections dealing with causes of death other than drowning, SCUBA deaths, and manner of death issues.

Keywords Drowning · Water deaths · SCUBA

Introduction

Drowning can be considered a specialized form of asphyxia in which environmental oxygen (air) is displaced by a liquid (usually water). Unfortunately, death due to drowning is relatively common. Many victims include infants, toddlers, and young children. Intoxicated adolescents and adults are another group of individuals who succumb to drowning at a rate greater than the general, non-intoxicated population. Drowning can occur indoors or outdoors. Several different bodies of water may be involved in drowning, including bathtubs, water troughs, swimming pools, ponds, lakes, the ocean, fountains, wells, and even buckets containing water. Several myths exist regarding drowning: one is that persons who are excellent swimmers cannot drown; another is that all drowning deaths represent accidental deaths; another is that a person's body must be totally submerged in order for drowning to occur. An additional myth is that there are positive findings at an autopsy that allow pathologists to definitely determine that drowning occurred.

Physiology and Mechanism of Death in Drowning

Drowning may involve fresh water, salt water, or, rarely, some other form of liquid. Obviously, the frequency of each of these types of drowning depends on the local environment. Hence, salt water drownings are much more common in ocean coastal areas as compared to non-coastal locales. In salt water drowning, the high concentration of dissolved salt within the water may draw additional fluid from the bloodstream into the alveoli. This results in massive pulmonary edema (fluid in the lungs), thus preventing adequate oxygen from reaching the bloodstream.

In contrast, in freshwater drowning, some of the water that is “breathed into” the lungs is actually drawn into the blood stream. This results in a massive increase in blood volume, with associated hemolysis (destruction of red blood cells). Obviously, fluid also remains in the lungs, both as the water that was aspirated, as well as pulmonary edema fluid. Ultimately, the final mechanism of death is thought to be related to cardiac arrhythmias (irregular heart rhythms).

An alternative theory thought to apply in certain drowning cases involves the concept known as “laryngeal spasm” (or “laryngospasm”). The theory is that a reflex constriction of the laryngeal structures prevents a substantial amount of water from entering the lungs. Obviously, at the same time, air is unable to enter the lungs. Laryngeal spasm is thought to explain cases of “dry drowning,” in which the lungs are not particularly heavy or laden with pulmonary edema fluid.

In some cases, resuscitation efforts may prevent immediate death. Such an occurrence is sometimes referred to as “near drowning.” If sufficient brain anoxia (lack of oxygen) has occurred, brain death may eventually ensue. In such cases, the near-drowning event ultimately caused death by causing brain death. Such cases are variably described as “drowning,” or “sequelae of near drowning,” or “anoxic brain injury due to near drowning.”

Occasionally, a person who nearly drowned but was rescued will initially appear as if they have completely recovered from the event. This may actually occur within minutes of the event. In some of these victims, subsequent severe respiratory compromise develops, and death can occur. This is referred to as the “post-immersion syndrome” or “secondary drowning” or “dry drowning.” Consequently, persons who have experienced a near-drowning episode should be closely monitored medically after the initial event.

As an aside, in presumed drowning cases involving extremely cold water, some incredible stories of complete recovery following many minutes or even hours of submersion have been reported. Apparently, hypothermia and the “diving response” (a reflex in response to cold water that results in marked oxygen conservation) somehow prevent lethal hypoxic brain injury. This has led to the following credo within emergency medicine: “a person is not dead until they are warm and dead.”

Scene Investigation

As with any other scene investigation, investigators should consider the possibility that the setting of a drowning death may pose significant risk for rescuers, investigators, and bystanders. This is particularly true in drowning deaths involving rapidly flowing water, such as occurs in certain rivers and in flash-flood situations. Persons, as well as entire vehicles, can be easily washed away in flood waters, only to be found many days later (Figs. 16.1, 16.2, and 16.3). Murky water, with very little or virtually no visibility, creates a tremendous challenge for persons involved in search and rescue or recovery operations. The importance of safety measures for those involved in these activities cannot be stressed enough. Other potential hazards that may be present in certain drowning cases, besides the water itself, include electricity (downed power lines, lightning, etc.), avalanche/mudslide concerns, and blunt trauma related to the structural failure of buildings.



Fig. 16.1 A scene photograph showing a full-sized automobile that had previously been swept away in a flash flood, only to be discovered after flood waters receded

It is relatively obvious for the casual observer to understand that recovering a body from a murky pond, lake, or river can be difficult, because the body cannot be easily seen from the surface. What is less widely understood is the fact that certain “clear, clean” recreational bodies of water, such as swimming pools and hot-tubs, can present similar difficulties. Depending on the cleanliness of the water and the chemical content, swimming pool and hot-tub water may actually appear quite “blue,” but still be extremely cloudy, such that visibility is not possible beyond a few inches or feet from the surface. As such, a body could be at the bottom of a swimming pool, yet not be visible from the surface. Alternatively, the foaming and bubbling within a hot-tub can make it difficult or impossible to see a submerged person, particularly a small child.

Fig. 16.2 The driver of the motor vehicle depicted in Fig. 16.1

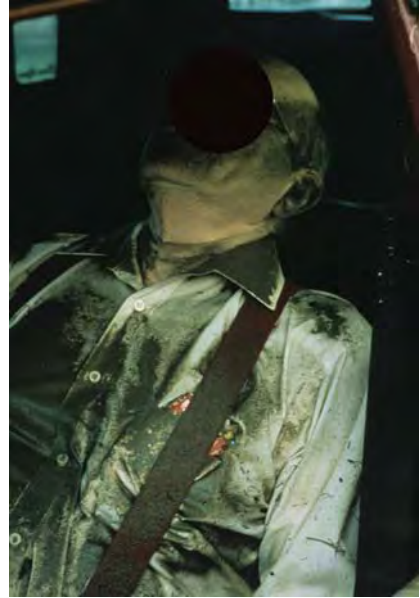


Fig. 16.3 Another individual who was swept away by a flash flood and drowned

Autopsy Findings

As alluded to in the introduction, there is typically no specific autopsy finding in drowning victims that absolutely allows the pathologist to make a diagnosis of drowning. As such, forensic pathologists include drowning amongst a variety of case types that are considered “diagnoses of exclusion.” In other words, the autopsy is negative or has various non-specific findings (see below), such that there is no other adequate anatomic explanation for death, and the circumstances of the death are such that drowning is the likely explanation. As described elsewhere in this text, other case types that represent “diagnoses of exclusion” include certain electrocution deaths, drug/toxin deaths, cardiac conduction system abnormalities, seizure-related deaths, hypothermia, and hyperthermia, among others. In each, the autopsy is usually negative regarding an anatomic explanation of death, and the circumstances are such that the underlying cause of death can be ascertained.

Some important issues to address in any drowning death include the following. Could the person have been dead before entering the water? What if there is underlying severe natural disease that might otherwise explain death? A thorough death investigation usually provides the answer to the first question, although occasionally this question cannot be answered with certainty. Regarding the second question, the circumstances in which death occurred, coupled with the details of the autopsy, will probably provide the answer. Just because a person has a disease that could explain death (such as severe coronary artery atherosclerosis) does not mean that they died from that disease. The disease process may simply represent an “incidental” finding. In contrast, certain natural disease processes, such as a ruptured myocardial infarct, do not represent incidental findings. If a body is found in the water with a ruptured myocardial infarct, it is likely that they experienced the natural death and then fell into the water.

Occasionally, a drowning will be witnessed by other persons, with the victim pulled from the water relatively soon after submersion. If the person is already dead, rigor mortis may already be evident. Such “instantaneous” rigor results from the fact that the drowning victim, while struggling to survive, used up much, if not all, of their ATP during the struggle to avoid drowning. Recall that rigor mortis becomes apparent after death as ATP is depleted within the muscles. If the ATP is used up prior to death, rigor mortis will appear very quickly. In some drowning victims, vegetation from the bottom of the body of water (lake, pond) will be seen, clutched in the victim’s hands. Such a finding is sometimes referred to as “cadaveric spasm,” and is another term that is used to describe the rapid onset of rigor mortis that frequently accompanies drowning deaths (Fig. 16.4 and Disc Image 16.1).

Various nonspecific findings may be present in drowning deaths. The most consistent of these is probably the presence of pulmonary edema fluid, although it is not universally present in all drowning deaths. Pulmonary edema is characterized by heavy lungs, as well as frothy fluid within bronchi, and sometimes the trachea, larynx (Fig. 16.5), oropharynx and nasal passages. It is not unusual to see frothy fluid exuding from the mouth and/or nose (Fig. 16.6 and Disc Images 16.2 and 16.3). Hemorrhage within the lungs may accompany the pulmonary edema. It should be



Fig. 16.4 “Cadaveric spasm,” or “instantaneous rigor mortis,” in a drowning victim. Note the vegetation clutched in the hand

Fig. 16.5 Frothy pulmonary edema fluid within the larynx of a drowning victim



noted here that prominent pulmonary edema fluid (with or without associated hemorrhage), with the production of “foam cones” coming out of the mouth and/or nose, can be seen in a variety of other types of death, including certain drug-related cases, cases with severe heart failure, and a variety of other situations. Occasionally in drowning deaths, water can actually be seen within the bronchi of the lungs, but this is the exception rather than the rule. If specific substances (such as small vegetation)



Fig. 16.6 Pulmonary edema fluid exuding from the nose of a drowning victim

are present within the body of water in which drowning occurred, gross visual identification of these substances within the lungs at autopsy is possible (Disc Image 16.4).

Another nonspecific finding in many drowning cases is an intense congested appearance of the middle/inner ear region, as visualized through the basilar skull, after brain and dura removal. This is characterized by a dark purple appearance of the petrous ridge of the basilar skull (Fig. 16.7 and Disc Image 16.5). The petrous ridge is part of the temporal bone, and forms the dividing region which separates the middle cranial fossa from the posterior cranial fossa. Like pulmonary edema, this finding is nonspecific; it is known to occur in a variety of other death types, including electrocutions, seizure disorders, and various other natural and non-natural deaths.

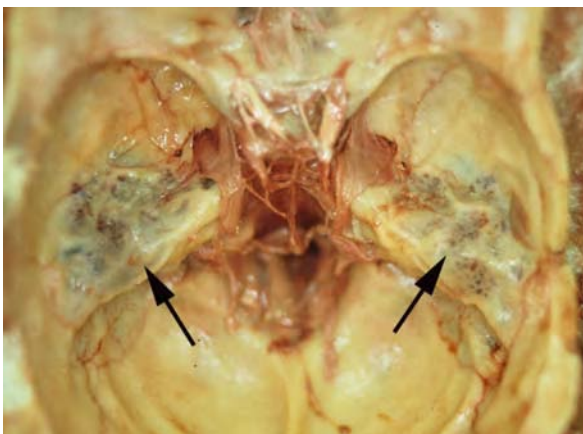


Fig. 16.7 Petrous ridge discoloration/hemorrhage present in a drowning victim (*arrows*)

Finding water within the sphenoid sinus (seen at autopsy, after brain removal, via uncovering the basilar skull which overlies the sinus, just medial and posterior to the eyes) is considered, by some, to represent evidence that is suggestive of drowning (Fig. 16.8). Dark red staining of the intima (inner lining) of the aorta, near the heart, as a result of hemolysis (red blood cells breaking apart), without similar staining of the pulmonary artery, has been recently reported as a possible indicator of fresh water drowning, although it is only found in a small percentage of cases, and the finding is a well-known and common decomposition change.

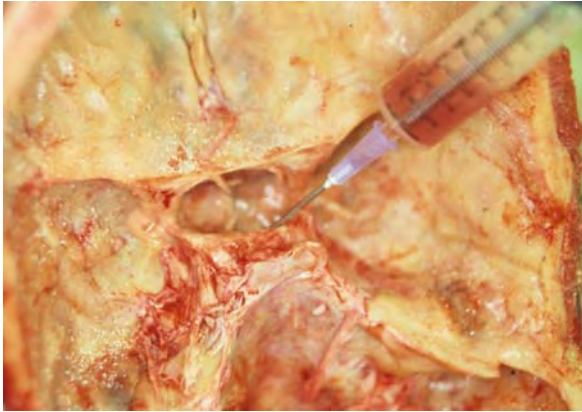


Fig. 16.8 Water being withdrawn by a needle and syringe from within the sphenoid sinus of a drowning victim. Note that a “window” of bone overlying the sphenoid sinus has been previously cut away, thus exposing the sinus

Microscopic examination in drowning deaths also tends to be nonspecific. Some forensic literature in the past has suggested that performing specialized studies to identify and quantitate the number of diatoms (microscopic algae plants) within a body’s tissues can allow pathologists to differentiate cases of drowning from cases where death preceded immersion. Most forensic pathologists within the United States do not subscribe to this theory, as many studies have shown the presence of diatoms in tissues of persons who haven’t drowned and were not found in water. Although microscopic examination in most drowning deaths is nonspecific, it may be very important to perform histologic examination in certain drowning deaths. This is particularly true in individuals who drown who are otherwise considered excellent swimmers. Microscopic examination of the hearts of these individuals may reveal the presence of inflammation (a “myocarditis”), which may put the individual at risk for a cardiac dysrhythmia. The risk for dysrhythmia might be especially heightened if there is hypoxia associated with breath-holding. Thus, the underlying myocarditis, which is typically thought to be related to a viral infection, could play an underlying role in the drowning death.



Fig. 16.9 Markedly wrinkled hands (“washerwoman hands”) in a body recovered from water

Whether death is due to drowning or not, bodies within water will frequently demonstrate marked wrinkling of the palms of the hands and the soles of the feet (Fig. 16.9 and Disc Image 16.6). The outdated and politically-incorrect term “washerwoman hands” has been used to describe this wrinkled appearance. As most readers probably know by experience, similar changes can occur in living persons. The important thing to remember is that the finding has no bearing on whether or not the person died in the water. The change is frequent in drowning deaths, as well as in dead bodies that are placed in water.

Depending on the body of water in which the drowning occurred, as well as the length of time before the dead body is recovered from the water, there may be a variety of postmortem changes evident. In moderate to colder climates, the rate of decomposition tends to slow down in bodies submerged in water, but it is not uncommon for certain drowning victims to demonstrate relatively severe decomposition, primarily because the bodies are not discovered/recovered in a timely fashion (Figs. 16.10 and 16.11 and Disc Image 16.7). A variety of skin defects (skin splitting) may be present (Fig. 16.12 and Disc Images 16.8, 16.9, and 16.10). Marine wildlife, including fish and turtles, may be responsible for some of these defects, because these animals actually feed on dead bodies. Postmortem injuries can be superficial or fairly deep. Extravasation of blood into the subcutaneous tissues can occur in the scalp and neck, as with decomposed cases outside of the water. Claw marks and “beak” marks from certain large turtle species can mimic antemortem sharp force injury. Occasionally, some fairly bizarre findings may be seen at autopsy. A bloated, decomposing body in water may have part of the rectum inverted and protruding as a result of decomposition (Fig. 16.13). If a marine animal or water bird begins to feed on the protruding intestine, with continued pulling by the animal, a majority of the intestines can actually prolapse out of the anus and be consumed.

Fig. 16.10 Marked decomposition in a drowning victim who was recovered from a river approximately four weeks after he was witnessed jumping into the river. Note the extensive skin slippage, as well as river “slime” on the body surface



The surprise occurs during autopsy, when the body is opened and there are virtually no intestines to be found.

Besides the fact that postmortem injuries can occur in submerged bodies, it is also important to note that changes can occur in true, antemortem injuries such that they take on a postmortem appearance. This occurs when the constant “washing” of



Fig. 16.11 Decomposition with unusual color variation of the skin, in another river drowning victim



Fig. 16.12 A postmortem defect (skin split) on a decomposing river drowning victim. The defect may have been produced by a marine animal attempting to feed on the body

external wounds results in blood being “washed out” of the wounds (Fig. 16.14 and Disc Image 16.11).

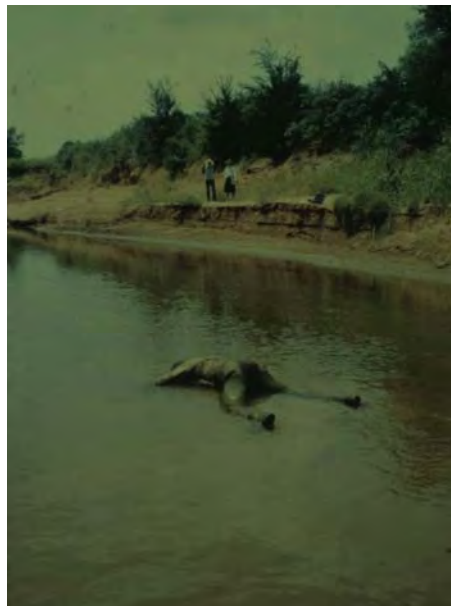


Fig. 16.13 A scene photograph of a drowning victim who has come to rest on the floor of the riverbed near the shoreline. Note how the victim is face-down, with the bloated body’s buttock region higher than the remainder of the body



Fig. 16.14 True antemortem (occurring before death) sharp force injuries on the chin of a homicide victim whose body was “dumped” into water after death

Causes of Death Other Than Drowning

As discussed previously, it is important for pathologists to attempt to determine whether a decedent found in water died in the water or was dead prior to being placed in the water. The possible causes of death for those persons who die before entering the water are quite numerous. The important question in these cases is how did they get into the water? In most instances, someone had to place them there, so homicide should be suspected.

A vast majority of such deaths in persons who actually die while in the water are a result of drowning. Occasional drowning deaths have underlying contributing factors, such as the myocarditis case described above. Other natural disease processes may contribute in a similar fashion, including other cardiovascular disorders that predispose an individual to an arrhythmia, and seizure disorders. It is not uncommon for seizure-prone individuals to drown in a bathtub. Non-natural, traumatic processes may likewise contribute to the drowning or even be the sole cause of death. Examples of contributing factors include blunt force injury, with subsequent injuries that make it impossible to swim, or subsequent unconsciousness. Persons jumping from a bridge may or may not have significant blunt force trauma, depending on the height of the bridge. Persons diving in shallow water or participating in horseplay can sustain neck fractures, typically at the C1–2 level of the neck. Occasional electrocutions occur, when a boathouse electrical or other power supply has electrified the water (Fig. 16.15). Carbon monoxide inhalation from boat or other watercraft exhaust can lead to unconsciousness and subsequent drowning.



Fig. 16.15 A scene photograph showing a swimming pool in which a combined drowning/electrocution death occurred. The electric wiring of the pool lights was corroded such that the water was electrified

SCUBA Deaths

In persons participating in SCUBA (self-contained underwater breathing apparatus) diving, a variety of mechanisms of death may be at play, including drowning (the most common cause of death in SCUBA divers), lack of oxygen (due to running out of air, contaminated tanks, or some type of equipment failure), and various pressure-related processes (barotrauma). Specifically regarding pressure-related issues, it is important to understand that, as a diver descends within the water, non-oxygen gases normally present within the air we breath (and in the SCUBA tank), such as nitrogen, dissolve into the body's tissues.

If a diver ascends (comes to the surface) too rapidly, these dissolved gases can become gaseous again and cause all sorts of problems. In the lungs, the expanding gases can burst the lung, resulting in a pneumothorax. In addition, air can escape into the soft tissues of the central chest and even the skin. When dissolved gas comes out of tissues, tiny gas bubbles develop and travel within veins. These venous gas emboli result in "decompression sickness," characterized by joint pain (the "bends"), difficulty breathing (the "chokes"), etc. If the diver has a defect in their heart that allows venous emboli to travel into the arterial system, fatal arterial gas embolism can occur. Fatal arterial gas embolism can also occur if the lung's alveoli rupture and allow air to directly enter blood vessels within the lungs. This catastrophic occurrence typically happens after a diver has completed the ascent, and is evident when they simply collapse.

When investigating a SCUBA-related death, it is advisable to consult with unbiased experts who can perform a detailed examination of the SCUBA gear as well as the circumstances of the death. Special autopsy procedures for identification of pneumothorax and gas emboli should be performed. This will typically include chest X-ray and careful dissection techniques, such as opening the chest cavity and heart under water, and examining the brain prior to the rest of the body.

Manner of Death

A large percentage of drowning deaths are accidental. Infants, toddlers, and young children represent a large population at risk for such deaths. Bathtub drownings frequently involve the very youngest in this age group. Leaving a young child unattended (or with an older but still young sibling) in a bathtub can be considered a disaster waiting to happen (Fig. 16.16). Despite the warnings present on most containers, young children continue to die when they accidentally plunge head-first into water-filled containers, such as a 5-gallon paint bucket. Small children wandering into un-secured swimming pools or other bodies of water is another frequent occurrence (Disc Image 16.12). In older age groups, accidental drownings typically occur in association with intoxication, or in situations where someone does not properly anticipate the danger of the particular body of water where the drowning occurs. This latter scenario is particularly common with flowing bodies of water (rivers), where, on the surface, the river may appear quite calm, but in reality, under the surface, very strong and dangerous currents can sweep away the best of swimmers. Alternatively, persons may break through ice (again, particularly over flowing water) and subsequently drown. Occasionally, an underlying natural disease process, such as myocarditis, or a seizure disorder, initiates the accidental drowning. In a like manner, accidental trauma can initiate the drowning process.



Fig. 16.16 An infant who was the victim of an accidental bathtub drowning

Homicidal drownings are rare, but known to occur. Obviously, persons who do not know how to swim are the most vulnerable to such killings. As such, perhaps the most vulnerable age group is infants and young children. Others at risk would be those who are disabled or intoxicated.

Suicidal drowning deaths are rare, but are probably more common than most people might think. As with other suicides, careful scene investigation, as well as overall death investigation, are important in identifying such cases. A small percentage of suicidal drowning victims will actually attempt to use some sort of anchoring device or weight. As such, the mere presence of something like a concrete block

tied to a drowning victim should not eliminate the possibility of suicide, although such a finding should definitely raise concern of a possible homicide.

Disc Image Legends

- Disc Image 16.1 Another example of cadaveric spasm.
- Disc Image 16.2 Another example of pulmonary edema fluid exuding from the mouth of a victim of a flash flood drowning.
- Disc Image 16.3 Another example of a “foam cone” in a drowning victim. This boy drowned in a river.
- Disc Image 16.4 Plant material present within the tracheobronchial tree of a drowning victim.
- Disc Image 16.5 Another example of petrous ridge congestion/hemorrhage in a drowning victim. Note also that the basilar skull overlying the sphenoid sinus has been removed. Water was evident within the sphenoid sinus.
- Disc Image 16.6 An example of “washerwoman hands” in a decomposed body.
- Disc Image 16.7 Another example of a drowning victim recovered from a river after several weeks in the water. Note the extensive river “slime” on the body.
- Disc Image 16.8 Postmortem skin defects on the trunk region of a decomposing drowning victim. These may be misinterpreted as true injuries.
- Disc Image 16.9 Additional postmortem skin defects on a decomposed drowning victim. These may have been produced by marine animals attempting to feed on the body.
- Disc Image 16.10 A further example of postmortem skin defects (arrows) on a decomposing drowning victim (the same decedent as shown in Disc Image 16.7).
- Disc Image 16.11 Additional examples of true antemortem (before death) sharp force injuries sustained prior to the homicide victim’s body being dumped in the water.
- Disc Image 16.12 A toddler who was found at the bottom of the family swimming pool. Autopsy revealed no evidence of non-drowning related injuries.

Selected References

- Carter N, Ali F, Green MA. Problems in the interpretation of hemorrhage into neck musculature in cases of drowning. *Am J Forensic Med Pathol* 1998;19:223–5.
- Caruso, JL. The pathology of diving accidents, In *Bennett and Elliott’s Physiology and Medicine of Diving*, 5th edition, edited by Brubakk, AO and Neuman, T, 2003.
- Davis JH. Bodies found in water: an investigative approach. *Am J Forensic Medicine Pathol* 1986;7(4):291–7.
- Dix J, Graham M, Hanzlick R. *Asphyxia and Drowning – An Atlas*. Boca Raton, FL: CRC Press; 2000.
- Golden FStC, Tipton MJ, Scott RC. Immersion, near-drowning and drowning. *Br J Anaesth* 1997;79:214–25.
- Martin TG. Near-drowning and cold water immersion. *Anns Emerg Med* 1984;13:263–73.

- Pachar JV, Cameron JM. Submersion cases: a retrospective study – 1988–1990. *Med Sci Law* 1992;32:15–7.
- Tsokos M, Cains G, Byard RW. Hemolytic staining of the intima of the aortic root in fresh water drowning: a retrospective study. *Am J Forensic Med Pathol* 2008;29(2):128–30.
- Wirthwein DP, Barnard JJ, Prahlow JA. Suicide by drowning: a 20-year review. *J Forensic Sci* 2002;47(1):131–6.

Chapter 17

Electrical Deaths

He unleashes his lightning beneath the whole heaven and sends it to the ends of the earth.

Job 37:3

Abstract Chapter 17 begins by providing a basic, general description of electricity, followed by a discussion of “electrocution.” Another section deals specifically with the various mechanisms of death implicated in electrical deaths. The importance of a careful and thorough scene investigation is emphasized. Next, autopsy findings in low-voltage and high-voltage electrocutions are presented, followed by sections dealing with lightning deaths, non-lethal electronic shock devices, and death certification issues.

Keywords Electricity · Electrocution · Low-voltage · High-voltage · Lightning

Introduction

Electricity can be considered the movement of electrons through a circuit. During this movement, electrons attempt to move toward the “ground.” If something is “grounded,” it is connected to the ground, thus allowing the movement of an electrical current to the ground. If something is “insulated,” a path to ground is prevented. *Resistance* is defined as an object’s ability to avoid becoming part of an electrical circuit. Resistance is measured in “ohms.” Things with a high resistance do not easily become part of an electrical circuit. Substances with an extremely high resistance, such as rubber and glass, are used to insulate parts of electrical circuits. Items with low resistance, such as metal wires, are purposely used as the components of an electrical circuit.

Voltage is defined as the potential difference between two points within an electrical circuit. It is measured in “volts.” Low voltage is defined as ≤ 1000 V, while high voltage is defined as >1000 V. The common household voltage in the United States is 120 V (Fig. 17.1), while residential power lines (7500 V) (Fig. 17.2) and high tension lines ($>100,000$ V) (Fig. 17.3) are high voltage.



Fig. 17.1 Common household electrical outlet (receptacle) (120 V)



Fig. 17.2 Residential power lines (around 7500 V). Note that, in most instances, these power lines are *not* insulated (they are *not* coated with rubber)

Current is defined as the number of electrons flowing within an electrical circuit. Current is measured in “amperes” or “amps.” The amount of current flowing within an electrical circuit depends on two things, the voltage and the resistance. The relation between current, voltage, and resistance is defined by “Ohm’s law,” which states that the current is equal to the voltage divided by the resistance.

There are two basic types of “manmade” electricity, “alternating current” (AC) and “direct current” (DC). Alternating current is a current that alternates its direction within a circuit at regularly occurring intervals. The frequency at which this occurs is measured in “cycles per second” or “hertz.” Most household and industrial electricity is AC, with the common household frequency measuring 60 hertz.



Fig. 17.3 High-tension power lines (greater than 100,000 V)

Direct current is an electrical current that flows in one direction within a circuit. Battery-operated electrical devices, as well as various other specialized electrical devices, including cardiac defibrillators and certain welding machines, utilize DC. Generally, DC is considered less dangerous than AC; however, death can still result from DC. Static electricity and lightning can be considered natural forms of DC electricity.

Electrocution

By some individuals' definition, the term "electrocution" implies that death has occurred. A more generalized definition, and that which will be used here, uses the term to describe any situation where a person becomes incorporated into an electrical circuit, whether or not death or visible injury occurs.

In order for an electrocution to occur, a victim's body must become incorporated into an electrical circuit. For this to happen, the source of electricity must be of sufficient force to overcome the resistance of the body, and a relatively low-resistance pathway to ground must be present. Whether or not an electrocution results in death depends on the amount of current passing through the body and the effects of that current on the anatomic structures through which the current travels. The most susceptible organ is the heart, in which electrocution can disrupt the internal electrical activity and induce a fatal arrhythmia (abnormal heart rhythm). Other mechanisms of death may also occur (see below). Since an electrical current tends to take the shortest pathway between the point of entry and ground, the location of the entry and exit (ground contact) are very important in determining whether or not a fatal electrocution will occur. Ultimately, the amount of current flowing through a vital body structure determines whether or not the electrocution is fatal, and the time

required for a fatal electrocution to occur is typically only a matter of seconds or less. The extent of tissue damage occurring during an electrocution also depends on the amount of current passing through that particular tissue. Consequently, fatal electrocutions can occur with low voltage (≤ 1000 V) sources where there is sufficient current to induce electrical damage, but absolutely no external or internal physical evidence of electrocution.

Since the overall effects of an electrocution are proportional to the amount of current, it is appropriate to provide some perspective regarding the clinical effects of various amounts of current. A very small amount of current, such as 0.001 amps, causes only a tingle to be perceived on the skin surface. Twenty times that amount can induce muscular paralysis. At 0.04 amps, loss of consciousness can occur, while the fatal cardiac dysrhythmia known as “ventricular fibrillation” can result from 0.1 amps. When 2.0 amps of electricity enter the body, ventricular standstill (abrupt halting of the heartbeat) can occur. For comparison purposes, a common household fuse (or circuit breaker) blows (or trips) around 20 amps. These safety devices are present to prevent fires, not electrocutions. Ground Fault Circuit Interrupters (GFCIs) (Fig. 17.4) are designed to prevent electrocution, and monitor current flow. If a >0.005 amp difference exists, the circuit is instantly broken. These devices are installed in all modern bathrooms and kitchens and wherever moisture is present.



Fig. 17.4 A ground-fault circuit interrupter (GFCI or GFI), designed to prevent electrocution

Recall that according to Ohm’s law, the total amount of current that flows during an electrocution depends on the voltage, the resistance, and the time in which the current is flowing. Since most sources of electricity tend to have a constant voltage, such as the common household 120 V circuits, and the time required for most fatal electrocutions is only a matter of a few seconds or less, the major influence as to whether or not a fatal electrocution is possible is the resistance of the body part that makes contact with the electrical circuit. With this in mind, it is instructive

to consider the relative resistance values for various anatomic structures. Internal organs and tissues have very low resistances because of their high water content, with some as low as 500 ohms. Dry thick skin, such as occurs on the palm of the hand, has a substantial amount of resistance, about 100,000 ohms, whereas dry thin skin has a resistance around 30,000 ohms. In contrast, wet palm skin and wet thin skin have resistances of 1000 and 100 ohms respectively. Plugging the above numbers into Ohm's law in the setting of a typical 120 V household current, we get the following: for dry thick skin, current equals 120 V divided by 100,000 ohms, which is 0.001 amps (a tingle). In contrast, for wet thin skin, current equals 120 V divided by 1000 ohms, which is 0.12 amps (ventricular fibrillation)!

Mechanism of Death in Electrocutions

As implied by the discussion above, many electrocution deaths result from a fatal cardiac dysrhythmia induced by the electrical current entering the body. For such a death to occur, the path of current must travel through the heart. It should be noted that after a person sustains a fatal electrical shock, the victim may remain conscious for several seconds, giving them time to shout out, move away from, or even unplug or turn off the source of the electrocution.

Although many electrocution deaths result from an electrically-induced cardiac arrhythmia, such is not always the case, especially in high voltage electrocutions. In some instances, muscular paralysis (including the diaphragm) may contribute or cause death, via an asphyxial mechanism. If the head (and brain) are the primary sites of the electrical current, central nervous system respiratory center paralysis, with cessation of breathing, may occur. High-voltage electrocution can also result in severe electrothermal injuries, thus causing death. In some cases, entire body parts can essentially be totally consumed during the electrothermal insult. Finally, in some cases, including low-voltage electrocutions, a shock that might not be lethal in and of itself may lead to secondary injury and death.

Scene Investigation

As in many other case types, the scene investigation in electrocution deaths is very important in discovering and understanding how death occurred. An additional extremely important issue related to electrocution deaths is the fact that *the scenes may not be safe for first-responders or death investigators*. It is vitally important, at any scene, for witnesses, first-responders, death investigators, and anyone else to be certain that the conditions at the scene are safe. In some cases, the body may actually still be electrified. Alternatively, the source of "live" electricity will frequently be close by and still capable of causing lethal electrocution. The wise death investigator will always be on the look-out for the possibility of an electrocution

death (Fig. 17.5). As discussed in the next section, not all low-voltage electrocutions will demonstrate physical/anatomic evidence of electrocution. For this reason, a thorough analysis of the death scene may be the only clue that an electrocution has occurred. If questions exist regarding the safety of a scene, it may be necessary to call the local electrical company and/or fire department in order to ensure a safe scene.



Fig. 17.5 Scene photo showing a microwave oven on which an electrocuted man had been working when he was shocked. Note that the appliance is now unplugged; however, when the electrocution occurred, the unit was connected to power

If a source of electrocution is identified, the offending device, wire, equipment, etc. should be sequestered and evaluated by an unbiased expert. For certain sources, it is simple enough for the death investigator to unplug the device, and seize and secure it until such time that it can be properly evaluated (Fig. 17.6). For other sources, such as a several-ton transformer or other large or permanently-fixed object, the scene must be secured and an independent analysis performed as soon as possible. Whatever the offending object, it should be evaluated by an unbiased person with expertise in evaluating electrical equipment. Power company employees, electricians, and certain forensic engineers are able to perform such examinations. In straightforward cases, death investigators or forensic pathologists can perform such examinations, so long as appropriate safety precautions are taken. A voltmeter (Disc Image 17.1) and/or circuit tester (Disc Image 17.2) can aid in the evaluation of various electrical devices. Evidence of electrocution can sometimes be seen on or within the metal contacts of the device; blackening or charring (“braising”) can occasionally be seen in these locations (Fig. 17.7). The device should be evaluated for the presence of a short circuit. If the device is working properly with no evidence of a short circuit, it still might be the source of electrocution because of faulty safety features, or because the victim had removed or moved the safety devices, or simply because inappropriate contact was made with an electrified portion of the device.

Fig. 17.6 A light fixture implicated in a low-voltage electrocution death



Fig. 17.7 Braising on the metal contacts of the plug of the light fixture shown in Fig. 17.6



Autopsy Findings

Low Voltage

A significant number of low-voltage electrocution victims have no or very subtle electrical injuries. When injury is totally absent, ruling the case as an electrocution death is a diagnosis of exclusion, based on the absence of another explanation for death, and the presence of scene/investigative information suggesting electrocution. Examples of cases where electrical injuries are not present include electrocution deaths occurring in water, such as a bathtub or swimming pool (Fig. 17.8). While



Fig. 17.8 An electrocution death that occurred in a bathtub. Numerous electrical cords and devices were present in the bathroom, including a television set. Although there were no electrical burns on the victim's body, examination of the scene, the electrical devices, and the fuse-box revealed evidence of an electrocution having occurred

water-related electrocutions are the classic example of cases with no evidence of injury at autopsy, a certain percentage of non-water-related low-voltage electrocution deaths will also have no evidence of injury. In order for a low-voltage electrocution to occur, contact must be made with the electrical circuit. If enough energy is transferred onto the skin for a long enough period of time, electrical "burns" can result. These typically occur at the site of contact with the electrified object (the entry site), but they can also occur at the site of grounding (exit site).

Injuries in low-voltage electrocution cases can vary from quite subtle to rather obvious. Subtle injuries can include small white blisters (Fig. 17.9) and dark pinpoint sites of burnt or melted keratin (Fig. 17.10). Larger, more obvious lesions include larger blisters, red-yellow abrasion-like injuries, and injuries that have a



Fig. 17.9 Small white blisters at the site of entry in a low-voltage electrocution



Fig. 17.10 Multiple crater-like areas of melted keratin in the low-voltage electrocution case shown in Fig. 17.5

target-like appearance, with alternating concentric zones of white tissue, red tissue, and possibly even brown or charred tissue (Figs. 17.11, 17.12, and 17.13 and Disc Images 17.3, 17.4, 17.5, and 17.6). A peripheral zone of blanched (white or light) skin frequently surrounds any of the above lesions. Clothing examination is of great importance, since burns and other electrical defects can occur on the clothing (Fig. 17.14 and Disc Images 17.7 and 17.8).



Fig. 17.11 Another site of electrical injury in the case depicted in Figs. 17.5 and 17.10. Note the light and dark areas, including areas of charring. Note also the white lesions on the index fingertip

High Voltage

All high-voltage electrocutions result in the presence of electrical injuries. It should be noted that high-voltage electricity may actually “jump” from its source. Consequently, contact with an electrical source is not required for a high-voltage



Fig. 17.12 An example of a white blister in a different low-voltage electrocution case



Fig. 17.13 A larger white blister in another low-voltage electrocution case

electrocution to occur. The distance that the electricity can “jump” increases with voltage, such that 100,000 V can “jump” about 35 cm.

High-voltage electrical burns tend to be relatively obvious (Figs. 17.15 and 17.16 and Disc Images 17.9 and 17.10). Frequently, they are characterized by charred tissue, surrounded by an area of blanching and a peripheral rim of red skin (Fig. 17.17 and Disc Image 17.11). In some cases, dark pinpoint “spark” injuries may be evident (Fig. 17.18). Some electrical injuries have a dry, yellow appearance, simulating a postmortem injury (Disc Image 17.12). As mentioned above, complete combustion (absence) of body parts can occur (Disc Image 17.13).

As with certain low-voltage electrocution cases, clothing examination in high-voltage electrocution is of great importance. Burn marks are common and typically occur in association with underlying skin injuries (Figs. 17.19 and 17.20 and Disc Images 17.14–17.16).

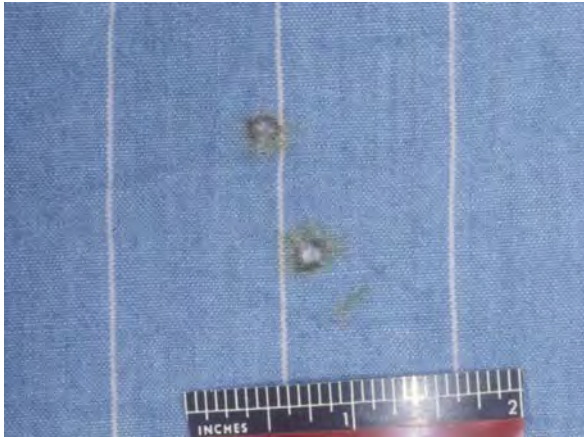


Fig. 17.14 Two electrical burn marks on the shirt of an individual who died of a low-voltage electrocution



Fig. 17.15 Extensive high-voltage electrical burns in a power company employee who came into contact with a powerline

Other Features

A process known as “metallization” may occur, in which small traces of metal from the electrified source are transferred to the skin (or clothing). Applying potassium ferrocyanide to the skin will result in black/purple discoloration, indicating that metal has, in fact, been deposited on the skin.

Microscopically, skin and other tissues damaged by electricity may demonstrate changes consistent with thermal damage. Frequently, the nuclei of cells become thinned, elongated, and aligned in parallel with adjacent nuclei; this is sometimes referred to as “nuclear streaming” (Fig. 17.21). Such changes are sometimes seen in cells within the wall of the aorta. Special microscopic stains for metals such as iron and copper can be used to identify areas of metal deposition.



Fig. 17.16 A high-voltage electrical burn on the index finger and thumb of the man depicted in Fig. 17.15

Fig. 17.17 A classic high-voltage electrical burn, with a central charred area surrounded by pale skin blanching and a peripheral rim of red discoloration



So-called “electrical petechiae” represent pinpoint hemorrhages occurring in various locations in some electrocutions, both low- and high-voltage types. They may occur on the eyelids, the conjunctivae, and on the pleural (lung) surfaces and the epicardium (surface of the heart) (Disc Image 17.17).

Postmortem electrical injuries are known to occur. Unfortunately, postmortem electrical injuries cannot be distinguished from antemortem electrical injuries.

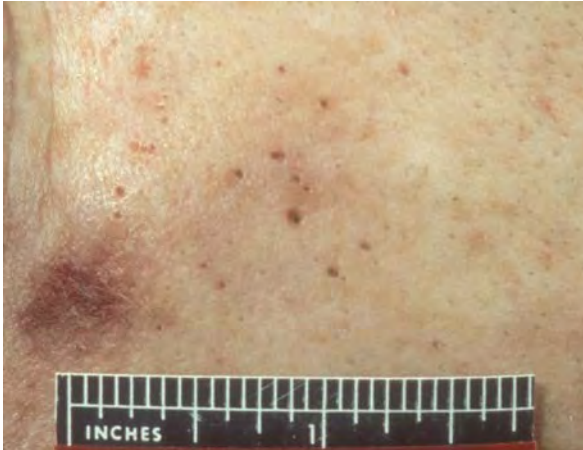


Fig. 17.18 Dark, pinpoint electrical burns in a high-voltage electrocution case

Fig. 17.19 The boot of a crane operator whose crane cable made contact with an overhead high-voltage wire. Note the defects and burning on the boot, as well as the charring of the skin immediately above the boot



Lightning

Lightning can be considered a form of very high-voltage direct current electricity. A lightning bolt is produced when the charged undersurface of a storm cloud discharges its electrical charge to earth. There are around 100 lightning strike deaths per year in the USA. Lightning can kill via a direct strike, a “side flash,” wherein the lightning strikes another object and then “jumps” to the victim, or by conduction through another object. Finding a body outdoors after a thunderstorm should raise

Fig. 17.20 Extensive high-voltage thermal injuries on the foot underlying the boot shown in Fig. 17.19

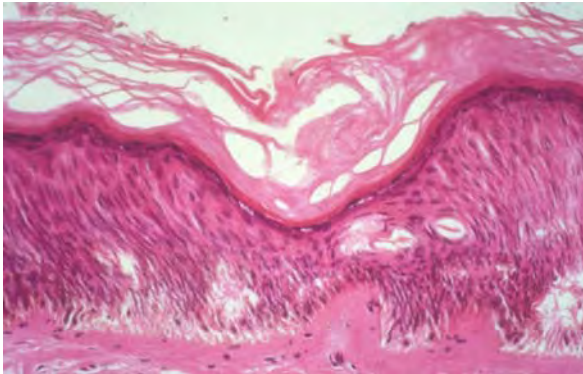


Fig. 17.21 The microscopic appearance of a skin electrical injury. “Nuclear streaming” is most evident on the left side of the image, where the nuclei of the epidermis cells are “lined-up” in a somewhat parallel fashion

the suspicion of a possible lightning strike death. Most lightning strike deaths are thought to be due to cardiac arrest and/or electrothermal injuries. Most lightning strikes occur outdoors; however, rare cases occur indoors.

The force of a lightning strike can literally rip the clothing off of a victim (Fig. 17.22). Metal objects on or in the clothing, or on the body (jewelry) can show evidence of “arc marks,” charring or even melting (Disc Image 17.18). Skin adjacent to metal objects is frequently burnt (Fig. 17.23). The spectrum of injuries that



Fig. 17.22 Scene photo showing a man who was struck by lightning. Note the torn clothing, which was produced by the lightning strike



Fig. 17.23 Autopsy photograph of the same individual shown in Fig. 17.22. Note the extensive abrasion-like injuries of the skin. These injuries were yellow in appearance

can occur is quite large, ranging from virtually none to near total obliteration from massive electrothermal forces, although severe charring and deep burns are relatively uncommon. Injuries can mimic other electrical injuries. Relatively superficial injuries, some of which are discolored yellow, are not infrequent (Fig. 17.24). Hair is frequently singed (Disc Image 17.19). Burns similar to thermal burns or electrical injuries may be seen. A lesion that is said to be characteristic of lightning strikes is the “arborescent” pattern of the skin, representing a fern or tree-like pattern of pink skin discoloration (Fig. 17.25 and Disc Image 17.20). It is frequently transient, and may represent intravascular hemolysis within the subcutaneous capillary/venous blood vessels.



Fig. 17.24 A different lightning strike victim, showing electrical burns adjacent to the metal belt buckle

Fig. 17.25 A classic arborescent (fern-like) lightning strike patterned injury



Non-Lethal Electronic Shock Devices

So-called “non-lethal electric shock devices” are available for a variety of purposes and range from electric fences to “conducted energy devices” (also known as conducted electrical weapons), such as Tasers, electronic capture shields, and stun guns. While these devices are unquestionably safer than something as potentially lethal as an unprotected AC circuit, it is probably unwise to consider them as being totally safe. Some believe that, in the appropriate circumstance and setting, each of these “non-lethal” electrical sources may cause or contribute to death, whether it is via

electrocution or the stress accompanying discharge. For example, if a person has a propensity to develop cardiac arrhythmias because of underlying heart disease or the influence of an illegal drug known to increase one's chance of experiencing a fatal arrhythmia, a shock from one of these devices that travels across the chest could theoretically initiate a chain of cardiac dysrhythmias ending in death. Alternatively, the stress associated with the shock might induce such a dysrhythmia. In general, the devices are considered safe for use on healthy subjects, with the caveats as mentioned above. Further discussion of some of these devices is provided in Chapter 21 under the section entitled "In-Custody Deaths."

Death Certification and Manner of Death

A vast majority of deaths due to electrocution are accidental; however, rare cases of suicide and homicide are encountered. Lightning deaths are typically ruled as accidental. If a death is related to a conducted energy device, the manner of death ruling may be controversial. This issue is discussed in greater detail in Chapter 21 under the heading of "In-custody Deaths."

Disc Image Legends

Disc Image 17.1 A voltmeter can be very helpful in evaluating an electrical device or circuit thought to be involved in an electrocution.

Disc Image 17.2 A circuit tester can also be helpful when evaluating an electrical device or circuit thought to be involved in an electrocution.

Disc Image 17.3 Two electrical injuries on the fingers of a child who was electrocuted while placing a metal kitchen utensil into an electric toaster. Note the crater-like lesion with a raised central area of melted keratin on the index finger. The lesion on the middle finger simulates an abrasion.

Disc Image 17.4 Two additional electrical injuries in a different low-voltage electrocution case.

Disc Image 17.5 Another example of a low-voltage electrical injury. Note that a portion of the injury appears to be a white blister, while a portion appears like an abrasion.

Disc Image 17.6 Another example of a low-voltage electrical injury.

Disc Image 17.7 Electrical burn defects on the shirt of the man who was electrocuted while working on the microwave oven shown in the textbook Fig. 17.5.

Disc Image 17.8 The appearance of the electric injury underlying the shirt that is shown in Disc Image 17.7.

Disc Image 17.9 Massive high-voltage electrical injuries sustained when a crane cable contacted an overhead electrical line (the same case depicted in textbook Figs. 17.19 and 17.20).

- Disc Image 17.10 Scene photograph from the electrocution shown in Disc Image 17.9, showing the contact of the cable and the overhead electrical lines.
- Disc Image 17.11 Another classic high-voltage electrical injury.
- Disc Image 17.12 A dry yellow high-voltage electrical injury occurring on the chest of an electrician who was standing on an aluminum ladder when he made contact with a live wire.
- Disc Image 17.13 A high-voltage electrocution case with extensive tissue destruction. The two men were carrying a metal antenna pole when the pole made contact with overhead high-voltage power lines. Note that the pole appears to have melted into the chest of one victim, while the head and right forearm and hand of the other victim are totally absent. (Photo courtesy of Dr. John Pless, MD, Indiana University School of Medicine (retired), Indianapolis, IN)
- Disc Image 17.14 A work boot with electrical burn marks from an electrical utility worker who accidentally made contact with a transformer. (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)
- Disc Image 17.15 The sock associated with the work boot shown in disc image 17.14. (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)
- Disc Image 17.16 Electrical injuries underlying the boot and sock depicted in disc images 17.14 and 17.15. (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)
- Disc Image 17.17 Numerous “electrical petechiae” present on the epicardium of the heart of a high-voltage electrocution victim.
- Disc-Image 17.18 “Arc marks” on coins in the pocket of a lightning-strike victim. (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)
- Disc Image 17.19 Singed hair occurring in a lightning strike death. (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)
- Disc Image 17.20 Another example of the characteristic fern-like patterned injury associated with lightning strikes.

Selected References

- Adekoya N, Nolte KB. Struck-by-lightning deaths in the United States. *J Environ Health* 2005;67:45–50.
- Al-Alousi LM. Homicide by electrocution. *Med Sci Law* 1990;30:239–46.
- Blanco-Pampin JM, Penaranda JMS, Boquete RR, Carro LC. An unusual case of death by lightning. *J Forensic Sci* 1997;42:942–4.
- Bligh-Glover WZ, Miller FP, Balraj EK. Two cases of suicidal electrocution. *Am J Forensic Med Pathol* 2004;25:255–8.
- Centers for Disease Control and Prevention (CDC). Lightning-associated deaths – United States, 1980–1995. *MMWR* 1998;47:391–4.

- Denton JS, Donoghue ER. Deaths due to electrocution and lightning. In: Froede RC, ed. *Handbook of Forensic Pathology*. 2nd ed. Northfield, IL: College of American Pathologists; 2003:213–218.
- Eriksson A, Ornehult L. Death by lightning. *Am J Forensic Med Pathol* 1988;9:295–300.
- Goodson ME. Electrically induced deaths involving water immersion. *Am J Forensic Med Pathol* 1993;14:330–3.
- Jacobsen H. Electrically induced deposition of metal on the human skin. *Forensic Sci Int* 1997;90:85–92.
- Karger B, Suggeler O, Brinkmann B. Electrocution – autopsy study with emphasis on “electrical petechiae.” *Forensic Sci Int* 2002;126:210–3.
- Lee WR. The mechanisms of death from electric shock. *Med Sci Law* 1965;5:23–8.
- Lifshultz BD, Donoghue ER. Deaths caused by lightning. *J Forensic Sci* 1993;38:353–8.
- Loomis D, Dufort V, Kleckner RC, Savitz DA. Fatal occupational injuries among electric power company workers. *Am J Industrial Med* 1999;35:302–9.
- Marc B, Baudry F, Douceron H, Ghaith A, Wepierre JL, Garnier M. Suicide by electrocution with low-voltage current. *J Forensic Sci* 2000;45:216–22.
- Mellen PF, Weedn VW, Kao G. Electrocution: a review of 155 cases with emphasis on human factors. *J Forensic Sci* 1992;37:1016–22.
- Peng Z, Shikui C. Study on electrocution death by low-voltage. *Forensic Sci Int* 1995;76:115–9.
- Strote J, Range Hutson H. Taser use in restraint-related deaths. *Prehospital Emerg Care* 2006;10:447–50.
- Study of Deaths Following Electro Muscular Disruption: Interim Report. U.S. Department of Justice, Office of Justice Programs, National Institute of Justice, Washington, DC, 2008.
- Taylor AJ, McGwin G Jr, Valent F, Rue LW 3rd. Fatal occupational electrocutions in the United States. *Injury Prevention* 2002;8:306–12.
- Wick R, Gilbert JD, Simpson E, Byard RW. Fatal electrocution in adults – a 30-year study. *Med Sci Law* 2006;46:166–72.

Chapter 18

Temperature-Related Deaths

When the sun rose, God provided a scorching east wind, and the sun blazed on Jonah's head so that he grew faint. He wanted to die, and said, 'It would be better for me to die than to live.'

Jonah 4:8

Abstract Chapter 18 begins by providing a general description of the normal temperature-regulation mechanisms in humans, along with an introduction to the concepts of hyperthermia and hypothermia. The remainder of the chapter is devoted to relatively in-depth examination of hypothermia and hyperthermia, with emphasis on scene investigation, autopsy findings, and death certification issues.

Keywords Temperature · Hypothermia · Hyperthermia

Introduction

The normal human body temperature is between 37.0 and 37.6°C (98.6–99.7°F), with temperatures up to 38.6°C (101.5°F) considered normal in infants and the elderly. There are slight daily cyclical variations, with the lowest temperatures occurring in the early morning and the highest in the afternoon. Vigorous exercise may cause a several-degree increase in temperature. The body temperature is under the control of feedback neural mechanisms involving the brainstem and the hypothalamus. Human tissue can survive only within a range of tissue temperatures between 20 and 44°C (68 and 111°F).

