VADEMECUM

Perioperative Care in Cardiac Anesthesia and Surgery

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Preface

The Peter Munk Cardiac Centre at Toronto General Hospital and University of Toronto is the largest cardiac center in Canada. Over 2,500 patients undergo open-heart operations each year. The cardiac surgical program has gained an international reputation for its academic productivity and attracts fellows and visitors from all parts of the world.

The nineties were characterized by cost containment, accountability, and value-based health care. We have responded to these obligations and have been able to provide the highest quality of care at the lowest possible cost. Surgery on the same day of admission, early tracheal extubation, improved operative techniques, and earlier rehabilitation have reduced the median intensive care stay and hospital stay. In addition less than 10% of our patients are transferred to a convalescence center.

This handbook describes the perioperative management of adult patients who undergo cardiac surgery at our centre. Although it was written by numerous authors, each chapter was carefully edited to reflect the collective view of our health care team. The purpose of this handbook is to provide a succinct, problem oriented source of practical information on cardiac surgical patients based on the clinical and research experience of our fast track cardiac surgery program. It was written for physicians, nurses and other allied health care personnel who are involved in the management of patients undergoing open-heart surgery.

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Tirone E. David, MD

Abbreviations

2,3-DPG	2,3-Diphosphoglyceraldehyde
AAA	Abdominal Aortic Aneurysm
ABG	Arterial Blood Gases
ACB	Aorto-Coronary Bypass
ACE	Angiotensin Converting Enzyme
ACT	Activated Clotting Time
AF	Atrial Fibrillation
AI	Aortic Insufficiency
AICD	Automatic Implantable Cardioverion Defibrillator
APS	Acute Physiology Score
ARDS	Adult Respiratory Distress Syndrome
ASA	Acetylsalicylic Acid
ASD	Atrial Septal Defect
AT	Anti-thrombin
AV	Atrio-ventricular
BCPS	Bi-directional Cavo-pulmonary Shunt
BID	Twice A Day
BP	Blood Pressure
BUN	Blood Urea Nitrogen
CABG	Coronary Artery Bypass Grafting
CAD	Coronary Artery Disease
cAMP	Cyclic Adenosine Monophosphate
CAPD	Continuous Abdominal Peritoneal Dialysis
CAVHD	Continuous Arterial-Venous Hemodialysis
CBC	Complete Blood Count
CBF	Cerebral Blood Flow
cGMP	Cyclic Guanine Monophosphate
CHF	Congestive Heart Failure
CI	Cardiac Index
CK	Creatine Kinase
CK-MB	Creatine Kinase Isoenzyme Fraction
CNS	Central Nervous System
CO	Cardiac Output
COPD	Chronic Obstructive Pulmonary Disease
CPAP	Continuous Positive Airway Pressure
CPB	Cardiopulmonary Bypass
CSF	Cerebral Spinal Fluid
CT	Computed Tomography

CVA	Cerebral Vascular Accident
CVICU	Cardiovascular Intensive Care Unit
CVP	Central Venous Pressure
CVVHD	Continuous Venous-Venous Hemodialysis
CXR	Chest X-Ray
DFT	Defibrillator Thresholds
DLCO	Diffusion Coefficient
DVT	Deep Vein Thrombosis
ECG	Electrocardiogram
ECMO	Extracorporeal Membrane Oxygenation
EDP	End Diastolic Pressure
EEA	Early Extubation Anesthesia
EF	Ejection Fraction
ETVC	Endotracheal Ventilation Catheter
FEV	Forced Expiratory Volume
FFP	Fresh Frozen Plasma
HAT	Heparin Associated Thrombocytopenia
HATT	Heparin Associated Thrombocytopenia Thrombosis
Hb	Hemoglobin
HIPA	Heparin Induced Platelet Activation
HIT	Heparin Induced Thrombocytopenia
HOCM	Hypertrophic Obstructive Cardiomyopathy
HR	Heart Rate
HTN	Hypertension
IABP	Intra-aortic Balloon Pump
ICU	Intensive Care Unit
ID	Infectious Disease
IDDM	Insulin Dependent Diabetes Mellitus
IHSS	Idiopathic Hypertrophic Subaortic Stenosis
INR	International Normalized Ratio
ITA	Internal Thoracic Artery
IVC	Inferior Vena Cava
KIU	Kallikrein Inhibitor Units
LAP	Left Arterial Pressure
LBBB	Left Bundle Branch Block
LITA	Left Internal Thoracic Artery
LM	Left Main
LMWH	Low Molecular Weight Heparin
LOS	Length of Stay
LSVC	Left Superior Vena Cava
LVAD	Left Ventricular Assist Device