Ultimately, body temperature depends on a balance between “heat load” and “heat loss.” A body’s heat load represents the heat produced by normal metabolism plus any heat gained from the environment. Heat loss occurs via four mechanisms, each of which involves the transfer of heat from warmer objects to cooler objects. The four mechanisms of heat loss are “conduction,” “radiation,” “evaporation,” and “convection.” Conduction represents the direct transfer of heat from one object to an adjacent object. Radiation is heat loss via the emission of infrared rays. Evaporation

is heat loss that occurs when liquid water vaporizes from the body. Convection is heat loss that occurs when air currents moving around the body foster heat loss via conduction and evaporation.

The primary means by which an overheated body cools itself is evaporation, and there are two ways in which evaporation occurs in the body. The most obvious is via the process of sweating. Sweat represents water that is expelled from sweat glands within the skin; as the sweat evaporates from the skin surface, heat loss occurs. A human can sweat up to approximately 700 mL/h. The second type of water loss that allows heat dissipation via evaporation is referred to as “insensible water loss,” and refers to water loss through the skin and lungs via simple diffusion. In skin, this is distinct from sweat production. Approximately 600 mL of insensible water loss occurs daily.

As long as heat gain and heat loss are balanced, normal body temperature will be maintained. If heat gain exceeds heat loss, hyperthermia can result. If heat loss exceeds heat gain, hypothermia can result. Increased heat gain can occur with exercise, infection-induced fever, any other cause of increased metabolism, and a very warm environment. When environmental temperature exceeds body temperature, the body actually begins to gain heat via radiation and conduction. Decreased heat gain occurs when metabolism slows. Increased heat loss can occur in a variety of settings, including decreased environmental temperature, increased convection, a wet skin surface, and increased blood flow to the skin. Decreased heat loss can occur with increased skin insulation (fat, clothing, blankets), high humidity (which interferes with evaporation), and increased environmental temperature. When environmental temperature exceeds body temperature, evaporation is the only means by which cooling (heat loss) can occur.

If the temperature of localized tissues becomes markedly elevated, actual thermal tissue injuries (burns) can result. Various types of thermal injuries can occur, including scalding injuries (from hot liquids or gases), dry burns (from hot solids), radiant burns (from radiant heat sources, including fires), and fire burns. Burns are discussed in the next chapter (Chapter 19).

Hypothermia

General Features

Hypothermia may occur when heat loss exceeds heat load. Clinically, hypothermia is defined as a body core temperature of less than 35°C (95°F). A variety of factors are associated with an increased risk for hypothermia, including low environmental temperature, extremes in age (very young and very old), immersion in water, wet clothing, certain pre-existing diseases, such as hypothyroidism and atherosclerosis, dementia, inadequate nutrition, and ethanol intoxication, which leads to excessive heat loss via dilation of skin blood vessels.

The human body has a variety of defense mechanisms that come into play when facing the threat of hypothermia. The blood vessels of the skin and skeletal muscles

constrict, thus conserving heat by reducing the amount of heat loss via blood flow in these areas. Increased heat production occurs via reflex muscle shivering and biochemical thermogenesis.

As hypothermia sets in, the body's response includes decreased respiratory rate and heart rate, mental confusion with eventual euphoria, hyperglycemia (increased blood sugar), and eventual paralysis of the hypothalamus (a deep structure within the brain responsible for temperature regulation among other things), atrial fibrillation (a heart arrhythmia), and ventricular fibrillation (a lethal heart arrhythmia).

Scene Investigation

Although hypothermia-related deaths are more likely in extremely cold environmental temperatures, the environmental temperature does not even have to be below freezing in order for hypothermia to occur. Recording the local environmental temperature is an appropriate exercise when any dead body is discovered. When hypothermia is suspected, investigators should attempt to determine temperatures during the previous days and nights as well. Particularly in certain geographical locations, there can be great fluctuations in temperature from daytime to nighttime. The daytime temperature may be rather warm and pleasant when an investigator is called to the death scene, even though the previous nighttime temperatures were low enough to induce hypothermia.

Two specific scenarios may occur in cases of hypothermia. One is referred to as the "hide and die syndrome," in which a person who is experiencing hypothermia is found in a location that gives the impression that they might have been attempting to "hide." Examples include bodies being found in a closet, under a bed, or behind a piece of furniture.

The second scenario that is occasionally encountered in hypothermia deaths is referred to as "paradoxical undressing." In this phenomenon, which is likely related to hypothermia-induced disruption of hypothalamic function, persons dying of hypothermia begin to remove their clothing. Bodies may be found totally naked or partially undressed (Fig. 18.1 and Disc Image 18.1). Because of these findings, it is important to rule out sexual assault in such cases; however, the mere fact that someone is partially or totally undressed does not mean that there is anything whatsoever related to sexual activity, or hypothermia for that matter (Disc Image 18.2).

Autopsy Findings

There are no definitive autopsy findings which allow a pathologist to state with absolute certainty that a person died of hypothermia; however, there are several features that, taken together, and in the presence of the correct scene and circumstances, allow pathologists to be fairly confident in their diagnosis. Not all cases will demonstrate each of these features, but most hypothermia cases will have at least some of them.



Fig. 18.1 An example of “paradoxical undressing” occurring in an individual who died from hypothermia. (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)

Paradoxical undressing, as described above, is the first of the autopsy features that suggest the possibility of hypothermia. A second feature that is common in hypothermia-related deaths is a bright red-pink discoloration of livor mortis (lividity), which mimics, to an extent, that seen with carbon monoxide deaths. Some believe that this bright red color may be related to cold-induced retention of oxygen on hemoglobin molecules within red blood cells. Another feature visible on external examination is the presence of prominent red-brown skin discoloration evident on exposed skin surfaces, most notably over the extensor surfaces of large joints such as the knees and elbows (Figs. 18.2 and 18.3 and Disc Image 18.3), but also elsewhere, including the face. Finally, depending on the circumstances and timing



Fig. 18.2 Prominent red discoloration of the knees in a case of hypothermia, a 48-year-old intoxicated individual found dead outdoors in early January, with temperatures in the 20–30°F range



Fig. 18.3 Prominent red discoloration of the elbow in the same hypothermia case depicted in Fig. 18.2

of the death, there may be evidence of frostbite. This is typically evident as blue-black discoloration of fingers, toes, or other susceptible body parts, such as the nose and ears.

Depending on the circumstances, portions of the body or the entire body may be frozen solid. If the entire body is frozen solid, the performance of an autopsy is difficult and must be delayed until the body thaws. Unfortunately, as this happens, decomposition occurs at an accelerated rate, and will probably make the internal examination less than optimal.

The internal examination of hypothermia cases may or may not show features which suggest the cause of death. A nonspecific finding that is occasionally present is pulmonary edema. In cases where death did not occur rapidly, there may be evidence of bronchopneumonia. In some cases of hypothermia, a hemorrhagic pancreatitis is identified (Fig. 18.4). Perhaps the most diagnostic finding that is encountered in some hypothermia cases is the presence of “Wishnewski ulcers,”



Fig. 18.4 The gross appearance of hemorrhagic pancreatitis

which actually represent relatively evenly dispersed, consistently-sized superficial hemorrhagic gastric mucosal (stomach) erosions, ranging up to several millimeters in greatest dimension (Figs. 18.5 and 18.6 and Disc Images 18.4 and 18.5). Although their presence can be extremely helpful, Wishnewski ulcers are not required in order to make a diagnosis of hypothermia.



Fig. 18.5 An example of Wishnewski ulcers (superficial gastric mucosal erosions) in a case of hypothermia



Fig. 18.6 Another example of Wishnewski ulcers occurring in a case of hypothermia

It is not uncommon for the tissues of hypothermia victims to be remarkably well-preserved, such that microscopic examination reveals little autolysis. If death is relatively prolonged, various other features may be seen microscopically, including pulmonary edema, bronchopneumonia, hemorrhagic pancreatitis, acute tubular necrosis within the kidney, cardiac muscle degeneration, and perivascular hemorrhage. Finally, there may be evidence of other underlying disease processes.

Toxicology testing should be performed in any suspected hypothermia death. Persons acutely intoxicated with ethanol are at increased risk of hypothermia. Vitreous chemistry testing may reveal an elevated glucose level.

Death Certification

As alluded to above, the diagnosis of hypothermia at autopsy is considered a diagnosis of exclusion. In other words, so long as the death scene and circumstances suggest the possibility of hypothermia, and the autopsy reveals no other definite cause of death such that hypothermia can be ruled out, then it is appropriate to rule hypothermia as the cause or a contributing cause of death. It is certainly not unusual for persons dying of hypothermia to have underlying significant natural disease processes. This is particularly true in elderly persons: a relatively common occurrence involves an elderly patient with Alzheimer's disease who wanders away from the nursing home facility and dies during the night. It is entirely appropriate to list both hypothermia and the underlying natural disease(s) on the death certificate. Some prefer to use the term "exposure" rather than hypothermia.

Hyperthermia

General Features

Clinically (during life), hyperthermia is defined as a body core temperature equal to or greater than 40.5°C (104.9°F). It occurs when heat load exceeds heat loss. When hyperthermia occurs as a result of a body's inability to compensate for the heat of the environment, the term "heat stroke" is sometimes used.

It is important to note that there are a variety of non-environmental causes of hyperthermia, including brain hemorrhage (from natural disease or injury), the so-called "malignant hyperthermia syndromes" (caused by exposure to certain anesthetic or neuroleptic drugs), salicylate overdose, and reactions to certain drugs of abuse, such as cocaine (as occurs in excited delirium syndrome).

A variety of factors may be associated with environmental hyperthermia (heat stroke). These include high environmental temperature or heat index, extremes of age (infants and the elderly), obesity, alcoholism, other underlying natural disease, such as atherosclerotic cardiovascular disease, and physical exertion.

As described above, the body's main defense against hyperthermia is the process of evaporation via sweating. In dry air, a nude human can maintain a normal body temperature in environmental temperatures up to 54.4°C (130°F). Humidity decreases the body's ability to dissipate heat via evaporation.

The physiologic effects of hyperthermia include eventual hot, dry skin (because sweating ceases), an overall general feeling of warmth, nausea, vomiting, muscle cramps, and dyspnea (labored breathing). Eventually, central nervous system dysfunction occurs, as does cardiac failure.

Scene Investigation

A careful scene investigation is of great importance when evaluating cases that may represent hyperthermia deaths. Noting the environmental temperature at the scene is absolutely essential. Unfortunately, persons discovering the body (or first responders) may have altered the environmental temperature (by opening windows or turning on fans or air-conditioning) before investigators arrive. For this reason, it is important to ask these individuals about the temperature when the body was discovered. If resuscitative efforts are attempted on an individual suspected of hyperthermia, recording the body temperature in the ambulance or the emergency department may provide very useful information for eventual death investigation.

Recording the body temperature at the scene of death is advocated by some within the forensic community. Several potential problems exist with this recommendation. The first is that simple methods of recording body temperature tend to measure temperatures of superficial parts of the body, which may actually be quite cool compared to the underlying body core temperature. The second problem is that the most commonly employed method to accurately record the body's core temperature requires cutting into the body. The method involves making a small, full-thickness abdominal incision over the liver, with an associated underlying puncture defect of the liver, and subsequent insertion of a thermometer. Whether or not such a technique should be employed depends on weighing the benefits of obtaining the core temperature against the potential artifacts and other issues introduced by cutting into the body at the scene. Protocols should be established by each death investigation agency.

Heat-stroke scenarios that are most commonly encountered by the death investigation community include individuals undergoing extreme physical exertion in high-temperature settings, the elderly during prolonged heat-waves (Fig. 18.7), unattended infants and children in automobiles, and drug-induced hyperthermia (Fig. 18.8).



Fig. 18.7 When an elderly individual is found dead at home during a heat wave, there is no air-conditioning in the home, and the scene temperature is extremely hot, hyperthermia should be considered a possible cause or contributing cause of death



Fig. 18.8 A 24-year-old man, found dead in his backyard in the middle of July. His naked body, along with other scene findings, including a bathtub with running cold water, the presence of ice cube trays strewn about the kitchen, and overturned furniture, suggested the possibility of hyperthermia in association with drug-induced excited delirium. Autopsy was essentially negative, except for the presence of cocaine. The cause of death was the toxic effects of cocaine, with associated hyperthermia and excited delirium

Autopsy Findings

As with deaths due to hypothermia, deaths related to hyperthermia have no specific autopsy findings (Fig. 18.9). Intrathoracic petechiae may be present, particularly in infants and children (Fig. 18.10). Underlying natural disease processes frequently play a contributory role in death, particularly in the elderly. Persons surviving for a



Fig. 18.9 An autopsy photograph of a 3-year-old who was found unresponsive in an enclosed automobile in the middle of the summer. He was transported to an emergency department, where his body temperature was recorded at 108°F. All resuscitative efforts failed. Autopsy was essentially negative, disclosing only rare, superficial abrasions and a swollen brain



Fig. 18.10 Intrathoracic thymic petechiae (pinpoint hemorrhages) in an infant who died of hyperthermia. Such petechiae are not diagnostic of hyperthermia, especially in infants less than 1 year old, as the nonspecific finding is frequently encountered in asphyxial deaths and sudden infant death syndrome (SIDS) cases

period of time following the initial effects of hyperthermia may develop pneumonia, adrenal gland hemorrhage, liver necrosis, acute tubular necrosis within the kidneys, degenerative changes in the heart, and problems with coagulation.

Toxicology testing is absolutely essential in cases of suspected hyperthermia. As described above, a variety of legal and illegal substances can be associated with hyperthermia, including ethanol, salicylate (aspirin), anesthetic agents, neuroleptic drugs, and cocaine.

Death Certification

Like hypothermia, a diagnosis of hyperthermia at autopsy is a diagnosis of exclusion. If the scene and circumstances are such that hyperthermia is likely to have occurred, and autopsy findings reveal no underlying disease, or disease that would likely have been exacerbated by excessive heat, then hyperthermia is a reasonable ruling. If it is determined that a high environmental temperature caused or significantly contributed to death, “hyperthermia” is an appropriate cause of death. If an antemortem core body temperature is documented at or above 40.6°C (105°F), the term “heat stroke” is acceptable. Accident is the most common manner of death in hyperthermia cases; however, homicides may also occur.

In some cases, such as those where a person is discovered before they die, rushed to hospital, have a clinical diagnosis of hyperthermia, and then succumb to the process, a definitive diagnosis can be rendered. In many cases, the person is already dead at the scene. If the environmental temperature is markedly elevated, it is reasonable to conclude that hyperthermia caused death, with any underlying disease (such as heart disease) being a contributing factor. Sometimes, there is a decent amount of evidence to suggest that hyperthermia occurred, but also severe natural

disease. In such cases, some forensic pathologists will list the disease in part I of the death certificate, “probable hyperthermia” as a contributing cause of death, and “accident” as the manner. In cases where there is less evidence of hyperthermia, but it is still possible, the term “possible hyperthermia” can be listed as contributory, and the manner can be ruled “undetermined.”

Disc Image Legends

Disc Image 18.1 Another example of “paradoxical undressing” occurring in a hypothermia case (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC).

Disc Image 18.2 Although the decedent in this scene photograph is partially undressed, the death was not related to hypothermia. There was no indication of sexual activity or assault. The cause of death was a mixed drug overdose. A component of drug-induced hyperthermia could not be excluded.

Disc Image 18.3 Another example of skin discoloration occurring in a case of hypothermia (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC).

Disc Image 18.4 An elderly woman who wandered away from a nursing home during the winter. Her decomposing body was found outdoors, in a wooded area, several days later. Decompositional changes with associated animal feeding activity are noted on the arm. The internal findings in the stomach (see Disc Image 18.5) confirmed the suspicion of hypothermia.

Disc Image 18.5 Wishnewski ulcers in the decomposing stomach of the woman shown in Disc Image 18.4.

Selected References

- Birchmeyer MS, Mitchell EK. Wishnewski revisited – the diagnostic value of gastric mucosal ulcers in hypothermic deaths. *Am J Forensic Med Pathol* 1989;10:28–30.
- Christiansen LR, Collins KA. Pathologic findings in malignant hyperthermia – a case report and review of literature. *Am J Forensic Med Pathol* 2004;25:327–33.
- Coe JI. Hypothermia: autopsy findings and vitreous glucose. *J Forensic Sci* 1984;29:389–95.
- Denton JS, Fusaro AJ, Donoghue ER. Deaths due to heat and cold exposure. In: Froede RC, ed. *Handbook of Forensic Pathology*. 2nd ed. Northfield, IL: College of American Pathologists; 2003:225–30.
- Donoghue ER, Graham MA, Jentzen JM, Lifschultz BD, Luke JL, Mirchandani HG. Criteria for the diagnosis of heat-related deaths: National Association of Medical Examiners. *Am J Forensic Med Pathol* 1997;18:11–4.
- Krous HF, Nadeau JM, Fukumoto RI, Blackbourne BD, Byard RW. Environmental hyperthermic infant and early childhood death – circumstances, pathologic changes, and manner of death. *Am J Forensic Med Pathol* 2001;22:374–82.
- Mant AK. Autopsy diagnosis of accidental hypothermia. *J Forensic Med* 1969;16:126–9.
- Prahlow JA, Davis GJ. Death due to cocaine intoxication initially thought to be a homicide. *S Med Journal*. 1984;87:255–8.

- Ruttenber AJ, Lawler-Heavner J, Yin M, Wetli CV, Hearn WL, Mash DC. Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. *J Forensic Sci* 1997;42:25–31.
- Sheil AT, Collins KA, Schandl CA, Harley RA. Fatal neurotoxic response to neuroleptic medications – case report and review of the literature. *Am J Forensic Med Pathol* 2007;28:116–20.
- Wedin B, Vanggaard L, Hirvonen J. “Paradoxical undressing” in fatal hypothermia. *J Forensic Sci* 1979;24:543–53.
- Wolfe MI, Kaiser R, Naughton MP, Mirabelli MC, Yoon SS, Hanzlick R, Henderson AK. Heat-related mortality in selected United States cities, summer 1999. *Am J Forensic Med Pathol* 2001;22:352–7.

Chapter 19

Burns and Fire-Related Deaths

So all the men cut branches and followed Abimelech. They piled them against the stronghold and set it on fire over the people inside. So all the people in the tower of Shechem, about a thousand men and women, also died.

Judges 9:49

Abstract This chapter addresses deaths related to burns, as well as fire-related deaths. It begins by describing the various types and extent of burns that can be encountered in such cases, along with specific sub-types of burns, including dry burns, radiant burns, scald burns, chemical burns, and fire-related thermal burns. Next, the chapter discusses issues of importance when dealing with a body found at a fire scene. It concludes by addressing other important issues, including autopsy findings, mechanism of death, death certification, fire investigation, and cremation.

Keywords Burns · Thermal injuries · Scalding · Fire · Chemical burns

Introduction

The tissues of the body, including the skin, can sustain burn injuries by several mechanisms. The most common types of burns are related to thermal damage, which occurs when a tissue's cooling ability fails to compensate for externally applied heat. The extent of damage depends on the applied temperature, the ability of the tissue to conduct away excess heat, and the time for which the heat is applied. With these concepts in mind, it should be evident that thermal burns can occur very quickly when contact is made with very hot objects/substances. Conversely, objects/substances that are hot, but not extremely hot, can still cause thermal burns so long as contact with the object is long enough to overcome the tissue's ability to cool itself. Another important concept is that skin surfaces of different body regions, and skin from individuals of different ages, can vary greatly in their ability to withstand thermal insult. The classic example involves infant skin being burned by hot water that does not readily burn adult skin.

Besides thermal injury, burns can result from contact with chemical substances or as a result of exposure to various forms of radiation. In each of these situations, a thermal component may play a role in tissue damage, but other mechanisms are also present.

Burn severity as seen on the skin surface may be categorized as follows. First degree burns demonstrate a red discoloration of the skin surface. Physicians may frequently describe this as “erythema.” Such burns are limited to the epidermis (Fig. 19.1). Second degree burns damage not only the epidermis, but also the upper dermis. These burns are characterized by blisters and skin “slippage” (Fig. 19.2). In third degree burns, full-thickness skin injury occurs (Fig. 19.3). Fourth degree burns are characterized by charring of the skin and underlying subcutaneous tissues (Fig. 19.4). If one considers the skin as an organ, it represents the largest organ



Fig. 19.1 The intact, dark-appearing skin (centrally) represents a first degree burn. The adjacent burns with skin slippage are second degree burns



Fig. 19.2 Example of second degree burns seen at autopsy. Note the slippage of skin



Fig. 19.3 Example of third degree burns seen at autopsy. Note the full-thickness skin injuries, with the underlying subcutaneous fat (*lighter areas*) visible. Note also the residual remnants of charred, adherent clothing (*darker areas*)

Fig. 19.4 Example of fourth degree burns seen at autopsy. Note the presence of extensive charring of soft tissues



of the body. Its integrity is absolutely essential for life; if a significant portion of the skin is burnt, death can result. Certainly, death can occur relatively rapidly; however, it is not unusual for burn victims to survive the initial injury, only to die several days, weeks, or even months later. Metabolic complications following burns include electrolyte disturbances, shock, infection, and the release of substances into the bloodstream as a result of inflammation that is induced by the burn injuries. It is not necessary for a majority of the skin surface to be burned in order for death to occur. For example, if a child sustains burns that are localized only to the legs, this can be enough to cause major metabolic disturbances such that death can occur.

Estimating the surface area of a thermal injury is important clinically, when attempting to treat those who are suffering from burns. The “rule of nines” is frequently employed by physicians when they attempt to estimate the percentage of body surface area affected by thermal injuries. The following general percentages are applied to each of the listed body regions: head – 9%; anterior torso – 18%; posterior torso – 18%; right arm – 9%; left arm – 9%; right leg – 18%; left leg – 18%; genitalia – 1%. Some forensic pathologists use the rule of nines, others prefer to diagram, photograph, and describe the extent of burn.

In this chapter, each of a variety of burn injuries will be presented, followed by a more general description of fire-related deaths, where death may or may not be related to thermal injury.

Burn Types

Dry Burns

A dry burn is a thermal burn that results from contact with a dry heat source. This is in contrast to a “wet burn,” or a scalding injury, that results from contact with a wet heat source. Strictly speaking, fire-related (flame) injuries are at least partially a type of dry burn, but fire burns will be discussed separately below.

Examples of dry burns include skin burns from contact with hot surfaces, such as a clothes iron, a hot dish or pan, a stove top, an oven, a heating pad, a furnace, a hot engine, or a muffler. In the preceding list, although the heating pad does not generate nearly as much heat as the other examples, it still can result in thermal injury, so long as the time of exposure is long enough.

A subtype of dry burn is an electrothermal burn, where heat is generated at a very concentrated location during electricity flow. Such “electrical burns” are discussed in greater detail in Chapter 17.

Dry burns can result in first, second, third, or fourth degree burns. Depending on the source of heat, dry burns may cause body hair to singe. This is particularly true of fire burns, but can occur in other situations as well. Figure 19.5 represents an example of a dry burn.

Radiant Burns

A radiant burn results from heat or electromagnetic energy that is radiating away from an energy source. Two general types can be described: radiant heat burns that are associated with a local heat source in relatively close proximity to the victim (which commonly occur in fire-related burns), and other electromagnetic energy burns, such as those occurring in association with ultraviolet and ionizing radiation.

In contrast to dry burns as defined above, where direct contact with the heat source is required, radiant heat burns do not require direct contact. Strictly speaking,



Fig. 19.5 A postmortem dry burn caused by the decedent's leg being adjacent to an extremely hot exhaust pipe. Differentiating postmortem and antemortem dry burns can be difficult if death occurs at or shortly after the burn is sustained

radiant heat burns represent, to an extent, a type of dry burn, where the “object” in direct contact with the tissue is represented by the superheated air. Such burns are common in fire deaths (see below).

Burns from ultraviolet, ionizing, and other sources of radiation are actually very common, but rarely result in death. Probably the most common example is sunburn, wherein ultraviolet rays from the sun cause localized skin injury. Probably the most common example of ionizing radiation burns occurs in the setting of radiation therapy for internal cancers, where it is not uncommon for a small amount of sunburn-like skin burning to occur (Disc Image 19.1).

Scald Burns

Scald burns are created by moist, or wet, heat, such as occurs when skin comes into contact with a hot liquid or gas (steam). The most common substance involved with scald burns is water (in the form of hot water or steam); however, other substances may also cause these injuries. Examples include hot oils, tar, and molten rubber or metal (Disc Images 19.2 and 19.3).

Typically, scald burns do not result in charring of the skin or singeing of hair, but first, second, and third degree burns can occur. Extremely hot molten metals may produce burns that are similar to dry burns.

In certain cases, the pattern of scalding injuries may provide useful information regarding how the injury was sustained. The classic scenario involves cases of alleged child abuse. Occasionally, a child is forcibly placed into extremely hot water as a form of punishment. When this happens, an “immersion injury” results. “Fluid level lines” occur on the skin surface, typically on the legs but sometimes on the hands/arms, at levels that correspond to how deep the child was placed into the

water. These are sometimes referred to as having a “stocking pattern” or a “glove pattern.” A child’s reflexive reaction to being placed into the water causes them to flex at the knees and waist, as he/she tries to keep from entering the water. As such, there may be areas of “sparing” (lack of burns), where skin has been protected from exposure to the water, because the skin in certain areas is compressed against other skin. This occurs at points of flexion at the joints, including the popliteal fossa (back of knee joint), the inguinal region (groin), and the antecubital fossa (bend of elbow joint). While immersion injuries are classically described as child abuse injuries, it is important to recognize that it is within the realm of possibilities for such injuries to be accidental, depending on the specific details of the case. As with many forensic cases, investigation of the death scene and circumstances is absolutely essential in making such determinations (Fig. 19.6).

Fig. 19.6 Child abuse death related to a bathtub scalding. (Photo courtesy of Dr. Patrick Lantz, MD, Department of Pathology, Wake Forest University School of Medicine, Winston-Salem, NC)



The other classic scalding injury is typically described as being indicative of an accidental injury. If a pot of boiling water is accidentally pulled from the top of a stove by a curious toddler, scalding injuries will not have fluid-level lines, but will instead have a “drip” or “splash” pattern, with the severity of burns decreasing as the liquid flows downward. As with the immersion injuries, the mere presence of drip or splash scalding injuries should not result in an automatic manner of death determination. Certainly, it is within the realm of possibilities for an abusive adult to intentionally splash a child with boiling water.

Other settings in which accidental scalding injuries and death may occur include various industrial settings, such as the case depicted in Disc Images 19.2 and 19.3, as well as in settings involving naturally-occurring heat sources. Examples of the latter include hot lava from volcanoes and hot springs, mud pots, and fumaroles, such as those that occur in Yellowstone National Park.

Chemical Burns

As mentioned above, a component of thermal injury may play a role in certain types of chemical burns; however, most involve other mechanisms of injury, including acid-induced protein precipitation, alkali-induced protein destruction or fat saponification, oxidation or reduction reactions, salt formation, corrosion, protoplasmic poisoning, metabolic competition or inhibition, desiccation (drying), and vesication (blistering). It is important to note that certain chemical burns may not become evident for several hours. The extent and form of the tissue damage depends on the mechanism of action as described above. Ultimately, whether or not tissue damage will occur depends on the concentration of the chemical, the quantity of the chemical, the time of exposure, and the depth of tissue penetration. As such, the most important preventive measure for most chemicals is to remove the chemical as quickly as possible and to flush the area of exposure with water for an extensive period of time. Medical evaluation and consultation with the Material Safety Data Sheet (MSDS) for the specific chemical will allow for the most appropriate measures to be implemented.

Probably the most common chemical burn-related death scenario that is encountered by forensic pathologists involves the suicidal ingestion of a chemical such as lye. In such a setting, severe skin, intraoral, digestive system (esophagus), and upper airway alkali burns are identified.

Infrequently, chemical burns on the skin may be of sufficient severity to result in death. The extent of the burn depends on the chemicals involved. With extremely caustic chemicals, it may be necessary to “decontaminate” the body prior to examination and autopsy, so as to avoid causing similar injuries to investigators, pathologists, and assistants. Figure 19.7 is an example of a death related to extensive chemical skin burns.



Fig. 19.7 Extensive chemical skin burns occurred in this mentally-handicapped woman when she rolled around in a bleach-containing cleaning solution on the floor at a nursing facility. Although she was “washed off” and put into bed, the chemicals were not sufficiently washed from her skin surface, such that she developed severe chemical burns

Chemical warfare agents such as sulfur mustard, lewisite, and phosgene oxime (CX), are described as “vesicants” (blistering agents). Skin blistering is one mechanism by which these agents kill; however, respiratory and other mechanisms are frequently more severe and debilitating.

Fire-Related Burns

Fire-related burns are the most common type of burns encountered in death investigation. The typical setting is a structural fire with associated fatalities. Fire-related burns in which death occurs can range from very localized, small areas of first or second degree skin burns (where death is related to smoke and soot inhalation) (Fig. 19.8), all the way to near-complete combustion of a human body, with only a charred mass of tissue remaining (Fig. 19.9).



Fig. 19.8 A small area of second degree burns on a fire victim who succumbed to smoke and soot inhalation



Fig. 19.9 A badly burnt body, consisting of charred remnants of the torso and head. Note that a majority of the extremities have been totally consumed by the fire

Fire burns represent a spectrum and combination of dry burns and radiant burns. They may involve radiant heat injury alone, injury resulting from tissues being in contact with burning items (clothing, bedding), or actual combustion of tissues themselves (the tissues are actually “on fire”). As will be described below, it is usually impossible to differentiate antemortem (before death) thermal burns from postmortem (after death) thermal effects.

Fire Deaths

Questions to Address

When a body is discovered in a structural fire or a vehicular fire, or if a burnt body is found anywhere, several important questions must be addressed. The first question is: who is it? Identification of the decedent may be the single most important issue in a given case. Depending on the extent of thermal damage, fingerprint, dental, X-ray, and perhaps even DNA identification may be required.

The next very important question is: did the person die from the fire or were they dead before the fire started? A person who dies from the effects of the fire and a person who was dead prior to the fire starting can look absolutely identical when first discovered. The typical situation is the discovery of a badly-charred body, one that is “burnt beyond recognition” (Disc Image 19.4). As stated above, it is very difficult, if not impossible, to differentiate antemortem and postmortem burns. When a very badly burned body is sent for autopsy, many persons unfamiliar with the examination of such a body find it somewhat amazing that an autopsy can still be performed. In fact, on internal examination, a great deal of very useful information can usually be obtained.

There are several important things that pathologists look for when attempting to determine whether or not a victim died from a fire. Three of these in particular are evidence that the person was actually breathing during the fire, since then it stands to reason that they were not dead before the fire. When a pathologist is able to determine that a person was, in fact, breathing during the fire, the cause of death (or at least a contributing cause) is usually able to be determined as well. In other words, the three findings that indicate that a person was breathing during the fire also tend to provide evidence that the victim died from smoke and soot inhalation.

The first indicator is the presence of bright red discoloration of the tissues of the body, which suggests the possibility of an elevated blood carbon monoxide (CO or carboxyhemoglobin) level. If skin is visible, bright red lividity may be present (Disc Image 19.5); however, thermal skin burns can sometimes mimic this, so this indicator is probably the least reliable of the three. Bright red discoloration of internal tissues is fairly classic (Disc Image 19.6). Occasionally, a fire death victim does not have an elevated carbon monoxide level (see below), in which case this indicator may be absent.

The second indicator that a person was breathing during a fire relies on toxicology tests that show an elevated CO level within blood samples collected at

autopsy. In many offices, CO levels can be determined during the performance of the autopsy, such that results are available before the autopsy is completed. In most fire deaths where smoke and soot inhalation are considered the cause or a contributing cause of death, CO levels are greater than 40–50%. Normal CO levels are typically less than about 5%, with very heavy smokers sometimes having levels approaching 10%. Therefore, if a CO level in a fire victim is somewhere between 10 and 40%, it indicates two things: (1) the person was, in fact, alive prior to (and during) the fire; (2) there is probably another contributing cause of death, besides smoke and soot inhalation. Other contributing causes may include thermal injuries, underlying severe natural disease, and/or intoxications of various types. Occasionally, other traumatic injuries are also encountered. This is more likely in vehicular incidents, where blunt force injuries are common. As mentioned above, there are occasional fires where CO levels are not significantly elevated but the death was absolutely related to the fire (see below). In such instances, pathologists rely on the fire investigator's description of the fire, as well as the third and final indicator that the person was breathing during the fire.

The third indicator that provides evidence of breathing during a fire is the identification of soot and/or thermal injuries within the upper airways of fire victims at autopsy. In bodies that are not badly burned, one can occasionally see soot within and around the nostrils (Fig. 19.10 and Disc Image 19.7). Such a finding is particularly important if there is little soot elsewhere on the body; however, it becomes less meaningful if dense soot covers the body. On internal examination soot is commonly found in the oral cavity, including on the tongue (Disc Image 19.8). Again, if the mouth is open, and there is dense soot present, such a finding is nonspecific. Finding soot within the trachea and bronchi can be considered definitive proof that the person was breathing during the fire (Fig. 19.11 and Disc Image 19.9). The only exception to this statement would be if the soft tissues of the neck are partially destroyed by fire, such that there is direct communication between the environmental air and



Fig. 19.10 Soot within the nostrils of a fire victim, indicating that he was probably breathing during the fire

Fig. 19.11 Dense soot coating the mucosal surface of the trachea and mainstem bronchi, indicating that the victim was breathing during the fire



the mucosal surfaces of the larynx/trachea, so that soot might be deposited directly on these surfaces. In such a situation, the discovery of soot within bronchi, distant from the trachea, would still be sufficient to indicate breathing during the fire. In some fires, there is very little soot, but extremely hot gases/air, such that the airway mucosal surfaces are scorched, rather than coated with soot (Disc Image 19.10). Occasionally, there will be very little or no soot (Fig. 19.12), as well as absence of scorching, but there is a markedly elevated CO level.



Fig. 19.12 An example of a fire-related death where there is minimal soot evident within the trachea

Since there are exceptions to each of the three indicators used to determine whether or not a person was breathing during a fire, it is theoretically possible that a person was breathing but does not have any of the indicators present. In such a case, concern should be raised as to whether the person was actually dead before the fire started. Additional autopsy findings and scene/fire investigation findings should help to clarify such cases.

If a person is actually dead before a fire starts, a variety of possibilities exist. As mentioned numerous times already, fire investigation is extremely important in these cases. Persons have been known to die a natural death, and then the house burns down because the stove was left burning. If a fire is determined to be an arson, and a body within the fire scene is determined to have been dead prior to the fire, it is a very strong possibility that someone is attempting to “cover up” a homicide. Alternatively, occasional suicide victims intentionally set themselves on fire, or intentionally set a fire immediately prior to taking their own life.

Autopsy

External Examination

A variety of procedures should be considered during the external examination of a fire victim. If a body is badly burned, to the point of being charred, X-ray examination may help to identify the decedent as well as a cause of death (e.g. bullets). The identification of burnt fragments of clothing, personal effects, and jewelry can assist in preliminary identification. In addition, fragments of clothing may be retained within sealed containers in case subsequent evaluation for the presence of accelerants must be performed. The presence and extent of thermal injuries should be documented, as described above. In some cases, burns are absent, as the death is totally related to smoke and soot (carbon monoxide) inhalation. In other cases, the body (including clothing) is covered with soot. In persons who have thermal injuries, it is not uncommon for unburned skin as well as multiple burns of differing severity to be present (Fig. 19.13 and Disc Images 19.11, 19.12, and 19.13). Clothing, or the floor (when a person is lying on the floor), can prevent burns from forming (Fig. 19.14 and Disc Image 19.14).

Severe combustion of tissues can result in absence of tissue, including bone. It is not uncommon to find badly burnt remains in a fire in which a majority of the distal extremities have been totally consumed by fire. External examination may reveal bright red lividity as described above, as well as a variety of heat-induced artifacts (see below). Clouding of the corneas may be evident (Disc Image 19.15). Finally, in badly burned (charred) remains, it is very common for the bodies to have what is referred to as a “pugilistic” posture (also referred to as a “boxer’s stance”) (Fig. 19.15). As muscles become superheated after death, the flexor muscles tend to contract more than the extensors. This results in postmortem flexion of the extremities (bending at the hips, knees, shoulders, and elbows). Occasionally, thermal injuries destroy the soft tissue (and associated bones) to such an extent that



Fig. 19.13 Multiple burn severities. Note areas of skin with no thermal injury, areas with first and second degree burns, and areas with third degree burns, approaching the charred stage



Fig. 19.14 Thermal injuries consisting of second degree burns, primarily of the extremities. Note the sparing of the sock, shorts, and most of the shirt region

internal organs are visible on external examination (Fig. 19.16). If a person survives in the hospital following thermal injury, a variety of medical artifacts may be evident, including evidence of tissue debridement (wherein burned tissue is surgically cut off of the body) (Disc Image 19.16) and the presence of “fasciotomy” incisions (surgical cuts to relieve underlying pressure (Disc Image 19.17)).

Internal Examination

On internal examination, bright red discoloration of tissues and the presence of soot and/or scorching of the airways may be evident, as detailed above. In some cases, with extensive postmortem burning, internal organs can become “heat-fixed,” or “cooked.” In addition, antemortem injuries and pre-existing disease should also be evident. Particularly in cases where it appears that the victim was dead prior to the fire, the identification of an underlying cause of death is of paramount importance.

Fig. 19.15 The classic “pugilistic” appearance of a badly burnt body. Note that the hands have been totally consumed by thermal injury



Fig. 19.16 Note that a portion of intestines is protruding from a thermally-induced defect in the abdomen

Toxicology

As with other deaths, toxicology tests are vitally important in fire deaths. Indeed, as detailed above, the identification of elevated and/or lethal levels of CO in postmortem blood samples allows pathologists to determine that the victim was breathing during the fire and died as a result of smoke and soot inhalation. Before sending blood samples for CO testing, it is advisable to check with the lab to be sure the samples are collected in the appropriate containers (laboratories sometimes differ in the instruments used to detect CO; different instruments may require different collection protocols). Occasionally, the blood is of insufficient quality to perform

a CO test or an insufficient amount of blood may be available (typically in very severely burned bodies). Collecting other tissues, such as liver, can allow CO determination via reference laboratories. Occasionally, other poisonous gases, such as cyanide, may be present in the smoke produced in fires, and specialized testing may be able to detect such poisons. Unfortunately, there is no postmortem test that allows pathologists to determine that a person died from lack of oxygen.

Artifacts

A variety of heat-induced artifacts may be evident in fire deaths, and these can occur whether or not death is caused by the fire. A common finding in bodies that are charred or partially charred is the presence of “skin splitting.” As the tissues become superheated after death, the skin becomes heat-fixed and shrinks to an extent, resulting in the skin splitting open to reveal the underlying subcutaneous fat (Fig. 19.17 and Disc Images 19.18 and 19.19). This fat may also burn, but not necessarily.



Fig. 19.17 Fourth degree burns with extensive skin-splitting

Another artifact is the presence of long bones that appear to be fractured. In reality, the bones have been partially consumed, along with the associated soft tissues. The burnt portions of bones protruding from residual charred soft tissues can occasionally be misconstrued as traumatic injuries (Disc Image 19.20).

Heat-induced skull fractures are relatively common (Fig. 19.18). As a dead body becomes superheated, pressure builds within the enclosed cranial cavity. Eventually, the pressure causes skull fractures. A layer of brown, heat-fixed, sludgelike epidural blood is usually found immediately underlying and adjacent to the skull fracture sites. This skull fracture/epidural hematoma complex is considered a classic heat-induced artifact (Fig. 19.19 and Disc Image 19.21). It should be noted that subdural hemorrhage should never be considered artifactual (Fig. 19.20).

Finally, it should be reiterated that, in many instances, postmortem thermal damage cannot be distinguished from true antemortem injury. As such, when thermal injury accompanies findings that confirm smoke and soot inhalation as a cause or



Fig. 19.18 Heat-induced skull fracture. Such a fracture occurs when the intracranial contents become superheated after death and the pressure causes the overlying bone to fracture. Note that the dura is intact and there is a small amount of epidural hemorrhage present (*on the left*)

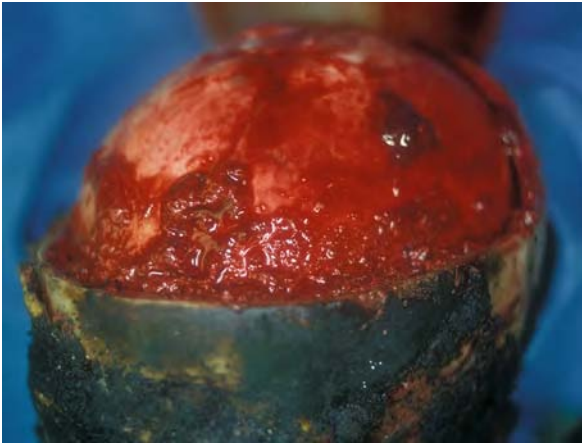


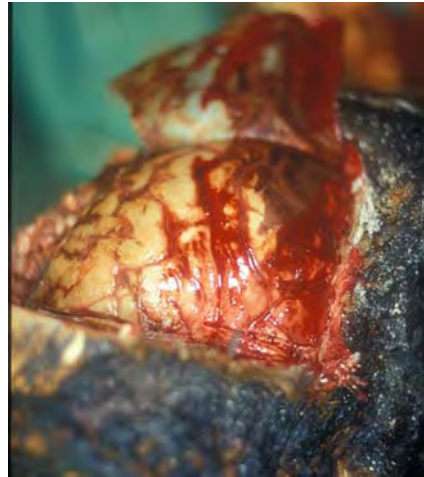
Fig. 19.19 A heat-induced, artifactual epidural hemorrhage, after skull cap removal. Note that the dura remains covering the brain. The sludge-like hemorrhage is between the skull and the dura

contributory cause of death, it is not usually possible to say with absolute certainty that thermal injuries did or did not contribute to death.

Mechanism of Death

In many fire-related deaths, death is ultimately at least partially related to chemical asphyxia due to carbon monoxide binding to hemoglobin within red blood cells, such that oxygen is not able to reach the cells of the body. Recall that hemoglobin binds to CO much more readily than to oxygen. The CO is present within the smoke

Fig. 19.20 A true antemortem (occurring before death) head injury in a person whose body subsequently burned. Note that the dura has been reflected upward, revealing underlying subdural (and subarachnoid) hemorrhage



and soot that is produced within the fire. In slow, smoldering fires, CO levels within the air tend to be very high. In some fires, abundant carbon dioxide displaces oxygen. In some rapid, so-called “flash fires,” little CO may be produced. Thus, even if CO levels are relatively low or absent, the lack of oxygen in the air can result in “simple asphyxia” (lack of environmental oxygen). When little or no oxygen is available to breathe, unconsciousness and subsequent death results. Conversely, the sudden intense heat associated with certain flash fires can be so rapidly fatal that, even if CO is produced, there is no time for a victim to breath it in. In such cases, death results from thermal injuries. In some fire deaths, other poisonous gases may be present and contribute to death. Cyanide gases are sometimes produced. Finally, the smoke and soot within a fire are frequently extremely hot, to the point of being able to scorch the airways. Reflexive airway closure in response to breathing in this superheated air may play a role in some deaths.

Thermal injuries are also very common in fire deaths. Injury extent varies from first to fourth degree, as detailed above. Even if the injuries are limited to first and second degree burns, as long as a sufficient percentage of skin surface is involved, subsequent metabolic insult can contribute to death. Occasionally, thermal injuries occur so quickly that they alone are considered the sole cause of death. In such cases, CO levels will be normal, and there will be no evidence of breathing smoke/soot.

In some cases, underlying natural disease, such as pre-existing lung and/or heart disease, injuries, and/or intoxicating substances may contribute to death. In cases where death precedes the fire, another cause of death (natural, drug-related, or traumatic) must be identified.

Death Certification

Since so many potential mechanisms are involved when smoke inhalation is considered a cause of death, including CO toxicity, lack of oxygen, other poisonous

gases, and superheated air, it is appropriate to certify such deaths as being due to “smoke and soot inhalation.” When thermal injuries are considered contributory, listing “thermal injuries” is likewise appropriate. Example: part I – Smoke and soot inhalation; part II – Thermal injuries.

If other factors, such as underlying natural disease, injury, or intoxication, are considered contributory, it is appropriate to list these in part II of the death certificate, with smoke and soot inhalation, thermal injuries, or both listed in part I.

As described above, in deaths that precede the fire, the manner of death can be natural, homicide, suicide, or accidental. In cases where victims die from the effects of the fire, then accidental deaths are, by far, the most common. If a person dies in an arson where the fire was intentionally set by another individual, it is appropriate to rule that death a homicide, whether or not the death itself was intentional. Rarely, a homicide is committed by setting the victim on fire. If a person dies in an arson that they themselves set, in the absence of other evidence of suicide such deaths are probably best ruled as accidental deaths. Occasional persons intentionally set themselves on fire in an act of suicide. The term “self-immolation” is sometimes used to describe such cases.

Fire Investigation

As mentioned numerous times in the preceding paragraphs, it is absolutely necessary for an official fire investigation to be performed whenever a fire-related death occurs. The responsibility for performing such an investigation is typically outside of the realm of the death investigation office, and often falls on the fire marshal or perhaps the state crime laboratory. It is important for death investigation agencies and fire investigators to work together and share information when investigating these cases. Important questions that fire investigators attempt to answer include how the fire started and where the fire started.

Cremations

Occasionally, forensic scientists will be asked to evaluate “cremains,” the ashes that remain after a body has been cremated. These cases are extremely difficult to evaluate and often require the combined efforts of pathologists, anthropologists, odontologists, and other forensic scientists. Since the process of cremation involves total or near-total incineration of all organic material, only the inorganic components of the human body, including parts of bone and teeth, and occasionally other tissues. The presence of various implanted structures, such as dental restorations or orthopedic hardware, provides the best route for positive identification.

Disc Image Legends

- Disc Image 19.1 Radiant burn related to radiation therapy for breast cancer. The red-pink area on the lower portion of the breast represents the radiation burn.
- Disc Image 19.2 Severe scalding burns occurred in this individual who accidentally fell into a creosote pit (Photo courtesy of John Pless, MD, Indiana University School of Medicine (retired), Indianapolis, IN).
- Disc Image 19.3 An autopsy photograph of the case depicted in Disc Image 19.2. Death was due to thermal injuries in combination with asphyxia/drowning (Photo courtesy of John Pless, MD, Indiana University School of Medicine (retired), Indianapolis, IN).
- Disc Image 19.4 In badly burnt bodies such as the one depicted in this photograph, visual identification is not possible. Depending on the condition of the remainder of the body, positive identification must be made via fingerprint comparison, dental examination, X-ray examination, or other means, such as DNA testing.
- Disc Image 19.5 Bright red discoloration of the lividity, indicating carbon monoxide poisoning. Note that there are also thermal injuries on the skin.
- Disc Image 19.6 Bright red discoloration of the muscles and soft tissues of the body as seen at autopsy after organ removal. The bright red discoloration results from high levels of carbon monoxide breathed in prior to death.
- Disc Image 19.7 Another example of soot within the nostrils, indicating that the victim was breathing during the fire.
- Disc Image 19.8 Soot on the tongue, within the larynx, and within the trachea, indicating that the person was breathing during the fire.
- Disc Image 19.9 Another example of dense soot deposition within the trachea and bronchi.
- Disc Image 19.10 A case with scorching of the trachea, with associated soot.
- Disc Image 19.11 Variable degrees of skin burns, including areas with no burns, and other areas with first and second degree burns.
- Disc Image 19.12 Areas of third and fourth degree burns.
- Disc Image 19.13 Various degrees of skin burns, ranging from none to fourth degree.
- Disc Image 19.14 Areas of skin sparing on the fire-victim's back, due to the fact that her body was found lying on the floor, on her back.
- Disc Image 19.15 Clouding of the corneas may be evident in burned bodies.
- Disc Image 19.16 This victim was intentionally set on fire while he was sleeping. He survived several days prior to death. Note where the burned skin has been surgically debrided (removed) on the upper chest/shoulder regions.
- Disc Image 19.17 A photograph of the arm of the case depicted in Fig. 19.16, showing a surgical fasciotomy incision used to relieve soft tissue pressure.
- Disc Image 19.18 Another example of fourth degree burns with associated skin splitting.
- Disc Image 19.19 An example of extensive skin splitting associated with fourth degree burns.
- Disc Image 19.20 An example of how bones may appear to protrude in a badly burned body. Note that the entire right foot, as well as a majority of the soft

tissues of the lower right leg have been consumed by fire, leaving the bones of the lower leg readily visible.

Disc Image 19.21 Another example of a heat-induced epidural hematoma. In this example, the dura remained over the brain when the skull cap was removed.

Selected References

- Adair TW, DeLong L, Doberson MJ, Sanamo S, Young R, Oliver B, Rotter T. Suicide by fire in a car trunk: a case with potential pitfalls. *J Forensic Sci* 2003;48:1113–6.
- Bohnert M, Rost T, Pollak S. The degree of destruction of human bodies in relation to the duration of the fire. *Forensic Sci Int* 1998;95:11–21.
- Bohnert M, Werner CR, Pollak S. Problems associated with the diagnosis of vitality in burned bodies. *Forensic Sci Int* 2003;135:197–205.
- Bonavilla JD, Bush MA, Bush PJ, Pantera EA. Identification of incinerated root canal filling materials after exposure to high heat incineration. *J Forensic Sci* 2008;53:412–8.
- Eckert WG. The medicolegal and forensic aspects of fires. *Am J Forensic Med Pathol* 1981;2:347–57.
- Fanton L, Jdeed K, Tilhet-Coartet S, Malicier D. Criminal burning. *Forensic Sci Int* 2006;158:87–93.
- Gerling I, Meissner C, Reiter A, Oehmichen M. Death from thermal effects and burns. *Forensic Sci Int* 2001;115:33–41.
- Henry RE, Graham MA. Deaths associated with fires and burns. In: Froede RC, ed. *Handbook of Forensic Pathology*. 2nd ed. Northfield, IL: College of American Pathologists;2003:219–24.
- Hill IR. Immediate causes of death in fires. *Med Sci Law* 1989;29:287–92.
- Hirsch CS, Adelson L. Absence of carboxyhemoglobin in flash fire victims. *JAMA* 1969;210:2279–80.
- Hirsch CS, Bost RO, Gerber SR, Cowan ME, Adelson L, Sunshine I. Carboxyhemoglobin concentrations in flash fires – report of six simultaneous fire fatalities without elevated carboxyhemoglobin. *Am J Clin Pathol* 1977;68:317–20.
- Kane AB, Kumar V. Environmental and nutritional pathology. In: Kumar V, Abbas AK, Fausto N, eds. *Robbins and Cotran Pathologic Basis of Disease*. 7th ed. Philadelphia, PA: Elsevier Saunders; 2005:415–68.
- Lee KAP, Opeskin K. Fatal alkali burns. *Forensic Sci Int* 1995;72:219–27.
- Leth P, Hart-Madsen M. Suicide by self-incineration. *Am J Forensic Med Pathol* 1997;18:113–8.
- Mallak C. Miscellaneous and special topics in forensic pathology – blast injuries, aircraft mishaps, and radiation injury. In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:157–61.
- Mettler FA Jr, Voelz GL. Major radiation exposure – what to expect and how to respond. *N Engl J Med* 2002;346:1554–61.
- Prahlow JA. Chemical burns. *Forensic Pathology Program*. Northfield, IL: College of American Pathologists; 2001. FR-B#9.
- Warren MW, Falsetti AB, Hamilton WF, Levine LJ. Evidence of arteriosclerosis in cremated remains. *Am J Forensic Med Pathol* 1999;20:277–80.
- Warren MW, Maples WR. The anthropometry of contemporary commercial cremation. *J Forensic Sci* 1997;42:417–23.
- Warren MW, Schultz JJ. Post-cremation taphonomy and artifact preservation. *J Forensic Sci* 2002;47:656–9.
- Warren S. Forensic pathologic criteria for radiation death. *J Forensic Sci* 1971;16:137–43.
- Zaloga WF, Collins KA. Pediatric homicides related to burn injury: a retrospective review at the Medical University of South Carolina. *J Forensic Sci* 2006;51:396–9.

Chapter 20

Deaths in Infancy and Childhood

During the night this woman's son died because she lay on him.
1 Kings 3:19

Abstract Chapter 20 covers the important topic of childhood death. It not only addresses deaths in infancy and childhood, but also fetal deaths and birth-related deaths. A section entitled “Infant Deaths” provides discussion of sudden infant death syndrome (SIDS) and similar cases, accidental asphyxial deaths, and the topics of “co-sleeping” and “bed-sharing.” Additional sections of the chapter deal specifically with natural death in childhood, accidental childhood death, suicides in children, childhood homicides, and pediatric autopsy considerations.

Keywords Infant death · Childhood death · Fetal death · Sudden infant death · Natural childhood death

Introduction

Pediatric (childhood) forensic pathology encompasses a spectrum of case types, from discarded fetuses/infants, through birth-related deaths, sudden infant death syndrome (SIDS) and similar deaths, natural childhood deaths, to traumatic childhood deaths, including deaths from accidental trauma, deaths from homicidal trauma, and even deaths from suicidal trauma. Childhood deaths include neonatal deaths, deaths in infancy, deaths of young children, and deaths of older children, including adolescents. The upper age of “childhood” or adolescence varies, depending on the researcher. Different manners of death tend to affect different ages. This chapter will be divided into several sections, based on type of death or age of death. It is important to remember that children, particularly infants and young children, do not simply represent “little adults.” There are a variety of significant anatomic, developmental, behavioral, and physiologic differences between children and adults. These differences can make children more susceptible to certain stressors. At the

same time, these differences can make the recognition of certain types of deaths much more difficult in children as compared to the adult population.

Discarded Fetuses/Infants and Fetal/Infant Deaths in Unattended Births

One of the most challenging types of case for a forensic pathologist is that of a dead newborn fetus/infant where the birth was not attended by medical personnel or witnessed by other individuals. Depending on the circumstances, the body may or may not be decomposing. A variety of scenarios occur. The fetus/infant has frequently been discarded or otherwise hidden, indoors or outdoors (Fig. 20.1 and Disc Image 20.1). In many cases where the infant has been discarded, the identity of the mother is not known. Decomposition is frequent in these cases. Another relatively frequent scenario involves a teenage mother who has concealed her pregnancy and then gives birth. She or another family member eventually reports the delivery, sometimes after many days (with associated decomposition), but whether or not the fetus/infant was born alive is in question.

Fig. 20.1 A discarded newborn found in a garbage bag



In these types of cases, there are typically several questions that forensic pathologists attempt to answer. First, who is the mother? Second, was the baby of sufficient gestational age to survive outside of the womb (in other words, was it “viable”)? Third, was the baby born alive, was it a stillbirth, or did the baby die during delivery? Fourth, what was the cause of death?

Who is the Mother?

The identity of the mother is sometimes obvious; however, in cases where the fetus/infant has been discarded away from anything that might associate the child with a mother, mother identification may prove to be quite challenging. A thorough investigation might result in finding a possible mother. DNA testing can provide a conclusive answer to the identification question, but only if a presumptive mother can be identified and legal channels are available for obtaining a DNA sample from her.

Was the Fetus/Infant Viable?

By weighing the infant and measuring the height (crown–heel length), various other measurements (crown–rump length, head circumference, chest circumference) and the foot length (the best measurement), an approximation of the gestational age can be made. Microscopic examination of various organs can also assist a pathologist in estimating the gestational age of a fetus/infant, using tables from pediatric pathology textbooks. In general, fetuses having a gestational age of around 22–26 weeks are considered potentially viable, although survival usually requires medical support. Survival of younger infants has been reported. In general, the greater the gestational age, the more likely the child is able to survive extra-uterine (outside of the womb) life.

Was the Fetus/Infant Live-Born or Stillborn?

The question as to whether or not a fetus/infant was born alive is the issue that makes these types of deaths quite difficult for forensic pathologists, particularly so in the setting of decomposition. When decomposition is *not* present, a variety of features can be useful in attempting to answer this question. As mentioned above, there are essentially three possibilities: (1) the baby died in utero (it was a stillbirth); (2) the baby was born alive; (3) the baby died during the birth process. Unfortunately, it is frequently not possible to differentiate between these possibilities.

Stillbirths

The term “maceration” refers to the in-utero postmortem breakdown of the fetus. It may be thought of as in-utero decomposition, but since the uterus is typically a sterile (germ-free) environment, the process typically occurs without micro-organism putrefaction, so in a strict sense, the term “decomposition” is not synonymous with “maceration.” The presence of maceration in combination with a lack of lung aeration (air within the lungs) are the two features that are most frequently used to identify a dead infant as a stillbirth. The earliest changes of maceration include dark discoloration of the normally yellow-tan umbilical cord (Disc Images 20.2

and 20.3), and skin slippage, both of which may develop within several hours of death in utero (Fig. 20.2). However, the absence of these changes does not necessarily mean that the child was born alive (Disc Image 20.4). Eventually, there are extensive, multifocal areas of skin slippage, along with skull plate hypermobility, and internal changes, including the presence of decomposition-like fluid in the body cavities, and liquefaction of the brain. Maceration indicates that the death occurred in utero. Unfortunately, decomposition can develop in dead infants, whether they were live-born or stillborn. Therefore, decomposition occurring after birth can make the identification of pre-birth maceration impossible.

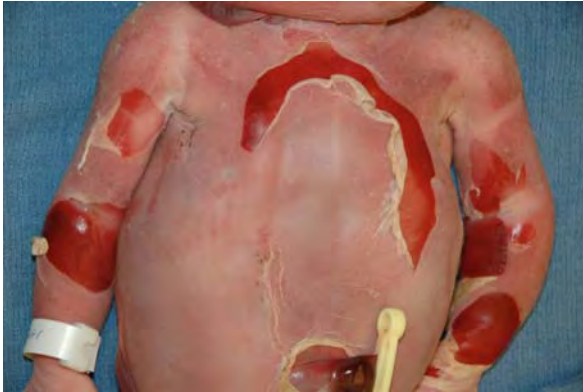


Fig. 20.2 Fetal maceration characterized by skin discoloration and slippage. Note also the presence of a darkened umbilical cord stump. This case was a hospital stillbirth in which the mother and physician knew that the fetus was dead prior to delivery

Aeration of the lungs is frequently used as an indication that breathing has occurred, thus indicating live birth. In macerated cases with no overlying decomposition, the lungs will demonstrate what is referred to as “primary atelectasis” (“atelectasis” refers to collapse of the lung; “primary” refers to the fact that they were never filled with air). Grossly, the lungs appear collapsed within the chest cavities. Microscopically, the alveoli (air sacs) are not expanded, and the lungs will not float in water (this is sometimes referred to as the “flotation test” – see below). Decomposition makes evaluation for lung aeration difficult at best, since with decomposition, putrefaction can result in extensive production of gas, such that the lungs will float. In decomposed cases, the lungs will frequently float in water, but in some instances so can certain solid organs, such as the liver.

Live Births

In newborn infants who die after live birth, there will be no maceration. Another finding that is frequently (but not necessarily always) present is lung aeration; when an infant breathes following birth, the lungs expand with air. This is readily visible grossly and microscopically at autopsy. In the flotation test, the lungs (or portions thereof) float and the liver (and other organs, or portions thereof) sinks (Disc

Image 20.5). Air may also be present within the gastrointestinal tract. Occasionally, a baby may be born alive (with heart beating), but fail to breath, due to mucus obstruction of airway, poor tone, or other circumstances. These cases are indistinguishable from intrapartum death cases, as they will have no lung aeration and no maceration. As previously described, decomposition can create difficulties in interpretation: decomposed lungs will float in water. Another situation where lungs may be artifactually filled with air involves rescue breathing. In other words, if resuscitation is attempted on a stillborn fetus, air can be present within the lungs at autopsy.

A few other findings have been reported as being useful in determining that a baby was born alive, although each is uncommon. If acute inflammatory cells are present on the fetal/infant side of the umbilical cord transection site, but not on the placental side, then this finding indicates that there was a live birth. If there is food within the gastrointestinal tract, then this indicates that the infant was fed prior to death, and thus was a live birth. It is important to note that newborn (live and stillborn) infants may have a small amount of mucus within their stomachs.

Intrapartum Deaths

The third possibility for deaths occurring in children whose births are unattended is that the fetus/infant died during the birth process. The findings which suggest that a death is an intrapartum death are lack of maceration and lack of lung aeration. However, in most instances, there is no possible way by autopsy findings alone to differentiate these cases from deaths that occur in-utero prior to birth but have not yet developed maceration or from deaths that occur after birth where there is little or no aeration of the lungs. In addition, when a precipitous (very fast) birth occurs, it has been shown that air can enter the lungs, via the chest compression followed by rapid chest expansion that occurs during passage through the birth canal, even if the infant does not actively inhale. This artifactual aeration of the lungs is said to typically involve the upper parts of the lungs, while active inspiration tends to first involve the lower parts of the lungs (because the diaphragm creates the forces that allow inhalation).

What Was the Cause of Death?

The cause of death in neonatal deaths where the birth was not attended by medical personnel or witnessed by others is quite varied and includes many of the causes and mechanisms discussed below. The cause of death may be related to pregnancy problems, including maternal/placental diseases, birth trauma, natural disease processes, or exogenous trauma, either accidental or homicidal. Particularly in cases where some type of pregnancy-related problem is suspected (but actually in *every* neonatal death), it is important to obtain the placenta for gross and microscopic examination, as such an examination can provide very important information regarding the cause of death. Ultimately, if a definitive cause of death cannot be determined

for a stillborn, the phrase “intrauterine fetal demise of undetermined etiology” is appropriate.

Birth-Related Infant Deaths

Infant deaths related to the birth process include some that are specifically related to maternal-placental pregnancy issues, those that are related to “natural” complications of the birth process, and deaths that are “iatrogenic” (due to complications of medical therapy). In all birth-related fetal/infant deaths, it is important to obtain all birth records, and consider conducting interviews with those in attendance at the birth. In addition, it is important for the placenta to be collected and examined, both grossly and microscopically. The following paragraphs will present birth-related infant deaths based on two basic categories: asphyxia and trauma. In each, various “endogenous” (or natural) and “exogenous” (or non-natural) causes can be responsible for the asphyxia or trauma.

The term “asphyxia” as used in relation to fetal/infant deaths refers to the general lack of oxygenation of the fetus/infant. Unlike in many other instances when the term “asphyxia” is used within the forensic pathology community, birth-related asphyxia does not necessarily imply a non-natural underlying cause. In other words, there are a variety of natural diseases or conditions that can result in insufficient oxygenation of a fetus. When asphyxia occurs in a fetus during labor/birth, a common occurrence is that the fetus defecates within the bag of waters. Meconium (the baby’s first bowel movement) present within the amniotic fluid, and possibly staining the child’s skin, can be considered an indicator that asphyxia or some other stress occurred. An example of birth asphyxia is the insufficient transfer of oxygen from the mother to the placenta, and thus to the fetus, as a result of disease processes that specifically affect the placenta (for example, pre-eclampsia/eclampsia and placenta abruption (premature separation of the placenta from the uterus)). Another endogenous cause of birth asphyxia is prolonged labor secondary to “cephalopelvic disproportion” (CPD), in which the infant’s head is relatively large (and the mother’s pelvis is relatively small) such that the baby cannot easily move through the birth canal. Compression of the umbilical cord, via a variety of causes, including trapping the cord between the infant and the pelvis, can lead to a lack of blood flow to the infant. Another cord-related process involves the wrapping of the umbilical cord around a vital infant structure, such as the neck (referred to as a “nuchal cord”) (Disc Image 20.6). If severe enough, this can lead to a more typical traumatic type of asphyxial death (strangulation), although the underlying process is endogenous, meaning that it is unrelated to an exogenous event or intervention.

A number of exogenous causes of birth-associated asphyxia can also occur. These may or may not be related to medical intervention. Non-medical exogenous causes include traumatic injuries of the mother, with secondary lack of oxygenation of the placenta and fetus. Occasionally, maternal trauma can result in traumatic placental

abruption or even uterine rupture. When an exogenous event is related to medical therapy, the term “iatrogenic” is sometimes used. It is beyond the scope of this text to provide detailed commentary about these sometimes controversial issues. The reader is asked to recognize that debate exists with regard to the value of various medical interventions regarding management of labor and birth. These range from issues as seemingly basic as the body positioning of the mother, to the use of medications (such as epidurals), to the use of various maneuvers, instrumentation, and surgical interventions. Various studies exist which clearly document that certain medical interventions can result in prolongation of the labor and birth process. Theoretically, prolongation of labor by medical means or drug-induced fetal effects might contribute to birth asphyxia.

The other category of birth-related deaths is related to traumatic fetal/infant injuries or other (placenta or fetal membrane) injuries. As with birth asphyxia, there can be both endogenous (natural) and exogenous causes for these traumatic injuries. Premature rupture of the membranes (PROM) refers to a situation where the fetal membranes (bag of waters) breaks an extended amount of time before the actual birth. When PROM occurs, the mother and fetus are at risk for infection. Such infection can lead to fetal death or to various other complications that can ultimately lead to the infant’s death. PROM can occur following spontaneous rupture of the membranes (an endogenous cause) or on purpose, following artificial rupture of the membranes by the obstetrician (an exogenous cause).

Another example of an endogenous traumatic cause of fetal death is referred to as “occipital osteodiastasis,” which is defined as a birth-related injury in which the relatively mobile occipital bone (at the back, base of the skull), under forces related to passing through the birth canal, is forced forward, causing injury (and sometimes transection) of the brainstem. This entity can be difficult to recognize. Lateral (from the side) X-rays of the head can help to identify this injury.

Scalp injuries are relatively common during the birth process. There are various names for these, depending on the exact location of the injury within the scalp and skull. A common type is a “cephalohematoma;” another example is a “caput succedaneum.” Both are considered minor. Occasionally, a skull fracture can occur. Small, incidental subdural hemorrhages may also occur. In rare cases of severe CPD, emergency attempts at removing the “stuck” fetus, using forceps or other instruments, with or without manual manipulation, can result in cervical vertebral column dislocation, or similar skeletal disruption, with associated lethal upper spinal cord or brainstem injury. This is an example of an exogenous, iatrogenic traumatic birth-related death.

An example of an exogenous, trauma-related birth related death that is not iatrogenic would be the case of a mother who is the victim of an assault, which results in premature labor, delivery of an extremely premature infant who survives a few hours, but then dies because of extreme prematurity. Such a case could be considered a homicide, and criminal charges could well be pursued. In similar cases where the fetus dies in-utero, whether or not criminal charges are able to be pursued depends on local statutes.

Infant Deaths

For the purposes of this chapter, an “infant death” is defined as a death occurring within the first year of life. This definition includes cases that occur during the “neonatal period,” which, by definition, is within the first 28 days of life. The deaths occurring in newborns that were described in the preceding section would be considered infant deaths as well as neonatal deaths.

A variety of natural causes of death may occur during infancy. With many of these, the child is hospitalized prior to death, and a diagnosis has been rendered prior to death. As such, these cases tend not to be referred to the local medicolegal death investigation agency. Occasionally, an infant experiences a sudden illness, with subsequent medical evaluation, but death occurs prior to a diagnosis being made. These cases are appropriately referred to the medical examiner or coroner. Likewise, certain sudden, unexpected deaths (where death occurs without any medical intervention) result from underlying natural disease. Some of these natural disease processes are relatively specific for infancy, while others (described below under “Natural Death in Childhood”) can occur in infancy or at other times during childhood.

Natural disease processes that can cause death and are more-or-less specific to infancy include various congenital abnormalities or anomalies. In general, these may or may not be associated with symptoms. Various congenital anomalies of the kidneys/urinary tract, lungs, or diaphragm, can result in “pulmonary hypoplasia” (“hypoplasia” means that something does not grow enough; there is “under-growth”) wherein the lungs do not develop sufficiently in utero. The fetus tends to do relatively well in utero in these cases, since the placenta functions to oxygenate the fetus. Once birth occurs, if the lungs are extremely “hypoplastic,” the newborn will not do well, and will frequently die. Certain kidney and urinary tract abnormalities, including multicystic renal dysplasia, and anything that can result in urinary outflow obstruction, can result in inadequate fetal urine production, and thus insufficient amniotic fluid production (“oligohydramnios”), and subsequent pulmonary hypoplasia. A congenital diaphragmatic hernia can also cause pulmonary hypoplasia (because abdominal organs protrude through the defect in the diaphragm and prevent lung growth), with severe respiratory distress after birth, and subsequent neonatal death (Fig. 20.3).

Heart (cardiac) anomalies can also result in death occurring soon after birth. It is beyond the scope of this text to describe all such conditions, although several are described briefly in Chapter 10. Suffice it to say that there are many “congenital heart anomalies” that can cause infant death. In a like manner, there are a variety of congenital anomalies in virtually every other organ system that can result in death of an infant. Other natural diseases in childhood that can result in death are described in the section entitled “Natural Childhood Deaths.”

The general term “SIDS-like” is sometimes used to describe infant deaths where there are no or minimal injuries evident on the external aspect of the body, and initial scene investigation fails to reveal an obvious cause of death. A term that is being used more frequently to describe such deaths is “sudden unexplained infant death”

Fig. 20.3 A diaphragmatic hernia in a newborn infant who died shortly after birth. Note that the left lobe of the liver is within the left chest cavity (*arrow*), as a result of the absence of a majority of the left side of the diaphragm. The right side of the diaphragm is indicated by the arrowhead



or SUID. In every such case, a detailed scene investigation is of utmost importance (Fig. 20.4). Frequently, the baby has been transported by emergency personnel to the hospital, so that the body is no longer at the scene. A scene investigation must be performed in these cases as well. One of the best tools to use in such cases is a doll re-enactment, where the investigator asks the person who found the infant to place the doll in the exact position where the infant was found. Photographs can then be taken (Fig. 20.5). An infant death investigation form (Sudden Unexplained Infant Death Investigation or SUIDI) is also a useful tool in the investigation in any infant death.

The term “sudden infant death syndrome” (SIDS) is a term traditionally used to describe the death of an infant less than one year old where a complete



Fig. 20.4 A scene investigation must be performed in every childhood death



Fig. 20.5 If the child's body is no longer at the scene, a doll re-enactment should be performed, with photographic documentation

autopsy, including a scene investigation, complete external and internal autopsy examinations, X-ray examination and toxicology testing, fails to reveal a definite cause of death. In general, it is believed that a variety of underlying natural disease processes or predilections are the underlying conditions responsible for many SIDS deaths; however, it is also believed that many SIDS cases may represent unrecognized accidental asphyxial deaths, or hyperthermia (overheating), or perhaps even allergic reactions. Since there is no recognizable cause of death identified in these cases, the term "SIDS" is actually a technical way of saying that the cause of death is undetermined. As mentioned previously, some forensic pathologists have moved away from using "SIDS" for death certification. Instead, the term "sudden unexplained infant death" is utilized. There are no specific autopsy findings in SIDS cases. Frequently, there are petechial hemorrhages on the organs of the thorax, especially the thymus (Fig. 20.6), although this finding does not have to be present, and it can certainly occur in non-SIDS deaths.

The classic SIDS case involves an infant found dead in his or her crib. As described below, this classic scenario is less common than it used to be. Statistical studies have shown that infants are more likely to die in their sleep if they lie on their stomachs (the "prone" position). The exact physiologic reason for this is not known, although some suggest that it involves an asphyxial component. Others suggest an element of hyperthermia. Whatever the underlying explanation, it is currently recommended that infants be placed on their backs (the "supine" position). With various issues of concern involving accidental asphyxia and/or hyperthermia, different forensic pathologists may differ on how to interpret the findings in a particular case. In other words, some pathologists might call a death where the infant is stomach-down with lots of blankets a SIDS death, whereas another pathologist might call this an undetermined death or even an accidental asphyxia death. For pathologists who subscribe to the "SUID" terminology, some will also list (either in part II of the cause of death section, or elsewhere on the death certificate) any potential "risk factors" that are identified. In the example just provided, "prone

Fig. 20.6 The presence of thymic and other thoracic petechiae are common in SIDS-like cases. Note the numerous petechiae on the thymus. Some larger areas of hemorrhage are evident on the right lung (lower left of photograph). The heart (pericardial) sac has not yet been opened



sleeping position” and “abundant bedding” could be listed as risk factors. Further discussion of accidental asphyxial deaths and accidental hyperthermia deaths occur in the following paragraphs.

In recent years, more attention has been paid to the exact nature of infants’ sleeping positions and other potential risk factors in cases of infant death. As indicated above, the trend within the forensic pathology community is to attempt to identify various risk factors which may be associated with an increased risk of sudden infant death. Listing the cause of death as “sudden unexplained infant death,” including a list of potential risk factors (such as prone sleeping position, abundant bedding, bedsharing with others, formula-feeding, cigarette smokers in house, etc.), and certifying the manner of death as “undetermined” is one method of approaching these cases. If one of the “risk factors” is of sufficient severity or so obvious that it becomes more than simply a risk factor, then it is appropriate to implicate that factor as being instrumental in the cause of death. This situation is of particular importance in possible asphyxial deaths.

Many believe that if an infant’s face (mouth and nose) is pressed against a very soft, flexible surface, such as a pillow, abundant bedding material, or a stuffed animal, this represents a potentially dangerous sleeping position, and the cause of death is best called “accidental asphyxia,” or some other similar descriptor. These types of potentially dangerous items may be present within a crib or in any other location. Sleeping environments outside of a crib are also frequently considered dangerous for infants. This is particularly true regarding sofas. The back corner of a sofa, where the seat cushions and back cushions meet, is a location that should be considered extremely dangerous for infants. In cases of infant death that occur via some type of external airway compromise, there are typically no external or internal injuries identified. Lividity patterns may be useful in ascertaining the exact location of the infant (Fig. 20.7).



Fig. 20.7 The lividity pattern in this case clearly indicates that the infant was face-down, with the mouth and nose potentially obstructed

There are also certain other situations where accidental asphyxia is the obvious cause of death. If an adult, or older child, or even a part (leg, arm, etc.) lays on top of an infant, an asphyxial death can occur. These cases are sometimes referred to as “overlay” deaths. The lividity pattern may provide clues in these cases. There may or may not be petechiae on the skin and conjunctiva. If an infant becomes wedged between a mattress and a wall, or between a too-small mattress and the crib frame, an asphyxial death can result. These cases are sometimes referred to as “wedging” deaths. As above, lividity patterns may be useful in recreating the scene, and petechiae may or may not be evident in these cases. Entrapment of the infant’s neck or chest or abdomen in the crib framing or any other location can result in hanging or strangulation or positional asphyxia. Facial and conjunctival petechiae may be present in these cases. Obstruction of the face (nose and mouth) with plastic or other non-porous objects can result in smothering. Typically, petechiae are not evident in these cases.

In certain asphyxial deaths, microscopic examination of the lungs may disclose evidence of “hyper-inflated” or “hyper-expanded” alveolar spaces; however, this finding is not specific for an asphyxial event, since such findings may be induced by resuscitative efforts (CPR, rescue breathing). The presence of blood within alveoli may similarly be explained by an asphyxial mechanism versus resuscitation attempts. Evidence of hemosiderin (a breakdown product of the hemoglobin contained in red blood cells) suggests the possibility of prior episodes of asphyxia.

“Co-sleeping” is a term used to describe a situation where an infant sleeps in close proximity to another person. This may be an adult, such as a parent, or a sibling, or multiple persons. In fact, if an infant sleeps in a crib or bassinette next to their parents’ bed, this is also considered a form of co-sleeping. Studies have shown that co-sleeping is beneficial to infants, and may even be protective against SIDS. Bed-sharing is a sub-type of co-sleeping in which the infant actually shares a bed with someone. Bed-sharing remains a controversial topic amongst pediatricians, forensic

pathologists, and others. It should be noted that there are two basic populations who participate in infant bed-sharing.

Bed-sharing with an infant is particularly common amongst mothers who are breastfeeding (Disc Image 20.7). Generally, within this population, bed-sharing can be accomplished in a very safe manner, and many argue that the benefits can far outweigh the risks. In addition, many of the risks can be minimized by using various specially-created products (firm, angled pillows to place between parent and infant) and by using common-sense (using a very firm mattress, avoiding abundant bedding, remaining cognizant of the fact that the infant is in the bed with the parent, etc.). Breastfeeding mothers tend to be very “in tune” with their infants, and the bonding and other benefits of breastfeeding can be extremely worthwhile for both mother and child. While the risk of overlay, wedging, and other forms of accidental asphyxia are not totally excluded in this population, they are very much reduced compared to the second population of mothers who bed-share with their infants. If breastfeeding parents are over-tired (more than usual) or under the influence of medication or drugs, it is advisable to avoid bed-sharing. Obese parents may also consider avoiding this type of sleeping arrangement.

The other population of mothers who bed-share with their infants are typically not breast-feeding, and they frequently bed-share because it is simply easier for them to bring the infant to bed with them than to place their infant safely in a crib. Some within this population do not even own a crib, but for those who do, it remains easier for them to bring the infant into bed with them (and anyone else who happens to be there). Various subsets of these individuals may have additional risk factors, such as frequently being intoxicated, being over-tired, being obese, or being in the habit of having other children or adults in bed with them. Each of these factors can substantially increase the risk of accidental infant asphyxia. For this reason, it is generally not advisable for non-breastfeeding mothers to bed-share with their infants.

Natural Death in Childhood

Numerous childhood natural diseases are known to be able to cause death. It is beyond the scope of this text to provide a detailed discussion of all of these entities. A general synopsis of some diseases will be provided.

Children are considered somewhat resistant to many adult disease types, including many diseases that are considered diseases related to aging. Unfortunately, children are not immune from cancer. The cancer types are different than many adult cancer types, but virtually every organ system of the body can be affected by a childhood cancer. The most frequent types of cancer are leukemias (blood cancers), brain (central nervous system) cancers, bone cancers, and endocrine cancers. Certain cancers, while not particularly common, are classically described in children. Some of these include Wilm’s tumor (kidney), hepatoblastoma (liver), and retinoblastoma (eye).

Children are commonly afflicted with various infectious diseases, including many viral diseases. In most instances, when the child is otherwise healthy, the child's immune systems fend off the infectious organisms, and they recover just fine. Unfortunately, this is not always the case. Some children have an inherited "immune deficiency" disease that makes them more susceptible to infection. In others, an infectious disease can occasionally lead to severe illness and death. A particularly serious example involves the Gram-negative bacteria *Neisseria meningitides*, which can cause bacterial meningitis, as well as adrenal gland hemorrhage and a diffuse, hemorrhagic rash (this constellation of findings is called the Waterhouse–Friderichsen syndrome). Many other bacteria, viruses, fungi, and other micro-organisms have been known to cause death in children.

A few developmental/congenital disorders were described previously in the "Infant Death" section above, but there are numerous others that have been described as causing childhood deaths. Examples include many that have already been presented in Chapter 10 (Natural Deaths), such as coronary artery anomalies, hypertrophic cardiomyopathy, and arrhythmogenic right ventricular dysplasia (cardiomyopathy). Other examples include cerebral palsy, where various physical and developmental abnormalities can result in complications that lead to death, spina bifida, which can result in numerous infectious and other complications, and various forms of mental retardation, which can contribute to death in a variety of settings, including via a seizure disorder. Seizure disorders in children as well as adults are associated with an increased incidence of sudden death.

Some other diseases that classically manifest during childhood and can cause a significant amount of disability and possibly death include asthma, autism, juvenile diabetes mellitus, cystic fibrosis, sickle cell disease, and a multitude of "inborn errors of metabolism." Several of these are also discussed in Chapter 10.

Two eating disorders that are particularly common in teenage girls are anorexia nervosa and bulimia. These disorders are discussed further in Chapter 21 (Miscellaneous Topics).

Accidental Childhood Deaths

Accidental death in childhood represents a diverse group of entities that tend to vary depending on the age of the individual. The following paragraphs provide descriptions based on age, beginning with infants and ending with adolescents.

Infants – Most forms of infant accidental asphyxial death were addressed previously in the section entitled "Infant Deaths." Drowning deaths are particularly common in infants, with many occurring in infants left unattended in bathtubs. Parents should be cautioned against leaving infants unattended in bathtubs, even if it is for only a few seconds. Other causes of accidental infant death include virtually any other mechanism of injury, including blunt force, sharp force, temperature-related, electrical, and even gunshot wounds.

Toddlers – Like infants, toddlers are relatively frequently involved in drowning mishaps, although with toddlers, other bodies of water, such as swimming pools,

ponds, creeks, rivers, and lakes, are more common than in infancy. Accidental asphyxial deaths also occur in this age-range, with neck compression by entanglement by some sort of ligature (such as a child-restraint seatbelt or a window blind cord) being responsible for many such deaths (Fig. 20.8), and choking on food or other foreign objects occurring in others (Disc Image 20.8). Compared to other childhood age ranges, accidental poisoning deaths are much more frequent in toddlers. Deaths related to blunt force, sharp force, temperature, etc. also occur in this age range (Disc Image 20.9). Accidental deaths from falls can occur. Accidental blunt injuries tend to be on overlying bony prominences (the parts of the body most susceptible to injury, because they “stick out” more than other, more recessed parts).

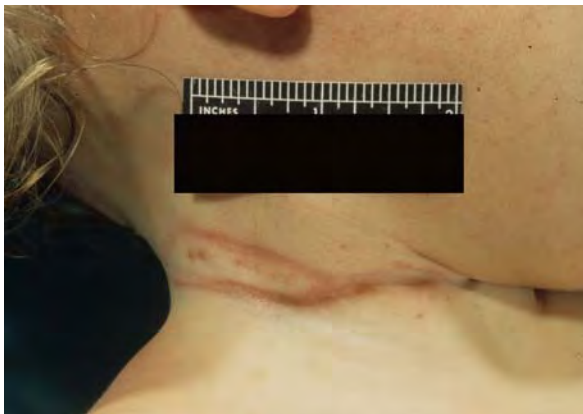


Fig. 20.8 A toddler who was found hanging by a mini-blind cord

Older Children – School-age childhood accidental deaths include drownings and other asphyxial mechanisms, blunt force injuries from bicycle, skateboarding, rollerblading and other accidents, and a wide range of other childhood mishaps. As children approach their teenage years, a variety of risky behaviors tend to develop. Accidental deaths related to recreational drug abuse are not unheard of in this age range. The “choking game” (known by numerous other names; see Chapter 15) typically involves some sort of neck compression, in order to induce “blacking out,” and the sensations associated with that experience. This activity is distinct from autoerotic activity. Many cases of accidental death, typically related to a hanging mechanism, have been reported with the choking game.

Adolescents – Adolescent accidental deaths occur in a variety of settings. Sports-related deaths are rare, but occur with increasing frequency as children become involved with more physically demanding and competitive sporting events. Concussio cordis (a fatal cardiac arrhythmia induced by a sudden blow to the chest) can occur in a variety of athletic events, including baseball, football, and hockey. Severe head injuries can occur in many sports. Accidental asphyxial deaths include deaths related to various forms of the choking game, as well as autoerotic asphyxia.

Accidental drug-related deaths become more frequent in this age range, and as teenagers become old enough to drive, there is a significant increase in accidental deaths related to motor vehicle collisions. A significant number of cases involve the use of ethanol. Although many forensic pathologists prefer to rule the manner of death as suicidal in cases of Russian roulette, some will rule these deaths as accidental.

Suicidal Childhood Deaths

Suicides in children tend to occur in older age ranges, particularly in teenagers; however, suicides have been reported in children as young as nine years. As with suicides occurring in other age groups, the investigation surrounding the death is frequently very important in making the MOD determination. Interviews with family members and friends, classmates, and acquaintances may reveal extremely useful information. Recent suicidal thoughts and/or gestures provide a certain amount of credible evidence for a suicide ruling. “Copycat” suicides may occur.

Common causes of death in childhood suicides include drug or toxin overdose, hanging, and gunshot wounds. Others, such as sharp force injury, various forms of blunt force injury (jumping from a height, intentional motor vehicle collision, etc.), other forms of asphyxia, including drowning, electrocution, and self-immolation (setting oneself on fire), may all occur.

As detailed above, care must be taken when the COD involves neck compression, as many such deaths actually represent accidental deaths related to the “choking game” or autoerotic asphyxia.

Many forensic pathologists consider self-inflicted gunshot wounds occurring in the setting of Russian roulette as suicidal. The argument is that the activity is considered extremely dangerous and the person engaging in the activity presumably recognizes this fact. Knowing the inherent dangers of the activity, the person performs a deliberate act (pulling the trigger) that may result in death. As such, the argument goes, the act can be considered an intentional act to harm oneself; therefore, suicide is a reasonable MOD ruling. Others argue that, like drinking and driving, Russian roulette is certainly risky, but it is not absolutely always lethal; therefore, the proper MOD ruling is accident.

Homicidal Childhood Deaths

Remembering that childhood extends through, or at least well into, the teenage years, depending on one’s definition, it is not too surprising that homicide is one of the leading causes of childhood death. A vast majority of these cases occur in older children and are related to issues that are responsible for many of the homicides that occur in young adults. Included here are domestic and intoxication issues, as well as various illegal activities, most notably the drug culture. Various mechanisms are involved in drug-related childhood homicides, and they essentially parallel those

occurring in adults. The reader is referred to various other sections of this text for more details regarding various specific causes of death, such as gunshot wounds. A majority of the remainder of this chapter will deal specifically with “child abuse” homicides.

The reader should be reminded that detailed investigation is of vital importance in all cases suspected of being homicides. It cannot be stressed strongly enough that the mere presence of lethal injuries in a child does not automatically mean that the injuries were intentionally inflicted. Certainly, there are various patterns or stories that “raise a red flag;” but every case must be evaluated on its own merits, taking into consideration all investigational information. Examples of “red flags” that should immediately raise suspicion include: injuries that are “out of proportion” for the explanation being offered, descriptions of how injuries were sustained that are impossible or improbable (for example, a one-month old is said to have climbed into the bathtub), changing stories by the care provider, the absence of any type of credible explanation for the severe injuries that exist, the presence of excessive injuries or certain unusual injuries (see below), and the presence of severe injuries in various stages of healing.

There are various terms used to describe childhood homicides. The term “neonaticide” refers to the killing of a newborn baby. The typical perpetrator is the mother. The term “infanticide” refers to the killing of an infant. The killer is typically a parent, a care provider, or a friend of the parent (frequently the boyfriend of the mother). The term “filicide” means the killing of a child by his or her parent.

A relatively common injury type in child abuse homicides is blunt force trauma. Particularly common is head/brain injury. If there is no associated skull fracture, the term “closed head injury” is sometimes used to describe these injuries. If a skull fracture is also present, the term “craniocerebral” (skull and brain) trauma may be used. On external examination, there may or may not be evidence of injury (abrasions, contusions, lacerations) (Fig. 20.9). Sometimes the hair conceals



Fig. 20.9 Blunt force head injuries in a child abuse victim

subtle scalp injuries. When the scalp is reflected during autopsy, contusions will be evident (Fig. 20.10 and Disc Image 20.10). Occasionally, there is no or very little external evidence of injury even in the presence of extensive subscalpular trauma (Disc Images 20.11 and 20.12). A subscalpular hemorrhage that is not associated with an underlying skull fracture is evidence that a scalp impact has occurred, even in the absence of skin findings. Skull fractures may or may not be present (Fig. 20.11 and Disc Image 20.13). Hemorrhages around the brain, particularly subdural (Fig. 20.12) and subarachnoid (Fig. 20.13) hemorrhages, are typically present, although their size is not always large. Depending on the survival time following injury infliction, there may be significant brain swelling (Disc Image 20.14). Brain contusions, lacerations and/or other hemorrhages may be present. In infants, especially young infants, brain swelling from severe head trauma may be of such an extent that the skull's bones separate from one another, with marked widening of



Fig. 20.10 Subscalpular hemorrhage related to blunt force head trauma

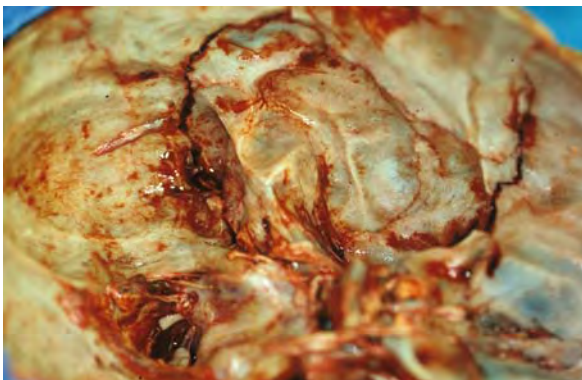


Fig. 20.11 Multiple skull fractures

Fig. 20.12 The presence of relatively extensive subdural hemorrhage on the left side. Note also the presence of relative scanty subarachnoid hemorrhage overlying the brain on the left

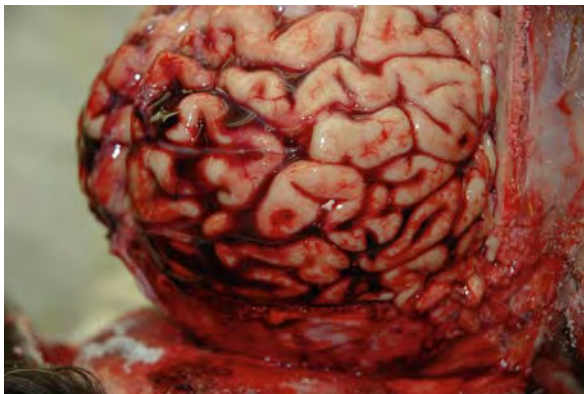


Fig. 20.13 Subarachnoid hemorrhage overlying the posterior (back) portion of the brain

the “sutures” (where the bones join one another). Such fractures are referred to as “diastatic fractures” (Disc Image 20.15). If survival following injury infliction exceeds several hours, microscopic identification of “diffuse axonal injury” within the brain may be possible (Disc Images 20.16 and 20.17).

The presence of subarachnoid hemorrhage (SAH) and subdural hemorrhage (SDH) indicate that severe brain injury has probably occurred, even if no gross or microscopic brain injuries are evident. The lack of identifiable brain injuries is explained by the relatively quick death; as indicated above, it takes time for the brain injuries to become identifiable. Because of this, SAH and SDH can be considered “markers” for underlying lethal brain trauma. Occasionally, massive cerebral

edema (brain swelling) may be the sole intracranial (inside the skull) finding with lethal brain injury.

Bleeding around the optic nerves (perioptic nerve hemorrhage) (Disc Image 20.18) and retinal hemorrhages are commonly encountered (Fig. 20.14 and Disc Images 20.19 and 20.20) in head/brain trauma cases. Particularly with regard to retinal hemorrhages, it must be emphasized that the mere presence of retinal hemorrhages does not indicate traumatic injury. There are numerous non-traumatic causes of retinal hemorrhages. It should also be reiterated here that, while the combined findings of scalp impact sites, subarachnoid and subdural hemorrhage (often with retinal hemorrhages) are sufficient to determine that the cause of death is related to head/brain injury, it is not true that such findings are diagnostic of *inflicted* head trauma (sometimes referred to as “non-accidental” injury). Accidental head/brain trauma can produce similar findings. As indicated above, each case must be evaluated on its own merits.

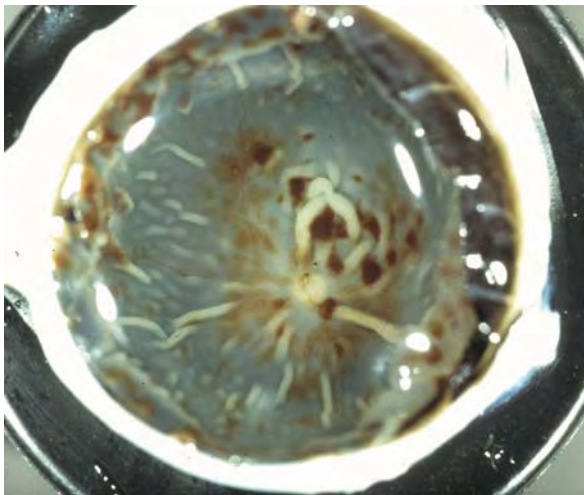


Fig. 20.14 The front (anterior) portion of this formalin-fixed eye has been removed in order to visualize the back inner surface (the retina). Note the presence of numerous dark “spots” on the lighter-colored retina. The spots represent retinal hemorrhages

In many cases of lethal head/brain injuries in children, especially those involving infants, a suggestion of “shaken baby syndrome” may occur. This may originate from the police, the parents or care providers, or medical personnel. In fact, the discovery of retinal hemorrhages along with brain injuries and subarachnoid and/or subdural hemorrhage within an injured infant at the hospital is considered by many physicians as being diagnostic of the “shaken baby syndrome.” At one time, the presence of retinal hemorrhages by themselves was considered a suspicious finding. As mentioned above, retinal hemorrhages are *NOT* diagnostic of shaking, head/brain injury, or abuse. Also, even if a perpetrator admits to having shaken an infant, it does not mean that the entire injury complex is related to shaking. In point of fact,

in a majority of cases with a clinical diagnosis of “shaken baby syndrome” that eventually come to autopsy, there is definite evidence of scalp impact (subscalpular contusions); however, there are occasional cases where there is no external or subscalpular evidence of impact (Disc Image 20.21). The presence of scalp impact sites suggests that blunt force impact caused or contributed to the brain injuries. Some experts are quite vocal in their argument that lethal infant brain injuries cannot occur by shaking alone. Many others maintain more of an open mind about the possibility, recognizing that “pure shaking” cases are rare, at best. If one considers upper spinal cord trauma as a potential mechanism of death in pure shaking cases, then most agree that such cases could occur. Despite the debate amongst experts with regard to shaking, the fact remains that a vast majority of supposed shaken baby cases have autopsy evidence of scalp impact. As such, some prefer to use the term “shaken impact syndrome.” The term “shaken baby syndrome” is used infrequently, if at all, by many forensic pathologists.

Other child abuse homicide deaths are related to blunt force injuries sustained below the head. Abdominal trauma is particularly common, whether inflicted by the blow from a fist, a stomp from a foot, or squeezing of the fingers while in the grasp of an adult. Organ and/or tissue lacerations, with associated hemorrhage, are most common. The liver, the intestines, and the mesentery of the intestines are particularly prone to lacerations (Fig. 20.15), although virtually any organ/tissue may be injured (Fig. 20.16 and Disc Images 20.22, 20.23, and 20.24). Occasionally, deaths are delayed and due to peritonitis following rupture of the intestine.

Other blunt force injuries that should be considered suspicious include any external injury (abrasion, laceration, contusion) that is in a location not normally injured during accidental trauma or normal childhood activities. Injuries of the inner, recessed aspects of the external ear represent such injuries (Fig. 20.17). Lacerations of the frenulum (inside of upper or lower lip, in the midline) can occur accidentally, but there is usually a history of a definite event that explains the injury



Fig. 20.15 A section of small intestine demonstrating a relatively large tear (or laceration) within the mesentery (*arrows*)



Fig. 20.16 A liver laceration (*arrow*)

Fig. 20.17 Numerous external ear contusions in a child abuse victim



(Fig. 20.18). In the absence of such a history, such an injury should be considered suspicious. Occasionally, resuscitative efforts are implicated in causing such an injury. Interviewing emergency medical personnel is of utmost importance when mouth and inner lip injuries are seen at autopsy. Typically, the medics are able to relate to investigators whether or not pre-existing injury was noted. Obviously, such questions should be asked immediately after discovering the injuries, rather than waiting weeks or longer.

On rare occasions, severe soft tissue hemorrhage, with or without associated skeletal trauma, may be severe enough to induce the systemic inflammatory response syndrome, shock and death. In such cases, cutting into the skin of the extremities (arms and legs) and the back and buttocks will reveal extensive



Fig. 20.18 A torn frenulum

Fig. 20.19 Posterior skin incisions with abundant subcutaneous (under the skin) and muscular bleeding from inflicted trauma



hemorrhage within the subcutaneous fat tissue and muscles (Fig. 20.19 and Disc Image 20.25).

Bite marks may be evident in certain childhood deaths. Bite marks tend to have a rounded, or oval, shape, composed of two semicircular marks corresponding to the upper and lower jaws (Disc Image 20.26). If a bite mark is suspected, and it appears fresh, pathologists should attempt to collect saliva/DNA samples from the mark. Photographic documentation should be performed, and consultation with a forensic odontologist should be considered.

Skeletal blunt force injuries (fractures) are relatively common in child abuse homicide, although their presence is not universal. In chronically battered children, old (remote), healed fractures, and fractures at varying stages of healing, are frequently discovered, particularly involving the ribs (Figs. 20.20 and 20.21 and Disc Images 20.27 and 20.28). Two long-bone fracture types are considered suspicious for inflicted trauma. One is referred to as a “corner” fracture, or a “bucket handle” fracture, depending on the orientation of the fracture on X-ray examination (Fig. 20.22 and Disc Images 20.29 and 20.30). This type of fracture involves the epiphyseal plate (growth plate). The other is referred to as a “spiral” fracture and has a twisted, or spiral, appearance along the shaft of a long bone (Fig. 20.23). Virtually



Fig. 20.20 A healing rib fracture (*arrow*) present in a victim of repeated child abuse

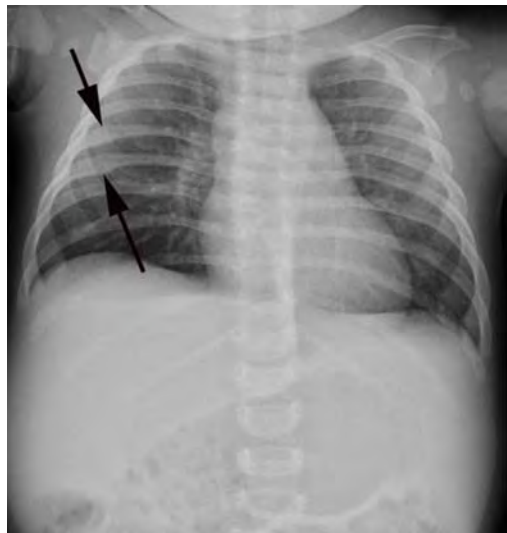


Fig. 20.21 Multiple healing rib fractures (*arrows*) seen on X-ray (courtesy of Department of Radiology, Indiana University School of Medicine)



Fig. 20.22 A so-called “bucket-handle” fracture (*arrows*) involving the growth plate of the bone (courtesy of Department of Radiology, Indiana University School of Medicine)

Fig. 20.23 A spiral fracture of the femur (thigh bone). (courtesy of Department of Radiology, Indiana University School of Medicine)



any long bone can be fractured (Disc Image 20.31). The presence of unexplained healing fractures should raise suspicion for abuse (Disc Images 20.32 and 20.33).

Other injury types that are encountered in child abuse homicides include scalding and other burn injuries (Fig. 20.24) (also see Fig. 19.6), other temperature-related



Fig. 20.24 A scalding injury on the hand, wrist and forearm

deaths, such as exposure (see Chapter 18), as well as various asphyxial mechanisms (see Chapter 15). It is particularly important to perform an intraoral examination during a pediatric autopsy, looking for evidence of lip, tongue, or inner cheek injuries (see Figure 15.3 and Disc Image 15.5). Some parents/caregivers have been known to purposefully asphyxiate infants and young children, in order to “calm them down,” or “get them to go to sleep.” Other injury categories, such as sharp force injury, electrical injury, drug/toxin cases, and gunshot wounds may also be present, but these are less commonly encountered.

Classification of Childhood Homicides

There are no absolute rules regarding child abuse homicides. While some of these tragic deaths occur at the hands of individuals unknown to the child (such as in certain child abduction cases), many of them occur at the hands of individuals well-known to the child, such as a parent, a babysitter, or a parent’s significant other. The following paragraphs will present different types of filicide (the killing of a child by its parent), based on the underlying motive, reason, or explanation for the killing. It should be emphasized that care providers or other significant adults in a child’s life may kill children for similar reasons as a parent, so the following classification scheme can be applied to these deaths as well. Finally, children killed by a person that they do not know are frequently killed for reasons similar to some of those presented below. In essence, then, the following filicide classification system can be applied to virtually any child homicide, recognizing that certain types are more or-less restricted to parents, while others are more widespread. The system is based on classification schemes first described by psychiatrists and then elaborated upon by forensic pathologists. It should be noted that the categories are not necessarily mutually exclusive. In other words, a particular death may be included within more than one category. Some of the latter categories are extremely rare.

Violent Outburst (Angry Impulse)

This category probably represents the most common type of child abuse homicide. In such cases, the child, who is usually rather young, suffers lethal trauma, most commonly blunt force trauma. Common locations of blunt force trauma are head/brain injury and abdominal injury. Skeletal injury (broken bones) may be present. In infants, a component of shaking injury may also be present. Other injury types may be involved, such as asphyxia. Whatever the injury type, the infliction of the injury is related to a violent outburst on the part of the adult perpetrator. Two subtypes of this form of homicide exist. The first involves a child where there is no historical, clinical, or autopsy evidence of prior injury. It is not uncommon in these cases for the perpetrator to immediately seek medical attention for the injured child. Although the injury results from an intentional act, it is unusual for the perpetrator to have actually intended the injury to be lethal. The second subtype of violent outburst child abuse homicide involves a child where there is evidence of prior abusive injuries. The latter scenario is sometimes referred to as the “battered child syndrome.” In it, there is typically autopsy evidence of old (remote), healing injuries, in addition to the acute injuries that caused death (Fig. 20.25 and Disc Image 20.34).



Fig. 20.25 Numerous bruises in a “battered child.”

Negligence/Neglect

Negligence may be defined as conduct that falls below the standard established by law for the protection of others against the unreasonable risk of harm. Neglect may be defined as prolonged failure to provide adequate care. Childhood deaths related to negligence/neglect occur as a result of acts of commission or acts of omission. Examples of acts of omission include leaving an infant in an enclosed, hot car with resultant death by hyperthermia, and leaving an infant unattended in a bathtub with

resultant drowning. An example involving an act of commission is the intentional overmedication of a crying infant in order to quiet them, with resultant death due to drug toxicity. Certifying the manner of death in these cases can be particularly difficult. In fact, many are certified as “accident” or “undetermined,” deferring any further legal action to law enforcement authorities and the courts.

Sadistic Acts of Punishment

These deaths may involve an isolated incident or long-term, repetitive abuse. The underlying motivation of the perpetrator in these cases is a deliberate attempt to inflict pain or discomfort on the child. They are distinct from violent outburst deaths by the fact that they involve deliberate planning. Examples include forced salt ingestion, asphyxiation due to an object or substance being forced into the child’s mouth, intentionally inflicted burns, including scalding burns, withholding food and water (Fig. 20.26 and 20.27), and deaths related to prolonged restraint. Children



Fig. 20.26 A child who was starved to death

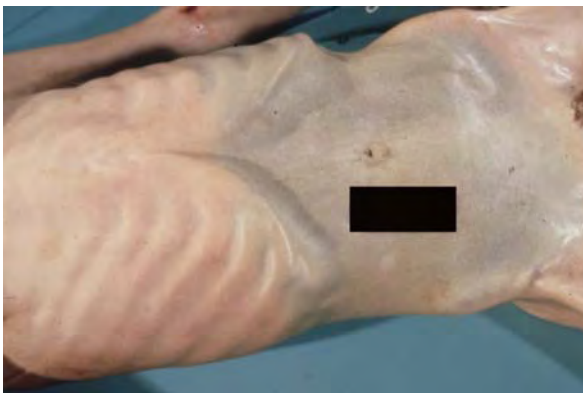


Fig. 20.27 Another photograph of the child depicted in Fig. 20.26

dying from such injuries may also demonstrate classic findings of the battered child syndrome.