LVF	Left Ventricular Function
LVOT	Left Ventricular Outflow Track
MAO	Monamine Oxidase
MAP	Mean Arterial Pressure
MAPCA	Major Aorto-Pulmonary Collateral Arteries
MH	Malignant Hyperthermia
MI	Myocardial Infarction
MIDCAB	Minimally Invasive Direct Coronary Artery Bypass
MR	Mitral Regurgitation
MRI	Magnetic Resonance Imaging
MVV	Maximal Voluntary Ventilation
NG	Nasogastric
NIDDM	Non-insulin Dependent Diabetes Mellitus
NIF	Negative Inspiratory Force
NO	Nitric Oxide
NSAID	Non-steroidal Antiinflammatory Drug
NTG	Nitroglycerin
OLV	One Lung Ventilation
OR	Operating Room
PAP	Pulmonary Artery Pressure
PAPVD	Partial Anomalous Pulmonary Venous Drainage
PCA	Patient Controlled Analgesia
PCSU	Post Cardiac Surgical Unit
PCWP	Pulmonary Capillary Wedge Pressure
PDA	Patent Ductus Arteriosis
PE	Pulmonary Embolism
PEEP	Positive End-Expiratory Pressure
PGE	Prostaglandin
PI	Pulmonary Insufficiency
PLV	Partial Left Ventriculectomy
POD	Postoperative Day
PR	Per Rectum
PRBC	Packed Red Blood Cell
PS	Pulmonary Stenosis
PSV	Pressure Support Ventilation
PT	Prothrombin Time
PTCA	Percutaneous Transluminal Coronary Angioplasty
PTT	Partial Thromboplastin Time
PVC	Premature Ventricular Contraction
PVR	Pulmonary Vascular Resistance
Qp:Qs	Pulmonary to Systemic Blood Flow Ratio

RAP	Right Arterial Pressure
RATS	Rabbit Anti-thymocyte Serum
RBC	Red Blood Cells
RCA	Right Coronary Artery
RT	Respiratory Therapist
RVOT	Right Ventricular Outflow Tract
RVOTO	Right Ventricular Outflow Tract Obstruction
SAM	Systolic Anterior Motion
SAP	Systemic Arterial Pressure
SDA	Same Day Admission
SNP	Sodium Nitroprusside
TA	Tranexamic Acid
TCCCA	Toronto Congenital Cardiac Centre for Adults
TEE	Transesophageal Echocardiography
TEF	Transesophagel Fistula
TGA	Transposition of Great Arteries
TIA	Transient Ischemic Attack
TIF	Tracheoinnominate Fistula
TLC	Total Lung Capacity
TOF	Tetralogy of Fallot
TPN	Total Parenteral Nutrition
TR	Tricuspid Regurgitation
TGH	Toronto General Hospital
VC	Vital Capacity
VSD	Ventricular Septal Defect
WBC	White Blood Cell

Toronto General Hospital Cardiac Surgical Program

Tirone E. David

Toronto General Hospital cardiac surgical program was created in 1990 after the merger of the Toronto General and Toronto Western Hospitals. Since then, the number of open-heart procedures increased from approximately 2,000 to 2,700 per year. Nine heart surgeons perform these operations plus 500 pacemaker and defibrillator implantations each year.

Our cardiac surgical program is a provincial and national referral center for cardiovascular diseases. In addition to a large number of cases of coronary artery and heart valve disease, we also treat numerous patients with complex and unusual cardiovascular problems. This variety of cases offers an unique opportunity to learn and investigate new surgical approaches.

This is an academic program but the primary emphasis is patient care. Education and research are important components of our mission but they come second to our responsibilities to our patients.

A detailed database form containing numerous clinical, hemodynamic, operative and postoperative variables is completed for every patient who undergoes heart surgery and is entered into a computer. This database is indispensable to monitor clinical outcomes, utilization trends, indirect costs, and individual surgeon's performance. It is also used for clinical research. Patients' satisfaction surveys are conducted twice yearly. Problems identified during these assessments of quality are discussed among caregivers and recommended changes are implemented and the outcome reassessed.

From 1993 to 1997, 11,112 patients underwent cardiovascular operations with cardiopulmonary bypass. The overall operative mortality was 3.4%. Table 1.1 gives a summary of operations performed and the operative mortality rates.

Postoperative complications are serious and expensive problems in cardiovascular surgery. Table 1.2 shows our complications rates from 1993 to 1997.

Approximately 90% of our patients are discharged within 7 days from surgery. The remaining 10% utilize as much resource as approximately one-half of all others.