Munchausen's Syndrome by Proxy

This syndrome exists when a parent or caregiver, often the mother, attempts to gain attention, praise, or sympathy by intentionally inflicting illness, injury, or even death on a child. The underlying motive is the emotional and psychological attention that the perpetrator gains from the process. Two subtypes exist. In the classic Munchausen's Syndrome by Proxy case, the parent intentionally causes or mimics illness in their child, in order to gain attention from persons within the medical community, as well as others. Numerous actions can be involved, including the injection of foreign substances into the child, feeding them foreign substances or sublethal doses of poisons, or smothering the child to the point of unconsciousness. Death in these cases is usually inadvertent and not desired by the parent. The second subtype involves a parent who intentionally kills their child in order to gain the subsequent sympathy and attention associated with losing a child. The parent may have previously lost a child, although the first death might have been unrelated to Munchausen's Syndrome by Proxy. In other words, the first child's death may have been a natural death or an accidental death. Certainly, the possibility exists that the first death was also homicidal, but this is not an automatic given.

Unwanted Child

These filicides are typically committed because of the parent's selfishness: the child is simply not wanted. They might be related to an underlying malformation, illness, or behavioral problem in the child. Conversely, they may be related to cultural or superstitious reasons, such as in communities where female infants are killed because of the preference for males. Another example is when a female child is killed by her father after "disgracing" the family by being raped (a so-called "honor killing"). Others may occur because of the financial burden that the child represents, or the social burden (a single mother who wants to be free to date someone who doesn't like kids), or because a father doubts that he is the father.

Unwanted Pregnancy (Neonaticide)

This can be considered a subcategory of the unwanted child category. Neonaticide is defined as the killing of a newborn child by its parent within the first 24 hours after birth. The mother is usually the perpetrator: she is usually young, unmarried, and received no prenatal care. She may have attempted to conceal her pregnancy. Birth typically occurs in isolation. The dead newborn may or may not have been hidden

or discarded. Discussion of the difficult forensic issues involved in such cases has been addressed previously in this chapter.

Spouse Revenge

These murders are committed in a deliberate attempt to torment or punish one's spouse, the parent of the murdered child. A variety of precipitating events might initiate such a killing, the most common being the discovery of infidelity by the spouse. Many cases are related to child custody battles. For example, if a mother figures that her soon-to-be ex-husband is likely to gain custody of the child, she may intentionally kill the child as a form of revenge toward her husband. Differentiating certain of these cases from those involving altruistic motives (see below) can sometimes be difficult.

Postpartum Mental Disorder

These filicides occur during the postpartum period (after birth) as a result of a mental disorder within the mother related to pregnancy (postpartum depression or other related conditions). These cases may involve "command hallucinations" ("voices" telling her to do it) or delusions within the mother. In other words, there may be a psychotic component, but this is not always the case.

Acute Psychosis

These filicides are committed during an acute psychotic episode in the parent in which there is no other obvious discernible motive. This category is similar to, but distinct from, postpartum mental disorder. Command hallucinations and/or delusions may play a role in these killings.

Altruism

"Altruism" refers to the unselfish concern for the welfare of others. Altruistic filicide typically occurs when a suicidal parent, out of genuine love and concern for their child, intentionally kills the child, so that when the parent commits suicide, the child will not be left parentless. It is truly one of the most bewildering and sad situations that a death investigator may encounter. Ultimately, the underlying reason for the killing is as varied as the reasons for the parent's suicide. In general, the parent does not want to leave the child to face whatever it is that the parent is trying to escape. Often, the parent's thoughts are delusional. Despite the fact that these types

of filicides are referred to as altruistic, in many cases there does appear to be a truly selfish motive on behalf of the parent.

Euthanasia (True Mercy Killing)

In contrast to altruism, where the “unavoidable” situation that the child faces is often related to depressed or delusional thoughts of the parent, in euthanasia cases the child is truly suffering from a real illness or an adverse situation. The child is killed in order to protect them from further or eventual horrors. The killing of a newborn infant with a severe, incapacitating genetic disease is an example. The killing of a child who is in imminent danger of being serially raped by militant terrorists is another example.

Sexual Abuse

These child killings occur under various circumstances, each involving sexual abuse directed toward the child. The murder may be intentional, such as when a chronically sexually abused child threatens to inform others of the abuser’s actions. Alternatively, death may be unintentional, occurring during or as a result of injurious sexual activity. Examples include a violent rape with associated strangulation, or a delayed death in a small child, due to peritonitis following traumatic rupture of the rectum/colon. It should be noted here that perceived dilation (a stretched-open appearance) of a child’s anus does not necessarily imply that some type of abuse



Fig. 20.28 Postmortem laxity (relaxation) of the anus, which can sometimes be misinterpreted as evidence of abuse

has occurred (Fig. 20.28). Postmortem anus dilation is a recognized entity. Only if associated trauma (bleeding, bruising) is identified should the finding raise suspicion. Microscopic identification of inflammation is not sufficient to prove abuse, considering the location. Similarly, the presence of a non-intact hymen should not necessarily be considered evidence of abuse, although associated fresh or remote trauma should raise concern.

Violent Older Child

These cases involve some type of altercation between the parent and an older child (usually a teenager), often related to drug or alcohol problems in the child. The situation is precipitated by the child. For example, an intoxicated teenager arrives home and begins shouting at his mother. The father enters the argument, tempers flare, and the teenager begins to attack his father. Eventually, the son produces a knife, but the son ends up dying from stab wounds. These cases essentially mimic cases that would otherwise be considered “domestic violence” cases.

Drug and Alcohol Abuse

These cases involve drug and/or alcohol abuse issues within the parent, rather than within the child. It is not uncommon for other filicide category types to involve parents who are intoxicated. In addition, cases of infant overlay associated with maternal (or paternal) intoxication may be placed in this category.

Seizure Disorder

This is perhaps the least common (and least believable) category of filicide explanations. The classic example that is described in the literature involves an epileptic mother, who, following a seizure, placed her baby on the fire and her kettle in the baby’s crib. A more believable example is a case wherein a friend stops by the home of an epileptic mother to find her on the floor, obviously in a post-ictal (post-seizure) state, lying atop her dead infant.

Innocent Bystander

These cases involve the inadvertent death of a child, usually during an attempt by one parent to kill his or her spouse, or another person. An example involves a toddler, being held by his mother, when her boyfriend shoots her multiple times, killing both mother and child. A subtype would be a feticide, in which an unborn child is killed when the mother is killed.

Pediatric Autopsy Considerations

A variety of autopsy findings are either common in or relatively unique to childhood or infancy. A so-called “Mongolian spot” over the sacrum of dark-skinned individuals (Fig. 20.29) can be mistaken for a bruise. The presence of an “umbilical hernia” (Disc Image 20.35) is occasionally misinterpreted as some form of trauma. Diaper rash can sometimes be misinterpreted as some form of inflicted injury (Disc Image 20.36). Postmortem laxity of the anus (or vagina) can occasionally be misinterpreted as evidence of some sort of sexual abuse (Disc Image 20.37; also see Fig. 20.28). Children, and especially infants, who have undergone emergency medical therapy frequently have “intramedullary” (IM) lines instead of intravenous lines. IM lines consist of IV tubing connected to a plastic hub that attaches to a sharp, hollow, metal needle that is inserted through the skin of the shin into the tibia (shin bone) (Disc Image 20.38).

Fig. 20.29 A Mongolian spot over the sacral region (lower back/upper buttocks) (indicated by *arrows*). This pigment change is sometimes mistaken as a bruise



A variety of examinations and special techniques may be performed on pediatric deaths, depending on the age of the child, the circumstances of death, and the pathologist. In perinatal deaths, gross and microscopic examination of the placenta should be performed. In most infant deaths, many pathologists perform a variety of additional measurements and/or procedures. Measurement of the infant's total length (the “crown–heel” length), the “crown–rump” length, the head circumference, the chest circumference, and the foot length are routine in many offices. These enable the pathologist to compare the measurements to standardized pediatric growth and development charts. Some of these measurements (obviously body length, but also head circumference if indicated) should be taken in older children as well. Particularly in perinatal deaths, it is important to check whether or not the external ear canals, the nasal passages, and the anus are patent (appropriately open).

Also in perinatal deaths, it is important to look for any subtle external (and internal) malformations, such as webbing of the spaces between fingers or toes, wide-spread nipples, low-set ears, wide-set eyes, and various other features that may indicate a syndrome or genetic abnormality.

In many infant deaths, pathologists will perform a sterile spinal tap, collecting cerebrospinal fluid for culture. Some collect the sample from the lower back (Fig. 20.30), others prefer the lower posterior skull. Depending on the case, office protocol, and pathology preference, infant (and sometime young child) cases will routinely include blood bacterial cultures, blood or lung viral cultures, blood testing for heavy metals (such as lead) and carbon monoxide, and specialized testing for an array of metabolic abnormalities.



Fig. 20.30 Postmortem cerebral spinal fluid (CSF) collection from an infant

On internal examination in childhood autopsies, besides measuring the weights of the brain, heart, lungs, liver, spleen, and kidneys, it is common for pathologists to also measure the weight of the adrenal glands and the thymus. Whereas many adult autopsies are performed by removing individual organs one at a time, it is not uncommon for pediatric autopsies, especially infant cases, to be performed via removal of most of the organs *en bloc* (in one large interconnected mass) (Disc Image 20.39). Particularly in infant cases, since infant brains tend to be very soft, pathologists frequently choose to fix the brain in formalin solution for several weeks prior to sectioning (cutting) it. In perinatal deaths and in young infant deaths, an alternative procedure for uncovering the brain is commonly employed. In contrast to the usual method of cutting the skull with a bone saw, when the skull plates are flexible enough, the suture lines (where the skull plates abut one another) can be cut, and the plates can be bent away from the head/brain, like flower petals opening. This method is typically referred to as the “rose petal” technique (Disc Image 20.40). In certain infant cases, the eyes are removed and retained for further examination (Disc Image 20.41). In some cases, the middle and inner ears are removed for culture and/or subsequent examination.

Disc Image Legends

Disc Image 20.1 A “dumped” newborn found outdoors.

Disc Image 20.2 A stillborn child with early skin discoloration and a dark umbilical cord.

Disc Image 20.3 Two placentas with attached umbilical cords. The one on the left is from a macerated stillbirth. Note the dark discoloration of the umbilical cord. The one on the right is “normal” placenta. Note the yellow color of the cord.

Disc Image 20.4 A stillborn fetus with no maceration or discoloration of skin/umbilical cord.

Disc Image 20.5 The flotation test. A photograph looking down on a container of water containing small sections of multiple organs. Note that several pieces of lung are floating on the surface, whereas the other organ tissue sections have sunk to the bottom of the container.

Disc Image 20.6 A tight “nuchal cord” wrapped about the neck of a stillborn infant.

Disc Image 20.7 A diagram advocating a potentially safe bed-sharing scenario. (Illustration reprinted from *“Sleeping with your Baby: A Parent’s Guide to Cosleeping,”* by James J. McKenna, Ph.D. Courtesy of Platypus Media, LLC, Washington, DC.)

Disc Image 20.8 A choking death in an autistic child, with food (arrows) occluding the airway.

Disc Image 20.9 A child that was accidentally run over by a motor vehicle. The rapid loss of blood pressure related to severe internal injuries resulted in tire-tread imprint abrasions which appear postmortem (they are yellow).

Disc Image 20.10 Multiple scalp impact sites.

Disc Image 20.11 An infant with massive head injuries, but little, if any external evidence of trauma. See Disc Image 20.12 for comparison. (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)

Disc Image 20.12 The posterior (back) head of the infant shown in Disc Image 20.11, after scalp reflection. Note the extensive subscalpular hemorrhage, as well as the skull fractures. (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)

Disc Image 20.13 A skull fracture in a child abuse case.

Disc Image 20.14 Marked cerebral edema (brain swelling).

Disc Image 20.15 Diastatic fractures (separation of the skull suture lines) (arrows) resulting from severe brain injury and associated swelling.

Disc Image 20.16 The microscopic appearance of diffuse axonal injury, as seen via routine H&E staining. Note the rounded “retraction balls,” signifying areas of damaged axons, with accumulated intracellular substances. In order for these changes to be evident on routine H&E staining, post-injury survival time must be many hours to days.

Disc Image 20.17 The microscopic appearance of diffuse axonal injury, using an immunoperoxidase stain that specifically stains beta-amyloid precursor protein (bAPP) brown. The retraction balls seen in Disc Image 20.16 are evident, as are other areas of axonal bAPP staining. Use of the bAPP

stain can indicate axonal injury after only a few hours of post-injury survival.

Disc Image 20.18 Periosteal nerve hemorrhage immediately behind the eyeball (arrow). Note that the front (anterior) portion of the eye has been previously removed.

Disc Image 20.19 Retinal hemorrhages (arrows) as visualized by “backlighting.”

Disc Image 20.20 A small retinal hemorrhage visualized under the microscope.

Disc Image 20.21 Lack of scalp trauma.

Disc Image 20.22 Two small intestine contusions (arrows), with associated injuries of the mesentery.

Disc Image 20.23 Two sections of an injured adrenal gland, compared to a section of a non-injured adrenal gland (below).

Disc Image 20.24 Contusions of the pancreas.

Disc Image 20.25 Dissection of the back showing absence of major injuries. There is a small contusion of the upper right back (arrow).

Disc Image 20.26 A bite mark on the arm.

Disc Image 20.27 A rib cut in half lengthwise, showing a fracture with early healing (arrows).

Disc Image 20.28 Another rib cut in half lengthwise, showing a different rib fracture (arrows), with more healing than in Disc Image 20.27.

Disc Image 20.29 Another example of a “bucket-handle” fracture (arrow). (courtesy of Department of Radiology, Indiana University School of Medicine)

Disc Image 20.30 A “corner” fracture (arrow). (courtesy of Department of Radiology, Indiana University School of Medicine)

Disc Image 20.31 A fractured humerus (upper arm bone) in a child abuse victim.

Disc Image 20.32 A femur (thigh bone) fracture that has healed inappropriately. Note how the bone is bent.

Disc Image 20.33 Multiple healing fractures in a child abuse victim, including a spiral fracture of the humerus (upper arm), a fracture of a forearm bone, and multiple rib fractures. (courtesy of Department of Radiology, Indiana University School of Medicine)

Disc Image 20.34 Multiple injuries on the upper extremity of a “battered child.”

Disc Image 20.35 An umbilical hernia.

Disc Image 20.36 Diaper rash can sometimes be misinterpreted as inflicted injury.

Disc Image 20.37 Postmortem dilation of the vagina. There are no injuries. The finding in this case is meaningless.

Disc Image 20.38 An intramedullary (IM) line used to provide fluids to the child.

Disc Image 20.39 Removal of organs *en bloc*. The tongue is on the left and the pelvic organs are on the right.

Disc Image 20.40 The “rose-petal” method of skull bone reflection (after the brain has been removed) utilized in young infants for brain removal.

Disc Image 20.41 Eye removal via removal of the base of the skull overlying the eyeballs.

Selected References

- Baker A. Investigating asphyxial deaths in infants and small children. American Society for Clinical Pathology Teleconference; March 14, 2008.
- Byard RW, Kohle SD. *Sudden Death in Infancy, Childhood, and Adolescence*. Cambridge, England: Cambridge University Press; 1994.
- Byard RW, Krous HF. *Sudden Infant Death Syndrome – Problems, Progress, & Possibilities*. London, England: Arnold; 2001.
- Collins KA. Sudden infant death syndrome (chp 11). In: Froede RC, editor. *Handbook of Forensic Pathology*, 2nd edition. Northfield, IL: College of American Pathologists; 2003. pp 105–10.
- Collins KA, Knight LD. Pediatric deaths (chp 17). In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006. pp135–56.
- Corey TS. Investigation of child abuse (chp13). In: Froede RC, editor. *Handbook of Forensic Pathology*, 2nd edition. Northfield, IL: College of American Pathologists; 2003. pp 125–38.
- Corey TS, Hanzlick R, Howard J, Nelson C, Krous H. A functional approach to sudden unexplained infant deaths. *Am J Forensic Med Pathol* 2007;28:271–7.
- Davis GJ. Accidental and natural deaths in children (chp 10). In: Froede RC, editor. *Handbook of Forensic Pathology*, 2nd edition. Northfield, IL: College of American Pathologists; 2003. pp 99–104.
- Dolinak D, Reichard R. An overview of inflicted head injury in infants and young children, with a review of B-amyloid precursor protein immunohistochemistry. *Arch Pathol Lab Med* 2006;130:712–7.
- Fierro ME. Infanticide, abandoned newborns, and feticide (chp 12). In: Froede RC, editor. *Handbook of Forensic Pathology*, 2nd edition. Northfield, IL: College of American Pathologists; 2003. pp 111–24.
- Gilbert-Barness E, Debich-Spicer DE. *Handbook of Pediatric Autopsy Pathology*. Totowa, NJ: Humana Press; 2005.
- Krugman SD, Lantz PE, Sinal S, DeJong AR, Coffman K. Forced suffocation of infants with baby wipes: a previously undescribed form of child abuse. *Child Abuse Neglect* 2007;31:615–21.
- McKenna JJ, Ball HL, Gettler LT. Mother-infant cosleeping, breastfeeding and sudden infant death syndrome: what biological anthropology has discovered about normal infant sleep and pediatric sleep medicine. *Yrbk Phs Anthropol* 2007;50:133–61.
- McKenna JJ, McDade T. Why babies should never sleep alone: a review of the cosleeping controversy in relationship to SIDS, breast feeding and bedsharing. *Pediatric Respiratory Reviews* 2005; 6: 134–152.
- O’Neal BJ. *Investigating Infant Deaths*. Boca Raton, FL: CRC Press; 2007.
- Pasquale-Styles MA, Tackitt PL, Schmidt CJ. Infant death scene investigation and the assessment of potential risk factors for asphyxia: a review of 209 sudden unexpected infant deaths. *J Forensic Sci* 2007;52:924–9.
- Plunkett J. Fatal pediatric head injuries caused by short distant falls. *Am J Forensic Med Pathol* 2001;22:1–12.
- Schwartz LL, Isser NK. *Endangered Children – Neonaticide, Infanticide, and Filicide*. Boca Raton, FL: CRC Press; 2000.
- Sharma BR. Sudden infant death syndrome – a subject of medicolegal research. *Am J Forensic Med Pathol* 2007;28:69–72.

Chapter 21

Miscellaneous Topics

They die in an instant, in the middle of the night; the people are shaken and they pass away; the mighty are removed without human hand.

Job 34:20

Abstract Chapter 21 encompasses numerous topics that are either too overlapping in their content to fit nicely into a previous chapter, or too narrowly focused or uncommon to deserve a chapter by themselves. This relatively lengthy chapter includes discussions on a wide array of topics, including aircraft crashes, allergic reactions, animal attacks, artifacts and mimics, emboli, exhumations, explosions and blast injuries, high-profile cases, homicide by heart attack, in-custody deaths, mass fatality incidents, cases with multiple causes of death, nutrition and hydration disorders, food poisoning, occupational deaths, organ and tissue procurement issues, postmortem chemistry tests, postmortem cultures, pregnancy-related maternal deaths, product-related deaths, radiation, sexual assault, terrorist agents, and therapy-related deaths.

Keywords Aircraft · Allergic reaction · Animals · Artifacts · Emboli · Exhumation · Explosions · High-profile cases · Homicide by heart attack · In-custody · Mass fatalities · Multiple causes of death · Nutrition · Hydration · Food poisoning · Occupational · Work-related · Organ donation · Tissue donation · Postmortem chemistry · Cultures · Pregnancy · Product-related · Radiation · Sexual assault · Terrorist agents · Therapy-related

Introduction

This chapter is a compilation of a variety of specific issues and/or case types that do not easily fit into any other chapter. The topics are arranged alphabetically. Each topic is presented in general terms. The reader is referred to more detailed descriptions as delineated in the “Selected References” section at the end of the chapter.

Aircraft Crashes

Fatal aircraft crashes can range from a single occupant (pilot) plane crash to a jet airliner crash with hundreds of victims (Fig. 21.1). Ultimately, in most instances, it is the local medical examiner or coroner who has jurisdictional responsibility for investigating such deaths; however, several federal government agencies will also be involved in the overall investigation. While the local death investigation office has jurisdiction over the dead bodies, the National Transportation Safety Board (NTSB) has legal jurisdiction over the wreckage and crash site and is charged with determining the probable cause of all civil (non-military) aircraft crashes. As part of NTSB's investigative responsibilities, it may order autopsies and toxicology tests. The Federal Aviation Administration (FAA) is specifically concerned with determining the existence of any human factors that may have played a role in incapacitation of the pilot/crew. The FAA's Civil Aeromedical Institute (CAMI) provides consultative services, including toxicology services. Whenever an aircraft crash occurs and the pilot/crew is killed, a CAMI "tox box" should be collected during autopsy and sent to the CAMI toxicology laboratory for analysis. The box provides instructions for collecting, packaging, labeling and submitting numerous body tissues and fluids for analysis. If an aircraft crash is so large that it overwhelms the resources of the local death investigation office, the Disaster Mortuary Operational Response Teams (D-MORT) of the National Disaster Medical System (NDMS) can be called to help (1-800-USA-NDMS).



Fig. 21.1 The scene of a passenger airliner crash

The term "aerospace pathology" represents a discipline that applies the principles of medicine and pathology to the evaluation of diseases and injuries of pilots and other aircraft occupants. An "aeromedical investigation" attempts to identify factors that may have led to human errors associated with an aircraft crash. Such human factors may include underlying natural disease, a medical emergency, toxin inhalation, or the toxic effects of drugs or alcohol. In contrast, a "safety investigation" searches for non-human factors that may be associated with the crash, including equipment

operation and function. Human error is the underlying cause of aircraft mishaps in a majority of cases (80%), with mechanical and environmental circumstances accounting for the remainder.

An aircraft crash typically involves major forces. As such, massive injury can result, such that bodies can become severely disfigured or fragmented (Fig. 21.2 and Disc Image 21.1). A general rule of thumb is that if the aircraft is recognizable, there is a good chance that the body(ies) will be intact, or at least partially intact. In contrast, if the aircraft is in pieces, the body(ies) will be in pieces. Thermal injuries can further obscure bodies. It should be noted that persons on the ground at the site of the crash may also be killed. Positive identification of all fatalities is absolutely essential. In all cases, documentation of the wreckage and bodies should be made before anything is moved. All bodies and body parts, as well as personal effects, should be tagged and documented by diagram, photography, and video. Particularly when multiple fatalities are involved, a grid system (see Mass Fatality Incidents section below) can provide a somewhat organized method of documenting the wreckage and victims. Death investigators should obtain the passenger manifest (list of all passengers and crew) as soon as possible after the crash. In commercial airlines, the manifest should provide the exact seat location for all passengers; however, as any frequent flyer knows, “seat swapping” occurs fairly routinely. Identification should be scientific if at all possible. Clothing and personal effects can be very useful in making preliminary identifications.



Fig. 21.2 Massive body injury in a pilot who crashed at high speed (after clothing removal)

The main purpose of postmortem examination of passengers is for identification purposes; however, the identification of pre-crash injuries or evidence of a pre-crash cabin fire (elevated carbon monoxide level) may provide valuable information regarding the cause of the crash. Of particular importance is the identification of the pilot (and co-pilot and crew), as complete autopsies should be performed on these individuals. The autopsy should include whole-body X-rays, as well as complete toxicology testing. It is advisable for pathologists to perform their own toxicology tests, including carbon monoxide, in addition to collecting and submitting the

CAMI tox box specimens, as the results from the tox box are not usually reported very quickly. Identification at autopsy of significant disease, intoxication, or possible pre-crash injuries may provide clues to the cause of the crash. The presence of fractured hands/wrists/forearms and/or feet/ankles may indicate that the pilot (or co-pilot) (Fig. 21.3 and Disc Image 21.2) was conscious and at the controls when impact with the ground (or other structure) occurred, giving rise to the term “control surface injuries.” It should be noted that similar injuries may exist in passengers.



Fig. 21.3 Pilot control injuries involving the hand

A major part of the entire evaluation of an aircraft crash is the post-crash analysis of survivability. Autopsy findings aid in addressing several of the issues involved in this analysis. In general, an aircraft crash occupant’s ability to survive depends on (1) the person’s ability to tolerate crash forces, (2) maintenance of adequate occupiable space, and (3) a non-lethal post-crash environment. As little as 25 G of force can cause lethal neck injuries. Approximately 80–100 G of force are required to transect an aorta. Body fragmentation occurs at about 350 G.

Many other important details exist regarding the investigation of aircraft fatalities. It is beyond the scope of this text to address all of these. The reader is referred to an excellent review of the entire topic of aerospace pathology and aircraft crash investigation for a more complete discussion.

Allergic Reactions (Anaphylaxis)

Allergic reactions, or “anaphylactic reactions,” occur in response to exposure to a variety of “allergens” (also referred to as “antigens”). An allergen is something that can induce hypersensitivity in a person’s immune system. If a person becomes “sensitized,” their immune system produces antibodies that react to and bind to the allergen, as part of the immune system’s attempt to rid the body of the allergen. Most anaphylactic reactions involve a specific antibody type called an IgE antibody (“Ig” stands for “immunoglobulin,” a technical name for antibody) attached to mast cells.

If a previously sensitized person is subsequently exposed to the allergen, an allergic reaction can occur. This results from the allergen binding to the IgE antibodies on mast cells, which causes the mast cells to release numerous substances that result in the symptoms of an allergic reaction. Allergic reactions vary in their intensity from a localized reaction (rash, itching, sneezing fit) to a severe, systemic, lethal reaction characterized by marked respiratory compromise and severe hypotension (lowering of blood pressure) with eventual cardiorespiratory collapse and death. At autopsy, laryngeal edema (swelling) may or may not be present (Fig. 21.4). Evidence of exposure to a specific allergen (drug, insect sting, radiocontrast dye) may or may not be evident during autopsy, but a credible history of such exposure should be confirmed.

Fig. 21.4
Anaphylaxis-associated
laryngeal edema



Certain laboratory tests may be helpful in making a postmortem diagnosis of anaphylaxis. A substance referred to as tryptase is one of the many substances contained in mast cells, and measurement of total tryptase levels in the blood provide a measure of the total number of mast cells. Measurement of a subtype referred to as “beta-tryptase” (or “mature tryptase”) provides an indication of mast cell activity. Hence, when an allergic reaction occurs, beta-tryptase levels will be elevated. Unfortunately, if samples are not drawn within the correct time during and/or after an allergic reaction, the levels may be normal. Also, levels may occasionally be elevated in situations that are clearly not related to anaphylaxis.

If a specific allergen is suspected (such as bee venom or an antibiotic), tests showing an elevation of the specific IgE antibody directed against the allergen will provide further evidence that anaphylaxis has occurred. Unfortunately, not all allergens have antibody tests available. Also, a person who is sensitized to any number of allergens will tend to have elevated levels of IgE antibodies to each of these allergens, even if an anaphylactic reaction has not occurred.

To summarize, elevated IgE levels indicate the existence of sensitization to a specific allergen. Elevated beta-tryptase levels indicate the possibility of an anaphylactic event. Autopsy and/or investigative evidence of recent exposure to the presumed allergen, and subsequent symptoms and anatomic changes that are characteristic of anaphylaxis, should be documented. Autopsy findings may or may not be present, but autopsy should not show evidence of a different cause of death.

The manner of death in anaphylactic deaths is somewhat controversial within the forensic community. Many suggest that most allergen exposures involve an external, or exogenous, substance. As such, it is appropriate to rule these deaths as “accidents.” Others contend that, since the underlying problem involves a hypersensitive reaction within the body’s natural defense system, then the underlying problem is a natural disease. Consequently, the manner of death should be ruled “natural.”

Animal Attacks

Occasional deaths result from animal attacks. Some involve insect stings, with subsequent anaphylaxis, as described above. Occasional insect stings or arthropod bites result in localized infection, tissue necrosis, and subsequent sepsis and death. Some arthropods, insects, and other animals act as vectors for micro-organisms or parasites.

Certain mammals can transmit rabies or other infections via their bites. Poisonous animals, such as rattlesnakes, can kill via their venom (Disc Images 21.3 and 21.4).

Larger animals, whether domesticated or wild, can kill humans via a variety of mechanisms. Large animals, such as livestock, certain wildlife, and zoo animals, can cause significant blunt force and crushing type injuries. Others, particularly predatory animals, such as cougars, wolves, and bears, can kill by a combination of sharp and blunt force injuries. If sufficient neck compression is involved, an asphyxial component may also be at play. Death from dog attacks involve similar mechanisms (Fig. 21.5). Large constrictor-type snakes can kill by mechanical/compressive asphyxia.

When multiple mechanisms are involved, it is sufficient to simply rule the cause of death as “multiple injuries from animal attack” or some similar terminology. The manner of death ruling in animal attack cases can also be controversial, although many forensic pathologists rule such deaths as accidents.

Artifacts and Mimics

There are a variety of entities or conditions that may alter the appearance of the decedent, obscure the body such that evaluation for injuries and/or disease is less than optimal, or mimic true antemortem findings. Many of these have been presented elsewhere in this text. Common artifact types are those that accompany the



Fig. 21.5 Multiple injuries from an attack by three large dogs. (photo courtesy of the Dallas County Medical Examiners Office)

decomposition process, including postmortem drying (Disc Image 21.5), Tardieu spots (Disc Image 21.6), and even lividity (Fig. 21.6), as well as tissue damage which results from insect and/or animal activity, and those related to resuscitation efforts and medical intervention. Particularly problematic for forensic pathologists are cases in which surgeons cut through injuries, such as bullet wounds or stab wounds (see Disc Image 14.32). Other classic artifacts include heat-induced skull fractures, epidural blood sequestration, and skin-splitting in fire deaths. A wide variety of artifacts can be misinterpreted as true injuries. In addition, some true antemortem injuries can be misinterpreted as another, different type of injury.



Fig. 21.6 Lividity pattern on neck mimicking ligature marks

Artifacts that represent postmortem injuries are classically described as having a yellow appearance because of the lack of a “vital tissue reaction” (lack of bleeding) (Disc Image 21.7). While this is true for many postmortem injuries, depending on the case, postmortem wounds can sometimes lack a yellow appearance. This

typically occurs if the wound is located in a region that is subject to livor mortis formation. In other words, if a postmortem wound occurs on the skin surface where blood pools due to gravity, the postmortem injury can appear like a wound that occurred prior to death (antemortem) (Disc Image 21.8).

Postmortem burns and electrical injuries can appear identical to antemortem wounds. A variety of artifacts and injuries can mimic gunshot wounds or other injury types, including penetrating blunt force trauma, sharp force injuries, insect activity, and medical intervention (Fig. 21.7). Various entities can mimic ligature marks, including skin creases in combination with lividity (with or without associated decomposition), clothing compression marks, and even true injuries, such as abrasions and contusions.

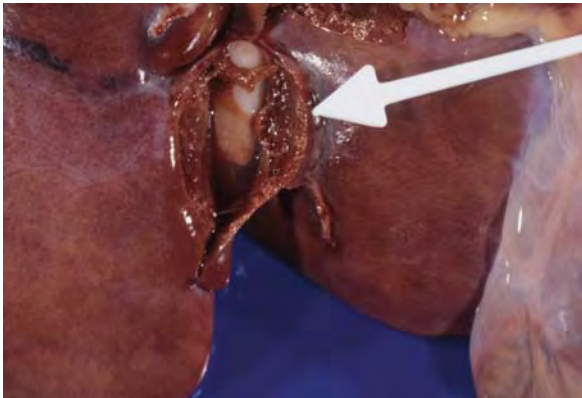


Fig. 21.7 A liver laceration which resulted from aggressive cardiopulmonary resuscitation (CPR)

Decomposition can result in bloody discoloration of soft tissues, such that optimal examination for true injury is compromised. Usually, such decompositional changes are most severe in dependent portions of the body. For example, a decomposing body is found face-down in bed. At autopsy, the sclera (whites) of the eyes appear markedly hemorrhagic (Fig. 21.8). In addition, anterior neck dissection reveals strap muscles that have bloody discoloration, primarily near blood vessels, without any definite muscular hemorrhage (Disc Image 21.9). Similar soft tissue discoloration is present within the anterior and upper subscalpular tissues (Disc Images 21.10 and 21.11).

Emboli

An “embolus” is a substance (solid, liquid, or gas) that travels within the blood vessels from one point to a different point. The plural for “embolus” is “emboli.” An embolus can be “endogenous” (arising from within the body) or “exogenous” (something that comes from outside the body). The most common type of embolus is a thrombus (a “blood clot”). It is sometimes referred to as a “thromboembolus.”



Fig. 21.8 Decomposed sclera (white part of eyes)

Pulmonary Thromboemboli

Thromboemboli typically arise as thrombi (blood clots) within the deep veins of the legs, although occasionally they involve the pelvic veins, or even within the dural sinuses. When thrombi occur in these deep veins, the condition is called “deep venous thrombosis” (DVT). Persons are at increased risk for developing DVT in a variety of situations, including various hereditary conditions that predispose to clotting (factor V leiden mutations, protein S deficiency, protein C deficiency), obesity, pregnancy, any condition that leads to decreased mobility, and trauma. If a DVT breaks away from the blood vessel wall, it can travel (“embolize”) through the venous system, into and through the right side of the heart, and then become lodged within the pulmonary arteries (Fig. 21.9). If the thromboembolus is large enough, it can cause sudden collapse and death. Such thromboemboli are usually

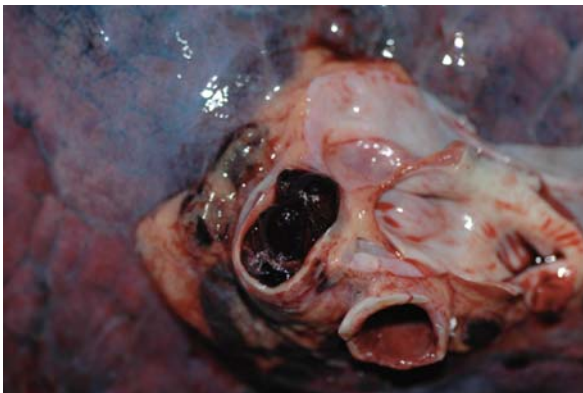


Fig. 21.9 Pulmonary embolism characterized by blood thrombi (clots) blocking the pulmonary artery

quite obvious within the lung arteries at autopsy. Particularly large thromboemboli can be present within the main pulmonary artery, just after it arises from the heart and divides into the right and left pulmonary arteries. This is sometimes referred to as a “saddle embolus.”

If pulmonary thromboemboli are discovered at autopsy, it is advisable to dissect the leg veins in order to attempt to identify the site of origin of the thromboemboli (Disc Image 21.12). Ultimately, the explanation for the cause of the underlying DVTs should also be ascertained (as listed above), as such a determination may have far-reaching implications regarding the manner of death. For example, if a person sustains a gunshot wound of the leg when he is shot by another person during a robbery attempt, survives the initial trauma, but develops DVT and dies from subsequent pulmonary thromboemboli, the manner of death is “homicide.”

Systemic Thromboemboli

In persons with infectious endocarditis (heart valve infection; usually as a complication of intravenous drug abuse) or heart disease with the presence of intracardial thrombosis (blood clots), thromboemboli can break away from their attachment sites on the valves or in the heart and embolize to distant organs and tissues, including the brain. These emboli can totally block an arterial branch, resulting in an “infarct” of the tissue or organ. If sufficiently large and in the brain, death can result.

Bone Marrow and Fat Emboli

In cases of severe skeletal (bone) trauma, a significant amount of bone marrow and fat (from within the bone marrow) can enter the blood vessels and embolize within the veins, through the heart, and into the microvasculature of the lungs. Occasionally, isolated fat emboli can occur without any skeletal trauma, presumably from massive soft tissue crushing injury.

The “fat embolism syndrome” is a specific clinicopathologic entity that typically occurs within a couple of days of severe skeletal trauma. It is characterized by the sudden onset of respiratory difficulties, often with associated neurologic deficits and subsequent death. At autopsy, the brain and lungs demonstrate intravascular fat emboli. In classic cases, gross examination of the brain reveals the presence of numerous petechial hemorrhages within the white matter (see Disc Image 12.48). Fat can be visualized microscopically in the lungs (see Disc Image 12.49) and sometimes in the brain.

Amniotic Fluid Emboli

Amniotic fluid is the water-like fluid in which a developing fetus resides within the “bag of waters” in the pregnant uterus. In rare pregnancies, which have typically

been normal and uneventful, usually at or near delivery, the pregnant woman experiences sudden cardiorespiratory collapse and dies. If the woman survives for any length of time following the initial event, she typically develops severe bleeding and clotting problems (a disorder called “disseminated intravascular coagulation”). At autopsy, small amniotic fluid debris (including fetal hairs and skin cells) is found within the microvasculature (small blood vessels) of the lungs by microscopic examination (Disc Image 21.13). Occasionally, a small laceration is found in the uterus or cervix. The proper cause of death in these cases is “amniotic fluid embolism.” It is not known with absolute certainty what the underlying mechanism is in these cases, but it is generally believed that obstruction (such as occurs in pulmonary thromboemboli) is not a major factor.

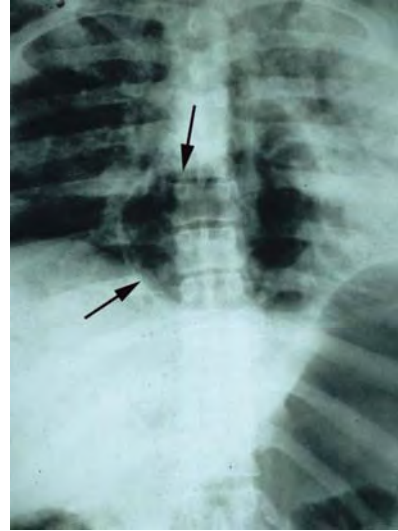
Gas/Air Emboli

Gas bubbles within blood vessels can cause blockage in a fashion analogous to “vapor lock.” In arteries, even a very small amount of gas can be very serious or even fatal (if the brain is involved). In veins, a larger amount of gas is necessary, but it doesn’t have to be a great volume. Gas can be introduced into blood vessels in a variety of ways, including trauma, sexual activity, and via atmospheric pressure changes.

Traumatic causes include injuries of the neck and/or arms, including sharp force injuries or gunshot/shotgun wounds, and a variety of medical interventions (intravenous lines, central lines, laparoscopic surgery). Women who are recipients of oral sexual activity with air being blown into the vagina can experience massive air embolism. Pregnant women are particularly susceptible. Atmospheric pressure changes that occur in SCUBA divers during rapid ascent (see Chapter 16) can experience nitrogen gas emboli.

If an air embolism is suspected based on investigative information, there are a variety of procedures that pathologists can employ at autopsy to more readily detect and document an air embolism. A simple chest X-ray will reveal a radiolucent (dark) mass of air trapped within the right side of the heart (Fig. 21.10). Some advocate the examination of the brain prior to any other internal organs in cases of suspected air embolism. The scalp should be dissected very carefully, followed by extremely careful removal of the skull, followed by the dura. If present, air bubbles can be visualized within the superficial meningeal blood vessels (Disc Image 21.14). Next, careful dissection of the skin and subcutaneous tissues of the chest allows the pathologist to then cut a “window” in the anterior chest wall to access the heart. If the heart sac (pericardium) is carefully opened anteriorly, then the sac can be filled with water, and a small incision made within the submerged right atrium will result in the release of multiple air bubbles that are easily seen. An inverted, water-filled graduated cylinder can be used to “catch” the bubbles so that actual measurement can be made. The remainder of the autopsy should involve the identification and documentation of any other pertinent findings (site of blood vessel transection, if present).

Fig. 21.10 A chest X-ray demonstrating a radiolucent (*dark*) area of air within the heart (*arrows*)



Bullet and Other Foreign Body Emboli

Occasionally, bullets (or other items) can enter the blood vessels and embolize to distant sites. Most forensic pathologists have experienced cases where a bullet cannot be easily found, even on X-ray exam. In such cases, pathologists should remember that a bullet embolism may have occurred. For example, a victim with a penetrating small caliber gunshot wound of the chest is found to have no bullet visible on chest X-ray. On internal examination, the bullet pathway passes into the left ventricle. A subsequent X-ray of the legs reveals the presence of a bullet within the left femoral artery.

Exhumations

“Exhumation” is the word used to describe a situation where a previously-buried dead body is “dug up,” “unearthed,” or “disinterred” (Fig. 21.11). The usual situation involves a body that was not originally autopsied but which, for some reason, must be exhumed in order for an autopsy to be performed. Occasionally, a body is exhumed for a second (or third, etc.) autopsy to be performed. While most exhumation cases that are evaluated by forensic pathologists are cases that are disinterred from legally-authorized burial sites (cemeteries), occasional bodies that are “exhumed” represent remains that have been buried as a result of some type of disaster (mine collapse, avalanche, etc.), or buried secretly by another person. While the latter examples actually represent “exhumations” in the strict sense of the word, most forensic pathologists limit the use of the word to cases that have been legally



Fig. 21.11 An exhumation

buried in cemeteries. In many jurisdictions within the United States, bodies that are to be buried must first be embalmed. As such, most exhumed bodies will show evidence of having been embalmed. The embalming process is performed to preserve the body tissues; however, the process also creates a variety of obstacles to overcome when performing an autopsy (see below). In bodies that are buried without having been embalmed, decompositional changes are typically evident. In soil, the decomposition process tends to slow down considerably. If an unembalmed body happens to be buried within an air-tight casket, as is legal in some jurisdiction, the decomposition that ensues eventually becomes totally “anaerobic” (without oxygen), with the production of incredibly putrid odors (Fig. 21.12).



Fig. 21.12 Anaerobic decomposition occurring in an unembalmed woman who was buried in a sealed casket for seven months

The embalming process is performed in order to preserve the tissues of the body, especially for the short term, so that viewings of the body can occur for up to several days following death. Embalming within the United States generally involves two basic procedures. In one, commonly referred to as “arterial embalming,” the mortician cuts through the skin to identify blood vessels (both artery and vein). This tends to occur in the lower neck region, on one side or the other, although other locations, such as the groin, can also be utilized (Disc Image 21.15). Once the vessels are identified, tubes are inserted into the vessels, and embalming fluid is injected into the artery, while blood is drawn out of the vein. Embalming fluid contains a variety of chemical substances, including formaldehyde, which functions to “fix” tissues, thus slowing down the decomposition process. The second procedure used for embalming involves the use of a “trocar,” which is a relatively large, pipe-like metal object with a sharp, pointed end. A puncture hole is made within the abdominal wall, the pointed end of the trocar is inserted through this hole, the opposite end of the trocar is connected to a hose that supplies embalming fluid, and the trocar is repeatedly moved into and partially out of the trunk region, via the abdominal hole, such that the internal organs of the chest and abdomen are perforated and bathed in embalming fluid. The trocar embalming process creates numerous perforations throughout the internal organs (Fig. 21.13 and Disc Image 21.16). If, for some reason, it is known that an autopsy will be performed after a body is to be embalmed, it is best to ask that only the arterial method be used, in order to avoid these perforations and the confusion that they might cause.

Fig. 21.13 Trocar puncture marks in a heart



Other items of note that may be present in previously embalmed bodies include the presence of make-up, plastic “eyecaps” covering the eyes (Disc Image 21.17), a metal wire that keeps the mouth from gaping open (the wire attaches the maxilla

and mandible) (Disc Image 21.18), various locations, including the mouth, nose, and sites of skin incision, containing cotton and/or other packing material (Disc Image 21.19), and the presence of a plastic “button” within the trocar puncture hole in the abdominal skin (Fig. 21.14 and Disc Image 21.20).

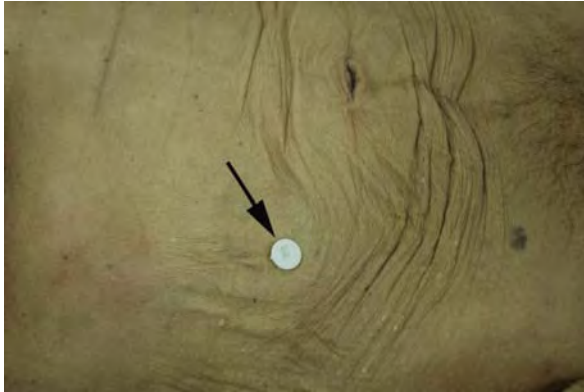


Fig. 21.14 Trocar or embalming button (*arrow*)

Other findings in embalmed bodies tend to depend on the length of time that a person has been buried and the extent and quality of embalming (Fig. 21.15). In poorly-embalmed persons, continued decomposition may occur relatively quickly, with anaerobic odors as described above. In very well-embalmed persons, remarkable body preservation may be observed several years following death. Even in relatively well-embalmed persons, the skin tends to darken and become somewhat dry after years of being buried (Disc Image 21.21). It is also very common for



Fig. 21.15 An embalmed body, three months after having been buried. Note the relatively good preservation of the body. The substance on the face is mold, which can be washed away

various types of mold to develop on the skin of embalmed bodies. This can actually occur relatively quickly (months), even in very well-embalmed bodies. In cases that have been buried for many, many years (decades and longer), there may be little or no soft tissue remaining on the body. Disintegration of clothing also occurs.

Explosions and Blast Injuries

For most forensic pathologists in the United States, encountering fatalities related to explosions occurs infrequently. Exceptions include the forensic pathologists working within the Armed Forces. In the non-military sector, explosion deaths are infrequent, but still common enough that most forensic pathologists have examined at least several cases during their careers.

Perhaps the most common type of non-military explosion involves the explosion of some type of petroleum product. This may occur as part of a vehicular accident or when a structure explodes following a natural gas leak. Terrorist and military attacks may involve a variety of explosive types, including both low order and high order explosives (see below).

There are four categories of “blast injuries” associated with explosions. A “primary blast injury” is caused by a blast wave striking the body. Blast waves that strike a body via the air tend to produce ear and airway injuries, and patterned contusions on the lungs that mimic the rib/intercostal space pattern may be produced. Blast waves that strike a body in water (underwater explosion) tend to produce intra-abdominal injuries, including perforations of hollow organs (stomach, intestines). A “secondary blast injury” occurs as a result of flying debris striking the body. A “tertiary blast injury” describes injuries caused when the moving body, propelled by the blast, strikes another object. A “quaternary blast injury” is any other injury not defined as primary, secondary, or tertiary. Examples include crushing injuries from a collapsing building and burn injuries.

Low order explosives include gunpowder, pipe bombs, and oil- or petroleum-based explosives. Low order explosives result in subsonic pressure waves that are able to produce blast injury types 2–4. High order explosives produce supersonic pressure waves and can cause all four blast injury types. High order explosives include ammonium nitrate (AN), nitroglycerin (NG), trinitrotoluene (TNT), nitrocellulose (NC), cyclotrimethylene trinitramine (RDX), pentaerythritol tetranitrate (PETN), C4 (RDX and oil), Semtex (RDX and PETN), and dynamite (NG, NC, sodium nitrate, and combustibles).

High-Profile Cases

A so-called “high-profile case,” from a forensic death investigation standpoint, can be said to include any case that involves (or *may* involve) intense publicity or scrutiny related to the autopsy itself, the specific circumstances of the case, or the

identity of the decedent or other persons involved in the death. Examples of cases that should be considered “high-profile” include in-custody deaths, police-involved deaths, deaths involving well-known persons (politicians, professional athletes and entertainers, and other public figures), and deaths involving unusual, bizarre, highly public, or politically charged circumstances. An example of the latter would be a death resulting from a “hate crime,” which is a crime in which the perpetrator’s conduct is motivated by hatred, bias, or prejudice, based on the actual or perceived race, color, ethnicity, national origin, religion, sexual orientation, gender, gender identity, or disability of another individual or group. Mass fatality incidents and in-custody deaths are two subtypes of high-profile cases that have specific sections within this chapter. The comments that follow apply to these, as well as to other high-profile cases.

When a high-profile case is encountered, all those involved in the investigation of the case should anticipate intense scrutiny. As such, an attempt should be made to predict and address all issues that might become important in such a case. Obviously, this is not necessarily an easy task. As has been advocated elsewhere in this text, detailed and complete investigation and documentation should be part of every death investigation. It is an extremely good idea to perform an autopsy on these cases, even if one might only perform an external examination in a similar case that is not considered high-profile. In high-profile cases, as well as certain other cases, and depending on the specifics of the case at hand, a variety of ancillary autopsy procedures should be considered. These include taking additional photographs, photographically documenting “negative” findings (lack of injuries), collecting and retaining clothing and trace evidence, collecting a sexual activity kit, performing radiologic examination, performing anterior and posterior neck dissections, performing additional skin incisions looking for subcutaneous trauma, performing additional specialized dissections (eyes, spinal cord, etc.), performing additional special studies (microbiology, chemistry, etc.), documenting presence or absence of genital trauma, performing extensive toxicology testing, collecting and saving extra blood and tissue samples, performing histologic examination, retaining important organs/tissues for further dissection/examination, inviting impartial witness to observe autopsy, videotaping autopsy, delaying death certification until additional follow-up information is obtained, deciding to delay releasing the body so that additional examination can be performed in subsequent days, keeping family members updated regarding results and progress, and being careful and truthful when dealing with the media. Certainly, some of these suggestions should be implemented in every case; however, in high-profile cases, deliberate attention should be paid to these issues.

Homicide by Heart Attack

The term “homicide by heart attack” is used by some forensic pathologists to describe a situation where the final mechanism of death is related to underlying heart disease via an arrhythmia which is induced by the physical and/or emotional

stress associated with the criminal activity of another individual. Physical contact and/or injury does not have to occur in such cases, and the victim does not necessarily have to be specifically threatened by the perpetrator. The action of the perpetrator should be of such severity and have sufficient intent to frighten, injure, or kill, so as to lead to a charge of homicide if the event had led to lethal injury. Additionally, the victim should have realized that the threat to personal safety was present, either to themselves or a loved one or friend. The circumstances of the event should be of a highly emotional nature. The collapse, and often the subsequent death, must occur during the emotional response period, even if the criminal act had already ended. Autopsy should demonstrate heart disease of a type that is commonly associated with a predisposition to a lethal arrhythmia (irregular heart rhythm) (Disc Image 21.22). In the absence of such a disease, the case may involve a functional cardiac disorder, such as a conduction system disorder.

Certain other natural disease processes may contribute to the mechanism of death in a homicide; ruptured cerebral artery aneurysms or arteriovenous malformations secondary to head trauma are the most common examples.

In-Custody Deaths

An “in-custody death” refers to any death occurring while a person is in the custody of a police agency, jail, detention center, or other penal institution. Some expand the definition to include psychiatric patients (and others) who have been placed in an institution or hospital against their will. By their very nature, in-custody deaths should be considered high-profile cases, and as such, those involved in the official investigation of such deaths should consider implementing the suggestions as described above in the section entitled “High-Profile Cases.”

In persons who are in police custody or incarcerated, it is generally a good idea to photographically document the presence or absence of injuries. This should include overall external photographs of the entire body, as well as some close-ups and some special shots. Many pathologists take close-up photographs of the wrists and soles of the feet, both before and after cutting very superficially into the skin and subcutaneous tissues (Figs. 21.16 and 21.17). If injuries are present (from ligatures or handcuffs on the wrists or from baton strikes to the soles of the feet), they should be evident, especially after skin incision. Another photograph that is commonly taken is a shot of the external genitalia, including one with the scrotum raised (to view the back and underside) (Disc Image 21.23), again for the purpose of showing presence or absence of injury. Internally, photographs of the subscalpular area after scalp reflection, the brain after skull cap removal, and anterior and posterior neck dissections, will likewise document the presence or absence of injuries.

Occasionally, a person in police custody or a recently-jailed individual will die of a massive drug overdose, because they have attempted to “hide the evidence” by swallowing a large quantity of an illegal drug. On internal examination at autopsy, it is important to inspect and possibly save the gastric (stomach) and/or duodenum

Fig. 21.16 Wrist incision in a custody death, showing absence of injury



Fig. 21.17 An incision of the sole in a custody death, showing absence of injury



contents. Persons in jail or prison may die of natural disease, accidents, including drug overdoses, suicides, and homicides.

In persons who die during or shortly after confrontation with police, a number of additional issues may arise. This is particularly true if the decedent was physically subdued by police. It is common for an individual who is resisting arrest to require several police officers to subdue him. A variety of weapons, maneuvers, and strategies have been employed by the police in such situations. Many of these

Fig. 21.18 A man who died of cocaine-induced excited delirium. Death occurred many minutes following subdual by police. During the confrontation with police, the man was shocked with an electronic shock device, the darts of which remain in the decedent's back



require physical contact with the victim, and most if not all of them contribute to the physical and emotional stress of the ensuing struggle. Included here are various neck holds as described in the asphyxia chapter (choke hold, carotid sleeper hold), pepper spray, Mace, police batons, bean bag guns, various wrestling-type maneuvers, including placement of the individual face down (on his belly), with his hands behind his back, with or without one or more officers applying pressure to his back, the “hogtie” position (face down, on belly with hands behind back and tied to the ankles, which are in close proximity to the hands because the knees are flexed), and electronic stun guns and similar devices (Figs. 21.18 and 21.19 and Disc Images 21.24, 21.25, and 21.26). When respiratory compromise is considered



Fig. 21.19 Close-up of electronic shock device darts. It is important for pathologists to note the exact location of the darts and their depth of penetration



Fig. 21.20 In-custody death reconstruction

to be a result of police action, some pathologists prefer to use the term “restraint asphyxia.” Others simply prefer to use the more general terms “police restraint” or “subdual.” When a specific weapon type or maneuver is considered contributory, it is acceptable to specifically mention these.

Each of the above-mentioned devices and maneuvers has been implicated as a contributory factor in certain deaths that occurred during or shortly after police confrontation. It cannot be emphasized enough that reconstruction of the events is absolutely essential in these cases (Fig. 21.20 and Disc Image 21.27). If possible, death investigators and pathologists should be called to the scene, so that interviews and event reconstruction can take place immediately. Review of police videotapes and audiotapes can be very valuable. Constructing a minute-by-minute, or even a second-by-second timeline of the event is desirable. Ultimately, it is up to the pathologist to decide whether or not police actions caused or contributed to death. In some cases, this decision is relatively straightforward. For example, if police officers held a baton across the victim’s neck and applied force until the victim stopped struggling, and at autopsy, there are facial petechiae, neck muscle hemorrhage, and a fractured hyoid bone, with no other explanation for death, then there is little question that the police action was related to death. Another example of a relatively straightforward case is that of a victim who is successfully apprehended, handcuffed, and allowed to sit on the ground, where he is conversant and in no apparent distress for 20 minutes following the event, but subsequently suddenly collapses and dies. In this situation, since the emotions and stress of the event have subsided many minutes previously, it is not reasonable to conclude that the police action contributed to death. Difficult cases exist between these two extremes, and each case must be evaluated on its own merits.

In many cases, the victim is found to have probably experienced “excited delirium” during the police confrontation. This condition may be induced by several drugs, most notably cocaine. Postmortem toxicology tests will show evidence of recent cocaine use. Other drugs that may induce excited delirium include

amphetamines, LSD and PCP. Conversely, it can also occur in various psychiatric conditions. In excited delirium, the victim experiences major cardiovascular manifestations (increased heart rate and blood pressure and an increased risk for arrhythmias, or abnormal heart rhythms, some of which may be lethal), paranoid behavior, marked agitation, superhuman strength, and hyperthermia, among other symptoms. This condition can result in death even without police or other intervention; however, when police intervention does occur, certain interventional maneuvers and/or devices can, depending on the timing and circumstances, be considered contributory to death. It can be tempting to single out a particular device or maneuver to include on the death certificate, and it may occasionally be appropriate to do so; however, if an exact mechanism cannot be attributed to the device or maneuver, it is probably best to use a more general description of the restraint/subdual, thus implicating the stress involved with the police intervention. An example of a cause of death ruling in such a case might be: I – Cocaine-induced excited delirium; II – Physical restraint by police. If police action does contribute to death, it is appropriate to rule such deaths as homicides. It should be emphasized that such a ruling is a medical ruling, not a legal ruling, and in many such instances, the police action is subsequently determined to be reasonable and justified, especially considering the alternatives (not apprehending an individual who is likely to harm someone else, or simply shooting the individual, which is far more likely to end in death). Others prefer to rule the MOD as “accident” or “undetermined.” A final note is that some do not prefer to use the term “excited delirium;” instead, they prefer the term “sympathomimetic poisoning syndrome” or “sympathomimetic toxicity syndrome” or some similar terminology to describe the massive neurologic reaction that occurs in these people.

Mass Fatality Incidents

Mass fatality incidents, sometimes referred to as “multiple fatality incidents,” can range from relatively small incidents, with a relatively small number of victims, to major catastrophic events, such as occurred with the Indian Ocean tsunami in December 2004. Mass fatality incidents may involve natural disasters or man-made events. Examples include weather-related and other environmental disasters (heat, cold, tornadoes, floods, conflagrations (fires), mudslides, hurricanes, volcanic eruptions, and earthquakes), infectious diseases, toxic environmental conditions (natural or man-made), transportation events (motor vehicles, trains, planes, ships), fires and explosions, mass shootings, and acts of terrorism or war. In any mass fatality incident, multiple different agencies and institutions, including many from all different levels of government, will be involved, including police, fire and rescue, healthcare, etc. Usually, the local death investigation agency will have jurisdiction over the dead bodies. Cooperation between all of the groups involved is an absolute necessity in these cases, as is involving local funeral directors.

Planning is the key to being adequately prepared for a mass fatality incident. Each death investigation office/jurisdiction should have a detailed mass disaster plan, with

sufficient supplies and workforce available to implement the plan if required. Mass disaster drills should occur periodically. Unfortunately, even after several major disasters that have occurred over the past decade, far too many local disaster drills do not include death investigation offices. Although the Disaster Mortuary Operational Response Team (D-MORT) is available for certain disasters, local authorities should not automatically assume that D-MORT will always be available. In other words, if an office's mass disaster plan relies on D-MORT, the plan is not sufficient.

Some of the major issues of concern related to mass fatality incidents include search and recovery, tagging and documenting remains, possible decontamination concerns, body transportation, body storage (frequently available within pre-arranged refrigerated semi-trailers), temporary morgue sites, ability to operate without power, identification of bodies, identification of body parts (in certain disasters), documentation of injuries, body disposition, incident reconstruction, security issues, communication with family members, and media relations. It is beyond the scope of this text to provide detailed discussion of these and other important issues; however, a few will be addressed below. The reader is referred to several excellent resources for further details.

The major concerns within the death investigation community regarding most mass fatality incidents include identification of victims and determination of cause of death. It is imperative that obviously dead bodies be left alone be rescue workers. The bodies should not be undressed or moved. Depending on the disaster type, the exact location of a body can be extremely useful in working toward establishing positive identification. Scientific means of identification is essential in mass fatality incidents.

Another very important aspect in certain mass fatality incidents is the concept of decontamination. Unfortunately, depending on the contaminant involved (chemical, biological, radioactive), it is unlikely in many situations that dead bodies can be adequately decontaminated. For this reason, a better term and concept is that of containment. The goal should be to contain a potential contaminant as much as possible and to protect those who must work with the bodies. As such, the setting-up of temporary morgue facilities is essential in planning for a mass disaster incident. In a variety of potential mass disaster incidents, the utilization of the existing morgue facilities is not advisable.

Multiple Causes of Death

There are many deaths that result from a combination of multiple, separate entities, be they traumatic injuries or natural disease processes. The reader will recall from Chapter 5 (Death Certification) that the death certificate has space for two unrelated causes of death (parts I and II). In a case where there are two factors contributing to death, it is appropriate to list one of them in part I and the other in part II. When there are more than two contributing factors, an interesting challenge presents itself. Some choose to list as many possible factors (causes), separated by semicolons. Both part I and part II may be utilized in this format. For example, in a homicide

victim who dies from a combination of blunt force injuries, sharp force injuries, and strangulation, the death certificate might read as follows: “blunt force injuries; sharp force injuries; strangulation” (Fig. 21.21). Another option might be to use a more descriptive phrase, such as “combined blunt force, sharp force, and strangulation injuries.” Others choose to use a more general description, such as “multiple severe injuries.”



Fig. 21.21 Multiple injury types contributed to this victim’s death, including blunt force, sharp force, and strangulation

Nutrition and Hydration Disorders

Nutritional and hydration disorders encompass a wide range of disorders, including overindulgence (obesity), insufficient nutrition, various psychiatric disorders (anorexia and bulimia), dehydration, and overhydration. Many of these are properly considered natural diseases, and as such might be better placed in Chapter 10 (natural deaths); however, since some can be considered accidental, or even suicidal or homicidal, this topic is addressed here.

Proper nutrition requires sufficient amounts of energy (calories) in the form of carbohydrates, fats and proteins, essential amino acids and fatty acids, and vitamins and minerals. A diet deficient in any of these required components will lead to malnutrition. In “primary malnutrition,” one or several of these components is missing from the diet. In “secondary malnutrition,” each of the components is present in sufficient amounts within the diet, but malnutrition occurs because of problems associated with absorption, usage, storage, excessive losses, or increase requirements. Overabundance of calories can result in obesity, with or without associated deficiencies of other required nutritional components. In addition to the nutrients described above, normal metabolism requires sufficient amounts (but not excessive amounts) of water. A lack of water can result in “dehydration,” which can cause death if severe enough. In contrast, excessive water intake can lead to electrolyte disturbances and death.

Obesity

Obesity is considered by some to be an epidemic in the United States. In general terms, obesity refers to being overweight. Perhaps the best measure of obesity is the body mass index (BMI), which is calculated by dividing a person's weight in kilograms by the height in meters squared: $BMI = (\text{weight in kg})/(\text{height in m})^2$. Using common USA units: $BMI = ((\text{weight in pounds})/(\text{height in inches})^2) \times 703$. A normal BMI ranges between 18.5 and 24.9. Values between 25 and 29.9 designate those who are "overweight." Obesity is defined as values over 30. Obesity is of grave concern because there is an increased risk of morbidity and mortality in obese individuals (Fig. 21.22). Specific obesity-related diseases (those diseases that have an increased incidence in obese individuals) include diabetes mellitus, hypertension, and coronary artery disease, where obesity may be listed as a contributory factor in deaths due to these entities. Obesity can also be considered an underlying primary cause of death, for example in cases due to dilated cardiomyopathy or deep venous thrombosis with pulmonary thromboembolism.



Fig. 21.22 Morbid obesity. The weight of the decedent was in excess of 700 pounds

Malnutrition and Starvation

Nutritional deficiencies include protein–energy malnutrition, vitamin deficiencies, and trace-mineral deficiencies. Deficiencies can occur as a result of primary and secondary causes as detailed above. Ignorance, poverty, chronic alcoholism, acute and chronic illnesses, and self-imposed dietary restrictions can be at play, even in industrialized nations with "plenty of food." Protein–energy malnutrition (PEM) is very common worldwide, responsible for the deaths of far too many persons, many of whom are children. PEM represents a range of syndromes, including "marasmus," in which there is a severe reduction in calorie intake, and "kwashiorkor," in which protein deprivation is relatively greater than the reduction in total calories. In marasmus, the extremities (arms and legs) appear emaciated. In kwashiorkor, which is

more severe, various skin changes occur, along with widespread edema (swelling). Self-imposed starvation (as occurs in hunger strikes or anorexia; see below), or starvation as a result of imprisonment or various forms of abuse or neglect, may mimic either form of PEM (Fig. 21.23 and Disc Image 21.28). Dehydration may play a role in these deaths.



Fig. 21.23 An elderly individual who was starved by “caretakers.”

Vitamin Deficiencies

Deficiencies of specific vitamins or micronutrients are known to occur. Probably the most common that is encountered within forensic death investigations is thiamine (vitamin B₁) deficiency in the setting of chronic alcoholism. Central nervous system manifestations include hemorrhagic destruction or atrophy of a structure called the mamillary bodies. A syndrome associated with this is the Wernicke–Korsakoff syndrome. Table 21.1 provides a synopsis of vitamin deficiencies. The fat-soluble vitamins (A, D, E, K) can actually have toxicity states if too much of the vitamin is ingested. In contrast, the other vitamins, which are water-soluble, tend not to have toxic states associated with too much intake.

Anorexia

Anorexia nervosa can be considered self-induced starvation. It is considered an eating disorder (a psychiatric condition) and predominantly affects previously healthy young females who are obsessed with being thin. In severe cases, the physical findings are similar to those of PEM. Endocrine manifestations also occur. Skin changes consisting of a dry, yellow, scaly appearance are common. Most importantly from a death investigation standpoint is a definite increased risk of sudden death via an arrhythmia, possibly related to potassium electrolyte disturbances. Although the

Table 21.1 Vitamins and deficiency states

Vitamin	Deficiency state
A	Blindness
D	Rickets; osteomalacia (weak bones)
E	Spinocerebellar degeneration
K	Bleeding disorder
B ₁ (thiamine)	Beriberi (heart and CNS abnormalities)
B ₂ (riboflavin)	Eye, skin, and mouth inflammation
Niacin	Pellagra (dementia, dermatitis, diarrhea)
B ₆ (pyridoxine)	Mouth and skin inflammation; neuropathy
B ₁₂	Anemia; spinal cord degeneration
C	Scurvy (poor healing)
Folate	Anemia; neural tube defects in fetuses
Pantothenic acid	None recognized
Biotin	None recognized

proper manner of death ruling in anorexia-related deaths can be controversial, a “natural” ruling seems to be most appropriate.

Bulimia

Bulimia is another eating disorder that may occasionally be associated with sudden death. In this condition, which is also most common in young females, the affected individual binges (consumes a great deal of food), followed by self-induced vomiting. Typically, a bulimic’s weight remains near normal, but serious medical complications can be related to electrolyte disturbances due to repeated vomiting, aspiration of gastric contents into the airways, and esophageal or cardiac rupture related to severe retching. Although some controversy exists over the best manner of death ruling in bulimia-related deaths, “natural” seems most appropriate.

Dehydration

Dehydration occurs when there is insufficient water to maintain normal metabolism. Cases of dehydration encountered by the death investigation community frequently involve elevated levels of sodium (as measured in vitreous fluid; see below); however, it is important to remember that, from a clinical standpoint, dehydration can be hypernatremic (elevated sodium), hyponatremic (decreased sodium) or even isonatremic (normal sodium levels). Dehydration may play a role in certain deaths, particularly those involving individuals who are dependent on others for their nutritional and health care (the elderly and the very young), and in those experiencing various forms of malnourishment, as described above (Fig. 21.24). Forced salt ingestion occurring in child abuse can result in a severe hypernatremic dehydration.



Fig. 21.24 A case of severe dehydration, with “sunken eyes.”

Dehydration may occur as part of the final mechanism of certain natural deaths, including Alzheimer’s disease. Further discussion of dehydration in relation to postmortem chemistry tests is provided below.

Overhydration

Water intoxication can occur in a variety of situations. Probably the most important from a forensic standpoint are forced water ingestion occurring as a form of child abuse, and “psychogenic polydypsia,” a manifestation occurring in certain psychiatric disorders (including schizophrenia) wherein the affected person drinks so much water that a low-salt electrolyte disturbance occurs, resulting in death (see postmortem chemistry section below).

Food Poisoning

Food poisoning causes a considerable number of illnesses every year, as well as occasional deaths. Cases can be related specifically to seafood or to virtually any other type of food. The very young, the very old, and those who are otherwise debilitated are at most risk for death. While gastrointestinal symptoms are common in many cases, they do not necessarily have to occur. The causative agents include bacteria, viruses, and parasites, as well as various chemicals. Six pathogens (“germs”) account for greater than 90% of non-seafood-related food poisoning deaths: *Salmonella* (bacteria), *Listeria* (bacteria), *Toxoplasma* (parasite), Norwalk-like viruses, *Campylobacter* (bacteria), and *E. coli* 0157:H7 (bacteria). Unfortunately, in a majority of cases of suspected food-poisoning deaths, a causative organism cannot be identified.

Seafood may cause infectious or toxin-mediated diseases that can result in death. It is beyond the scope of this text to deal with these entities in detail. A variety of

infectious organisms can be transmitted by seafood, particularly uncooked seafood. The bacteria *Vibrio vulnificus* is particularly virulent (causing serious illness) and has been reported in persons consuming raw oysters and clams. The disease known as “ciguatera” is caused by consuming various fish, including barracuda, grouper, snapper, and sea bass, that have an elevated level of the neurotoxin produced by the dinoflagellate *Gambierdiscus toxicus*. “Scombroid” is a foodborne, toxin-related illness related to the improper refrigeration of fish that contain abundant histidine (mackerel, swordfish, moray eel, mahi-mahi, tuna, and bluefish). Bacteria interact with the histidine to produce a toxin called “saurine.” Consumption of mollusks (mussels, clams, oysters, scallops) contaminated with the dinoflagellate *Ptychodiscus brevis*, whether cooked or not, can result in “neurotoxic shellfish poisoning.” “Paralytic shellfish poisoning” is caused by consuming mollusks that are contaminated with a third dinoflagellate species, *Gonyaulax*, famous for being present in “red tides.” The toxin is also heat-stable (it will not be destroyed by cooking). “Tetrodotoxin fish poisoning” is associated with consuming improperly prepared puffer fish (fugu). The toxin is another heat-stable neurotoxin. It has been reported in other species as well, including octopus, salamanders, and the California newt.

Occupational Deaths

Deaths related to one’s occupation are not infrequent. There are three basic categories: deaths occurring on the job and related to the job, deaths occurring on the job but unrelated to the job, and deaths occurring as a result of long-term occupational exposures. Deaths occurring on the job and related to the job are usually related to accidental trauma sustained while the person is engaging in specific work-related activities. Construction and industrial workers are most likely to be the victims in such cases, although certain other highly-specialized vocations may be considered very risky. Whenever a death occurs on the job and is believed to be related to the job itself, or the job environment, then the Occupational Safety and Health Administration (OSHA) will probably be involved in the investigation.

Deaths occurring on the job but unrelated to the job are typically the result of underlying natural disease, although accidental, suicidal, and even homicidal deaths may occur at job-sites. Identifying the cause of death, and understanding the specific duties of the individual at the worksite, are of utmost importance in identifying such cases.

Work-related deaths that occur following many years of exposure to various toxic substances usually fall under the category of “occupational lung diseases.” These are discussed in slightly more detail in Chapter 10 (natural diseases). Like deaths related to chronic alcohol use, these deaths are frequently considered natural, even though an environmental factor causes or contributes to death. Pathologists investigating such deaths may wish to retain fairly sizable sections of lung tissue, in the event that specialized testing is eventually performed.

Organ and Tissue Procurement Issues

During the past several decades, the science of organ transplantation has revolutionized the treatment of several diseases such that, every year, thousands of people's lives are saved by organ donation/transplantation. In a similar way, the utilization of tissues (as opposed to entire organs) recovered from dead bodies has increased tremendously over the past several decades, such that great advances have been made in the treatment of numerous diseases and injuries. It should therefore come as no surprise that death investigation offices have frequent interaction with organ and tissue procurement agencies. In fact, a vast majority of organ donors fall under the jurisdiction of the local medicolegal death investigation office, since brain death usually results from a non-natural process (an injury). As such, in most states, not only does the organ or tissue procurement agency require consent from the legal next-of-kin (or prior consent from the decedent), but in medical examiner/coroner cases, the medical examiner/coroner must also give approval for procurement.

Before discussing this issue further, it is important to make a clear distinction between "organs" and "tissues" as these two terms are used within this arena. As mentioned, for both, legal consent (next-of-kin or self-designated, depending on state laws) must be obtained before organs or tissues can be recovered. An organ donation refers to the removal of an entire whole organ from a donor, with subsequent surgical insertion into a recipient. While certain organs (kidneys) can be removed from a living donor, for cases that involve the death investigation community, all organs (including kidneys) that are removed for "organ transplantation" can only be removed after the donor has been declared legally "brain dead." This determination occurs in a hospital setting, with the donor maintained on "life support" (a ventilator or "breathing machine"). The donor's heart remains beating on its own, even though the person has been declared legally "dead." Establishment of brain death requires various medical tests, with confirmation by more than one physician. After brain death has been established, an official "time of death" is declared, but the donor remains connected to the ventilator (with the heart beating). Subsequent organ removal for transplantation purposes must occur in a sterile operating room (Fig. 21.25). After brain death is established, the donor frequently remains on the ventilator for many hours before the procurement surgery is performed. During this time, various additional testing will typically occur on the donor, organ recipients will be contacted and prepared for surgery, and the transplantation surgical team must arrive at the hospital. It should therefore be evident that in organ donation cases it is impossible to perform an autopsy prior to organ recovery – it is not medically feasible. By definition, when an organ donation involves a medical examiner/coroner case, the donation occurs prior to the official postmortem exam (autopsy).

As the science of transplantation advances, attempts are being made toward procuring organs from patients who are officially declared dead (not just "brain dead," but also having no heart beat), so long as various criteria are met. In other words, the hope is that certain organs may still be utilized from patients who die, but are not "maintained" on a ventilator. Various names have been applied to these



Fig. 21.25 A decedent who was declared brain-dead is being operated on for removal of organs for transplantation. Organ removal for transplantation must occur in the sterile environment of an operating room

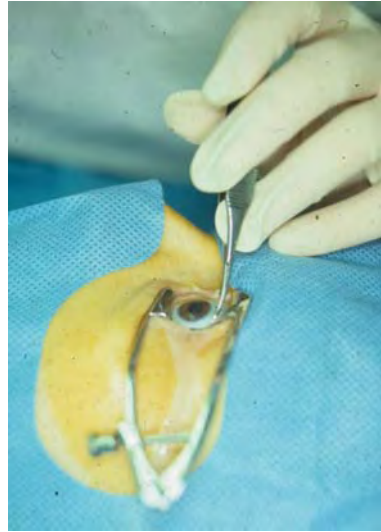
procedures, including “rapid organ recovery.” If this proves successful, it would have an huge impact on the availability of organs for transplantation. At the same time, it would markedly increase the number of cases requiring cooperative efforts between the organ procurement agencies and the death investigation community.

In contrast to “organ” donation, at the present time, “tissue” recovery can occur after the heart stops beating. The term “tissue” in this context refers to skin, bone, corneas (from the eyes) (Figs. 21.26 and 21.27), and some other tissues, including heart valves. With the exception of heart valve recovery, which must occur prior to autopsy (because the entire heart must be removed in as sterile (germ-free) an environment as possible, with subsequent valve removal) (Disc Image 21.29), the



Fig. 21.26 A body following skin and bone removal for transplantation purposes

Fig. 21.27 Cornea removal for transplantation purposes can be performed in the morgue, as can skin, bone, and heart valve recovery



other tissues can be recovered after autopsy, as long as this occurs within certain time restrictions. In many cases, these tissues can even be collected prior to autopsy, so long as it will not compromise the autopsy examination. Tissue recovery does not require the donor to remain on a ventilator and in the hospital, and it does not need to occur in an operating room. In fact, many larger medical examiner/coroner offices have specially designated space for tissue recovery.

Potential conflicts may occur if organ or tissue recovery efforts interfere with subsequent forensic evaluation of a particular case. Many death investigation offices have outstanding working relationships with their local organ procurement agencies, as well as their local tissue procurement agencies. Protocols have been established, and cooperative efforts from all sides allow for maximum donation while maintaining important forensic evidence. In many jurisdictions, it is virtually unheard of for a medical examiner to deny a request for organ donation, even in homicide cases (and even in child abuse homicide cases). To assist this, organ procurement agencies and hospital staff tend to do all they can to ensure the proper documentation of injuries and evidence, along with evidence collection, etc. Allowing death investigators and police to examine the body prior to organ recovery surgery, the performance of various imaging procedures (CT, MRI) to define and/or rule out injuries, and the overall recognition of the importance of forensic evidence by the transplantation team all go a long way to establish this cooperative effort.

Most forensic pathologists do all that they can to support and allow life-saving organ transplantation, including attending organ procurement surgery if need be. The good that results (saving lives) is worth the additional efforts. When it comes to tissue procurement cases, however, most forensic pathologists contend that the forensic evidence issues must take precedence over tissue recovery in certain cases. Although tissues certainly have life-enhancing effects, the situation that exists

with organs (people on waiting lists who may die if they do not receive a suitable organ), by and large does not apply to tissue recovery efforts. Unfortunately for the tissue procurement community, there is a small percentage of cases that cannot be disturbed prior to autopsy. Because of this, a small percentage of potentially suitable tissue donors will be unsuitable for tissue recovery after autopsy, because the time limitations for recovery will have been exceeded. Some of the cases that fall into this category include any case where the examination for and collection of trace evidence is required, including homicides, suspected homicides, cases with unknown circumstances, and pedestrian fatalities, to name a few. In these cases, the forensic issues must take precedence over the tissue recovery concerns.

A recent unfortunate occurrence involves an attempt by the tissue procurement community to override or amend existing laws via the Revised Uniform Anatomical Gift Act. In this model legislation that has been distributed to all state legislatures, language exists that essentially elevates “tissues” to equal status with “organs” by simply referring to them as “parts.” Efforts have been successfully made by the death investigation community in many states to eliminate or change this language so that it better maintains the necessary differences between organ and tissue cases. If the original language of this model legislation is adopted and implemented as written, there is no doubt whatsoever that valuable forensic evidence will be compromised or lost altogether, prosecutions of cases will fail, and, in some cases, pathologists will be unable to determine a cause of death.

In cases where organ or tissue recovery occurs prior to autopsy, autopsy performance can occur as close to usual as possible, recognizing that the bodies have obviously been previously “disturbed,” such that trace evidence and other evidence (clothing) is either no longer present or has been contaminated or compromised. Also, parts of the body (organs and/or tissues) will obviously be absent. As with the identification and documentation of medical therapy in other forensic cases, the extent of organ/tissue procurement should be documented, and the pathologist must be careful not to misinterpret findings that resulted from procurement as true injuries. In some instances, the procurement procedures may partially or totally obscure a particular part of the body, such that optimal evaluation is impossible at autopsy. It may be very important in such instances for those who performed the procurement to share with the pathologist any pertinent findings that were discovered during the procurement procedures. For this reason, it is recommended that documentation of the presence or absence of injuries be produced by procurement personnel and incorporated into the official medical record, with copies forwarded to the death investigation office.

As part of the tissue procurement procedure, the agency typically collects numerous samples of blood, in order to test for various infectious diseases to ensure the safety of the tissues. If autopsy occurs prior to tissue procurement, pathologists will typically collect these samples for the tissue procurement agency. In cases where tissue procurement is allowed to occur prior to autopsy, medical examiners/coroners should instruct tissue procurement agencies to avoid drawing blood from the neck/subclavian region. If blood collection from the neck/subclavian

region occurs prior to autopsy, postmortem bleeding into the soft tissues can be quite extensive, precluding optimal internal neck examination (Disc Image 21.30).

Postmortem Chemistry Tests

The term “postmortem chemistry” refers to a variety of postmortem tests that can be performed in the laboratory, excluding toxicology tests and microbiology tests (cultures). While blood and urine and other fluids can be utilized in these tests, vitreous humour (eye fluid) is one of the most common fluids used for these tests. Postmortem chemistry test results may assist in determining the cause of death, evaluating the physiologic effects of anatomic lesions, diagnosing antemortem biochemical abnormalities, and estimating the postmortem interval (time of death).

Many laboratory tests that can be performed on samples from living patients cannot be performed or are unreliable on postmortem samples. Of particular note is the fact that the following tests/diagnoses are not reliable within or based upon postmortem samples: oxygen content, carbon dioxide, pH, hypoglycemia (low blood sugar), and hyperkalemia (elevated potassium levels). The general rule of thumb is that certain postmortem chemistry tests (as indicated below) are fairly reliable in the “early postmortem period” (before decomposition is evident).

Glucose: Hypoglycemia cannot be diagnosed after death. Blood glucose levels are not reliable, and most of the time, as a result of postmortem glycolysis (breakdown of glucose), blood and vitreous glucose levels are low. Occasionally, postmortem glycogenolysis (breakdown of glycogen into glucose) within the liver results in elevated heart blood glucose. If vitreous glucose levels are elevated (>200 mg/dL), diabetic ketoacidosis should be considered as a cause. The presence of acetone confirms the existence of ketoacidosis. Intravenous glucose administration immediately prior to death may also explain an elevated vitreous glucose.

Nitrogen Compounds: Postmortem levels of urea nitrogen and creatinine remain stable in postmortem blood and vitreous, with vitreous levels essentially equivalent to blood levels. Elevated levels should lead to a suspicion of renal (kidney) failure or dehydration.

Electrolytes: Potassium levels within the blood begin to increase rapidly immediately after death. Consequently, a postmortem diagnosis of hyperkalemia (elevated potassium) is not possible. Vitreous levels increase, but not as fast as in blood. There are some mathematical equations that use vitreous potassium levels to estimate the postmortem interval; however, the answers have a huge margin of error, such that the results are highly suspect.

Postmortem sodium and chloride levels within the vitreous fluid mimic blood levels at the time of death. As a result, some disturbances of sodium and chloride levels can be diagnosed postmortem. As the postmortem period lengthens, sodium and potassium levels slowly decrease. As long as the vitreous potassium levels are under 15 mEq/L, the sodium and chloride levels are reliable. If potassium is greater

than 15, the low levels of sodium and chloride are explained by decomposition. If vitreous sodium and chloride levels are elevated, then this suggests the presence of hypernatremic (high sodium) dehydration. If the vitreous sodium and chloride levels are decreased (in the presence of “normal” postmortem potassium), then the condition is referred to as a “low salt vitreous electrolyte pattern,” which can be the sole explanation for a death related to electrolyte disturbances. It most frequently occurs in chronic alcoholics. Table 21.2 provides a synopsis of normal and abnormal “vitreous electrolyte patterns.”

Table 21.2 Vitreous electrolyte patterns

	Sodium (mEq/L)	Chloride (mEq/L)	Potassium (mEq/L)	Urea (mg/dL)	Glucose (mg/dL)
Normal	130–155	105–135	<15	<40	<200
Dehydration	>155	>135	Normal	40–100	Normal
Low-salt	<130	<105	Normal	Normal	Normal
Uremia	Normal to high	Normal to high	Normal	>100	Normal
Decomposed	<130	<105	>20	Normal	Normal
Diabetic	Variable	Variable	Variable	Variable	>200 *

Dehydration = hypernatremic type; * acetone is typically elevated in diabetic ketoacidosis

Table 21.3 provides a synopsis of some other chemistry tests that can be reliable in postmortem samples. Readers are encouraged to check with their laboratories before collecting samples or ordering tests, as some tests require specialized collection.

Table 21.3 Postmortem chemistry tests

Substance	Sample	Reliable?	Disorder	Result
Lipids and cholesterol	Blood	Sometimes	Hypercholesterolemia	Elevated
Proteins	Blood	Mostly	Many	Variable
Hemoglobin type	Blood	Yes	Sickle cell disease	Abnormal
Specific antibodies	Blood	Yes	Many	Present
Enzymes	Blood	Usually not	Many	Not reliable
Beta-tryptase	Blood	Yes	Anaphylaxis	Elevated
Cholinesterase	Blood	Yes	Insecticide poisoning	Decreased
Amylase and lipase	Blood	Sometimes	Pancreatitis	Elevated
Hormones	Blood	Some	Various	Variable
Insulin (free and total)	Blood	Yes	Diabetes mellitus	Variable
C-peptide	Blood	Yes	Diabetes mellitus	Variable

Regarding cases of diabetes mellitus, postmortem blood can be tested specifically for insulin. The usual situation involves attempting to detect whether or not an insulin overdose has occurred. In insulin-dependent diabetic patients, it is common for patients to have markedly elevated total insulin levels, with most of the insulin being bound (and unavailable) by anti-insulin antibodies. Consequently, free insulin levels are indicative of excess insulin levels. C-peptide is a substance that is formed along with insulin within the pancreas. It is not present in commercially available insulin preparations. If an insulin level is high, but the C-peptide is low, this confirms that the insulin is exogenous.

Postmortem Cultures

Microbiology cultures are typically performed on patients in order to identify the presence of and the specific microbiological organism responsible for an infection. It is very important to collect culture samples in as sterile (germ-free) an environment as possible. Morgues by their very nature are not considered sterile. In addition, since living people have a massive number of micro-organisms living on and within them (on the skin, in the mouth and other orifices, and within the gastrointestinal tract), dead people also have a great number of these micro-organisms. As decomposition ensues, overgrowth of these organisms can be substantial. Consequently, the reliability of postmortem cultures is guarded at best. Many forensic pathologists perform postmortem cultures if they suspect an infectious process. Protocols differ tremendously from one office to another, partly based on the availability and cost of such testing.

Bacterial, fungal, and viral cultures can be performed on postmortem samples. In addition, various “serologic” and molecular tests can be performed. Such tests can identify antibodies directed against micro-organisms, as well as the organisms themselves. Examples include antibodies directed against hepatitis viruses and nucleic acid molecular tests for the human immunodeficiency virus.

Particularly with regard to bacterial infections of the meninges (coverings of the brain), referred to as “meningitis,” it is prudent for pathologists to collect sterile swab samples of the purulent material (pus), send them to the microbiology laboratory, and ask specifically for a “stat gram stain.” Cultures take several days to complete; however, a stat gram stain allows laboratory technicians to look at the sample under the microscope, apply a special stain referred to as a Gram stain, and comment on whether or not bacteria are visualized. If they are seen, the Gram staining characteristics (Gram-negative or Gram-positive) and the shape of the bacteria can be immediately reported. A particularly contagious and deadly bacteria known as *Neisseria meningitidis* (or meningococcus) is a Gram-negative diplococcus. Discovery of this organism at autopsy should prompt all in attendance at the autopsy to receive prophylactic antibiotics. The health department should also be contacted so that all other contacts with the person can be treated.

Pregnancy-Related Maternal Deaths

Deaths related to pregnancy can be divided into fetal/infant deaths and maternal (mother) deaths. Deaths of the fetus/infant are presented in Chapter 20 (Deaths in Infancy and Childhood). Only maternal deaths will be considered here. This section could have reasonably been included in Chapter 10 (Natural Deaths); however, as there are occasional pregnancy-related deaths that are clearly not natural, it was decided to include this important topic here. As in the rest of this chapter, it is beyond the scope of this text to include all the details regarding this topic.

A pregnancy-related maternal death can be defined as the death of a woman resulting from or related to her own pregnancy. Included here are cases that are also related to the “postpartum” period, that period of time following pregnancy/birth, variably defined as lasting from 42 days to 1 year. Pregnancy is typically considered a normal biologic process, but it is associated with various physiologic and anatomic changes that increase a woman’s risk of death. Conditions that can result in death can be divided into those that are unique to pregnancy, such as pregnancy-induced hypertension, conditions associated with pregnancy, such as an increased risk of deep venous thrombosis with pulmonary thromboembolism, and conditions unrelated to but exacerbated by pregnancy, such as deaths related to underlying heart disease. Deaths can occur during the 1st, 2nd, or 3rd trimester of pregnancy, during labor/birth, during the postpartum period, or even many years after the postpartum period. Depending on the circumstances of death, the certainty of diagnosis, and various laws regarding which cases must be reported to the coroner/medical examiner, pregnancy-related deaths may or may not be referred to the local death investigation agency. In the following paragraphs, a synopsis of the causes of maternal death in pregnancy based on mechanism of death will be presented.

A number of disorders can occur in pregnancy wherein maternal death results from hemorrhage. An ectopic pregnancy represents a pregnancy occurring outside of the uterine/endometrial cavity. A common location is within the fallopian tube (a so-called “tubal pregnancy”) (Disc Image 21.31). As the embryo and placenta grow, the tube can rupture, and the woman can bleed to death internally. Various placenta implantation disorders can lead to hemorrhage, including placenta previa (the placenta implants abnormally low, over the cervix or uterine opening), placenta abruptio (the placenta prematurely separates from the uterus), retention of the placenta (placenta accreta, placenta increta, and placenta percreta), and other forms of postpartum hemorrhage, including uterine inversion and uterine atony. Non-traumatic, spontaneous rupture of the uterus can occur during labor. If this is identified at autopsy, the pathologist should consider the possibility of an underlying connective tissue disorder, such as Ehlers–Danlos syndrome (EDS). In addition to hemorrhagic disorders specifically related to the uterus/placenta, a variety of other hemorrhagic disorders can afflict a pregnant woman. These include various forms of spontaneous intracranial hemorrhage (strokes), rupture of the spleen, rupture of the gastrointestinal (GI) tract, and vascular dissection/rupture, which can involve a number of different sites, including the coronary arteries, the aorta, and other medium

and large arteries. When splenic, GI, or vascular rupture is identified, pathologists should again be suspicious of EDS.

Pregnancy-induced hypertension (PIH) encompasses a group of overlapping disorders, each characterized by new-onset, pregnancy-induced blood pressure elevation. Disorders included within this group include pre-eclampsia and eclampsia (also called “toxemia”), and the HELLP syndrome, characterized by hemolysis (red blood cell destruction), elevated liver enzymes, and low platelet counts. Women with PIH are at increased risk for many problems during pregnancy, including problems with the liver, hemorrhagic problems, and death. In the obstetric medical literature, PIH is the leading cause of maternal deaths. Many such deaths are probably not referred to the coroner/medical examiner community, since in many cases a definitive diagnosis is known prior to death.

Two other disorders that can affect the liver of pregnant women are “intrahepatic cholestasis of pregnancy” (ICP) and “acute fatty liver of pregnancy” (AFLP). ICP is rarely fatal for the mother, but may lead to fetal death. AFLP can be fatal, typically occurring in the 3rd trimester and presenting as fulminant liver damage.

Cardiac (heart) disorders in pregnancy include non-pregnancy-related heart disease, such as coronary artery atherosclerosis or congenital heart disease, which is exacerbated by the physiologic demands of pregnancy, and two specific pregnancy-associated disorders. The first, and more common, is referred to as “postpartum” or “peripartum” cardiomyopathy (Fig. 21.28). It is a form of dilated cardiomyopathy, where the heart becomes flabby and inefficient. Onset is typically in the postpartum period, although some cases develop late in pregnancy, hence the more appropriate designation of “peripartum cardiomyopathy.” The second is “peripartum myocarditis,” wherein the heart muscle becomes inflamed and dilated, putting it at risk for a fatal arrhythmia.

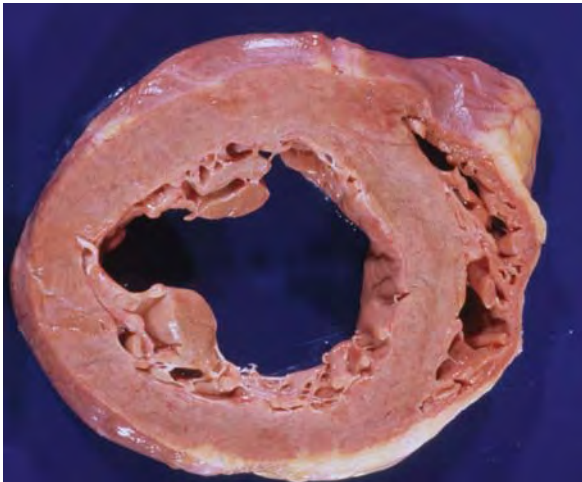


Fig. 21.28 Subtle dilation of the left ventricular cavity is evident in this case of peripartum cardiomyopathy

Several embolic disorders can occur in pregnancy. Each is more fully described above, in the section entitled “Emboli.” They include amniotic fluid embolism (AFE), pulmonary thromboembolism (PE), and air/gas embolism. Whereas AFE and PE typically represent natural deaths, air/gas embolism usually represents an accidental death.

The term “thrombotic microangiopathies of pregnancy” describes a group of primary and secondary disorders that have specific gross, microscopic, and laboratory findings and can lead to sudden unexpected death in pregnancy (Disc Image 21.32). The primary disorders include “thrombotic thrombocytopenic purpura” and “hemolytic uremic syndrome.” Disorders that can produce similar findings that are referred to as secondary thrombotic microangiopathies include PIH, HELLP syndrome, antiphospholipid antibody syndrome, AFLP, lupus, and disseminated intravascular coagulopathy (DIC).

In the industrialized world, where medical treatment is readily available, deaths in pregnancy related to infection are rare, but still occasionally occur. Certain endocrine disorders, such as pregnancy-induced diabetes mellitus (“gestational diabetes mellitus”), can lead to significant maternal morbidity (illness) and even mortality (death). The term “gestational trophoblastic disease” refers to neoplastic diseases (tumors) that arise from the placenta/fetus. Some of these are malignant and can lead to death.

A variety of pre-existing natural diseases can be exacerbated by pregnancy. Occasionally, these disorders were previously unrecognized. Besides heart disease, other disorders that can be exacerbated by pregnancy include “primary pulmonary hypertension,” asthma, seizures, and diabetes.

A majority of women within the United States choose to have medical intervention during labor and birth. As such, these obstetric procedures can be complicated by various medical-, anesthesia-, or surgery-related problems. Despite considerable advances in the world of modern medicine, such complications continue to account for a sizable number of maternal deaths. The reader is referred to the section entitled “Therapy-Related Deaths” later in this chapter.

A small percentage of maternal deaths occur from complications that are related to fetus number, size, or position within the uterus. Rare deaths occur in the setting of spontaneous abortion (miscarriage). At one time, a considerable number of maternal deaths resulted from the performance of illegal abortions, with many related to hemorrhage and/or infection. Occasional maternal deaths occur with elective (legal) abortions, including cases related to hemorrhage, infection, embolic events, and anesthesia complications.

With the exception of the air/gas embolism cases and those related to therapy, where the manner of death is usually accidental, the other causes of maternal deaths presented above represent natural deaths. Other accidental maternal deaths are known to occur, such as women who die in automobile collisions. Just as pregnancy does not protect a woman from accidental death, it does not protect her from suicide or homicide. In fact, “postpartum depression” is a well-recognized disorder that may well place a woman at increased risk for suicide. In a like fashion, for

some women, a pregnancy may well represent a motive for homicide, such as when a husband or boyfriend kills his wife/girlfriend when he discovers that the baby is not his.

Product-Related Deaths

Certain deaths are clearly associated with a specific product. Examples include deaths involving automobile collisions, deaths related to the toxic effects of pharmaceuticals (drugs), deaths related to failure of a medical device, accidental asphyxial deaths in an infant involving a small toy, etc. In these, and many other deaths involving specific products, the product itself may or may not have functioned properly. In some cases, although the product may have functioned properly, the death highlights a previously unforeseen dangerous aspect of the product. If a product is found to have functioned improperly, and this malfunction is thought to have been related to death, or if an inherently dangerous situation/characteristic is identified in a properly functioning product, it is very important to notify various governmental agencies so that other deaths may be prevented. Different agencies have different jurisdictional responsibilities. The two most likely agencies that should be notified in product-related deaths include the Consumer Product Safety Commission (CPSC) and the Food and Drug Administration (FDA).

Radiation

Radiation can be defined as energy in the form of waves or moving subatomic particles. Energy in the form of waves makes up the electromagnetic (EM) radiation spectrum, in which all forms travel at the speed of light but vary in their wavelength and frequency. From the low frequency/long wavelength end of the spectrum to the high frequency/short wavelength end, the following types of EM radiation occur: electrical energy, radio waves, microwaves, infrared, visible light, ultraviolet, X-rays, and gamma rays.

Ionizing Radiation

Ionizing radiation has enough energy to ionize atoms or molecules, whereas non-ionizing radiation does not. Within the EM radiation spectrum, only X-rays and gamma rays are ionizing. Something that is “radioactive” represents a physical material that emits ionizing radiation. There are several different types of ionizing radiation, including alpha particles (comprising two protons and two neutrons), beta particles (high-energy electrons), neutrons (high-energy particles from radioactive decay), X-rays (high frequency, short wavelength electromagnetic radiation), and gamma waves (very high frequency, very short wavelength electromagnetic

radiation). Alpha particles have the least ability to penetrate tissues, are able to be shielded by paper or clothing, but can result in serious health concerns if ingested. Beta particles can penetrate tissues to a moderate extent, are able to be shielded by plastic and light metals, and can induce serious health risks, including skin burns. Neutrons can be shielded by concrete, water and oil, and large doses can be fatal. Gamma waves are able to penetrate deeply into the body, are able to be blocked by lead and concrete, and can be highly lethal.

There are a variety of terms used to define specific amounts of ionizing radiation. A “roentgen” is a unit of charge produced by X-rays or gamma rays that ionize a specific volume of air. A “rad” is the dose of radiation that will produce absorption of 100 ergs of energy per gram of tissue. A “gray” (Gy) is the dose of radiation that will produce absorption of 1 joule of energy per kilogram of tissue. One Gy is equivalent to 100 rads. A “rem” is the dose of radiation that causes a biologic effect that is equivalent to 1 rad. A “sievert” (Sv) is the dose of radiation that causes a biologic effect that is equivalent to 1 Gy. One Sv is equivalent to 100 rem.

Ionizing radiation damage to the human body depends on the overall dose of radiation, the duration, and the mode of exposure. The cells that are most sensitive to ionizing radiation include the bone marrow (where blood cells are made), reproductive cells, and the cells that line the gastrointestinal tract. The least sensitive cells are muscle, bone, nerve, and red blood cells. In controlled settings, such as occurs in radiation therapy for cancer treatment, the radiation “beam” is directed at the malignancy, in the hope that the radiation will kill the cancer cells. Efforts are made to protect the surrounding normal tissues from the damaging effects of the radiation, but radiation injury can still occur (Disc Image 21.33). In situations where there is chronic (long-term) or subacute excessive exposure, such as may occur in persons working with radiation, a variety of effects can occur, including effects on growth and development, and the induction of genetic mutations, including those that cause cancer. When there is acute (quick), whole-body exposure, such as may occur in nuclear reactor accidents or in atomic/nuclear bombings, the damage depends on the overall whole-body dose.

The following effects occur with the corresponding whole-body dose: 25 rads – no effect; 100–200 rads – mild radiation sickness (“subclinical” nausea, vomiting, destruction of some lymphocytes); 200–600 rads – definite radiation sickness, primarily “hematopoietic” (marked reduction of white blood cell (WBC) numbers, infection, high incidence of death); 600–1000 rads – severe radiation sickness, primarily “gastrointestinal” (severe nausea, vomiting, diarrhea, hemorrhage, WBC destruction, infection, electrolyte disturbances, skin burns, death within 10–14 days); >1000 rads – very severe acute radiation sickness, with “central nervous system” effects (as above with confusion, convulsions, somnolence, coma, death within 2 days).

Before examination and autopsy of a person who has been exposed to ionizing radiation, death investigators and pathologists should first make certain that proper decontamination procedures have been instituted, such that the body (including surroundings and personal effects) does not pose a threat.

Non-ionizing Radiation

Non-ionizing radiation includes all of the EM radiation spectrum other than X-rays and gamma rays. The biologic effect of electrical energy is presented in Chapter 17. Microwaves, infrared waves, and visible light waves are each known to produce eye damage, including cataracts and retinal burns. “Solar radiation,” or radiation produced by the sun, includes the visible light spectrum, as well as infrared and ultraviolet radiation. Ultraviolet rays have the capability of producing severe, sometimes lethal, health effects.

Ultraviolet (UV) radiation can be divided into UVA, UVB, and UVC. Acute exposure to excessive UVA and/or UVB can result in skin burns (sunburn). Delayed effects of excessive exposure to UVA include skin tanning, whereas delayed effects of excessive exposure to UVB include skin tanning, age-related skin changes, and skin cancer, including the three major forms of skin cancer (squamous cell carcinoma, basal cell carcinoma, and melanoma). No definite changes are known to occur with exposure to UVC, although some evidence suggests a possible role in skin cancer with both UVC and UVA. Ozone within the earth’s atmosphere is protective against UVC and UVB.

Sexual Assault

Particularly in homicide or suspected homicide cases, it may be important to consider the possibility of a sexual assault having occurred on the decedent. Some relatively obvious scene and/or external examination findings suggest the possibility of a sexual assault, including torn clothing and absence of clothing. Victims can be female or male. Stabbing deaths and strangulation deaths require the perpetrator to be in close proximity to the victim. Therefore, whenever such case types are encountered, the pathologist and investigators should also consider the possibility of a sexual assault. Whenever an investigator believes that a sexual assault may have occurred, certain autopsy procedures should be performed. These include looking for and collecting trace evidence from the clothing and/or skin, collecting fingernail scrapings or clippings, saving clothing for further examination, and using a “sexual activity kit.” The sexual activity kit includes sterile cotton-tipped swabs for collecting samples from various body sites, including the oral cavity, the vagina, and the anus. Additional swabs are available for collecting other fluid samples from elsewhere, such as a presumed semen stain elsewhere on the body. Swabs should be left in the orifices for several minutes prior to collection, so that fluids can be absorbed into the cotton. The cotton tips can be swiped onto a glass slide so that microscopic evaluation for sperm can be performed. The cotton-tipped swabs are also retained for various other tests, including potential DNA testing. Other parts of the sexual activity kit include envelopes for collecting combed and pulled head and pubic hair samples. The hope is that some of the perpetrator’s hair might be intermixed with the victim’s hair. Another resource available in certain locations is an alternative light

source, which is used to aid in the collection of trace evidence or in identification of bodily fluids, such as semen.

Other autopsy considerations in cases of suspected sexual assault include careful external examination of the external genitalia for evidence of trauma (Disc Image 21.34) and possible external speculum examination of the vagina and/or anus. Some pathologists will remove a large portion of the external genitalia along with vagina, anus, and uterus for dissection and evaluation. Others prefer to perform a thorough external examination followed by routine internal removal and dissection of the vagina, uterus and rectum, without including the external genitalia and anus. Whenever a sexual assault is considered a possibility, it is usually a good idea to perform both a layer-by-layer anterior neck dissection and a posterior neck dissection.

Terrorist Agents

Although terrorist attacks may include a variety of injury types already addressed in different parts of this textbook, this section will specifically deal with biological and chemical agents. While many of the other injury types associated with terrorist activity will probably be associated with major events that will be obvious when they occur, biological and chemical terrorist attacks may occur in a more subtle fashion. As such, it may be the coroner/medical examiner/forensic pathology community, the emergency medical system, or both, that identify the occurrence of a biological or chemical attack. It is beyond the scope of this text to address these topics in detail. The reader is referred to an excellent review for further information.

Biologic Agents

Biological agents are micro-organisms, typically bacteria or viruses, that have the capability of causing severe, incapacitating infectious disease, with or without eventual death. The most effective organisms must be able to be easily distributed over a wide region, they must be highly infectious (easily “caught”), and they must be incapacitating or lethal.

Bacillus anthracis (anthrax) is a Gram-positive bacteria that can cause isolated skin disease (cutaneous anthrax), gastrointestinal disease, or inhalational disease (“hemorrhagic mediastinal lymphadenitis”). The cutaneous form results in the formation of a raised itchy lesion that eventually blisters and then forms a black center (eschar). Death occurs in a minority of cases. The gastrointestinal form results in nausea, vomiting, fever, loss of appetite, and abdominal pain with eventual bloody vomit and severe diarrhea. The risk of death is fairly high. The inhalational form, also referred to as “hemorrhagic mediastinal lymphadenitis,” begins like a cold, but progresses to severe respiratory compromise and death. Death is the rule, although survival has been reported with appropriate antibiotic therapy.

Yersinia pestis (plague) is a Gram-negative bacteria that is similar to anthrax in that it can produce cutaneous disease (bubonic plague), inhalational disease (pneumonic plague) and gastrointestinal or ingested disease (septicemic plague).

Francisella tularensis (tularemia) is a Gram-negative bacteria that is present throughout the environment. The typical case occurs when someone becomes accidentally exposed from a wild animal or carcass. Various forms occur, depending on the route of exposure. Theoretically, an aerosolized form could produce devastating effects.

Clostridium botulinum (botulism) is a Gram-positive bacteria that produces a deadly neurotoxin. If a suitable method of dispersing this bacterial toxin could be developed, it would be an extremely deadly terrorist agent.

Variola virus (orthopoxvirus) is the virus commonly known as smallpox. Certain forms are highly contagious and have a high mortality rate. It is believed to have been totally eradicated, partially as a result of worldwide vaccination efforts. As a matter of fact, the immunization is no longer given. As a consequence, it is feared that stockpiles of the virus may be utilized as a biological terror agent. The virus causes fever and pain associated with a rash that eventually becomes blistered and then pustular (filled with pus), with all lesions everywhere on the body appearing at the identical stage. Chickenpox lesions look similar, but chickenpox lesions of differing stages (rash, blister, pustule) can occur simultaneously.

Several viruses can cause viral hemorrhagic fevers, in which fever accompanies widespread hemorrhagic skin and other tissue hemorrhages. Included here are Ebola virus, Marburg virus, yellow fever, dengue fever, Hantaan virus, and Lassa virus, to name a few.

Chemical Agents

For a chemical agent to be considered a “good” terror agent, it must be able to be distributed widely so that numerous people are affected, and it must produce injury and/or death. Many chemical agents are not necessarily lethal, but the injuries that they produce can be incapacitating, both from a physical/medical standpoint and a resource/economic standpoint. Chemical agents may be categorized based on their mechanism of action.

Pulmonary agents include various gases that can cause localized airway/lung injury. Included here are chlorine, phosgene (CG), nitrogen oxides, HC and PFIB.

Cyanide agents include hydrogen cyanide and cyanogen chlorine, and occur as liquids or gases. Exposure may be via inhalation, ingestion, or skin contact. Cyanide blocks cellular respiration as described in Chapter 15.

Blistering agents, or vesicants, include sulfur mustard (H, HD), lewisite (L), and phosgene oxime (CX). They occur as liquids and vapors, with exposure via inhalation, ingestion, or contact. They produce chemical burns.

Nerve agents include G agents (tabun, sarin, and soman) as well as VX. They occur as liquids with or without associated vapors. Exposure is via inhalation,

ingestion, or contact, and the effects are primarily neurological, including seizure activity, paralysis, and muscle cramping, resulting from irreversible binding to acetylcholinesterase.

Ricin is a miscellaneous chemical agent derived from castor beans. It may be a powder, a mist, or a solid. Inhalation, ingestion, or percutaneous (via skin, including injection) routes can result in toxic/lethal effects. The poison blocks intracellular protein synthesis.

Therapy-Related Deaths

Along with in-custody/restraint-related deaths, deaths related to medical therapy (or those alleged to have been related to medical therapy) tend to be some of the most challenging cases encountered within forensic death investigation. Although the term “therapy” is used here, it is meant to encompass any type of medical intervention, whether diagnostic or therapeutic. Whenever such a death occurs, it is important that death investigators and pathologists attempt to gain as much information about the case prior to autopsy as possible. Therefore, attempts should be made to obtain copies of all medical records as well as any blood (or other samples) collected during hospitalization. If the death occurred during a procedure, the pathologist should learn as much about the specific procedure prior to autopsy. Occasionally, specialized autopsy procedures should be employed, depending on the procedure that was being performed. Careful documentation of findings on external and internal examination is essential, as the case may or may not eventually be scrutinized by others.

Deaths related to medical therapy can be classified in many different ways. One way is to consider the type of therapy that is related to the death. Examples include anesthesia, surgery, diagnostic procedure, therapeutic procedure, drug/medication reaction, drug/medication error, and medical device failure. Another classification scheme is based on the physiologic mechanism of death. Examples include airway compromise, vascular compromise (thrombus, embolus, compression), vascular disruption (puncture, transection), coagulation abnormality, infection, toxic reaction, allergic reaction, idiosyncratic reaction (unknown explanation), immune reaction, arrhythmia, metabolic alteration, hypoxia/ischemia, and neurologic compromise.

Some special considerations depend on the suspected underlying mechanism of death and include the following. If an anaphylactic (allergic) reaction is suspected, blood samples for tryptase and specific antibodies should be collected (see above). If a pneumothorax (air escaping the lungs into the pleural cavity, with subsequent compression of the lung) is suspected, a postmortem chest X-ray may show this. In addition, after the initial Y-shaped incision is made, the pathologist can create a “pocket” along the lateral chest wall, fill it with water, and then carefully puncture the chest wall/rib cage in order to visualize the escape of air bubbles, indicating the presence of a pneumothorax. Measurement of the amount of air is possible by “catching” the air bubbles in an inverted graduated cylinder that is initially full of

water (Fig. 21.29). An air embolus can be visualized by chest X-ray as a radiolucent (dark) area within the right side of the heart (see above). Careful dissection, followed by filling the heart sac with water, as described in the “Emboli” section above, should be performed.

Fig. 21.29 A case of pneumothorax, where a pocket is created between the skin and subcutaneous tissues and the rib cage and then filled with water. The pathologist inserts a scalpel blade through the muscles between two ribs and looks for air bubbles in the water. The inverted graduated cylinder was initially filled with water. As the air bubbles escape from the chest cavity, the bubbles can be “caught” and measured within the cylinder (photo courtesy of the Dallas County Medical Examiners Office)



One of the most difficult challenges in dealing with therapy-related deaths is how to properly certify the cause as well as the manner of death. Regarding the medical intervention and the cause of death, several possibilities exist. The intervention may have absolutely nothing to do with death; it might have possibly contributed to death; it might have probably contributed to death; or it might have definitely caused death. Depending on the case, careful medical chart review may be necessary to make such determinations. If the intervention is believed to have caused or contributed, however slightly, to the death, then it is appropriate to include the medical intervention within the cause of death ruling. Examples: Coronary artery dissection due to coronary artery angioplasty for atherosclerotic cardiovascular disease; I – Pneumonia, II – Post-operative state following appendectomy; Idiopathic reaction to general anesthesia during tonsillectomy.

Manner of death determinations can be equally problematic. Medical interventions that cause or contribute to death can be considered external factors in a death. As such, it would seem appropriate to rule such deaths something other than natural. Others argue that, since the underlying natural disease process was what initiated the chain of events that required the medical intervention, the manner of death should be considered natural. Unfortunately, there is no universally accepted way in which to deal with these issues. According to one proposed method, a disease-related death due to a medical intervention contributing to death can be ruled a natural death if the intervention-related contribution is considered common, inherent, chronic in

nature, or related to a non-traumatic, non-acute, natural physiologic response to appropriately-implemented therapy. In contrast, if the intervention that contributed to death is a true mistake or error, or if it is unexpected, acute, and temporally related to the intervention, associated with mechanical failure of a medical device, or traumatic in nature (Fig. 21.30), then the MOD ruling should be “accident.” Of note is the fact that in a few jurisdictions within the United States, the MOD choices include a sixth option: “therapeutic complication.” Unfortunately, such an option is not available in most jurisdictions.

Fig. 21.30 A case where chest tube placement inadvertently penetrated the lung, causing injury



Examples of cases where an intervention is believed to be contributory to death, but the MOD is ruled “natural,” include the following: an elderly patient with underlying heart and lung disease who develops post-operative pneumonia following otherwise successful surgery; an obese patient who develops post-operative deep venous thrombosis and dies suddenly from a pulmonary embolism, one week after otherwise successful knee replacement surgery; a patient with cancer who develops a severe infection and dies, following intensive chemotherapy. It should be noted that in jurisdictions where “therapeutic complication” is available on the death certificate, these cases can be ruled in this manner instead of “natural,” but when no such option exists, it is appropriate to rule them as “natural.” Examples of cases where an intervention is the cause or a contributory cause to death and the MOD ruling is appropriately considered “accident” include the following: a fatal transfusion reaction related to a clerical error; a patient dying from infusion of an intravenous line with the wrong medication; a patient dying from an air embolism during laparoscopic surgery; a patient dying from extensive internal hemorrhage initiated from a femoral artery perforation, performed for a cardiac catheterization procedure; death from peritonitis following dislodgement of a feeding tube; an idiosyncratic reaction

to general anesthesia; an allergic reaction to a medication. It should be noted that in some cases where “accident” is used, a true mistake (which theoretically might have been prevented) has occurred, but in others, a truly unforeseen (and theoretically unavoidable) event has taken place. The term “accident” as used in a MOD ruling should not be misinterpreted as suggesting that a mistake or “malpractice” has occurred.

Unfortunately, even when a pathologist attempts to employ a fairly detailed “protocol” or methodology when evaluating deaths related to medical intervention, there will always be cases that are quite problematic. Each case must be evaluated on its own merits. If a definitive ruling cannot be made, because of various factors or issues, it is appropriate to rule such cases as “undetermined.”

Depending on the case, a variety of special inquiries and procedures should be considered when a therapy-related death is suspected. These include: establish a protocol that asks hospitals to leave all medical devices on/in bodies after death, obtain copies of medical records, contact and discuss the case with involved persons (physician, nurse, etc.), document all conversations, consider collecting samples from the hospital lab for further testing, learn as much as possible about any procedure or device that is alleged to have been involved in death, anticipate questions, document (written and photograph) all findings (including pertinent negatives) at autopsy, consider retaining any medical device thought to be involved in death, consider collecting and retaining additional tissue/fluid samples in a possible toxicology case, consider implementing specialized X-rays or dissection procedures as described above, and avoid making statements regarding “standard of care” issues that are outside your area of expertise.

If a drug or medical device is implicated in death, it is appropriate to report the death to the Food and Drug Administration (FDA) or the Consumer Product Safety Commission (CPSC), depending on which agency has jurisdiction over the device. The contact information for each follows: US Food and Drug Administration, www.fda.gov, 1-800-FDA-1088. Consumer Product Safety Commission, www.cpsc.gov, 1-800-638-8095, or email: info@cpsc.gov.

Disc Image Legends

Disc Image 21.1 A pilot from an airplane crash. This is the same pilot depicted in Fig. 21.2, prior to clothing removal.

Disc Image 21.2 Pilot control injury hand X-ray.

Disc Image 21.3 Rattlesnake bite on index finger. Note the two fang marks (arrows) (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)

Disc Image 21.4 Hemorrhagic discoloration within the heart and lung of a rattlesnake bite victim. (courtesy of the Dallas County Medical Examiners Office, Jeffrey J. Barnard, Chief Medical Examiner)

- Disc Image 21.5 Postmortem drying of scrotum.
- Disc Image 21.6 Tardieu spots mimicking true injury.
- Disc Image 21.7 Postmortem injury. Note the yellow discoloration.
- Disc Image 21.8 Postmortem injury mimicking an antemortem injury. The wound, which was inflicted when the body was run over by a motor vehicle, was in a dependent (downward) position, thus accounting for the red color.
- Disc Image 21.9 Numerous areas of postmortem bleeding in the neck, related to intense lividity and decomposition.
- Disc Image 21.10 An early postmortem artifact of the reflected scalp, consisting of numerous areas of punctate bleeding.
- Disc Image 21.11 Postmortem blood extravasation (outside of vessels) occurring with decomposition.
- Disc Image 21.12 Deep vein thrombosis (blood clots in leg veins).
- Disc Image 21.13 Microscopic appearance of amniotic fluid embolism, with amniotic fluid debris contained within small blood vessels in the lungs.
- Disc Image 21.14 Air emboli within the blood vessels at the base of the brain. Note the numerous air bubbles within the basilar artery.
- Disc Image 21.15 A stitched arterial embalming incision.
- Disc Image 21.16 Trocar puncture marks in a lung.
- Disc Image 21.17 An eyecap covering the eye of an embalmed body.
- Disc Image 21.18 A jaw wire holding the mouth closed in an embalmed body.
- Disc Image 21.19 Packing material surrounding the site of arterial embalming in the case depicted in Disc Image 21.15.
- Disc Image 21.20 A trocar puncture mark after the button has been removed.
- Disc Image 21.21 A body with severe head trauma that has been embalmed and buried for 15 years. Note the relative preservation of the skin.
- Disc Image 21.22 A cross-section of the heart of an elderly victim of homicide by heart attack. Note the thin left ventricular wall (upper part of photo), indicating an old (remote) myocardial infarct (heart attack), a condition which predisposes to cardiac arrhythmias.
- Disc Image 21.23 A photo routinely taken in in-custody deaths, showing absence of injury to the scrotum.
- Disc Image 21.24 A close-up of an electronic shock device dart with its barbed tip embedded in the skin.
- Disc Image 21.25 An X-ray showing the appearance of two darts from an electronic shock device.
- Disc Image 21.26 An example of an electronic shock device.
- Disc Image 21.27 Another image of a reconstruction of events in an in-custody death.
- Disc Image 21.28 The internal examination appearance of an individual who died of starvation.
- Disc Image 21.29 Heart valve recovery taking place prior to autopsy.
- Disc Image 21.30 Extensive postmortem soft tissue bleeding following subclavian blood collection performed by tissue procurement personnel prior to autopsy.

Disc Image 21.31 A ruptured ectopic (tubal) pregnancy. Note the hemorrhagic area in the upper left portion of the photo, involving the fallopian tube. The uterus and opposite tube and ovary are unremarkable.

Disc Image 21.32 Numerous petechiae (pinpoint areas of bleeding) within the heart of a pregnant woman who died of thrombocytopenic purpura (TTP).

Disc Image 21.33 Skin “burns” resulting from radiation therapy.

Disc Image 21.34 Genital trauma in an elderly woman who was the victim of a sexual assault-associated homicide.

Selected References

Aircraft Crashes

Cimrmancic MA, Gormley WT, Cina SJ. Aviation pathology (Chapter 32). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003: pp 299–316.

Mallak C. Miscellaneous and special topics in forensic pathology – blast injuries, aircraft mishaps, and radiation injury. In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:pp 157–61.

Campman SC, Luzi SA. The sensitivity and specificity of control surface injuries in aircraft accident fatalities. *Am J Forensic Med Pathol* 2007;28:111–5.

Allergic Reactions (Anaphylaxis)

Ansari MQ, Zamora J, Lipscomb MF. Postmortem diagnosis of acute anaphylaxis by serum tryptase analysis: a case report. *Am J Clin Pathol* 1993;99:101–03.

Randall B, Butts J, Halsey JF. Elevated postmortem tryptase in the absence of anaphylaxis. *J Forensic Sci* 1995;40:208–11.

Schwartz LB, Yunginger JW, Miller J, Bokhari R, Dull D. Time course of appearance and disappearance of human mast cell tryptase in the circulation after anaphylaxis. *J Clin Invest* 1989;83:1551–55.

Schwartz LB, Metcalfe DD, Miller JS, Earl H, Sullivan T. Tryptase levels as an indicator of mast-cell activation in systemic anaphylaxis and mastocytosis. *N Engl J Med* 1987;316:1622–26.

Animal Attacks

Loewe CL, Diaz FJ, Bechinski J. Pitbull mauling deaths in Detroit. *Am J Forensic Med Pathol* 2007;28:356–60.

Prahlow JA. Deaths due to animals, plants, and other natural environmental hazards (Chapter 37). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003: pp 361–374.

Emboli

Sowell MW, Lovelady CL, Brogdon BG, Wecht CH. Infant death due to air embolism from peripheral venous infusion. *J Forensic Sci* 2007;52:183–8.

Exhumation

- Clark MA. Exhumations (Chapter 45). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003:pp 445–9.
- Hanzlick R. Embalming, body preparation, burial, and disinterment. An overview for forensic pathologists. *Am J Forensic Med Pathol* 1994;15:122–31.

Explosions and Blast Injuries

- Clark MA. Autopsy guidelines for explosion fatalities (Chapter 33). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003:pp 317–26.
- Mallak C. Miscellaneous and special topics in forensic pathology – blast injuries, aircraft mishaps, and radiation injury. In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:pp 157–61.

High-Profile Cases

- Gill JR, Pasquale-Styles M. Firearm deaths by law enforcement. *J Forensic Sci* 2009;54:185–8.
- Perper JA, Juste GM, Schueler HE, Motte RW, Cina SJ. Suggested guidelines for the management of high-profile fatality cases. *Arch Pathol Lab Med* 2008;132:1630–4.
- Prahlow JA. Hate crimes and the forensic pathologist. *Am J Forensic Med Pathol* 2007;28:284–7.

Homicide by Heart Attack

- Barnard JJ, Hirsch CS. Which came first, the rupture or the impact? *ASCP Check Sample* 1989;FP89–5:1–4.
- Davis JH. Can sudden cardiac death be murder? *J Forensic Sci* 1978;23(2):384–7.
- Prahlow JA. Homicidal cerebral artery aneurysm rupture. *J Forensic Sci* 2004;49:1082–5.
- Turner SA, Barnard JJ, Spotswood SD, Prahlow JA. “Homicide by heart attack” revisited. *J Forensic Sci* 2004;49(3):598–600.

In-Custody Deaths

- DiMaio TG, DiMaio VJM. *Excited Delirium Syndrome – Cause of Death and Prevention*. Boca Raton, FL: Taylor & Francis; 2006.

Mass Fatality Incidents

- Froede RC, Stahl CH, Wagner GN, Kelly C. Investigation of multiple fatality incidents (Chapter 30). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003:pp 275–92.
- Jensen RA. *Mass Fatality and Casualty Incidents – A Field Guide*. Boca Raton, FL: CRC Press; 2000.

- Leclair B, Shaler R, Carmody GR, Eliason K, Hendrickson BC, Judkins T, Norton MJ, Sears C, Scholl T. Bioinformatics and human identification in mass fatality incidents: the World Trade Center disaster. *J Forensic Sci* 2007;52:806–19.
- Schneid TD, Collins L. *Disaster Management and Preparedness*. Boca Raton, FL: Lewis Publishers; 2000.

Nutrition and Hydration Disorders

- Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. *Emerging Infect Dis*. 1999;5:607–625.
- Olsen SJ, MacKinnon LC, Goulding JS, Bean NH, Slutsker L. Surveillance for foodborne-disease outbreaks: United States, 1993–1997. *MMWR. CDC Surveillance Summaries*. 2000;49(1):1–62.

Occupational Deaths

- Graham MA, Henry TE, Weedn VW. Work-related deaths. In: Froede RC (ed). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003:pp 375–80.

Organ and Tissue Procurement Issues

- Pinckard JK, Wetli CV, Graham MA. National Association of Medical Examiners position paper on the medical examiner release of organs and tissues for transplantation. *Am J Forensic Med Pathol* 2007;28:202–7.

Postmortem Chemistry Tests

- Coe JI. Postmortem chemistries on human vitreous humor. *Am J Clin Pathol* 1969;51:741–50.
- Coe JI. Postmortem chemistry update: emphasis on forensic application. *Am J Forensic Med Pathol* 1993;14:91–117.
- Prahlow JA. Postmortem forensic chemistry (Chapter 40). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003:pp 393–400.

Postmortem Cultures

- Nolte KB, Guarner J, Shieh W-J, Zaki SR. Emerging infectious diseases and the forensic pathologist (Chapter 36). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003:pp 345–60.

Pregnancy-Related Maternal Deaths

- Prahlow JA, Barnard JJ. Pregnancy-related maternal deaths. *Am J Forensic Med Pathol* 2004;25:220–36.

Product-Related Deaths

United States Food and Drug Administration. www.fda.gov. Accessed on 10/13/18.
Consumer Product Safety Commission. www.cpsc.gov. Accessed on 10/13/08.

Radiation

Kane AB, Kumar V. Environmental and nutritional pathology. In: Kumar V, Abbas AK, Fausto N (editors). *Robbins and Cotran Pathologic Basis of Disease* 7th ed. Philadelphia: Elsevier; 2005:pp 436–42.

Mallak C. Miscellaneous and special topics in forensic pathology – blast injuries, aircraft mishaps, and radiation injury. In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:pp 157–61.

Mettler FA, Voelz GL. Major radiation exposure – what to expect and how to respond. *N Engl J Med* 2002;346:1554–61.

Warren S. Forensic pathologic criteria for radiation death. *J Forensic Sci* 1971;16:137–43.

Terrorist Agents

Byrnes ME, King DA, Tierno PM. *Nuclear, Chemical, and Biological Terrorism – Emergency Response and Public Protection*. Boca Raton, FL: Lewis Publishers; 2003.

Finelli LN. Miscellaneous and special topics in forensic pathology – biological and chemical agents. In: *Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006:pp 157–61.

Fowler DR, Nolte KB. Biologic and chemical terrorism: surveillance and response (Chapter 35). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003:pp 335–44.

Marty AM, Greenberg MI (eds). Biological weapons and bioterrorism. *Clinics Lab Med* 2006;26:1–551.

Therapy-Related Deaths

Caplan MJ. A Medical Examiner’s experience with the evaluation of deaths associated with complications of diagnostic and therapeutic procedures (therapeutic complications/periprocedural deaths) – revisited (abstract). American Academy of Forensic Sciences Annual Meeting Proceedings. 1999;164–5.

Hirsch CS, Flomenbaum M. Problem-solving in death certification. *ASCP Check Sample* 1995;FP 95–1:1–31.

Prahlow JA, McClain JL. Deaths due to medical therapy (Chapter 39). In: Froede RC (editor). *Handbook of Forensic Pathology* 2nd ed. Northfield, IL: College of American Pathologists; 2003: pp 381–92.

United States Food and Drug Administration. www.fda.gov. Accessed on 10/13/18.
Consumer Product Safety Commission. www.cpsc.gov. Accessed on 10/13/08.

Appendix: Additional Resources and Reference Books

- Adelson L. *The Pathology of Homicide*. Springfield, IL: CC Thomas; 1974.
- Basic Competencies in Forensic Pathology – A Forensic Pathology Primer*. Northfield, IL: College of American Pathologists; 2006.
- DiMaio VJM. *Forensic Pathology*. 2nd ed. Boca Raton, FL: CRC Press; 2001.
- Dolinak D, Matshes E. *Medicolegal Neuropathology – A Color Atlas*. Boca Raton, FL: CRC Press; 2002.
- Dolinak D, Matshes E, Lew EO. *Forensic Pathology: Principles and Practice*. Burlington, MA: Elsevier Academic Press; 2005.
- Froede RC, ed. *Handbook of Forensic Pathology*. 2nd ed. Northfield, IL: College of American Pathologists; 2003.
- Itabashi HH, Andrews JM, Tomiyasu U, Erlich SS, Sathyavagiswaran L. *Forensic Neuropathology – A Practical Review of the Fundamentals*. Amsterdam: Elsevier; 2007.
- Kumar V, Abbas AK, Fausto N. *Robbins and Cotran Pathologic Basis of Disease*. 7th ed. Philadelphia, PA: Elsevier Saunders; 2005.
- Leestma JE. *Forensic Neuropathology*, 2nd ed. Boca Raton, FL: CRC Press; 2009.
- Shkrum MJ, Ramsay DA. *Forensic Pathology of Trauma – Common Problems for the Pathologist*. Totowa, NJ: Humana Press; 2007.
- Silver MD, Gotlieb AI, Schoen FJ. *Cardiovascular Pathology*. 3rd ed. New York, NY: Churchill Livingstone; 2001.
- Spitz WU, ed. *Spitz and Fisher's Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation*. 4th ed. Springfield, IL: CC Thomas; 2006
- Stocker JT, Dehner LP, eds. *Pediatric Pathology*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001.
- Tsokos M, ed. *Forensic Pathology Reviews* (vols 1–4). Totowa, NJ: Humana Press; 2004 (vol 1), 2005 (vols 2–3), 2006 (vol 4).

Other Resources

American Academy of Forensic Sciences. www.aafs.org

American Board of Medicolegal Death Investigators, Inc. www.slu.edu/organizations/abmdi

American Society for Clinical Pathology. www.ascp.org

Armed Forces Institute of Pathology. www.afip.org

Centers for Disease Control and Prevention. www.cdc.gov

College of American Pathologists. www.cap.org

National Association of Medical Examiners. www.thename.org

National Institute of Justice. www.ojp.usdoj.gov/nij/

Index

Note: The letters ‘f’ and ‘t’ following locators denote figures and tables respectively.

A

- AAA, *see* Abdominal aortic aneurysm (AAA)
- AAFS, *see* American Academy of Forensic Sciences (AAFS)
- ABC, *see* American Board of Criminalistics (ABC)
- Abdominal aortic aneurysm (AAA), 215
- ABFDE, *see* American Board of Forensic Document Examiners (ABFDE)
- ABMDI, *see* American Board of Medicolegal Death Investigators (ABMDI)
- ABP, *see* American Board of Pathology (ABP)
- Abrasion collar, *see* Circumferential marginal abrasion
- Abrasions, blunt force injury deaths, 302
 abrasions on trunk of pedestrian, 303f
 hemosiderin, indicators of healing, 303
 patterned injuries, 302
 types, 302, 302f
- ACA, *see* Anterior cerebral artery (ACA)
- Accidental childhood deaths
 in adolescents, 515
 in infants, 514
 in older children, 515
 toddler hanging by mini-blind cord, 515f
 in toddlers, 514–515
- Accidental drownings, 446
- Accidental sharp force injuries, 393
- Accreditation Council for Graduate Medical Education (ACGME), 13, 43t, 45
- Accreditation process, forensic autopsy, 159
 checklist, specific areas addressed, 159
- ACF, *see* Anterior cranial fossa (ACF)
- ACGME, *see* Accreditation Council for Graduate Medical Education (ACGME)
- Acquired immunodeficiency syndrome (AIDS), 225, 242
- ACTH, *see* Adrenocorticotrophic hormone (ACTH)
- Acute asthmatic bronchitis, 233
- Acute/chronic alcoholism, 268
- Acute/chronic leukemias, 241
- Acute cocaine intoxication, *see* Cocaine intoxication
- Acute fatty liver of pregnancy (AFLP), 576–577
- Acute myocardial infarct, 69, 212
- Acute pancreatitis, 240
- Acute psychosis, 283, 530
- Adenine (A), 28
- Adipocere formation, 175, 176f
- Adrenal insufficiency (Addison disease), 244
- Adrenocorticotrophic hormone (ACTH), 117, 243–244
- Adrenogenital syndromes, 244
- Aeromedical investigation, 540
- Aerospace pathology, 540, 542
- AFIS, *see* Automated fingerprint identification systems (AFIS)
- AFLP, *see* Acute fatty liver of pregnancy (AFLP)
- AFTE, *see* Association of Firearm and Tool Mark Examiners (AFTE)
- AIDS, *see* Acquired immunodeficiency syndrome (AIDS)
- Aircraft crashes, 540–542
 ability to survive, 542
 aeromedical investigation, 540
 aerospace pathology, 540
 clothing and personal effects, preliminary identifications, 541
 massive body injury in a pilot, 541f
 pilot (hand) control injuries, 542
 safety investigation, 540
 seat swapping, 541

- Air embolism, 69, 366, 389–390, 394–395, 549, 585
- Alcoholism, acute/chronic, 268
- Algor mortis (cooling of the body), 163, 168, 180
- Alleles, 27
- Allergens/antigens, 542–543
- Allergic reactions
 - allergens/antigens, 542
 - beta-tryptase, 543
 - elevated IgE levels, 544
 - IgE antibody, 542
 - immunoglobulin, 542
 - tryptase, 543
- Alpha-1-antitrypsin deficiency, 234, 237
- Alpha-methyltryptamine (AMT), 284
- Altruism, 530–531
- Alzheimer's disease, 246, 475, 566
- Amanita phalloides*, 283
- American Academy of Forensic Sciences (AAFS), 14
- American Board of Criminalistics (ABC), 23, 28
- American Board of Forensic Document Examiners (ABFDE), 26
- American Board of Medicolegal Death Investigators (ABMDI), 54
- American Board of Pathology (ABP), 13–14, 43, 43t, 45–46
- The American Society for Clinical Pathology (ASCP), 14
- American Society of Crime Laboratory Directors (ASCLD), 23
- Amniotic fluid emboli, 548–549
 - disseminated intravascular coagulation, 549
- Amniotic fluid embolism (AFE), 549, 577
- AMT, *see* Alpha-methyltryptamine (AMT)
- Amyloidosis, 222, 246
- Anaphylaxis, *see* Allergic reactions
- Anatomic Pathology (AP), 6–7, 11, 13, 14t, 43, 45–46
 - anatomic, defined, 7
 - basic components
 - autopsy pathology, 7
 - cytology/cytopathology, 7
 - surgical pathology, 7
 - pathologists, role in, 7
- Anatomy and physiology
 - body regions and compartments, 92
 - gross anatomy, 82–90
 - histology, 90–91
 - physiology, 91–92
- specific organ systems
 - cardiovascular system, 104–106
 - endocrine system, 116–119
 - gastrointestinal system, 108–111
 - genitourinary system, 120–122
 - hepatobiliary system, 111–122
 - integumentary system, 92–94
 - musculoskeletal system, 94–97
 - nervous system, 97–104
 - respiratory system, 106–108
 - reticuloendothelial system, 113–116
 - special sensory structures, 122–123
- Angioplasty, 213–214, 584
- Anhalonium lewinii*, *see* *Lophophora williamsii*
- Animal attacks
 - asphyxia, mechanical/compressive, 544
 - multiple injuries, 545f
- Anorexia nervosa, 514, 562, 564–565
- Anoxic brain injury due to near drowning, *see* Sequelae of near drowning
- Antemortem (before death), 136, 178, 191, 194, 397, 442, 460, 478, 493, 495, 544–546, 572
 - dental X-ray, 489
 - neck, 192
 - record/standard, 186
 - sharp force injury, 441
 - view of, 193f
- Anterior cerebral artery (ACA), 82t, 103
- Anterior cranial fossa (ACF), 82t, 102, 314
- Anthropologic examination, 96, 174
- Anthropophagy, 169, 172
- Antipsychotic drugs, 287–288
 - neuroleptic malignant syndrome, 288
- Aortic aneurysms (abdominal and thoracic), 215–216
 - aortitis, 216
 - coronary artery ostia, 216
 - cystic medial necrosis, 216
 - hemopericardium/hemothorax, 215
 - hemoperitoneum or retroperitoneal hemorrhage, 215
 - obliterative endarteritis, 216
 - perivascular plasma cell inflammation, 216
 - syphilis (*Treponema pallidum*), 216
 - primary/secondary/tertiary syphilis, 216
- AP, *see* Anatomic Pathology (AP)
- Appendix (acute appendicitis), 110, 196, 238
- Apple green birefringence, 246
- Arachnoid membrane, 98, 99f–100f, 102, 315–317
- Arborescent pattern of the skin, 463
- Arc marks, lightning, 461–462

- Arnold–Chiari malformation, 223
- Arrhythmia, 208–209, 218, 221, 223, 235, 270, 276, 286, 444, 471, 555, 560, 564, 583
- cardiac, 267, 273, 434, 453, 465, 515
- lethal or fatal, 75, 143, 212, 217, 220, 247, 267, 277, 292, 323, 451, 465, 556, 576
- Arrhythmogenic right ventricular cardiomyopathy, 221
- Arrhythmogenic right ventricular dysplasia, *see* Arrhythmogenic right ventricular cardiomyopathy
- Arteriosclerosis, 248
- Arteriosclerosis, *see* Coronary artery atherosclerosis
- Arteriovenous Malformation (AVM), 226–228, 556
- Artifacts and mimics, 544–546
- decomposed sclera (white part of eyes), 547f
- decomposition, 546
- lack of vital tissue reaction, 545
- liver laceration, 546f
- lividity pattern on neck mimicking ligature marks, 545f
- postmortem/antemortem wounds, 546
- Artifacts, heat-induced, 495, 496f
- ASCLD, *see* American Society of Crime Laboratory Directors (ASCLD)
- ASCP, *see* The American Society for Clinical Pathology (ASCP)
- ASD, *see* Atrial septal defect (ASD)
- Asphyxia, definition, 401
- Asphyxial deaths, 70, 401–428
- chemical asphyxia, 419
- carbon monoxide, 419–422
- cyanide, 422–423, 422f
- hydrogen sulfide, 423
- issues, 423
- autoerotic asphyxia, 423–424
- choking game, 424–425, 426f
- combination, 427–428
- drowning, 428
- restraint asphyxia, 425–427
- neck compression, 411–412
- hanging, 412–415
- ligature strangulation, 416–417
- manual strangulation, 417–418
- non-ligature, non-manual, 418–419
- strangulation, 415–416
- suffocation
- choking, 406–408
- combination forms, 410–411
- mechanical asphyxia, 408–410
- positional asphyxia, 410
- simple asphyxia, 403–405
- smothering, 405–406
- Asphyxial mechanism, 427–428, 453, 512, 515, 526
- Asphyxia, mechanical/compressive, 544
- Aspiration pneumonia, 232, 247, 292
- cerebral palsy, 232
- chronic brain injury, 232
- neuromuscular disorders, 232
- Association of Firearm and Tool Mark Examiners (AFTE), 25
- Asthma, 233
- acute asthmatic bronchitis, 233
- bronchial asthma, 233
- chronic asthmatic bronchitis, 233
- mucous plugs, 233
- Asthmatic bronchitis, acute, 233
- Atherosclerosis, 211–215, 218, 248, 277, 437, 470, 576
- Atlas, 322
- Atrial myxomas/papillary fibroelastomas of valves, 223
- Atrial septal defect (ASD), 209
- Atrioventricular (AV) node, 83, 104, 106, 217, 223
- Autoerotic asphyxia, 423–424, 424f–425f
- Autoimmune disorders, 241
- autoimmune thyroid disease, 241
- collagen vascular diseases, 241
- connective tissue diseases, 241
- localized autoimmune disorders, 241
- systemic autoimmune disorders, 241
- systemic lupus erythematosus, 241
- Autoimmune hemolytic anemia, 241
- Autoimmune polyendocrine syndromes, 244
- Autoimmune thyroid disease, 241
- Autolysis/putrefaction, loss of soft tissues, 176, 176f
- Automated fingerprint identification systems (AFIS), 26
- Autonomic nervous system
- parasympathetic, 98
- sympathetic
- fight or flight system, 98
- See also* Nervous system
- Autopsy, 492–496
- ancillary procedures, 37
- chemistry testing, 39
- histology (microscopic) examination, 39, 40f

- Autopsy (*cont.*)
- laboratory testing, 39
 - microbiology testing, 39
 - radiologic examination, 39
 - retention of organs for further study, 39
 - toxicology testing, 39
- artifacts, 495–496
- classic heat induced artifact, 495
 - heat-induced, artifactual epidural hemorrhage, 496f
 - heat-induced skull fracture, 496f
 - skin splitting, 495
 - traumatic injuries, 495
- complete vs. limited autopsy, 39
- external examination, 37, 38f, 492–493
- bright red lividity, 492
 - clouding of the corneas, 492
 - fasciotomy incisions, 492
 - photography, means of documentation, 39, 132f
 - postmortem flexion, 492
 - preliminary identification, 492
 - pugilistic posture, 492
 - tissue debridement, 492
 - X-ray examination, 492
- internal examination, 37, 39f, 493
- heat-fixed/cooked, 493
 - postmortem burning, 493
- toxicology, 494–495
- blood samples for CO testing, 494
 - collecting other tissues, 494
- Autopsy assistants, 155, 158
- role of, variability in, 158
- Autopsy findings
- high voltage
 - dark pinpoint “spark,” 457
 - high-voltage electrical burn on index finger and thumb, 458f
 - low voltage
 - electrical burns, 456
 - electrocution deaths in water, 455
 - other features
 - electrical petechiae, 460
- Autopsy pathology
- basic types
 - forensic (medicolegal), 11, 12–13, 130t
 - hospital (medical), 11–12
 - process of autopsy, 11
- Autopsy performance standards, 159
- Autopsy, psychological, 22
- AVM, *see* Arteriovenous Malformation (AVM)
- AV node, *see* Atrioventricular (AV) node
- Avulsions, 302, 310
- avulsion injury of the skin on face, 311f
 - decapitation injury, 311f
- B**
- BAC, *see* Blood alcohol concentration (BAC)
- Bacillus anthracis* (anthrax), 581
- Bacterial brain abscesses, 224
- Bacterial endocarditis, *see* Infective endocarditis
- Bacterial meningitis, 72, 208, 514
- Bacterial pneumonia, 205, 231–232
- lobar/bronchopneumonia, 231
- Basilar skull with ACF/MCF/PCF, 102, 103f
- Bathtub drownings, 446, 446f
- Battery-operated electrical devices, 451
- BE, *see* Benzoylcegonine (BE)
- Benzoylcegonine (BE), 276–277
- Billiard ball effect, 360
- Biologic (terrorist) agents, 581–582
- Bacillus anthracis* (anthrax), 581
 - Clostridium botulinum* (botulism), 582
 - dengue fever, 582
 - Ebola virus, 582
 - Francisella tularensis* (tularemia), 582
 - Hantaan virus, 582
 - hemorrhagic mediastinal lymphadenitis, 581
 - Lassa virus, 582
 - Marburg virus, 582
 - Variola virus (orthopoxvirus), 582
 - yellow fever, 582
 - Yersinia pestis* (plague), 582
- Birth-related infant deaths
- asphyxia, 506
 - trauma, 506
- Black (or brown) tar heroin, 280f
- Blanching, 164, 415, 458
- Blast injuries, categories of, 554
- Bleeding esophageal varices (hematemesis), 237, 238f
- Blistering agents, 488, 582
- Bloated individual, advanced decomposition stage, 173f
- Blood alcohol concentration (BAC), 270–271
- Blood spatter analysis, 29, 30f
- Blood spot card, 28, 194, 195f
- Blue black discoloration, 473
- Blunt force injury deaths, 301
- aircraft fatalities, 330
 - control injuries, 330
 - blunt force head/neck trauma, 310–322
 - blunt force head injury, 312f

- closed head injuries, 311
- craniocerebral trauma, 311
- epidural, subdural, and subarachnoid hemorrhage, 315–316, 317–320, 320–321
- neck, spinal cord/vertebral artery injuries, 321–322
- skin/mucosal injuries, 312
- skull/facial bone injuries, 313–314
- subcutaneous injuries, 312–313
- blunt force trauma, death in, 322–323
- classification, 302–310
 - abrasions, 302–303
 - avulsions, 310
 - contusions, 304–305
 - fractures, 308–310
 - lacerations, 305–306
- clothing examination, 327
- defensive injuries, 331
- falls/jumps from heights, 331
- massive body trauma, 331
 - multiple traumatic injuries, 331f
- motor vehicle collisions
 - dicing injuries, 327
 - dicing injuries on the left cheek, 329f
 - impact with a steering wheel, 328f
 - injury in driver-side seatbelt shoulder strap, 328f
- patterned injuries, 325–326
- pedestrians, 329–330
 - homicides, 330
 - tire track marks on a murder victim, 330, 330f
- Blunt force trauma, death in, 322–323
 - abrasion in a blunt impact, 326f
 - cardiac tamponade, 323
 - commotio cerebri, 323
 - hemopericardium, 322
 - hemothorax, 323
 - malignant cerebral edema, 323
 - pneumothorax, 323
 - pulmonary embolism, 324
 - pulmonary thromboemboli evident, 325f
 - sepsis, 324
 - tissue perfusion, 322
 - tram track injury, 325f
- Body cooling, 56, 67
- Body mass index (BMI), 563
- Body packing syndrome, 262, 277
- Body stuffing syndrome, 262, 277
- Bone marrow and fat emboli, 548
 - fat embolism syndrome, 548
- Bone saw, 138, 139f, 147, 151, 534
- Bones, joints, and soft tissues, 245
 - inflammatory joint disorders (arthritis), 245
 - osteoarthritis (a wear and tear disorder), 245
 - osteomyelitis, 245
 - primary bone cancers, 245
 - rheumatoid arthritis, 245
 - sarcomas, 245
- Boxer's stance, 492
- Brain, bacterial infections in
 - brain abscess, 224
 - meningitis, 224
- Brain death, 67, 434, 548, 568
- Brain, nervous system, 98–102, 100f–101f
 - brainstem, 102
 - cerebellum, 101
 - cerebrum, 98, 101f
 - CSF, 101
- Breakdown product, *see* Metabolite
- Bridging veins, 98, 99f, 103, 316
- Bright red-pink discoloration of livor mortis, 472
- Bronchial asthma, 233
- Bronchopneumonia, 231, 473–474
- Bruises, *see* Contusions, Blunt force injury deaths
- Bucket-handle/corner fractures, 309
- Bullet and other foreign body emboli, 550
- Burns and fire-related deaths, 481–498
 - burn types, 484–489
 - chemical burns, 487–488
 - dry burns, 484
 - fire-related burns, 488–489
 - radiant burns, 484–485
 - scald burns, 485–486
 - fire deaths, 489–498
 - autopsy, 492–496
 - death certification, 497–498
 - fire investigation, 498
 - mechanism of death, 496–497
 - general percentages of listed body regions, 484
 - metabolic complications, 483
 - rule of nines, (forensic pathologists), 484
- Burn severity
 - first degree burns (erythema), 482, 482f
 - fourth degree burns (charring of skin), 482, 483f
 - extensive skin-splitting, 495f
 - multiple burn severities, 493f
 - second degree burns (blisters/skin slippage), 482, 482f

- Burn severity (*cont.*)
 succumbed to smoke and soot
 inhalation, 488
 thermal injuries, 493f
 “pugilistic” appearance of badly burnt
 body, 494f
 thermally-induced defect in abdomen,
 494f
 third degree burns (full-thickness skin
 injury), 482, 483f
- Burns, radiant, 484–485
 electromagnetic energy burns, 484
 ionizing radiation burns, 485
 postmortem dry burn caused by decedent’s
 leg, 485f
 radiant heat burns, 484
 sunburn, 485
- Burnt beyond recognition, 186, 186f, 489
- Burn types, 484–489
 chemical burns, 487–488
 dry burns, 484
 fire-related burns, 488–489
 radiant burns, 484–485
 scald burns, 485–486
- “But for” rule, COD, 45, 69, 77, 94, 157–158,
 198, 513
- C**
- CABG surgery, *see* Coronary artery bypass
 graft (CABG) surgery
- Cadaveric spasm, 166, 437, 438f
- CAMI, *see* Civil Aeromedical Institute
 (CAMI)
- Campylobacter (bacteria), 566
- CAP, *see* The College of American Pathologists
 (CAP)
- Caput succedaneum, 507
- Carboxyhemoglobin test, 421–422
- Cardiac arrest, 69, 462
- Cardiac arrhythmias, 273, 434, 465
- Cardiac conduction system abnormalities, 91,
 207, 222, 437
- Cardiac defibrillators, 451
- Cardiac dysrhythmia, 69, 70, 225, 440,
 452–453, 465
- Cardiac electric disorders, 222
- Cardiac hypertrophy/cardiomegaly, 218
- Cardiac tamponade, 69, 213, 323–324
- Cardinal signs of death, 163, 168
- Cardiomegaly (enlarged heart), 209, 218–219,
 267, 277
 causes, 209
- Cardiopulmonary arrest, 69
- Cardiopulmonary resuscitation (CPR), 403,
 512, 546
- Cardiovascular system
 aortic aneurysms (abdominal and thoracic),
 215–216
 atherosclerosis, AAA, 215f
 thoracic aortic dissection with
 hemorrhage, 216f
 cardiomegaly related to morbid obesity,
 209f
 cerebrovascular disease, 216
 conduction system abnormalities, 222
 congenital heart disease, 209–210
 aberrant origin of coronary artery, 210f
 congestive heart failure (heart failure),
 210–211
 coronary artery atherosclerosis, 211–214
 atherosclerotic plaque causing stenosis,
 211f
 blockage of artery, 212f
 blood filling in pericardial sac at
 autopsy, 213f
 coronary artery dissection, 214
 cross-section of heart
 with acute myocardial infarct, 212f
 myocardial infarct of interventricular
 septum, 213f
 deep venous thrombosis, 222
 dilated cardiomyopathy, 220–222
 hypertrophic cardiomyopathy or IHSS,
 221f
 left/right ventricular (dilated) cavities,
 221f
 fibromuscular dysplasia, 217
 heart/blood vessels, 104
 hypertensive cardiovascular disease, 218
 hypertrophic heart, with hypertension
 (high blood pressure), 218f
 hypertrophic cardiomyopathy, 220
 myocarditis, 217
 neoplastic heart disease, 223
 other coronary artery disorders, 214
 restrictive cardiomyopathy, 222
 SA/AV node, 104
 SVC/IVC, 104
 valve disorders, 219–220
 vasculitis, 217
- Carotid sleeper hold, 419, 425, 558
- Cartilage, *see* Fractures, blunt force injury
 deaths
- Cause of death (COD), 63, 66f, 226
 “but for” rule, 69
 definition, 68

- immediate, 68
- intermediary, 68
- possibilities, with/without anatomic or laboratory COD, 70
- proximate (underlying), 68
- CDC, *see* Centers for Disease Control (CDC)
- Centers for Disease Control (CDC), 63, 217, 225
- Central nervous system (CNS), 97, 223–229
 - cerebral palsy, 223–224, 232, 514
 - cerebrovascular disease, 226
 - congenital anomalies, 223
 - dural sinus thrombosis, 228–229
 - berry aneurysm, 229f
 - hypertensive disease, 226
 - infection, 224–225
 - meningitis, purulent exudate (pus) overlying brain, 224f
 - neoplasia, 229
 - ruptured AVM, 227–228
 - basilar subarachnoid hemorrhage, 228f
 - berry aneurysm, presence, 228f
 - ruptured berry aneurysm, 227
 - seizure disorders, 225–226
 - spontaneous intraparenchymal hemorrhage, 226, 227, 227f
- Central Nervous System (CNS), infections, 224–225
 - AIDS, 225
 - bacterial brain abscesses, 224
 - sepsis/bacterial endocarditis, 224
 - compromised immune system, 225
 - herpes and CMV, 225
 - in infants and children
 - Hemophilus*, 224
 - meningitis, 224
 - multiple brain abscesses, 224
 - in newborns
 - gram-negative bacilli (B *Streptococcus*), 224
 - parasitic infection
 - Naegleria fowleri*, 225
 - neurocysticercosis, 225
 - Prion diseases
 - CJD, 225
 - spongiform encephalopathies, 225
 - purulent material (pus), 224
 - in teenagers and young adults
 - Neisseria meningitidis*, 224
 - viral encephalitis, 224
- Central pontine myelinosis (CPM), 246–247, 270
- Cephalohematoma, scalp injury, 507
- Cephalopelvic disproportion (CPD), 506–507
- Cerebral artery berry aneurysm, 218, 227, 324
- Cerebral infarcts (bloodless or hemorrhagic), 226
- Cerebral palsy (CP), 45, 223–224, 232, 514
 - non-progressive neurologic motor (muscular) disorder, 223
- Cerebrospinal fluid (CSF), 83t, 98, 101–102, 154, 229, 230f, 266, 271, 315, 534, 534f
- Cerebrovascular accident (CVA), 216, 226
- Cerebrovascular diseases, 216, 226
 - cerebral infarcts (bloodless or hemorrhagic), 226
 - remote (old) strokes, 226
 - spontaneous intraparenchymal hemorrhages, 226
 - stroke or CVA, 216, 226
- Certifying agencies for MD/DO degrees
 - NBME, 14
 - NBOME, 14
- CF, *see* Cystic Fibrosis (CF)
- CFTR, *see* Cystic fibrosis transmembrane conductance regulator (CFTR)
- Chemical asphyxia
 - carbon monoxide, 419–422
 - automobile exhaust inhalation, 420
 - carboxyhemoglobin, 421–422
 - cause, necrosis, 420
 - cyanide, 422–423
 - autopsy feature/odor, 423
 - cytochrome oxidase, 422
 - oxidative phosphorylation, 422
 - hydrogen sulfide, 423
 - autopsy finding, 423
- Chemical asphyxiants, drugs, 269, 291
- Chemical burns, 487–488
 - caustic chemicals (decontamination), 487
 - chemical warfare agents
 - phosgene oxime (CX)/vesicants, 487
 - skin blistering, 487
 - sulfur mustard/lewisite, 487
 - extensive chemical skin burns, 487f
 - MSDS, 487
 - preventive measure, 487
 - suicidal ingestion of chemical (lye), 487
 - tissue damage, 487
 - types, 487
- Chemical (terrorist) agents, 581–582
 - blistering agents, 582
 - cyanide agents, 582
 - nerve agents, 582–583
 - ricin, 583

- Chemical warfare agents
 - phosgene oxime (CX)/vesicants, 487
 - skin blistering, 487
 - sulfur mustard/lewisite, 487
- Chemistry tests, postmortem, 572–574, 573t
 - electrolytes, 572
 - glucose, 572
 - low salt vitreous electrolyte pattern, 573, 573t
 - nitrogen compounds, 572
- Childhood deaths
 - accidental
 - in adolescents, 515
 - in infants, 514
 - in older children, 515
 - toddler hanging by mini-blind cord, 515f
 - in toddlers, 514–515
 - deaths in infancy, 501
 - deaths of older children(adolescents), 501
 - deaths of young children, 501
 - neonatal deaths, 501
 - natural death
 - types of cancer, 513
- Childhood homicides, classification, 526
 - acute psychosis, 530
 - altruism, 530
 - drug and alcohol abuse, 532
 - euthanasia (true mercy killing), 531
 - innocent bystander, 532
 - Munchausen's syndrome by proxy, 529
 - negligence/neglect, 527
 - postpartum mental disorder, 530
 - sadistic acts of punishment, 528
 - seizure disorder, 532
 - sexual abuse, 531
 - spouse revenge, 530
 - unwanted child, 529
 - unwanted pregnancy (neonaticide), 529
 - violent older child, 532
 - violent outburst (angry impulse), 527
- Choke-hold, 419, 425
- Choking deaths, 406–408, 406f, 407f
 - accidental, scenario, 406–407
 - game, 424–425, 426f
 - homicidal/natural/suicidal, 407
 - internal airway obstruction, 406f–408f
- Chop wounds, 379, 388
 - of scalp, 388f
- Chromosomal disorders, 250
 - cri-du-chat syndrome, 250
 - deletion syndromes, 250
 - Down syndrome, 250
 - Edwards syndrome, 250
 - Klinefelter syndrome, 250
 - Patau syndrome, 250
 - sex chromosome abnormalities, 250
 - trisomy syndromes, 250
 - Turner syndrome, 250
- Chromosomes, 27
 - base-pair sequence, 28
 - 46 chromosomes, human body, 27
 - 22 pairs of autosomes/1pair of sex chromosomes, 27
 - single X/single Y chromosome, males, 27
 - two X chromosomes, females, 27
- Chronic alcohol abuse, 268
- Chronic alcoholism, 71, 235, 237, 240, 246–247, 268, 270, 563–564
 - CPM, 246
 - ethanol-induced dilated cardiomyopathy, 247
 - spontaneous bacterial peritonitis, 247
- Chronic asthmatic bronchitis, 233
- Chronic blood loss (anemia), 236
- Chronic brain injury, 232
- Chronic hypoxia/right-sided heart failure, 234
- Chronic intravenous drug abuse, complications, 268
- Chronic lung disease, 235
 - chronic alcoholism or chronic drug abuse, 235
 - interstitial lung diseases/pneumoconiosis, 235
 - occupational lung diseases, 235
- Chronic obstructive pulmonary disease (COPD), 234–235
 - alpha-1-antitrypsin deficiency, 234
 - chronic hypoxia/right-sided heart failure, 234
 - emphysema/chronic bronchitis/bronchiectasis, 234
- Chronic pancreatitis, 240
- Chronic seizure disorder, 225
- Ciguatera, 567
- Cingulate gyrus herniation/subfalcine herniation, 319
- Circle of Willis, 103
 - See also* Skull, nervous system
- Circumferential marginal abrasion, 344, 358
- Circumstantial identification, 198
- Cirrhosis/esophageal varices, 237–238
 - bleeding esophageal varices (hematemesis), 237, 238f

- liver demonstrating cirrhosis, nodularity in, 237f
- portal hypertension, 237
- spontaneous bacterial peritonitis, 237
- Civil Aeromedical Institute (CAMI), 540, 542
- CJD, *see* Creutzfeld-Jakob disease (CJD)
- Clinical forensic medicine, 35–36
- Clinical pathology (CP), 6, 13–14, 14t, 43, 45, 223–224
- Closed head injuries, 311–312
- Clostridium botulinum* (botulism), 582
- Clothing examination, 327, 368, 383, 390, 457–458
 - hit-and-run pedestrian death, importance in, 134
- CMV, *see* Cytomegalovirus (CMV)
- CNS, *see* Central nervous system (CNS)
- Cocaine, amphetamines substances, 275–277
 - benzoylecgonine (BE), 276
 - cocaine-induced excited delirium, 276f
 - crack cocaine in pocket of homicide, 275f
 - crack pipe along with disposable lighter, 277f
 - erythroxylon coca*, 275
 - excited delirium, 276
- Cocaine intoxication, 69, 267
- COD, *see* Cause of death (COD)
- Collagen vascular diseases, 241
- The College of American Pathologists (CAP), 14, 44
- Columnar epithelium, 93
- Combination asphyxial deaths, 427–428
- Combined intoxication with cocaine and heroin, *see* Combined toxic effects of multiple drugs
- Combined toxic effects of multiple drugs, 267
- Commensal organisms or normal flora, 111, 207
- Commotio cerebri, 323
- Complement deficiency syndromes, 242
- Complete *vs.* limited autopsy, 39
- Compromised immune system, 225, 242
- Computer forensics, 30–31
- Conducted, energy devices/electrical weapons, 464
- Conduction system, 91, 104, 144, 207, 214, 222–223, 469–470, 556
 - abnormalities, 222
 - cardiac conduction system abnormalities, 222
 - cardiac electric disorders, 222
 - EKG or EKG, 222
 - long QT syndrome, 222
 - Wolff–Parkinson–White syndrome, 222
- Congenital anomalies, 143, 207, 223, 229–230, 236, 238, 508
 - anencephaly, 223
 - Arnold–Chiari malformation, 223
 - Dandy–Walker abnormality, 223
 - GI/liver systems, 236
 - Hirschprung disease, 236
 - holoprosencephaly, 223
 - incidental/internal anomalies, 236
 - Meckel’s diverticulum, 236
 - potentially lethal defects, 236
 - spina bifida, 223
 - tracheal bronchus, 230
- Congenital diaphragmatic hernia, 508
- Congenital disorder, 210, 227, 514
- Congenital heart disease, 209–210, 214, 576
 - ASD/PDA/VSD, 209
 - congenitally bicuspid aortic valve, 210
 - examples, 209–210
 - myocardial hypoxia, 210
 - systemic cyanosis, 209
- Congenitally bicuspid aortic valve, 210
- Congestive heart failure, 210–211, 231, 289
 - inefficient pumping, cause, 210
 - left/right-sided heart failure, 211
 - lung, or systemic disorders, 211
- Congo red stain, 246
- Connective tissue diseases, 241
- Consumer Product Safety Commission (CPSC), 578, 586
- Contral pontine myelinolysis (CPM), 246, 270
- Contrecoup contusion, 318–319
- Control surface injuries, 542
- Contusions, blunt force injury deaths, 304
 - contusion of the chest, 304f
 - ecchymosis, 304
 - mastoid process, 304
- Convection, 469–470
- COPD, *see* Chronic Obstructive Pulmonary Disease (COPD)
- CO poisoning, 420f, 421
- Corneas, clouded, 168
- Corner fractures, 309
- Coronary artery atherosclerosis, 211–214, 277, 437, 576
 - CABG surgery, 214
 - calcified coronary artery atherosclerotic plaques, 213
 - cardiac tamponade, 213
 - hemopericardium, 213
 - invasive therapies, 213

- Coronary artery atherosclerosis (*cont.*)
 angioplasty (compression with tiny balloon), 213
 bypass surgery, 213
 ischemic cardiomyopathy, 214
 myocardial infarcts (heart attacks), 212
 pericardial cavity (heart sac), 213
 thrombus (blood clot) formation, 211
- Coronary artery bypass graft (CABG) surgery, 214
- Coronary artery disease, 211
See also Coronary artery atherosclerosis
- Coronary artery disorders, 214
 aberrant coronary artery anatomy, 214
 intramyocardial tunneling or bridging, 214
 Kawasaki disease, 214
 small-caliber (diminutive) coronary artery, 214
- Coronary artery ostia, 216
- Coroner systems, 36, 41, 50–52
 Justices of the Peace, Texas, 51
 official death certification, 52
- Cortical adenomas (benign tumors), 244
- Cortical carcinomas (cancer), 244
- Co-sleeping, 512
- “Coup” contusion, 318–319
- CP, *see* Cerebral palsy (CP)
- CPD, *see* Cephalopelvic disproportion (CPD)
- CPM, *see* Central pontine myelinosis (CPM)
- CPR, *see* Cardiopulmonary resuscitation (CPR)
- CPSC, *see* Consumer Product Safety Commission (CPSC)
- Craniocerebral trauma, 311
- Cremations, 498
- Creutzfeld-Jakob disease (CJD), 225
- Cri-du-chat syndrome, 250
- “Crime of passion,” 178
- CSF, *see* Cerebrospinal fluid (CSF)
- Cultures, postmortem
 meningitis, 574
neisseria meningitidis (or meningococcus), 574
 stat gram stain, 574
- Curling/Cushing ulcers, 236
- ‘Current,’ definition, 450
- Cutaneous, *see* Integumentary system
- CVA, *see* Cerebrovascular accident (CVA)
- Cyanide
 agents, 582
 gases, 497
- Cystic Fibrosis (CF), 247, 250–251, 514
 sweat chloride test, 250
- Cystic fibrosis transmembrane conductance regulator (CFTR), 250
- Cystic medial necrosis, 216
- Cytology or cytopathology
 cells scraped off from a tissue surface, 11
 cytology slide (breast cancer) viewed under the microscope, 13f
- Cytomegalovirus (CMV), 225, 232, 243
- Cytosine (C), 28
- D**
- DAI, *see* Diffuse axonal injury (DAI)
- “Dandy–Walker” abnormality, 223
- Dead on arrival (DOA), 188
- Death
 definitions, 67–68
 types, 70
- Death certification, 63
 cause of death, 68
 circumstances of death, 65
 COD, 65, 66f
 COD/MOD, documentation of, 41
 death pronouncement, 66
 signs of death, 67
- decedent identification, criteria for, 65, 65f
- fire deaths, 497–498
 natural/homicide/suicide/accidental (MOD), 498
 self-immolation, 498
 smoke and soot inhalation, 498
 thermal injuries, 498
- forensic pathologist, role in, 41
 manner of death, 71
 MOD, 65, 66f
 ‘pending’ certificate, 66
 rules, 63
 types of death, 65
 asphyxial, 70
 drownings, 70
 electrocutions, 70
- Death investigation agency, 13, 36–37, 40–41, 43, 50–51, 54, 65, 157–158, 186, 189, 198, 476, 508, 560, 575
- Death investigation process, 40, 61
 county-based medical examiners
 systems/coroner systems, 36
 establishing/maintaining high-quality death investigation system, criteria for, 37
 steps involved
 performance of autopsy, 37
 scene investigation (initial), 37, 38f
 subsequent follow-up investigation, 37

- Death investigation systems, 44t, 49–50
agency, 50
death investigator, duties of, 53–60
 follow-up investigation, 60
 initial investigation, 54–55
 scene investigation, 55–60
grief counseling, 61
in US
 coroner systems, 51–52
 jurisdictional issues, 51
 medical examiner (ME) systems, 52–53
 mixed systems, 53
- Death investigator, 37, 131, 133, 153, 164, 167, 192, 257–259, 264, 368, 404, 453–454, 530–531, 550, 579, 583
duties, 53
 follow-up investigation, 60
 initial investigation, 54–55
 scene investigation, 55–60
- Death mechanism, 453
- Decalcification process, 95
- Decomposition, 169–177, 395
adipocere formation, 175, 175f–176f
advanced decomposition, 173
anthropophagy, 169, 172
bloated Caucasian individual, advanced decomposition, 173f
fatty decomposition fluid, 177
flies/maggots, 173, 175f
fluid, 169–170, 177
gastromalacia, 177
green discoloration, 170f
insects, 172
loss of soft tissues, autolysis/putrefaction, 176, 176f
marbling, 170, 171f
micro-organisms, 170
miliaria, 177
mummification, 177f
purge fluid, 170, 172f
putrefaction, 169
rate of, 441
saponification of the fat body, 174–175
skeletonization, 174, 174f
skin blistering, 171f
skin slippage, 170, 172f
'swiss cheese' appearance, 177
See also Postmortem changes
- Decompression sickness, 445
difficulty breathing (chokes), 445
joint pain (bends), 445
- Decontamination, concept of, 561
- Deep venous thrombosis, 222, 366, 547, 563, 575, 585
 pulmonary thromboembolism, 222
 thrombophlebitis, inflammatory process, 222
- Defensive wounds, 391
- Degree of certainty, 188
- Dehydration, 69, 249, 562, 564–566, 572–573
 severe dehydration, with sunken eyes, 566, 566f
- Deletion syndromes, 250
- Delta-9-tetrahydrocannabinol (THC), 282
- Dengue fever, 582
- Dental identification, 20, 191–192
 antemortem, 191
 dental chart, 191
 dental X-rays, 191
- Dependent hypostasis, 164
- Dextroamphetamine (Adderal), 273
- Dextromethorphan (DXM), 278, 284
- Diabetes Mellitus (DM), 240–241, 243–245, 247–248, 514, 563, 573t, 574, 577
 Armanni–Ebstein, 248
 arteriolosclerosis, 248
 diabetic nephropathy, 248
 endogenous insulin (C-peptide), 248
 free insulin levels, 248
 gestational DM, 247
 Kimmelstiel–Wilson lesion, 248
 long-term insulin replacement therapy, 248
 mucormycosis, 248
 pathologic disturbances, 247
 subendocardial myocardial infarcts, 248
 total insulin levels, 248
 transmural (full thickness) infarcts, 248
 type II-non-insulin dependent DM or adult-onset DM, 247–248
 type I-insulin-dependent DM or juvenile DM, 247
- Diabetic nephropathy, 248
- Diagnosis of exclusion, 70, 156, 226, 437, 445, 475, 478
- Diaphragm, 84t, 90, 92, 98, 107, 139
- Diaphysis, 84t, 94
- Dicing injuries, 327
- Dieners, *see* Autopsy assistants
- Diffuse axonal injury (DAI), 321, 323, 519, 535
- DiGeorge syndrome, 242
- Dilated cardiomyopathy, 220–222, 247, 270, 563, 576
 arrhythmic right ventricular cardiomyopathy, 221

- Dilated cardiomyopathy (*cont.*)
 hypertrophic cardiomyopathy (genetic mutations), 221
 peripartum cardiomyopathy, 221
- Dimethyltryptamine (DMT), 284
- Disaster Mortuary Operational Response Teams (D-MORT), 540, 561
- Discarded fetuses/infants, fetal/infant deaths in unattended births
 cause of death, 505
 discarded newborn, 502, 502f
 fetus/infant live-born or stillborn, 503
 fetus/infant viable, 503
 gestational age, 503
 maceration, 503
- Disciplines of forensic sciences
 blood spatter analysis, 29
 computer forensics, 30–31
 forensic anthropology, 19, 20f
 forensic artistry, 31
 forensic engineering, 31
 forensic entomology, 20–21
 forensic odontology, 19–20
 forensic pathology, 19
 forensic psychiatry, 22
 forensic toxicology, 22
 impression analysis, 30
- Disseminated intravascular coagulopathy (DIC), 577
- DM, *see* Diabetes Mellitus (DM)
- D-MORT, *see* Disaster Mortuary Operational Response Teams (D-MORT)
- DMT, *see* Dimethyltryptamine (DMT)
- DNA
 fingerprinting, 27
 identification, 192
- DNA molecule
 made of nucleotide, 28
 parts of, 28
 variable DNA regions
 PCR/RFLP, 28
 STR/VNTR, 28
- DOA, *see* Dead on arrival (DOA)
- DOM, *see* Methyl-2,5-dimethoxyamphetamine (DOM)
- Double-action, *see* Hammer cocked
- Down syndrome, 250
- Drip or splash pattern of burns, 486
- Drowning, 428, 433–447
 accidental, 446
 autopsy findings, 437–444
 cadaveric spasm/instantaneous rigor mortis, 438f
 decomposition with unusual color variation of skin, 442f
 diagnoses of exclusion, 437
 drowning victim, 443f
 frothy pulmonary edema fluid within larynx, 438f
 marked decomposition in drowning victim, 442f
 microscopic examination, 440
 nonspecific findings, 437, 439
 petrous ridge discoloration/hemorrhage, 439f
 pulmonary edema fluid from nose, 439f
 sphenoid sinus, water being withdrawn, 440f
 true antemortem sharp force injuries on chin, 444f
 washerwoman hands recovered from water, 441f
- combined drowning/electrocution death occurred, 445f
- MOD, 446–447
 accidental bathtub drowning, 446f
 accidental drownings, 446
 bathtub drownings, 446
 homicidal drownings, 446
 suicidal drowning deaths, 446
 swimming pools or other bodies of water, 446
- occasional drowning deaths, 444
 physiology and mechanism of death, 434
 dry drowning, 434
 freshwater drowning, 434
 ‘near drowning,’ 434
 post-immersion syndrome, 434
 salt water drownings, 434
 sequelae of near drowning, 434
- scene investigation, 435–436
 automobile, swept away in flash flood, 435f
 clean, clear recreational water bodies, 435f
 driver of motor vehicle, 436f
 murky water bodies, 435f
 person, swept away by flash flood and drowned, 436f
 potential hazards, 435f
- SCUBA deaths, possibilities, 445
 decompression sickness, 445
 lack of oxygen, 445
 pneumothorax, 445

- pressure related processes (barotrauma), 445
- Drowning deaths, accidental, 446
- Drug/alcohol abuse, childhood homicides, 532
- Drug/toxin-related deaths, 257, 437
 - amphetamines/similar substances, 273–277
 - barbiturate blisters, 275
 - cocaine, 275–277
 - examples, 273
 - globus pallidus, 275
 - Khat (cot), 274
 - autopsy findings, 261–263
 - body packing syndrome, 262
 - body stuffing syndrome, 262
 - foam cone, 262f
 - fresh needle puncture marks, 261f
 - needle tracks on arm, 261f
 - opened stomach at autopsy, 263f
 - sigmoid colon opened at autopsy, 263f
 - death certification, 267–269
 - ending others' suffering, 269
 - Florence Nightingale syndrome, 269
 - polypharmacy, 268
 - drugs/toxins, 269
 - chemical asphyxiants, 269
 - ethanol/related substances, 269–273
 - heavy metals, 294
 - arsenic, 294–295
 - cadmium, 295
 - iron, 295
 - lead, 295
 - mercury, 295–296
 - investigation, 258–260
 - closer view of the dumped body, 260f
 - death investigators inventory, 260f
 - drug-related death, 258, 258f–259f
 - dumped body, in an abandoned warehouse, 260f
 - mushrooms (psilocybin), 283–284
 - naturally-occurring toxins, 297
 - opiates/related substances, 277–279
 - chronic intravenous drug, 278f
 - fentanyl, 281
 - heroin, 279–280
 - methadone, 280–281
 - morphine, 279
 - narcotic describes, 277
 - Papaver somniferum*, 278
 - over-the-counter (OTC) drugs, 284–285
 - acetaminophen, 285
 - aspirin (salicylate), 285
 - ephedrine/herbal ecstasy, 286
 - poisons, 296
 - organophosphates, 296
 - strychnine, 296
 - prescription drugs, 286
 - anabolic steroids, 288
 - antidepressants, 286
 - antipsychotic drugs, 287–288
 - insulin, 288–289
 - nonbarbiturate sedative hypnotic drugs, 286–287
 - psychoactive drugs of abuse (Hallucinogens), 282
 - Lysergic Acid Diethylamide (LSD), 282–283
 - marijuana, 282
 - mescaline (peyote), 283
 - PCP, 283
 - toxicology issues, 263–267
 - breakdown product, 264
 - femoral blood collection at autopsy, 265f
 - metabolism, 263
 - pharmacogenomics, 267
 - postmortem redistribution, 266
 - test-tube rack of toxicology, 266f
 - toxicologist/a GC/MS, 265f
 - unwrapped plastic cellophane, 264f
 - volatiles inhalants, 289–291
 - carbon monoxide, 290
 - cyanide and hydrogen sulfide*, 291
 - foam cone inhalant abuse, 290f
 - helium/other simple asphyxiants, 293
 - hydrocarbons, 291–293
 - nitrous oxide, 291
 - paint on hands, 290f
- Dry burns, 470, 484–485, 489
 - electrical burns, 484
 - electrothermal burn, 484
 - examples, 484
 - fire-related (flame) injuries, 484
- Dry drowning, 434
- Dural sinuses, 228, 547
- Dural sinus thrombosis, 228–229
 - thrombus (blood clot) formation, 228
- Dura mater/dura, skull, 98f
 - falx cerebri, 102
 - subarachnoid space, 102
 - subdural space, 102
 - tentorium, 102
 - See also* Brain, nervous system
- Duret hemorrhages, 319–320
- Duties of forensic pathologist, 36t
 - autopsy, 37–40
 - consultation, 42–43

- Duties of forensic pathologist (*cont.*)
 death certification, 41
 documentation of findings, 40–41
 investigation, *see* Death investigator
 testifying, 41–42
 verification of identity, 40
- DXM, *see* Dextromethorphan (DXM)
- Dysrhythmia, *see* Arrhythmia
- E**
- Eastern equine encephalitis, 225
- Ebola virus, 582
- E. coli* 0157:H7 (bacteria), 566
- ECG, *see* Electrocardiograms (ECG/EKG)
- Ectopic pregnancy, 575
- EDS, *see* Ehlers-Danlos syndrome (EDS)
- Edwards syndrome, 250
- EEG, *see* Electroencephalogram (EEG)
- Ehlers-Danlos syndrome (EDS), 214, 250–251, 575
- EKG, *see* Electrocardiograms (ECG/EKG)
- Electrical burns, 484
- Electrical deaths, 449
 autopsy findings
 high voltage, 457
 low voltage, 455
 other features, 459
 death certification/manner of death, 465
 electrocution, 451
 lightning, 461
 mechanism of death in electrocutions, 453
 non-lethal electronic shock devices, 464
 scene investigation, 453
- Electrical injury
 electrical petechiae, 460
 microscopic appearance of a skin, 462f
- Electrically-induced cardiac arrhythmia, 453
- Electricity, description, 449
- Electrocardiograms (ECG/EKG), 67, 91, 222, 323
- Electrocution, 31, 70, 257, 439, 444, 451–461, 465, 516
 deaths, 437
 no evidence of injury, 456f
- Electroencephalogram (EEG), 67
- Electromagnetic energy burns, 484
- Electrothermal burn, 484
- Emboli
 amniotic fluid emboli, 548–549
 disseminated intravascular coagulation, 549
 bone marrow and fat emboli, 548
 fat embolism syndrome, 548
 bullet and other foreign body emboli, 550
 gas/air emboli, 549–550
 chest X-ray, with a radiolucent area of air within heart, 550f
 traumatic causes, 549
 vapor lock, 549
 pulmonary thromboemboli, 546–548
 blood thrombi blocking pulmonary artery, 547f
 DVT, 547
 saddle embolus, 548
 systemic thromboemboli, 548
 thromboembolus, 546
- Embolus, *see* Emboli
- Emergency medical services (EMS), 54, 58f, 60, 324
- Emphysema/chronic bronchitis/bronchiectasis, 234
- EMS, *see* Emergency medical services (EMS)
- En bloc conglomeration, 141–142, 534
- Endocrine system, 116–119, 243–244
 adrenal glands, 118, 119f, 244
 clinical disorders, 244
 metabolic processes, 116
 multisystem disorders, 243
 diabetes mellitus, 243
 pancreas, 119, 119f
 islets of Langerhans, 119
 parathyroid disorders, 244
 parathyroid glands, 118
 pineal gland, 117
 pituitary disorders, 243
 pituitary gland/hypophysis
 neurohypophysis, 117
 pituitary (master gland), 243
 ACTH/TSH/LH/FSH/hGH (hormones), 243
 primary adrenal disorders, 244
 thyroid disorders, 243
 thyroid gland, 117–118, 118f
- Endogenous insulin (C-peptide), 248, 288
- Entomology (study of insects), 20–21, 180–181
- Environmental asphyxia, *see* Simple asphyxia
- Ephedrine/herbal ecstasy, 286
- Epidermis, skin parts, 93
- Epidural, subdural, and subarachnoid
 hemorrhage, 315–316, 317–320, 320–321
 epidural hemorrhage, 315f
 middle meningeal artery, 315
 subarachnoid hemorrhage, 317f
 subdural hemorrhage, 316

- bridging veins, 316
 - dura in the inner aspect of the skull, 316f
 - hemosiderin, 316
 - neomembrane, 316
 - subdural space, 315–316
 - Epiphysis, 94
 - Epithelium/epithelial cells, *see* Histology
 - Erythrocytes, *see* Red blood cells (rbcs)
 - Erythroxylon coca* (Cocaine), 275
 - Ethanol, effects of, *see* Acute/chronic alcoholism
 - Ethanol-induced dilated cardiomyopathy, 247
 - Ethanol/related substances
 - breathalyzer, 270
 - calcium oxalate crystals in kidney, 272f
 - ethylene glycol, 273
 - isopropanol, 273
 - rubbing alcohol, 273
 - liver cirrhosis seen at autopsy, 271f
 - methamphetamine use (meth mouth), 274f
 - methanol, 272–273
 - wood alcohol, 272
 - Ethyl alcohol (EtOH), 269–272
 - EtOH, *see* Ethyl alcohol (EtOH)
 - Euthanasia (true mercy killing)
 - incapacitating genetic disease, 531
 - Evaporation, 469–470, 475
 - insensible water loss, 470
 - sweating process, 470
 - Evisceration, 137
 - bread-loafing, fashion, 143
 - techniques
 - Ghon (en bloc) method, 142
 - Rokitansky method, 142
 - Virchow method, 140
 - Examinations for MD training, *see* United States Medical Licensing Examination (USMLE)
 - Excited delirium, 283, 427, 559
 - cocaine-induced, 70, 78, 276, 559–560
 - syndrome, 475
 - Exhumation, 550–554, 551f
 - anaerobic decomposition in an unembalmed woman, 551f
 - embalmed body, 553f
 - other items, 552–553, 553f
 - trocár embalming process, 552f
 - trocár or embalming button, 553f
 - Explosions and blast injuries, 554
 - categories of blast injuries, 554
 - high/low order explosives, 554
 - non-military explosion, 554
 - Explosives, high/low order, 554
 - Exsanguination, 69, 322, 324
 - death mechanism, 69, 322
 - Extensive high-voltage
 - electrical burns in a power company employee, 459f
 - thermal injuries on foot underlying the boot, 462f
 - External airway obstruction, *see* Smothering deaths
- F**
- FAA, *see* Federal Aviation Administration (FAA)
 - Facial peel-down, 150, 313, 514
 - Fallopian tubes, 90, 120–121, 141, 196, 575
 - viewed under
 - high power magnification, 12f
 - low power magnification, 11f
 - medium power magnification, 11f
 - very high power magnification, 12f
 - Fasciotomy incisions, 493
 - Fatal arrhythmia, 75, 212, 217, 247, 277, 451, 465, 576
 - Fatal arterial gas embolism, 445
 - Fat necrosis, 240
 - Fatty decomposition fluid, 177
 - FDA, *see* Food and Drug Administration (FDA)
 - Federal Aviation Administration (FAA), 540
 - Fetal death, 507, 576
 - Fetal/infant deaths, births (unattended)
 - cause of death, 505
 - discarded newborn, 502, 502f
 - fetus/infant live-born or stillborn, 503
 - fetus/infant viable, 503
 - gestational age, 503
 - “maceration,” 503
 - Fibromuscular dysplasia, 217–218
 - conduction system, 217
 - AV/SA node, 217
 - Field agent, 52–53, 133
 - See also* Death investigator
 - Filicide, 517, 526, 529–532
 - Fingerprint cards, 188, 190
 - identification, 190
 - 10-print, 190
 - Fire
 - antemortem/postmortem burns, 489
 - breathing during fire, 489
 - autopsy findings, 490
 - first indicator (bright red discoloration of tissues), 489
 - occasional suicide victims, 490

- Fire (*cont.*)
- scene/fire investigation findings, 490
 - second indicator (CO level within blood samples), 489
 - third indicator (soot identification within upperairways), 490–491
 - burnt beyond recognition, 489
 - decident identification (thermal damage)
 - fingerprint/dental, 489
 - X-ray/DNA identification, 489
 - structural fire/vehicular fire, 489
- Fire deaths, 489–498
- autopsy, 492–496
 - artifacts, 495–496
 - external examination, 492–493
 - internal examination, 493
 - toxicology, 494–495
 - death certification, 497–498
 - dense soot coating the mucosal surface of trachea/mainstem bronchi, 491f
 - fire investigation, 498
 - mechanism of death, 496–497
 - minimal soot evident within trachea, 491f
 - soot within nostrils of a fire victim, 490f
 - thermal burns, 481
 - tissue damage, 482
- Fire investigation, 492, 498
- Fire-related burns, 484, 488–489
- charred remnants of torso and head, 488f
 - first/second degree skin burns, 488
 - (flame) injuries, 484
 - radiant heat injury, 489
- First degree burns (erythema), 482, 482f
- First responders, 55–56, 404, 408, 453, 476
- Flash fires, 497
- Flat bones, 94, 363
- Flies/maggots, 173, 175f
- Floppy mitral valve syndrome, *see* Myxomatous degeneration of the mitral valve
- Fluid level lines, 485–486
- Fluid malignancies, 241
- Fly maggots, 21f
- Foam cones, from mouth or nose, 438
- Follicle stimulating hormone (FSH), 117, 122, 243
- Follow-up investigation, 37, 49, 54, 60
- Food and Drug Administration (FDA), 286, 578, 586
- Food poisoning
 - campylobacter (bacteria), 566
 - ciguatera, 567
 - E. coli* 0157:H7 (bacteria), 566
 - gonyaulax* (red tides), 567
 - listeria* (bacteria), 566
 - neurotoxic shellfish poisoning, 567
 - non-seafood-related, 566
 - paralytic shellfish poisoning, 567
 - ptychodiscus brevis*, 567
 - salmonella* (bacteria), 566
 - saurine (toxin), 567
 - scombroid, 567
 - seafood, 566–567
 - tetrodotoxin fish poisoning, 567
 - toxoplasma* (parasite), 566
 - vibrio vulnificus*, 567
- Forensic anthropology, 19, 20f
 - Board certification via American Board of Forensic Anthropology, 19
- Forensic artistry, 31
- Forensic autopsy, components of, 130t
 - ancillary procedures, 153–154
 - postmortem chemistry tests, 154
 - radiographs, use of, 153
 - serology tests, 154
 - toxicology testing, qualitative/quantitative, 153–154
 - X-rays, use of, 153
 - autopsy report
 - ancillary procedures, listing of, 155
 - contents of, 155
 - evidence of injury section, 155
 - external/internal examination section, 155
 - pathologists' opinion statements, 156
- external examination, 134f
 - blood obscuring true nature of an injury, example, 136f
 - documentation of major/minor injuries, 135, 137f
 - evidence piece, hit-and-run pedestrian death case, 134
 - forensic issues in, 134
 - general body parts examined, 134
 - items of evidence, requirement for autopsy, 134
 - paper bags, use at the death scene, 135f
 - photographic documentation, vital factor, 136
 - toxicology samples, collection of, 136, 137f
- internal examination
 - anterior chest plate, removal of, 139f
 - bone saw to cut front of chest plate off of the chest wall, use of, 139f
 - brain removal, steps, 146f–148f

- brain, serially-sectioned in fresh (non-fixed) state, 149f
- classic “Y-shaped” incision during autopsy, 138f
- coronary arteries in removed heart, serial sectioning of, 143f
- epicardial fat with a coronary artery, section of, 144f
- evisceration process, 137
- heart removal, fashion of, 141f
- kidney, half longitudinal cutting, 145f
- liver cut away from the diaphragm, 142f
- liver, serially-sectioning of, 145f
- lungs, removal fashion, 141f
- pathologist policy/practice, variations in, 150f
- serially-sectioning of lungs, 144f
- small intestine, removal fashion, 140f
- special dissections, kinds of, 150–152
- spleen removal, 142f
- trachea, larynx, thyroid gland, removal of, 150f
- trunk injuries, case, 139–140
- trunk organs and diaphragm removed, 146f
- weight of lungs after dissection, importance of, 144
- people (‘selective’) for death investigation, role, 133
- Forensic DNA testing, *see* DNA
- Forensic engineering, 31
- Forensic entomology, 20–21, 180–181
 - Board certification via American Board of Forensic Entomology, 21
 - fly maggots on a decomposing body, 21f
 - postmortem examination of the teeth of burnt body, 21f
- Forensic medicine, definition, 35
- Forensic (medicolegal) autopsies, 11–13
 - sudden, unexpected, violent death cases, 13
- Forensic odontologists, 39, 192
- Forensic odontology, 19–20
 - bite-mark on the skin surface at autopsy, 20f
 - Board certification via American Board of Forensic Odontology, 20
 - types of cases, 19
- Forensic pathology, 19
 - definition, 35, 43–44
 - forensic pathologist, duties of, 36t
 - autopsy, 37–40
 - consultation, 42–43
 - death certification, 41
 - documentation of findings, 40–41
 - investigation, 37
 - testifying, 41–42, 42f
 - verification of identity, 40
 - programs, 43
 - training/qualifications of forensic pathologists, 43–46
 - education/training/examination requirements for Board-certified forensic pathologists (USA), 43t
 - guidelines for AP residency training, 44t
- Forensic psychiatry, 22
 - Board certification via American Board of Psychiatry and Neurology, 22
 - psychological autopsy, 22
- Forensic sciences
 - admissibility of forensic tests, legal standards
 - Frye v United States*, 18
 - Rule 702 of the Federal Rules of Evidence, 18
 - definition, 17
 - disciplines
 - blood spatter analysis, 29
 - computer forensics, 30–31
 - forensic anthropology, 19
 - forensic artistry, 31
 - forensic engineering, 31
 - forensic entomology, 20–21
 - forensic odontology, 19–20
 - forensic pathology, 19
 - forensic psychiatry, 22
 - forensic toxicology, 22
 - impression analysis, 30
 - document examination, 25–26
 - suicide cases, useful in, 25
 - evidence, maintaining ‘chain of custody,’ 18
 - expert witness, 18
 - fact witness, 18
 - fingerprint evidence, 26, 26f
 - certification via International Association for Identification, 26
 - class/individual characteristics, examination, 26
 - firearms and toolmarks examiners
 - AFTME, certifying organization, 25
 - deformed bullet identified at autopsy, 24f
 - evidence bullet/standard bullet fired from suspect weapon, 25f

- Forensic sciences (*cont.*)
- suspect weapon, 'class' characteristics of, 24
 - forensic, defined, 17
 - serology/DNA, 27–28
 - trace evidence, 22–23
 - crime laboratories, accredited by
 - ASCLD, 23
 - hair on fingers of a homicide victim, 23f
 - strand of evidence hair/hair standard obtained from suspect, comparison, 23f
 - types of evidence, 22
- Forensic toxicology, 22, 257, 264
- Board certification via American Board of Forensic Toxicology, 22
 - confirmatory (quantitative) test, 22
 - drug identification, example, 22
 - screening (qualitative) tests, 22
- Forensic vs. hospital autopsies
- documentation aspect, 131, 131f
 - external examination, importance, 131–132
 - organs/tissues fixation in formalin prior to dissection, 133
 - tests/procedures, importance, 132
 - radiography/toxicology tests, 132f
- Fourth degree burns (charring of skin), 482, 483f
- Fractures, blunt force injury deaths, 308
- basilar skull fractures, 310f
 - bucket-handle, 309
 - buckle fracture, 308
 - closed fracture, 308
 - comminuted fracture, 309
 - diastatic fractures, 310
 - fracture of the tibia (shin bone), 309f
 - greenstick fractures, 308
 - humerus (upper arm bone) fracture, 308f
 - linear and curvilinear skull fractures, 309f
 - open fracture, 308
- Francisella tularensis* (tularemia), 582
- Free insulin levels, 248, 289, 574
- Freshwater drowning, 434
- Frothy fluid, 278, 437
- FSH, *see* Follicle stimulating hormone (FSH)
- Full-blown sickle cell disease, 249
- Fungal organisms (histoplasmosis, aspergillus), 232
- Furrow mark, hanging deaths, 414f, 417
- G**
- Galactosemia, 250
- Gamma-hydroxybutyrate (GHB), 284, 287
- Gas/air emboli, 549–550
- chest X-ray demonstrating a radiolucent area of air within heart, 550f
 - traumatic causes, 549
 - vapor lock, 549
- Gas chromatography mass spectroscopy (GCMS), 264
- Gastritis, 236, 247, 270
- Curling/Cushing ulcers, 236
 - Wishnewski ulcers, 236
- Gastrointestinal and hepatobiliary system, 236–240
- cirrhosis/esophageal varices, 237–238
 - congenital anomalies, 236
 - gastritis, 236
 - hemochromatosis, 239–240
 - Mallory–Weiss Tears, 238
 - other GI abnormalities, 238–239
 - pancreatitis, 240
 - PUD, 236–237
- Gastrointestinal (GI) system, 108–111
- mesentery, 110, 110f
 - omentum, 109f
 - oral cavity/anus, 108, 108f
 - peristalsis, 110
 - purpose, 108
- Gastromalacia, 177
- Gaucher disease, 250
- GBM, *see* Glioblastoma multiforme (GBM)
- GCMS, *see* Gas chromatography mass spectroscopy (GCMS)
- Genes
- coding/non-coding regions, 28
 - gene's locus, 27
 - units of genetic information, 27
- Genetic disorders, 250–251
- autosomal dominant/recessive, 250
 - CF, 250–251
 - chromosomal disorders, 250
 - Ehlers–Danlos syndrome, 250–251
 - Marfan syndrome, 250–251
- Genitourinary system, 98, 120–122, 244–245
- clinical renal (kidney) failure, 245
 - kidney, 120f–121f
 - kidney infections (pyelonephritis), 245
 - kidney malignancies
 - Wilms tumor in children/renal cell carcinoma in adults, 245
 - retina, 123f
 - testicle, 122f
 - urinary bladder, 123f
 - urinary/sex organs, 120
 - uterus, 121f

- Gestational DM, 247
- Gestational trophoblastic disease, 577
- GFCIs, *see* Ground fault circuit interrupters (GFCIs)
- GHB, *see* Gamma-hydroxybutyrate (GHB)
- Ghon (en bloc) method, 142
- GI abnormalities, 238–239
 - appendix (acute appendicitis), 238
 - intussusception/volvulus, 239
 - Meckel's diverticulum (Congenital Anomalies), 238
 - strangulated hernia, 239, 239f
- Giant cell arteritis, 217
- GI system, *see* Gastrointestinal (GI) system
- Gliding contusions, 319
- Glioblastoma multiforme (GBM), 229
- Globus pallidus, amphetamines, 275, 421
- Gonyaulax* (red tides), 567
- Goodpasture syndrome, 241
- Goosebumps (cutis anserinus), 166
- Granulomatous inflammation, 114, 208
- Graves disease, 241
- Grief counseling, death investigation, 61
- Grooves, *see* Rifled, weapons/ammunition types
- Gross anatomy, 82–91
 - medical/scientific terms, 88–89, 88t–89t
 - morphology, 82
 - scientific/common names and descriptions, 82–88, 82t–88t
- Gross Brain Injuries, 317–320
 - cerebral cortical contusions in a formalin-fixed brain, 318f
 - cerebral edema (brain swelling), 320
 - cingulate gyrus, 319
 - contrecoup contusion, 318
 - coup contusion, 318
 - Duret hemorrhages, 319
 - gliding contusions, 319
 - herniation contusion., 319
 - intermediary contusions, 319
 - tonsillar herniation, 319
 - transcalvarial herniation, 319
 - traumatic axonal injury, 317
- Ground fault circuit interrupters (GFCIs), 452
- Guanine (G), 28
- Gunpowder stipple marks, 349–350
- Gunpowder tattooing, *see* Gunpowder stipple marks
- Gunshot wound deaths, 337–373
 - exit wounds
 - marginal abrasions absence, 352f
 - firing pin, 338
 - gunshot wounds, 343–362
 - atypical entrance wounds, 345
 - entrance wounds, 344–346
 - graze gunshot wound, 353
 - graze wounds, 351
 - head X-ray showing a bullet fragments, 344f
 - marginal microlacerations, 355f
 - miscellaneous features, 354
 - range of fire, 346–350
 - retained bullets, 344
 - shotgun wounds, 356–362
 - soot present on the hand, 354
 - stellate exit wound, 351
 - subcutaneous bullet, 353
 - high-velocity wounds, 354–356
 - lead snowstorm X-ray, 356f
 - rifle wound of the chin, 356f
 - with tissue damage, 355f
 - miscellaneous features, 354, 362–373
 - clothing examination, 368
 - cylinder-barrel gap mark, 354
 - documentation, 367
 - gunshot residue, 368–369
 - internal examination, 362–366
 - manner of death issues, 369–370
 - mechanism of injury, 366–367
 - special weapons, ammunition, and circumstances, 370–373
 - X-rays, 368
 - penetrating, 337
 - perforating injuries, 337
 - weapons/ammunition, types of, 337–343
 - abrasion collar, 344
 - birdshot, smaller shot, 342
 - buckshot, larger shot, 342
 - class characteristics, 338
 - cocked position (ready-to-fire), 340
 - cylinder-barrel gap, 341
 - discharging firearm, 340
 - hammer cocked, 340
 - handgun ammunition, 341
 - handgun cartridge(unfired), 339f
 - handguns, 340
 - handguns (semi-automatic and revolver)/a rifle, 338f
 - individual characteristics, 338
 - low-velocity vs. high-velocity, 340
 - miscellaneous handguns, 341
 - A 9 mm semi-automatic pistol, 342
 - A .357 revolver, 341
 - revolvers, 340
 - rifles, 342

- Gunshot wound deaths (*cont.*)
 sabot, 343
 semi-automatic (self-loading) pistols, 341
 shotguns, 342
 shotgun shells (gauges), 343
 single-action method, 340
- Gunshot wounds, 346–350
 angled entrance wound stippling on the skin, 350f
 close-range, 349
 contact gunshot wound of the neck, 348f
 contact range, 346
 distant (indeterminate) range, 350f
 gunpowder stipple marks, 349
 intermediate range, 349
 loose contact wound of the chest, 347f
 medium (intermediate) range, 349f
 muzzle imprint abrasion, 346
 scalp with a muzzle imprint abrasion, 348f
 scalp wound showing stellate shape, 347f
 starburst, 346
 stipple marks, 350
 stippling causes, 350
- H**
- Hammer cocked, 340
- Hanging deaths, 412–415
 full-suspension, 412f
 furrow mark, 414f
 partial-suspension, 413f
- Hantaan virus, 582
- Hara-kiri suicide, 392
- Hashimoto thyroiditis, 241
- Hassal's corpuscles, thymus, 116
- Hate crime/bias crime, 178, 555
- HbA molecule, *see* Hemoglobin A (HbA) molecule
- HbS, *see* Hemoglobin S (HbS)
- H&E, *see* Hematoxylin and eosin (H&E), stain type
- Heart, cardiovascular system, 104f–105f
- Heat stroke, 475–476, 478
- Helium/Other Simple Asphyxiants, 293
 death with natural gas, 292f
 helium tank, 294f
 simple asphyxiant, 293
- HELLP syndrome, 576–577
- Hematoxylin and eosin (H&E), stain type, 7, 90, 154
- Hemochromatosis, 239–240, 247, 295
 major manifestations, 239
 primary or hereditary hemochromatosis, 239
- Hemoglobin A (HbA) molecule, 248
- Hemoglobin electrophoresis, 114, 249
- Hemoglobinopathy, 248
- Hemoglobin S (HbS), 249
- Hemolysis, 434, 440, 463, 576
- Hemolytic uremic syndrome, 577
- Hemopericardium, 213, 215, 322
- Hemorrhagic mediastinal lymphadenitis, 581
- Hemorrhagic pancreatitis, 473–474
- Hemosiderin, 303, 316, 512
- Hemothorax, 215, 323
- Hepatobiliary system, 111–122, 236–237
 hepatocytes, 112, 113f
 liver/gallbladder, 112, 112f
- Herniation contusion., 319
- Herpes simplex virus (HSV), 232, 243
- Hesitation marks (tentative injuries)
 hesitation wounds on wrist, 392f
- hGH, *see* Human growth hormone (hGH)
- Hide and die syndrome, 471
- High blood pressure, *see* Hypertension
- High-profile cases, 554–555
 hate crime, 554
 mass fatality incidents and in-custody, 555
- High-tension power lines, 451f
- Hirschprung disease, 236
- Histology, 66, 90–91
 defined, 154
 routine histology, 154
 H&E, 90
 ultrastructural anatomy, 90
See also Microscopic anatomy
- Hit and run deaths, 76, 134, 329–330
- HIV, *see* Human immunodeficiency virus (HIV)
- Hodgkin disease/non-Hodgkin lymphoma, 241
- Homicidal childhood deaths
 bucket-handle fracture, 525f
 healing rib fracture (*arrow*) present in a victim of repeated child abuse, 524f
 liver laceration, 522f
 multiple skull fractures, 518f
 posterior skin incisions, 523f
 scalding injury on the hand, 525f
 small intestine demonstrating a large tear within the mesentery, 521f
 subarachnoid hemorrhage overlying posterior (back) of brain, 519f
 subdural hemorrhage on the left side, 519f
 subscalpular hemorrhage related to blunt force head trauma, 518f
- Homicidal drownings, 446

- Homicide(s), 444
 by heart attack, 555–556
 conduction system disorder, 555
 honor killing, 529
 MOD, 74–75
 by heart attack, 75
 homicidal violence, 76
 pedestrians, 330
- Hospital identification bands, 189
- Hospital (medical) autopsies, 11–12
- HSV, *see* Herpes simplex virus (HSV)
- Human body, 82, 92, 111, 130, 169, 206, 263, 287, 321, 384, 421
 cremations, 498
 defense mechanisms, 470
 gross anatomy, 82
 ionizing radiation, 578
 regions and compartments, 92
 temperature, 469
- Human body stress
 combination (multifactorial), 206
 external (environmental), 206
 internal (genetic), 206
- human growth hormone (hGH), 243
- Human immunodeficiency virus (HIV), 233, 242–243, 471, 574
- Human remains, identification of
 circumstantial identification, 198
 common/non-scientific methods, 188
 hospital identification, 188
 visual identification, 188
 other unique features, based on
 photograph of the artificial hip, 195
 policies, 188
 scientific methods, 190
 dental identification, 191
 DNA identification, 192
 fingerprint identification, 190
 radiologic identification, 192
 unidentified remains, 198
- Hydration disorders, 561–562
See also Nutrition and hydration disorders
- Hydrocarbons, volatiles inhalants, 291–293
 foam cone in man, 292f
 mixed hydrocarbons, 293f
 plastic garbage bag/helium tank were
 present in victim, 293f
- Hyoid bone, 149, 151, 415, 417, 559
- Hypercortisolism (Cushing syndrome), 244
- Hyper-IgM syndrome, 242
- Hyperplasia, 206, 233, 244–245
- Hypersensitive immune system, 73, 241
- Hypertension, 209, 211, 218, 226–227, 235, 237–238, 244–245, 247, 270, 283, 287, 563, 575–577
- Hypertensive cardiovascular disease, 218, 226–227
 cardiac hypertrophy/cardiomegaly, 218
 essential or idiopathic, 218
 hypertension or high blood pressure, 218
 hypertensive and atherosclerotic
 cardiovascular disease, 218
 spontaneous brain hemorrhages, 218
- Hypertensive disease, 226
- Hyperthermia, 437, 475–479
 autopsy findings, 477–478
 initial effects, 478
 intrathoracic petechiae, 477
 intrathoracic thymic petechiae (pinpoint
 hemorrhages), 478f
 superficial abrasions/swollen brain,
 477f
 toxicology testing, 478
 death certification, 478–479
 cause of death (hyperthermia), 478
 manner of death (accident, homicides),
 478
 possible/probable hyperthermia, 479
 general features, 475
 environmental hyperthermia, factors,
 475
 excited delirium syndrome, 475
 heat stroke, 475
 malignant hyperthermia syndromes,
 475
 physiologic effects, 475
 scene investigation, 476
 elderly individual is found dead at
 home, 476f
 first responders, 476
 potential problems, 476
 protocols, 476
 resuscitative efforts, 476
 toxic effects of cocaine and excited
 delirium, 477f
- Hypertrophic cardiomyopathy, 220
 genetic mutations, 221
 IHSS, 220
 myofiber disarray (myocytes), 220
 obstructive, 220
 septal hypertrophy or asymmetric left
 ventricular hypertrophy, 220
- Hypertrophy, 206
- Hypokalemia (low potassium), 244

- Hypothermia, 470–475
 autopsy findings, 471–474
 hemorrhagic pancreatitis, 473f
 microscopic features, 474
 paradoxical undressing, 472f
 prominent red discoloration of elbow, 473f
 prominent red discoloration of knees, 472f
 toxicology testing, 475
 vitreous chemistry testing, 475
 Wishniewski ulcers, 474f
 death certification, 475
 Alzheimer's disease, 475
 exposure, 475
 natural disease processes, 475
 diving response, 434
 general features, 470–471
 body's responses, 471
 definition of hypothermia, 470
 risk factors, 470
 scene investigation, 471
 daytime temperature, 471
 hide and die syndrome, 471
 paradoxical undressing, 471
- Hypoxia/anoxia, 207
- I**
- IAP, *see* International Academy of Pathology (IAP)
- ICP, *see* Intrahepatic cholestasis of pregnancy (ICP)
- Identification photograph, 188
- Idiopathic hypertrophic subaortic stenosis (IHSS), 220
- Idiopathic seizure disorders, 225
- IHSS, *see* Idiopathic hypertrophic subaortic stenosis (IHSS)
- IM, *see* Intramedullary (IM) lines
- Immediate COD, 68
- Immersion injuries (child abuse injuries), 486
- Immunodeficiency, 242–243
 AIDS, 242
 primary immunodeficiency syndromes, 242
 secondary immunodeficiency, 242
- Immunodeficient or immunocompromised, 242
- Immunoglobulin, 542
- Immunohistochemistry, 154, 321
- Immunostains, 208
- Impression analysis, 30
- Inborn errors of metabolism, 249–250
 galactosemia, 250
 Gaucher disease, 250
 Niemann–Pick disease, 249
 phenylketonuria or PKU, 249
 Pompe disease, 249
 Tay–Sachs disease, 249
- Incidental anomalies, 236
- Incised wounds, 385–387, 391–392, 394
 by broken bottle, 387f
 cluster across face, 386f
 of neck, 386f
- In-custody deaths, 55, 70, 277, 465, 556–560
 electronic shock device darts, 558f
 excited delirium, 559–560, 559f
 reconstruction, 559f
 restraint asphyxia, 559
 sympathomimetic poisoning syndrome, 560
 sympathomimetic toxicity syndrome, 560
 wrist/sole incision in custody death, showing absence of injury, 557f
See also High-profile cases
- Indian Ocean tsunami in December 2004, 560
- Infancy and childhood, deaths in
 accidental childhood deaths, 514
 birth-related infant deaths, 506
 childhood homicides, classification, 526–527
 acute psychosis, 530
 altruism, 530
 drug and alcohol abuse, 532
 euthanasia (true mercy killing), 531
 innocent bystander, 532
 munchausen's syndrome by proxy, 529
 negligence/neglect, 527
 postpartum mental disorder, 530
 sadistic acts of punishment, 528
 seizure disorder, 532
 sexual abuse, 531
 spouse revenge, 530
 unwanted child, 529
 unwanted pregnancy (neonaticide), 529
 violent older child, 532
 violent outburst (angry impulse), 527
See also individual
- discarded fetuses/infants and fetal/infant deaths in unattended births
 cause of death, 505
 fetus/infant live-born or stillborn, 503
 fetus/infant viable, 503
- homicidal childhood deaths, 516
- infant deaths, 508
- natural death in childhood, 513
- pediatric autopsy considerations, 533
- suicidal childhood deaths, 516

- Infant death, 501–502, 506–508
 abundant bedding, 511
 doll re-enactment, 510f
 hernia in a newborn infant died, 509f
 neonatal period, 501–502, 506–508
 pulmonary hypoplasia, 508
 thymic/thoracic petechiae, 511f
- Infarction, 207, 239
- Infectious disease, 72, 205, 207–208, 217, 514, 560, 571, 581
 commensal organisms or normal flora, 207
 infectious organisms, 207
 bacterial meningitis, 208
 mosquito bite or Rocky Mountain spotted fever, 208
 mycobacteria (tuberculosis), 208
 SIRS, 208
 tubercular and fungal, 208
 West Nile viral encephalitis, 208
- Pus or purulence or suppurative inflammation, 208
- sepsis, 208
- septicemia, 208
- systemic infection, 208
- tuberculosis, fungi, parasites, 232–233
 fungal organisms (histoplasmosis, aspergillus), 232
 parasites (pneumocystis), 232
 tuberculosis (TB, mycobacterium), 232
- white blood cell
 neutrophils/lymphocytes/macrophages, 208
- Infective endocarditis, 220
- Inferior vena cava (IVC), 92, 105, 112, 324
- Inflammation process, 89t, 114, 206
- Inflammatory joint disorders (arthritis), 245
- Initial investigation, 37, 49, 53–55, 60, 71, 185
- Injuries, postmortem, 177–178, 178f
 crime of passion, 178
 hate crime/bias crime, 178
 mechanism, 390
 overkill, 178
 perimortem, 178
 See also Postmortem changes
- Innocent bystander, childhood homicides, 532
- Insulin, prescription drugs, 288–289
 exogenous insulin, 288
- Integumentary system, 92–94
 functions, 93–94
 keratinocytes, 93
 types/parts of skin, 93
 dermis/epidermis, 93
- Intermediary
 COD, 68
 contusions, 319
- Internal airway obstruction (occlusion), *see* Choking deaths
- Internal anomalies, 236
- International Academy of Pathology (IAP), 14
- International Association for Identification, 26, 29–31
- Interstitial inflammation, 232
- Interstitial lung diseases or pneumoconiosis, 235
- Intrahepatic cholestasis of pregnancy (ICP), 576
- Intramedullary (IM) lines, 533
- Intramyocardial tunneling or bridging, 214
- Intrapartum deaths, 505
- Intrathoracic petechiae, 477
- Ionizing radiation burns, 485
- Ischemia, 206, 211, 214, 321, 583
- Ischemic axonal injury, *see* Vascular axonal injury
- Ischemic cardiomyopathy, 214
- IVC, *see* Inferior vena cava (IVC)
- J**
- Jurisdictional issues, 51
- K**
- Kacute cocaine intoxication, *see* Cocaine intoxication
- Kawasaki disease, 214
- Keyhole shaped defect, 363
- Kidney infections (pyelonephritis), 245
- Kimmelstiel–Wilson lesion, 248
- Klinefelter syndrome, 250
- Kwashiorkor, 563
- L**
- Laboratories (clinical), typical
 blood bank, 6
 chemistry laboratory, 6, 6f
 hematology laboratory, 6, 7f
 microbiology laboratory, 6
 molecular genetics laboratory
 identification of genes (DNA)/products, role, 6
- Laboratory medicine, *see* Clinical pathology (CP)
- Lacerations, 379, 393, 397, 398f
 aortic laceration sustained in deceleration/blunt impact, 307f
 of inguinal (groin) region in a pedestrian struck, 307f

- Lacerations (*cont.*)
 liver lacerations, 308f
 scalp with jagged appearance, 306f
 tissue bridging, 305
- Lands, *see* Rifled, weapons/ammunition types
- Laparotomy, surgical incision of abdomen, 397
- Laryngeal spasm, 434
- Laryngospasm, *see* Laryngeal spasm
- Lassa virus, 582
- Left-sided/right-sided heart failure, 211
- Lethal arrhythmias (irregular rhythms), heart, 143, 220
- Letulle (en masse) method, 142
See also Rokitansky method
- Leukemia/lymphoma, 241–242
 acute/chronic leukemias, 241
 fluid malignancies, 241
 Hodgkin disease/non-Hodgkin lymphoma, 241
- Leukemias, acute/chronic, 241
- Leukocytes, *see* White blood cells (wbcs)
- LH, *see* Luteinizing hormone (LH)
- Libman–Sacks endocarditis, 219
- Lifestyle factors, 205
- Ligature strangulation, 412, 415–419
 conjunctival petechiae, 417
- Lightning
 arc marks, 461
 classic arborescent (fern-like) lightning strike patterned injury, 463f
 side flash,” 461
- Liividity (livor mortis), 164f
 after several hours, 165f
- Listeria* (bacteria), 566
- Livor mortis, 56, 67, 94, 134, 163–165, 167, 179, 262, 472, 546
 “blanching,” 164
 “cardinal signs of death,” 163
 character and/or color of, 165–166
 “dependent hypostasis,” 164
 lividity (livor mortis), 164f
 after several hours, 165f
 tardieu spots (hemorrhage), 165, 165f
See also Postmortem changes
- Localized autoimmune disorders, 241
- Long bones, 94, 95f
 growth, 95f
- Long QT syndrome, 222
- Long-term insulin replacement therapy, 248, 288
- Lophophora williamsii*, 283
- “Low salt vitreous electrolyte pattern,” 247, 270, 573
- LSD, *see* Lysergic Acid Diethylamide (LSD)
- Lung or systemic disorders, 211
- Luteinizing hormone (LH), 117, 122, 243
- Lymphocyte, 7f
 types, 113
- Lysergic Acid Diethylamide (LSD), 282–284, 296, 559
- M**
- Maceration, 503–505
 fetal, characterized by skin discoloration and slippage, 504
See also Stillbirths
- Malignant cerebral edema, 320, 323
- Malignant hyperthermia syndromes, 475
- Mallory–Weiss tears, 238
- Malnutrition and starvation, 563–564
 kwashiorkor, 563
 marasmus, 563
 PEM, 563
 starved, elderly individual, 564f
- Malnutrition, primary/secondary, 562
- “Manmade” electricity, 450
 AC, 450
 DC, 450
- Manner of death (MOD), 63, 66f, 71, 226, 268, 446–447, 446f
 accidental deaths, 72
 accidental drownings, 446
 bathtub drownings, 446
 choices, 71
 “hit and run” deaths, 76
 homicidal drownings, 446
 issues, 73, 76
 ruling, 71
 autopsy/investigation, 71
 suicidal drowning deaths, 446
 swimming pools or other bodies of water, 446
 therapeutic complication, 78
 unclassified MOD, 78
- Manual strangulation, 417–418
- MAOIs, *see* Monoamine oxidase inhibitors (MAOIs)
- Marasmus, 563
- Marbling, 170, 171f
- Marburg virus, 582
- Marfan syndrome, 216, 220, 250–251
- Mass disaster drills, 561
- Mass fatality incidents, 61, 541, 555, 560–561
 decontamination, concept of, 561
 D-MORT, 540, 561
 Indian Ocean tsunami, 560
 mass disaster drills, 561

- Material Safety Data Sheet (MSDS), 487
- Maturity-onset diabetes of the young (MODY), 247
- MCA, *see* Middle cerebral artery (MCA)
- MCF, *see* Middle cranial fossa (MCF)
- MDA, *see* Methylenedioxyamphetamine (MDA)
- MDMA, *see* 3,4-methylenedioxyamphetamine (MDMA)
- Mechanical asphyxia, 408–410, 409f
traumatic compression, 409f
- Mechanism of death, 63, 68
“but for” rule, 69
examples, 69
fire deaths, 496–497
cyanide gases, 497
“flash fires,” 497
“simple asphyxia,” 497
thermal injuries, 497
true antemortem (occurring before death) head injury, 497f
- Mechanism of injury, 366–367
“stopping power,” 366
“temporary cavity,” 367
- Meckel’s diverticulum (Congenital Anomalies), 236, 238
- Meconium aspiration, 230
- Mediastinum, thymus, 92, 115
See also Reticuloendothelial system
- Medical examiner investigator, 52, 133
See also Death investigator
- Medical examiner (ME) systems, 4, 12, 36, 40, 46, 50–54, 52–53, 65–66, 130, 133, 157, 158, 200, 508, 540, 568, 570, 576, 581
lay death investigators, 36, 52
- Medical specialties, 3, 5t
types/categories
medical (non-surgical), 3
miscellaneous, 4
surgical, 4
- Medicolegal death investigation system, 186, 235
- Medullary bone, 94, 95
- “Mee’s lines,” chronic toxicity, 294
- Meninges membrane, *see* Arachnoid membrane
- Meningitis, 72, 208, 224, 514, 574
- “Metabolism,” toxicology issues, 263
- Metabolite, 264, 276–277, 282–283
- “Metallization,” 459
black/purple discoloration, 459
“nuclear streaming,” 459
- Metaphysis, 94
- Metaplasia, 206
- Methathinone, 273
- Methyl-2,5-dimethoxyamphetamine (DOM), 273
- Methylenedioxyamphetamine (MDA), 273
3,4-methylenedioxyamphetamine (MDMA), 273–274, 284
- Methylphenidate (Ritalin), 273
- Microlacerations, *see* Microtears
- Microscopic anatomy, 3, 4t, 7, 81, 90, 154
See also Histology
- Microscopic brain injuries, 320–321
- Microtears, 346
- Microtome, 9f
- Middle cerebral artery (MCA), 103
- Middle cranial fossa (MCF), 102, 314, 439
- Miliaria, 177
- Miscellaneous issues
death issues, manner of, 369–370
accidents, 370
homicides, 369
suicides, 369
internal examination, 362–366
internal beveling, 363
penetrating wound, 362
perforating wound, 362
periorbital ecchymosis, 363
- Missile/projectile injuries, 379
- Mitochondria, 27, 247
- Mitral valve prolapse (MVP), 219–220, 251
See also Valve disorders
- Mixed drug intoxication, 267
- Mixed systems, 50–53
types, 53
- MOD issues
NAME, 76
guidelines, 76
- MODY, *see* Maturity-onset diabetes of the young (MODY)
- Monoamine oxidase inhibitors (MAOIs), 286
- Morbid obesity, 563f
- Mosquito bite/Rocky Mountain spotted fever, 208
- Motor vehicle collisions, death cases, 327, 516
- MSDS, *see* Material Safety Data Sheet (MSDS)
- Multi-organ failure, MOD, 69
- Multiple brain abscesses, 224

- Multiple causes of death
 multiple injury types, 562
- Multiple “granulomas,” 251
- Multiple injuries, 544, 545f
- Multiple myeloma, 246
- Multiple sclerosis, 241
- Multiple sharp force injuries
 by barbed wire, 387f
 victim with decomposition, 395f
- Multisystem and other disorders, 245–251
 amyloidosis, 246
 Alzheimerdisease, 246
 “amyloid,” 246
 “apple green birefringence,” 246
 “Congo Red” stain, 246
 “multiple myeloma,” 246
 “polarized light,” 246
 chronic alcoholism, 246–247
 CPM, 246f
 DM, 247–248
 inborn errors of metabolism, 249–250
 other genetic disorders, 250–251
 psychiatric disease, 251
 sarcoidosis, 251
 sickle cell disease, 248–249
- Mummification (warm, dry environments),
 170, 175, 177f, 191
- Munchausen’s syndrome by proxy, 529
- Musculoskeletal system, 94–97
 cartilage, 95
 cortical bone, 95
 diaphysis, 94
 epiphysis, 94
 ligaments, 95
 medullary bone, 94
 metaphysis, 94
 muscular/skeletal system, 94, 96f, 97f
 myofilaments
 actin, 97
 myosin, 97
 osteoblasts, 95
 osteocytes, 95
 skeletal muscles, 96
 extensors, 96
 flexors, 96
 spongy bone, 94
 trabeculae, 94
- Mushrooms (Psilocybin), 282–284
 hallucinogens, 284
Salvia divinorum, 284
- MVP, *see* Mitral valve prolapse (MVP)
- Myasthenia gravis, 241
- Mycobacteria (tuberculosis), 207–208
- Myocardial hypoxia, 210
- Myocardial infarcts (heart attacks), 212
 “Myocarditis,” 217, 220, 440, 444, 446, 576
- Myxoid (mucinous) degeneration, *see* Cystic
 medial necrosis
- Myxomatous degeneration of the mitral valve,
 219
See also Mitral valve prolapse (MVP)
- N**
- NAME, *see* National Association of Medical
 Examiners (NAME)
- NAME guidelines, 76
- NamUS program, 200
- National Association of Medical Examiners
 (NAME), 14, 46, 76, 159
- National Board of Medical Examiners
 (NBME), 4, 14, 46
- National Board of Osteopathic Medical
 Examiners (NBOME), 4
- National Center for Health Statistics (NCHS),
 63
- National Disaster Medical System (NDMS),
 540
- National Institute for Justice, 200
- National Transportation Safety Board (NTSB),
 540
- Natural deaths, 63, 71, 205–251
 bones, joints, and soft tissues, 245
 cardiovascular system
 aortic aneurysms (abdominal and
 thoracic), 215–216
 cerebrovascular disease, 216
 conduction system abnormalities, 222
 congenital heart disease, 209–210
 congestive heart failure (heart failure),
 210–211
 coronary artery atherosclerosis,
 211–214
 coronary artery dissection, 214
 deep venous thrombosis, 222
 dilated cardiomyopathy, 220–222
 fibromuscular dysplasia, 217
 hypertensive cardiovascular disease,
 218
 hypertrophic cardiomyopathy, 220
 myocarditis, 217
 neoplastic heart disease, 223
 other coronary artery disorders, 214
 restrictive cardiomyopathy, 222
 valve disorders, 219–220
 vasculitis, 217
 in childhood, 513–514

- CNS, 223–229
 - cerebral palsy, 223–224
 - cerebrovascular disease, 226
 - congenital anomalies, 223
 - dural sinus thrombosis, 228–229
 - hypertensive disease, 226
 - infection, 224–225
 - neoplasia, 229
 - ruptured AVM, 227–228
 - ruptured berry aneurysm, 227
 - seizure disorders, 225–226
 - spontaneous intraparenchymal hemorrhage, 226–227
- endocrine system, 243–244
- gastrointestinal and hepatobiliary system, 236–240
 - cirrhosis/esophageal varices, 237–238
 - congenital anomalies, 236
 - gastritis, 236
 - hemochromatosis, 239–240
 - Mallory–Weiss tears, 238
 - other GI abnormalities, 238–239
 - pancreatitis, 240
 - PUD, 236–237
- genitourinary system, 244–245
- infectious disease, 207–208
- multisystem and other disorders, 245–251
 - amyloidosis, 246
 - chronic alcoholism, 246–247
 - DM, 247–248
 - inborn errors of metabolism, 249–250
 - other genetic disorders, 250–251
 - psychiatric disease, 251
 - sarcoidosis, 251
 - sickle cell disease, 248–249
- respiratory system, 229–235
 - aspiration pneumonia, 232
 - asthma, 233
 - bacterial pneumonia, 231
 - chronic lung disease, 235
 - congenital anomalies, 229–230
 - COPD, 234
 - neonatal conditions, 230
 - neoplasia, 235
 - other infections (tuberculosis, fungi, parasites), 232–233
 - pulmonary hypertension, 235
 - pulmonary thromboembolism, 231
 - sarcoidosis, 235
 - upper airway conditions, 230
 - viral pneumonia, 232
- reticuloendothelial and immune systems, 240–243
 - autoimmune disorders, 241
 - immunodeficiency, 242–243
 - leukemia/lymphoma, 241–242
 - “Natural disease,” 12–13, 41, 50, 54, 63, 65, 69, 71, 75, 78, 106, 157, 205–207, 232, 267, 304, 317, 324, 403, 422, 437, 444, 446, 475, 477, 490, 497–498, 505–510, 540, 544, 556
- Natural MOD, 71–72
- “Natural” or “normal” route of transmission, 205
- NBME, *see* National Board of Medical Examiners (NBME)
- NBOME, *see* National Board of Osteopathic Medical Examiners (NBOME)
- NCHS, *see* National Center for Health Statistics (NCHS)
- NDMS, *see* National Disaster Medical System (NDMS)
- “Near drowning,” 434
- Neck compression, 402, 411–412, 423–427, 515–516, 544
 - hanging, 412–415
 - ligature strangulation, 416–417
 - manual strangulation, 417–418
 - non-ligature, non-manual, 418–419
 - strangulation, 415–416
- Neck restraint maneuvers, 425
 - carotid sleeper hold, 419, 425
 - choke-hold, 419, 425
- Negligence/neglect, childhood homicides, 527
- Neisseria meningitidis* (meningococcus), 224, 574
- Neonatal conditions, 230
 - hyaline membrane disease, 230
 - “pulmonary surfactant,” 230
 - meconium aspiration, 230
- Neonaticide (unwanted pregnancy), 517, 529
- Neoplasia, 206, 229, 235
 - cerebral spinal fluid obstruction, 229
 - GBM, 229
 - neoplasms or tumors, 206
 - new growth, 206
- Neoplasms or tumors, 206
 - benign neoplasm
 - adjacent tissues or “metastasize,” 206
 - “malignant” neoplasm
 - invade and metastasize, 206
- Neoplastic heart disease, 223
 - atrial myxomas/papillary fibroelastomas of valves, 223
 - “cystic tumor of AV node” or “mesothelioma,” 223

- Nerve agents, 582–583
- Nervous system, 97–104
 - autonomic nervous system, 98
 - sympathetic/parasympathetic systems, 98
 - CNS/PNS, 97
 - nerve signals, 97
 - motor, 98
 - sensory, 97
 - parts of brain, 97
 - arachnoid membrane, 98, 99f–100f
 - basilar skull, 102, 103f
 - bridging veins, 98, 99f
 - CSF, 101
 - functions, 102
 - voluntary/involuntary pathways, 98
- Neuroblastomas in children, 244
- Neuroleptic malignant syndrome, antipsychotic, 288
- Neuromuscular disorders, 232
- “Neurotoxic shellfish poisoning”, 567
- Neutrophil, 7f, 86t, 113–114, 208, 213, 240
- Neutrophilic inflammation, 114
 - abscess, 114
- Niemann–Pick disease, 249
- Nitrous oxide (laughing gas), 291f, 293
- Nonbacterial thrombotic endocarditis, 219
- Non-lethal electronic shock devices, 464
 - See also* In-Custody Deaths
- Non-ligature, non-manual, 418–419
- Non-natural deaths, 65
- Non-natural, traumatic processes, 444
- Non-neoplastic hyperplasia (goiter), 244
- Non-scientific identification methods, 188
 - hospital identification, 188
 - visual identification, 188
- Non-seafood-related poisoning, 566
- Noxious stimulus, 67
- NTSB, *see* National Transportation Safety Board (NTSB)
- Nuclear streaming, 459
- Nucleotide bases
 - adenine (A), 28
 - cytosine (C), 28
 - guanine (G), 28
 - thymine (T), 28
- Nutrition and hydration disorders, 561–562
 - anorexia, 564–565
 - dehydration, 565–566
 - severe dehydration, with “sunken eyes,” 566
 - malnutrition and starvation, 563–564
 - kwashiorkor, 563
 - marasmus, 563
 - PEM, 563
 - starved, elderly individual, 564f
- obesity, 563
 - BMI, 563
 - morbid obesity, 563f
- overhydration, 566
- primary/secondary malnutrition, 562
- vitamin deficiencies, 564
 - thiamine (vitamin B1) deficiency, 564
 - Wernicke–Korsakoff syndrome, 564
- O**
- Obesity, 209, 220, 244, 248, 475, 547, 562–563
 - BMI, 563
 - morbid obesity, 563f
- Obliterative endarteritis, 216
- Occasional drowning deaths, 444
- “Occipital osteodiaschisis,” fetal death, 507
- Occupational deaths
 - categories, 567
 - “occupational lung diseases,” 567
 - OSHA, 567
 - “Occupational lung diseases,” 235, 567
- Occupational Safety and Health Administration (OSHA), 567
- Opiates/related substances, 279f
 - fentanyl, 281
 - patch found within gastrointestinal system, 281f
- Organ and tissue procurement issues, 568–572
 - cornea removal for transplantation purposes, 570
 - process of, 571
 - “rapid organ recovery,” 569f
 - Revised Uniform Anatomical jpegt Act, 571
 - skin and bone removal for transplantation purposes, 569f
- Organ donation, 568–570
 - See also* Organ and tissue procurement issues
- Organ transplantation, 67–68, 568, 570
- OSHA, *see* Occupational Safety and Health Administration (OSHA)
- Osteoarthritis (wear and tear disorder), 245
- Osteoblasts, 95
- Osteocytes, 95
- Osteomyelitis, 245
- OTC, *see* Over-the-counter (OTC)
- Overhydration, 562, 566
- Overkill, 178, 396, 396f

- Over-the-counter (OTC), 257–258, 284–286
 Oxidative phosphorylation process, 422–423
- P**
- Pancreatitis, 240, 270, 473–474
 acute, 240, 474
 chronic, 240
 fat necrosis, 240
 hemorrhagic pancreatitis at autopsy, 240f
 postmortem blood levels, 240
- Paper bags, advantage, 56, 135, 390
 use, death scene, 135, 135f
- Paradoxical undressing, 471, 472f
- Paraffin-embedded tissue, 194
- “Paralytic shellfish poisoning,” 567
- Paramethoxyamphetamine (PMA), 273, 284
- Parasites (pneumocystis), 232
- Parathyroid disorders, 244
- Parenchyma cells, *see* Histology
- Patau syndrome, 250
- Patent ductus arteriosus (PDA), 209
- Pathologists, forensic, 4, 11–14, 17, 19, 21–26, 28, 30–31
 qualities to be a, 43–46
 training/qualifications of, 43–46
 education/training/examination requirements for Board-certification, 43t
 guidelines for AP residency training, 44t
- Pathology, 3–14, 17, 19, 35–46, 49, 79, 81–82, 84, 96, 127, 129, 133, 140, 142, 154, 158, 169, 180, 194, 200, 220, 235, 501, 503, 506, 511, 534, 540, 542, 581
 Board-certified subspecialties, 14t
 branches of
 anatomic pathology, 6
 clinical pathology, 6
 clinical pathology laboratories
 automated chemistry machine in, 6f
 blood bank, 6
 chemistry laboratory, 6, 6f
 hematology laboratory, 6, 7f
 microbiology laboratory, 6
 molecular genetics laboratory, 6
 definition, 3
 histologic examination, stains used
 hematoxylin and eosin (H&E), 7
 medical degrees, types
 DO (doctor of osteopathy), 4
 MD (doctor of medicine), 4
 medical specialties, 5t
 medicine’s “basic sciences,” 3
 pathology disciplines, 14t
 physician, “Board-certification” criteria, 13
 physicians’ physician, 13
 traditional medical school curriculum, example, 4t
- Pathology assistants (PAs), *see* Autopsy assistants
- Pathology residency training programs, 5–6, 43
- PCA, *see* Posterior cerebral arteries (PCA)
- PCF, *see* Posterior cranial fossa (PCF)
- PCR, *see* Polymerase chain reaction (PCR)
- PDA, *see* Patent ductus arteriosus (PDA)
- Pediatric autopsy considerations, 501, 533, 534
 CSF collection from an infant, 534f
 Mongolian spot, 533, 533f
 “rose petal” technique, 534
 sterile spinal tap, 534
- Pediatric (childhood) forensic pathology, 501
- PEM, *see* Protein-energy malnutrition (PEM)
- Pending certificate, 66
- Peptic Ulcer Disease (PUD), 236–237
 chronic blood loss (anemia), 236
 destructive forces, 236
 “peritonitis,” 236
 protective factors, 236
 sepsis (systemic infection), 236
- Pericardial cavity (“heart sac”), 213
- Pericardial sac, 92, 139
- “Perimortem,” 178
- Peripartum cardiomyopathy, 221, 576
- “Peripartum myocarditis,” 576
- Peripheral nervous system (PNS), 97
- Peripheral rim of red skin, 458
- Peritoneal cavity, 90, 92, 111, 138–139, 177, 237–238
 “Peritonitis,” 236–237
- Perivascular plasma cell inflammation, 216
- Permanent record, dead body, 188
- Pernicious anemia, 241
- Petechiae/petechial hemorrhages, 136, 402–403, 403f, 408, 415, 510, 548
- “Pharmacogenomics,” toxicology issues, 267
- Phencyclidine (PCP), 283
- Phenylketonuria (PKU), 249
- Pheochromocytomas in adults, 244
- Phlebothrombosis, *see* Deep venous thrombosis
- Phosgene oxime (CX), 487–488, 582
 See also “Vesicants” (blistering agents)
- Physicians’ physician, 13

- Physiology, 3, 81–124, 91–92, 433–434
 ECG, 67, 91, 222, 276
- Pia mater, 98, 102
- Pituitary disorders, 243
- Pituitary gland, 103, 117, 122, 148, 243–244
 anterior cells type
 ACTH, 117
 FSH, 117
 LH, 117
 TSH, 117
 neurohypophysis
 antidiuretic hormone, 117
 vasopressin hormone, 117
- PKU, *see* Phenylketonuria (PKU)
- Platelets, 113–115, 241
- “Plumbism,” heavy metals, 294
- PMA, *see* Paramethoxyamphetamine (PMA)
- PMI, *see* Postmortem interval (PMI)
- Pneumonia, defined, 144, 205, 208, 231–232, 242, 247, 292, 295, 473–474, 478, 584–585
- “Pneumothorax,” 234, 323, 394–395, 445, 583
- PNS, *see* Peripheral nervous system (PNS)
- Pointed-tip “skin tags,” 351
- Polarized light, 246, 279
- Policies, 43, 52, 131, 185, 188
 blood sample, 188
 fingerprint cards, 188
 “identification photograph,” 188
 permanent record of particular body, 188
- Polyarteritis nodosa, 217, 241
- Polymerase chain reaction (PCR), 28
- Polypharmacy, death certification, 268
- Pompe disease, 249
- Portal hypertension, 237–238, 247, 270
- Positional asphyxia, 402, 410–411, 411f, 512
 wedging/burking, 410–411
- Positive based on circumstance, 198
- Positive identification, 40, 185–187, 197f
- Possible/probable hyperthermia, 479
- Posterior cerebral arteries (PCA), 103
- Posterior cranial fossa (PCF), 102
- Post-immersion syndrome, 434
- Postmortem (after death), 264, 489
- Postmortem blood levels, 240
- Postmortem changes, 37, 55, 94, 134, 163–182, 191, 441
 decomposition, 169–177
 definition, 163
 early postmortem changes, 163–169
 algor mortis, 168
 corneas, clouded, 168
 livor mortis, 163–166
 “postmortem suntan,” 169
 rigor mortis, 166–167
 tache noire (black line), 168
See also individual
- postmortem injuries, 177–178, 178f
 “crime of passion,” 178
 hate crime/bias crime, 178
 “overkill,” 178
 “perimortem,” 178
 time of death, 179–182
See also Time of death estimation
- Postmortem, detection
 dental X-ray, 191f
 hip X-ray in badly decomposed woman, 196f
- Postmortem electrical injuries, 460
- Postmortem forensic examination/autopsy, 129–159
 after-the-fact and in-absentia cases, 157–158
 autopsy assistants, 158
 external examination (without autopsy), 156–157
 cases that require an autopsy, 156f
 forensic autopsy, components of, 130f
 ancillary procedures, 153–154
 autopsy report, 154–156
 external examination, 134–137
 internal examination, 137–153
 investigation, 133
 forensic vs. hospital autopsies, 130–133
 major injuries, 135–136
 minor injuries, 136
 office accreditation/forensic autopsy standards, 159
 accreditation process, 159
 autopsy performance standards, 159
 inspection checklist, significance, 159
- Postmortem interval (PMI), 163, 179
- Postmortem/perimortem wounds, 395–396
- “Postmortem suntan,” 169
- Postmortem toxicology tests, 69, 137f, 266, 278, 421, 559
- Postpartum mental disorder
 command hallucinations, 530
- “Postpartum” (peripartum/cardiomyopathy), 576
- “Potato nodes,” 251
- Pre-existing dental record, 191
- Pregnancy-induced diabetes mellitus, 577
- Pregnancy-induced hypertension (PIH), 576
- Pregnancy-related maternal deaths, 575–578
 AFLP, 576

- Pregnancy-related maternal deaths (*cont.*)
 AFE, 577
 DIC, 577
 ectopic pregnancy, 575
 EDS, 575
 “gestational trophoblastic disease,” 577
 HELLP syndrome, 576
 “hemolytic uremic syndrome,” 577
 ICP, 576
 “peripartum myocarditis,” 576
 “postpartum”/“peripartum” cardiomyopathy, 576
 pregnancy-induced diabetes mellitus, 577
 PIH, 576
 “primary pulmonary hypertension,” 577
 PE, 577
 “thrombotic microangiopathies of pregnancy,” 577
 “thrombotic thrombocytopenic purpura,” 577
 “tubal pregnancy,” 575
See also Therapy-related deaths
- Preliminary/tentative identification, 185–186, 190
- Premature rupture of the membranes (PROM), 507
- Primary adrenal disorders, 244
- Primary atelectasis, stillbirths, 503–504
- Primary bone cancers, 245
- “Primary immunodeficiency” syndromes, 242
- Primary malnutrition, 562
- “Primary pulmonary hypertension,” 235, 577
- Prion diseases, 225
- Product-related deaths, 539, 578, 591
 CPSC/FDA, 578
- PROM, *see* Premature rupture of the membranes (PROM)
- Protein-energy malnutrition (PEM), 563
- Proximate (underlying) COD, 68
- Psychiatric disease, 251
 “acute exhaustive mania,” 251
 schizophrenia or manic-depression, 251
- Psychosis, acute, 283, 530
- Ptychodiscus brevis*, 567
- PUD, *see* Peptic Ulcer Disease (PUD)
- “Pugilistic” posture, 492
See also “Boxer’s stance”
- Pulmonary edema fluid, 262, 278, 434, 437–438
- “Pulmonary embolism,” 324
- Pulmonary hypertension, 235, 577
- Pulmonary thromboemboli, 152, 222, 231, 245, 366, 546–548, 546–549, 563, 575, 577
 blood thrombi (clots) blocking pulmonary artery, 547f
 DVT, 547
 “saddle embolus,” 548
- Pulmonary thromboembolism (PE), 222, 231, 577
- Purge fluid, 170, 172f, 179
- Purulent inflammation, *see* Neutrophilic inflammation
- Pus (purulence/suppurative inflammation), 114, 139, 208, 224, 238, 574, 582
- Putrefaction, 169, 174, 503–504
- R**
- “Raccoon’s eyes” (two black eyes), 304
 ecchymoses (raccoon’s eyes), 305f
- Radiant burns, 470, 481, 484–485, 489
 electromagnetic energy burns, 484
 ionizing radiation burns, 485
 postmortem dry burn caused by decedent’s leg, 485f
 radiant heat burns, 484–485
 sunburn, 485, 580
- Radiation
 definition, 578
 gray (Gy), 579
 ionizing radiation, 484–485, 578–580
 non-ionizing radiation, 580
 rad, 579
 rem, 579
 roentgen (unit), 579
 sievert (Sv), 579
 UV radiation, 579–580
- Radiologic identification
 skull X-ray, frontal sinuses, 193f
 X ray on a badly burned body, 192f
- Radiolucency, 395
- Rage killings, 396
- Rapid organ recovery, 569f
- Ratshot, handgun shot cartridges, *see* Snakeshot, handgun shot cartridges
- rbcs, *see* Red blood cells (rbcs)
- Red blood cells (rbcs), 7, 27, 113–115, 241, 249, 284, 316, 419, 434, 440, 472, 496, 512, 579
- Red-brown skin discoloration, 472
- Remote (old) strokes, 226
- Renal cell carcinoma in adults, 245
- Resistance, 248, 319, 449, 450–453
- Respiratory syncytial virus (RSV), 232

- Respiratory system, 92, 106–108, 207, 229–235
- asphyxial injuries, 44, 107, 135, 331
 - aspiration pneumonia, 232, 247, 292
 - asthma, 233–234, 514, 577
 - acute asthmatic bronchitis, 233f
 - bacterial pneumonia, 205, 231–232
 - alveolar spaces filled with polymorphonuclear wbc, 231f
 - chronic lung disease, 235
 - congenital anomalies, 143, 207, 223, 229–230, 236, 238, 508
 - colloid cyst of third ventricle of brain, 230f
 - COPD, 234–235
 - severe emphysema, with dilated air spaces (blebs or bullae), 234f
 - lungs/breathing passages, 106, 106f–107f
 - neonatal conditions, 230
 - neoplasia, 206, 229, 235, 242, 244
 - other infections (tuberculosis, fungi, parasites), 232–233
 - pulmonary hypertension, 235, 577
 - pulmonary thromboembolism, 231, 577
 - respiratory epithelium, 107–108
 - sarcoidosis, 235
 - upper airway conditions, 230
 - viral pneumonia, 232
- Restraint asphyxia, 425–427, 426f
- carotid sleeper/choke-hold, 425
 - excited delirium, 70, 78, 251, 276–277, 283, 427, 475, 477, 558–560, 589
 - police restraint, 426–427, 559
- Restriction fragment length polymorphism (RFLP), 28
- Restrictive cardiomyopathy, 222
- Resuscitation, 55, 67, 397, 403, 434, 505, 512, 545–546
- CPR, 403, 512, 546
- Reticuloendothelial and immune systems, 240–243
- “anaphylaxis” or “allergic reaction,” 241
 - autoimmune disorders, 241
 - hypersensitive immune system, 241
 - immunodeficiency, 242–243
 - leukemia/lymphoma, 241–242
- Reticuloendothelial system, 105, 113–116, 240–242
- bone marrow and blood, 113–115, 114f
 - hemoglobin electrophoresis, 113
 - neutrophilic inflammation, 114
 - immune system, 73, 84, 113, 116, 225, 240–242, 269, 514, 542
 - lymphatics and lymph nodes, 117f
 - functions, 116
 - spleen, 115, 115f
 - thymus, 115–116, 116f
- Retroperitoneum, 92, 142
- Revised Uniform Anatomical JpegT Act, 571
- RFLP, *see* Restriction fragment length polymorphism (RFLP)
- Rheumatic heart disease, 219
- Rheumatoid arthritis, 241, 245
- Ricin, 297, 583
- Rifled, weapons/ammunition types, 24, 338–342, 372
- Rigor mortis, 56, 67, 134, 163, 166–167, 170, 179, 437–438
- cadaveric spasm, 166, 437–438
 - goosebumps (cutis anserinus), 166
 - inappropriate, 167, 167f
 - See also* Postmortem changes
- Ring fracture, 314, 331
- Rokitansky method, 142–143
- Rolled-up skin, 386, 393
- Routine toxicology testing, 153, 157, 290
- RSV, *see* Respiratory syncytial virus (RSV)
- Rubbing alcohol (isopropanol), 273
- Rule of nines, 484
- Ruptured aneurysm, 69, 227
- Ruptured berry aneurysm, 226–227, 228f, 317
- cerebral artery berry aneurysm, 218, 227, 324
 - congenital disorder, 210, 227, 514
 - SAH, 227, 519
- Ruptured myocardial infarct, 213f, 437
- S**
- Sadistic acts of punishment, 528
- Safety investigation, 540
- SAH, *see* Subarachnoid hemorrhage (SAH)
- St. Louis encephalitis, 224
- Salmonella* (bacteria), 566
- Salt water drownings, 434
- SA node, *see* Sinoatrial (SA) node
- Saponification of fat body, 174–175
- Sarcoidosis, 222, 235, 251
- multiple “granulomas,” 251
 - “potato nodes,” 251
- Sarcomas, 245
- Saurine (toxin), 567
- Scald burns
- alleged child abuse, 485
 - “drip” or “splash” pattern, 486
 - “Fluid level lines,” 485
 - immersion injuries (child abuse injuries), 486

- industrial settings, 486
 - joints (points of flexion), 485
 - naturally-occurring heat sources, 486
 - popliteal fossa/inguinal
 - region/antecubital fossa, 486
 - “sparing” (lack of burns), 485
 - “stocking pattern”/“glove pattern,” 485
 - Yellowstone National Park, 486
- examples, 485–486
 - child abuse death related to a bathtub
 - scalding, 486f
 - first/second/third degree burns, 485
 - hot molten metals, 485
- Scalding, 470, 481, 484–486, 525, 528
- Scene investigation, 55–60, 55f, 56f
 - aspects, 58
 - blackening or charring (“braising”), 454
 - braising on metal contacts of plug of light fixture, 454
 - conditions, 56, 57f
 - electrical devices, evaluation of, 454
 - EMS, 58
 - man struck by lightning, 463f
 - medication/drug/weapon, 59f–60f
 - rules, 56
 - trace evidence, 56, 57f–58f
- Schizophrenia or manic-depression, 251
- Scientific identification, 186
 - antemortem (prior to death)
 - record/standard, 186
 - dental comparison, 186
 - DNA typing, 186
 - fingerprint comparison, 186
 - “match,” 186
 - X-ray comparison, 186
- Scientific methods, 190
 - dental identification, 191
 - DNA identification, 192
 - fingerprint identification, 190
 - radiologic identification, 192
- Sclera, decomposed (white part of eyes), 547f
- Scleroderma, 241
- Scombroid, 567
- Scrapes, *see* Abrasions, Blunt force injury
 - deaths
- Scratches, *see* Abrasions, Blunt force injury
 - deaths
- SCUBA, *see* Self-contained underwater
 - breathing apparatus (SCUBA)
- SCUBA deaths, 445
 - “decompression sickness,” 445
 - lack of oxygen, 445
 - pneumothorax, 445
 - pressure related processes (barotrauma), 445
 - SCUBA tank, 445
- SDH, *see* Subdural hemorrhage (SDH)
- “Seat swapping,” 541
- Secondary drowning or dry drowning, *see*
 - Post-immersion syndrome
- Second degree burns (blisters/skin slippage), 482, 482f, 497
 - succumbed to smoke and soot inhalation, 488
- Seizure disorders, 70, 91, 225–226, 257, 325, 439, 444, 514
 - childhood homicides, 532
 - chronic seizure disorder, 225
 - COD/MOD, 226
 - “diagnosis of exclusion,” 226
 - grand mal seizure, 225
 - idiopathic seizure disorders, 225
 - seizure-induced cardiac dysrhythmia, 225
- Seizure-related deaths, 437
- Selective serotonin reuptake inhibitors (SSRIs), 286
- Self-contained underwater breathing apparatus (SCUBA), 445
 - deaths, 445
 - decompression sickness, 445
 - lack of oxygen, 445
 - pneumothorax, 445
 - pressure related processes (barotrauma), 445
 - tank, 445
- Self-harm or self-injury (without suicidal intent), 392
- Self-immolation, 498, 516
- Self-inflicted wounds/suicide, 392
- Self-injurious behavior (cutting), 392–393
- Sella turcica, 98
 - See also* Nervous system
- Senile calcific aortic stenosis, 219
- Sensory structure system, 122–123
- Sepsis, 69
- Sequelae of chronic drug use, *see* Chronic
 - alcohol abuse
- Sequelae of near drowning, 434
- Serology
 - DNA
 - “blood spot” card from an autopsy, 29f
 - suspicious fluids, identified at crime scenes, 27
 - See also* DNA
 - tests, 208
- Sex chromosome abnormalities, 250

- Sexual abuse, childhood homicides, 531
- Sexual activity kit, 35, 135, 555, 580
- Sexual assault, 471, 580–581
 “sexual activity kit,” 580
- Shaken baby syndrome, 520–521
- Sharp force injury deaths, 379–398
 accidental, 393
 chop wounds, 388
 of scalp, 388f
 gaping stab wound, 380f
 incised wounds, 385–387
 by broken bottle, 387f
 cluster across face, 386f
 of neck, 386f
 reapproximation margins, 380f
 special issues, 388–398
 accidental sharp force injuries, 393
 artifacts, 397–398
 attacker, handedness, 393–394
 chest tube incision site, 397f
 clothing examination, 390
 decomposition, 395
 defensive wounds, 391
 hesitation marks (tentative injuries),
 391–392
 incised wound, direction, 393
 injury, mechanism, 390
 internal examination, 388–390
 knife blade embedded in body, 394f
 postmortem/perimortem wounds,
 395–396
 self-inflicted wounds/suicide, 392
 “self-injurious behavior” (“cutting”),
 392–393
 trace evidence, 390
 X-rays, 394–395
- stab wounds, 381–385
 of aorta, 389f
 chest/rib cage compression, 384
 cluster, neck, 383f
 double-edged, 382f
 flexibility of human body, 384
 of heart, 389f
 multiple superficial by fork, 385f
 of neck, 381f
 penetrating, 381
 perforating (through-and-through), 381,
 383
 puncture, 381, 385f
 by serrated knife, 383f
 single-edged, 382f
 by standard screwdriver, 384f
 of sternum (breast-bone), 396f
- Short tandem repeats (STR), 28
- Shotgun wounds, 356–362, 549
 absence of central defect, 360
 birdshot shotgun wound X-ray, 361f
 buckshot entrance wounds, 361f
 of chest, 358f
 of forehead, 357f
 pellet entrances, 360f
 petal strike marks, 359
 satellite pellet wounds, 359
 “scalloped,” 357–358
 “wad strike marks,” 358
- Sickle cell disease, 248–249, 514, 573t
 full-blown sickle cell disease, 249
 HbA molecule, 248
 HbS, 249
 “hemoglobinopathy,” 248
- Sickle cell trait, 249
- “Side flash,” lightning, 461
- SIDS, *see* Sudden infant death syndrome
 (SIDS)
- Signs of death
 body cooling, 67
 decomposition, 67
 livor mortis, 67
 rigor mortis, 67
- Simple asphyxia, 403–405, 404f, 497
- Sinoatrial (SA) node, 104
- SIRS, *see* Systemic inflammatory response
 syndrome (SIRS)
- Sjögren syndrome, 241
- Skeletal blunt force injuries, 524
- Skeletal muscles, 96, 166, 245, 470
- Skeletonization, 174, 174f, 179
- Skin
 adnexa, *see* Integumentary system
 blistering, 171f
 slippage, 170, 172f
 splitting, 495
- Skull, nervous system
 facial bone injuries, 313–314
 “hinge fracture,” 314f
 “ring fracture,” 314
 nervous system, 102
 ACA, 103
 ACF, 102
 circle of Willis, 103
 ganglion cells or ganglia, 104
 MCA, 103
 MCF, 102
 PCA, 103
 PCF, 102
 sella turcica, 103

- Smoke and soot inhalation, 420, 488–490, 498
- Smothering deaths, 405–406, 405f
accidental/suicidal/homicidal, 405–406
- Snakeshot, handgun shot cartridges, 371
- Solar radiation, 580
- Southern blot analysis, 28
- “Sparing” (lack of burns), 485
- Special dissections, approaches
body, storage in ‘coolers,’ 152f
complete autopsy of body, 152f
layer-by-layer anterior neck dissection, 151f
spinal cord removal, anterior (front) approach, 151f
- Special weapons, ammunition/circumstances, 370–373
dense soot with a black powder weapon, 371
“embolized,” 372
firing mechanics (black powder), 370
“frangible” bullets, 372
gilded/glazer bullet, 370
handgun shot cartridge, 371
“homemade,” 372
sabot ammunition, 372
Tandem bullets, 372
washed bullet, 370
zip guns, 370
- Spongy bone, *see* Medullary bone
- SponJPEGorm encephalopathies, 225
- Spontaneous bacterial peritonitis, 238, 247
- Spontaneous brain hemorrhages, 218
- Spontaneous intraparenchymal hemorrhage, 226–227
COD: part I/part II, MOD: natural, 227
occurrence, 226
- Spouse revenge, childhood homicides, 530
- Squamous epithelium, 87t, 91, 93, 111
- Squared-off (blunt) appearance, 382
- SSRIs, *see* Selective serotonin reuptake inhibitors (SSRIs)
- Staining techniques, highly-specialized, 154
- Standard Certificate of Death, 63, 64f
circumstances, 67f
COD, 66f
date and time of death, 65f
demographic information, 65f
MOD, 66f
- Standards, autopsy performance, 159
- Stat gram stain, 574
- Stellate exit wound, gunshot wounds, 351
- Sternal rub, 67
- Stillbirths, 503
- Stocking pattern/glove pattern, 485
- STR, *see* Short tandem repeats (STR)
- Strangulated hernia, 238–239, 239f
- Strangulation, 415–416
deaths, 415–416, 416f, 418f
ligature marks, 416f–417f
manual strangulation, 418f
types, 415
See also Neck compression
- Stroke or CVA, 216, 226
- Stroma cells, *see* Histology
- Strychnos nuxvomica*, 296
- Subarachnoid hemorrhage (SAH), 148, 227, 315, 317–318, 320, 322–323, 519
- Subcutaneous injuries, 312–313
facial subcutaneous tissues, 313
“facial peel-down,” 313
subgaleal hemorrhage of scalp impact, 313f
subscalpular subcutaneous tissues, 312
- Subcutaneous tissue, 93, 150, 312–313, 362, 380, 441, 482, 549, 556
microscopic view of skin, 93f
- Subcutis, *see* Integumentary system
- Subdural hemorrhage (SDH), 98, 316–317, 495, 507, 519–520
- Subendocardial myocardial infarcts, 248
- “Subfalcine” herniation, *see* Cingulate gyrus herniation/subfalcine herniation
- Subtle injuries, 136, 456
dark pinpoint sites of burnt, 456
melted keratin, 456
white blisters, 456
- Sudden infant death syndrome (SIDS), 70, 501, 508–510, 512
- Sudden unexpected death, 210, 223, 225, 251, 508, 577
- Sudden, unexplained infant death investigation (SUIDI), 59, 509
- Sudden unexplained infant death (SUID), 59, 70, 508–511
- Suffocation, 403
choking, 406–408
combination forms, 410–411
deaths, 403
choking, 406–408
combination forms, 410–411
mechanical asphyxia, 408–410
positional asphyxia, 410
simple asphyxia, 403–405
smothering, 405–406
- Suicidal childhood deaths, 516
- Suicidal drowning deaths, 446

- Suicide MOD, 73
 complex suicides, 74, 75f
- SUID, *see* Sudden unexplained infant death (SUID)
- SUIDI, *see* Sudden, unexplained infant death investigation (SUIDI)
- Sunburn, 485, 580
- Superinfection, 232
- Superior vena cava (SVC), 87t, 105
- Suppurative inflammation, *see* Neutrophilic inflammation
- Surgical pathology, 7
 dissection of specimen, 8f
 fallopian tube viewed under different magnifications, 11f–12f
 histologic examination, stains used
 H&E stain, 7
 histology laboratory, processing machines in, 8f
 microscopic anatomy, diagnoses based on, 11f
 microtome to cut tissue sections, use of, 9f
 patients' conditions analysis, dilemmas, 10
 placing tissue section on a glass slide, 10f
 slide via microscope, viewing, 11f
 tissue sample processing in plastic "cassettes," 8f
 tissue section embedded in paraffin wax, 9f
- SVC, *see* Superior vena cava (SVC)
- Sweat chloride test, 250
- Swimming pools or other bodies of water, 446
- Swiss cheese appearance, 177
- Systemic autoimmune disorders, 241
- Systemic "cyanosis," 209
- Systemic infection, 208
- Systemic inflammatory response syndrome (SIRS), 206, 208, 366, 522
- Systemic lupus erythematosus, 220, 241
- Systemic thromboemboli, 548
- T**
- Tache noire (black line), 168
- Tardieu spots (hemorrhage), 165, 165f, 415, 545
- Tardive dyskinesia, antipsychotic, 288
- Tay–Sachs disease, 249
- Temperature, 469
 heat gain exceeds heat loss (hyperthermia), 470
 heat load/heat loss, 469–470, 475
 heat loss exceeds heat gain (hypothermia), 470
- Temperature-related deaths, 469–479
- hyperthermia, 475–479
 autopsy findings, 477–478
 death certification, 478–479
 general features, 475
 scene investigation, 476
- hypothermia, 470–475
 autopsy findings, 471–474
 death certification, 475
 general features, 470–471
 scene investigation, 471
- See also* Temperature
- Terrorist agents
 biologic agents, 581–582
Bacillus anthracis (anthrax), 581
Clostridium botulinum (botulism), 582
 dengue fever, 582
 Ebola virus, 582
Francisella tularensis (tularemia), 582
 Hantaan virus, 582
 hemorrhagic mediastinal lymphadenitis, 581
 Lassa virus, 582
 Marburg virus, 582
 Variola virus (orthopoxvirus), 582
 yellow fever, 582
Yersinia pestis (plague), 582
- chemical agents
 blistering agents, 582
 cyanide agents, 582
 nerve agents, 582–583
 ricin, 583
- Tetrodotoxin fish poisoning, 567
- THC, *see* Delta-9-tetrahydrocannabinol (THC)
- Therapy-related deaths, 397f, 577, 583–586
 physiologic mechanism of death, 583
 pneumothorax, case of, 584
 "therapeutic complication," 585
- Thermal injuries, 94, 331, 422, 453, 462, 470, 481–482, 484, 487, 490, 492–493, 495–496, 498, 541
 dry burns, 470
 fire burns, 470
 radiant burns, 470
 scalding injuries, 470
- Thiamine (vitamin B1) deficiency, 247, 270, 564, 565t
- Third degree burns (full-thickness skin injury), 482, 483f, 485
- Thoracic aortic aneurysm, 215–216, 218, 251, 330
- Thoracotomy, surgical opening of chest cavity, 397
- "Thromboembolus," 88t, 115, 546–547

- Thrombophlebitis, inflammatory process, 222
- “Thrombotic microangiopathies of pregnancy,” 577
- “Thrombotic thrombocytopenic purpura,” 577
- Thrombus (blood clot) formation, 88t, 115, 211–215, 228–229, 546, 583
- Thymine (T), 28
- Thyroid disorders, 243–244
- Thyroid stimulating hormone (TSH), 117, 243–244
- Time of death estimation, 179–182
 - estimating PMI/entomology samples, 180–181
 - formula, 180
 - gastric content analysis, 180
 - “maggot motel,” 181–182
 - potassium concentration equations, 179–180*See also* Postmortem changes
- Tissue
 - bridging, 305, 379, 398
 - debridement, 492
 - donation, 568–572
- “Tonsillar herniation,” 319
- Tool marks (weapon cuts), 25, 389
- “Total insulin” levels, 248, 289, 574
- Toxicology issues, breakdown product, 263–267, 264
- Toxicology testing, 22, 39–40, 66, 132, 132f, 140, 145, 153–154, 157, 266, 290, 317, 421, 475, 478, 510, 541, 555
 - qualitative/quantitative, 153–154
 - sample types, 154
- Toxoplasma* (parasite), 566
- Trace evidence, types, 22
 - clothing, 134
 - fingernails, 135
 - hairs, fibers, gunshot residue, 135
 - sexual activity evidence, 135
- “Tracheal bronchus,” 230
- Track, (injury), 30, 74, 101, 134, 261, 278, 326, 362, 367–368, 373, 384, 388
- Transcervical herniation, 319
- Transmural (full thickness) infarcts, 248
- Transtentorial herniation, *see* Uncal
- Traumatic asphyxia, *see* Mechanical asphyxia
- Traumatic axonal injury, 317, 319, 321
- Traumatic injuries, 106, 324, 331, 410, 490, 495, 506–507, 561
- Trisomy syndromes, 250
- Trocar, 398, 552–553
 - embalming process, 552f
 - instrument, 398
 - (or) embalming button, 553f
- TSH, *see* Thyroid stimulating hormone (TSH)
- Tubal pregnancy, 575
- Tubercular and fungal, 208
- Tuberculosis (TB, mycobacterium), 114, 208, 232–233, 243
- Turner syndrome, 250
- Type II-non-insulin dependent DM or adult-onset DM, 247
- Type I-insulin-dependent DM or juvenile DM, 247
 - “diabetic pattern,” 248
 - insulin replacement (subcutaneous injection), 247
 - “ketoacidosis,” 247
- U**
- Ultraviolet (UV) radiation, 169, 272, 484–485, 580
- Uncal, 319
- United States and Canadian Academy of Pathology (USCAP), 14
- United States Medical Licensing Examination (USMLE), 4
- Unwanted pregnancy (neonaticide), 529–530
- Upper airway conditions, 108, 230, 407–408, 411, 487
 - inflammation from infection (epiglottitis), 230
 - swelling (allergic reaction), 230
- USCAP, *see* United States and Canadian Academy of Pathology (USCAP)
- USMLE, *see* United States Medical Licensing Examination (USMLE)
- V**
- Valve disorders, 219–220
 - degenerative aortic stenosis, 219
 - “senile calcific aortic stenosis,” 219
 - “wear and tear” valve disorder, 219
 - infective endocarditis, 220
 - “Libman–Sacks” endocarditis, 219
 - mitral valve prolapse, 219–220
 - myxomatous degeneration of, 219
 - redundant and “billowing,” 219f
 - nonbacterial thrombotic endocarditis, 219
 - “regurgitant,” 219
 - rheumatic heart disease, 219
 - “stenotic” or narrowed, 219
- Vapor-lock, 390, 549
- Variable number of tandem repeats (VNTR), 28
- Variola virus (orthopoxvirus), 582
- Vascular axonal injury, 321

- Vasculitis, 217, 241
 “giant cell arteritis,” 217
 infectious micro organisms
 Aspergillosis (fungus), 217
 Rickettsia rickettsii (bacteria), 217
 Treponema pallidum (spirochete
 bacteria), 217
 polyarteritis nodosa, 217
 terminology/subtypes, 217
- Venous thromboses (blood clots), 152
- Ventricular fibrillation, 452–453, 471
- Ventricular septal defect (VSD), 209
- “Vesicants” (blistering agents), 488, 582
- Vibrio vulnificus*, 567
- Violent older child, childhood homicides
 “domestic violence,” 532
- Violent outburst (angry impulse)
 numerous bruises in a battered child, 527
- Viral encephalitis, 208, 225
 Eastern equine, 224
 St. Louis, 224
 Western equine, 224
 West Nile viral, 224
- Viral pneumonia, 232
 CMV/HSV/RSV, 232
 interstitial inflammation, 232
 superinfection, 232
- Virchow method, 140–142
 alternative approach, 141, 142f
- Virchow, Rudolph (father of pathology),
 140–142
- Visual identification, 186, 188–189, 198, 439
 comparing the photo on a driver’s license,
 190
- Vital tissue reaction, lack of, 177, 395–396,
 545
- Vitamin deficiencies, 270, 563–564, 565t
 thiamine (vitamin B1) deficiency, 564
 Wernicke–Korsakoff syndrome, 564
- Vitamin D metabolism, 94
See also Integumentary system
- Vitreous chemistry testing, 475
- VNTR, *see* Variable number of tandem repeats
 (VNTR)
- Voir dire*, 18–19
- Voltage, 449
- VSD, *see* Ventricular septal defect (VSD)
- W**
- Washerwoman hands, 441
- Water deaths, *see* Drowning
- Waterhouse–Friderichsen syndrome, 244, 514
- wbcs, *see* White blood cells (wbcs)
- Wear and tear valve disorder, 219
- Wernicke–Korsakoff syndrome, 247, 564
- Western equine encephalitis, 224
- West Nile viral encephalitis, 208, 224–225
- White blister, 456
 in different low-voltage electrocution, 458f
 at site of entry in low-voltage electrocution,
 456f
- White blood cells (wbcs), 7f, 27, 113–114,
 208, 217, 241
 neutrophils/lymphocytes/macrophages, 208
- WHO, *see* World Health Organization (WHO)
- Wilms tumor in children, 245
- Wishnewski ulcers, 236, 473–474
- Wiskott–Aldrich syndrome, 242
- Wolff–Parkinson–White syndrome, 222
- Work-related death, *see* Occupational deaths
- World Health Organization (WHO), 63
- X**
- X-rays, 39, 132, 153, 191–193, 198, 200, 368,
 394–395, 507, 541, 578–579, 586
 examination, 492
 lead snowstorm, 368
 miscellaneous issues, 368
 use in ancillary procedures
 charred body cases, 153
 child abuse cases, 153
 gunshot wound case, 153
- Y**
- Yellow fever, 582
- Yellowstone National Park, 486
- Yersinia pestis* (plague), 582