We audited the problem of re-exploration for bleeding and the utilization of blood transfusion in our program. A multivariate logistic regression analysis identified several variables (the surgeon was one of them) predictive of re-exploration for bleeding and of blood transfusion. In addition, re-exploration for bleeding

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Table 1.1. Operations performed from 1993-1997

	Number	Mean age (mean +/-S.D.)	Timing Urgent	Emergent	mortality rate
Coronary artery bypass	7,371	62 ± 10	44%	3%	2.3%
Aortic valve surgery	1,070	63 ± 15	32%	2%	2.5%
Mitral valve surgery	704	59 ± 14	27%	6%	4.3%
Double/triple valve surgery	381	57 ± 16	23%	3%	9.0%
Ascending aorta ± arch ± aortic valve surgery	475	57 ± 16	26%	16%	6.6%
Congenital heart surgery in adults	473	42 ± 15	11%	1%	3.2%
Miscellaneous*	638	57 ± 14	33%	23%	10.2%

*Left ventricular aneurysms (254), heart transplantation (99), myectomy (94), mapping+ablation (44), postinfarction rupture of the septum (37), atrial myxoma (29), others (82).

Table 1.2. Complications rates from 1993-1997

	CABG	Valves	AA/A	CHD	Miscellaneous
Re-exploration for bleeding	1.5%	4.0%	6.5%	4.2%	4.2%
Perioperative stroke	1.4%	2.5%	6.6%	0.0%	2.2%
Perioperative myocardial infarction	2.4%	1.3%	1.7%	0.6%	1.6%
Deep sternal infection	0.8%	0.7%	1.1%	0.6%	0.2%
Superficial wound infection	1.7%	0.8%	1.7%	1.9%	1.4%
Sternal dehiscence	0.3%	0.1%	0.6%	0.0%	0.2%
Renal failure	0.3%	0.3%	0.6%	0.0%	1.6%
Mean ICU stay (days)	1.9	2.5	3.3	2.4	3.4

Abbreviations: CABG = coronary artery bypass graft; AA/A = ascending aorta +/-arch; CHD = congenital heart disease

was associated with a significant increase in operative mortality (12.2% vs. 2.7%), and hospital stay (14.8 vs. 9.4 days). The awareness of the problem alone resulted in an improvement of this type of complication. Careful reviews of perioperative complications and their implications in the clinical outcomes and costs frequently allow for changes that have a positive effect in the quality of care.

Fast Track Cardiac Surgical Program

Davy C.H. Cheng

INTRODUCTION

In this era of cost containment and physician report cards, we are being held accountable for patients' outcome in terms of mortality, morbidity, quality of life, length of stay (LOS) and costs of care. Intensive care unit (ICU) costs rank second only to operating room costs, thus it is economically appealing for early extubation to facilitate earlier ICU and hospital discharge of cardiac surgical patients.

Fast Track Cardiac Anesthesia

Fast track or early tracheal extubation anesthesia is a perioperative anesthetic plan which aims to facilitate tracheal extubation of patients within 1-6 h post cardiac surgery. Most centers consider fast track extubation up to 8-10 h postoperatively.

Fast Track Cardiac Surgery

This clinical pathway is a process of care including a multi-disciplinary approach aimed to improve the efficiency of care in cardiac surgical patients. Early extubation anesthesia is a key to the success of the fast track cardiac surgery pathways.

It is now feasible to accomplish early extubation because of improved anesthetic management, coupled with advancements in surgical, myocardial protection and normothermic cardiopulmonary bypass (CPB) techniques. Postoperative hemostasis is better controlled. We have demonstrated that early tracheal extubation anesthesia is safe, cost beneficial and can improve resource utilization in cardiac surgery.

RISK STRATIFICATION IN FAST TRACK CARDIAC SURGERY

Perioperative morbidity and mortality in cardiac surgery are strongly influenced by the severity of the patients' cardiac and non-cardiac illness. The

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postoperative acute physiology score (APS) is the most powerful predictor of mortality as it reflects the summation effects of surgical skill, anesthetic management, effectiveness of immediate postoperative care and the preoperative characteristics of the patient. The intraoperative anesthetic management, duration of CPB and of aortic cross clamp, the individual anesthesiologist and postoperative ICU management can all influence the outcome of patients.

An analysis of 1,250 cardiac surgical patients undergoing early extubation anesthesia included 74% coronary bypass, 22.6% valvular, and 9.6% combined and others procedures. Thirty-one patients died (2.5% mortality). The median time to extubation was 7 h (range 1-306 h).

- Delayed extubation: Age, intraoperative intra-aortic balloon pump (IABP), inotropes and antiarrhythmics appeared to be independent predictors of delayed extubation. However, intraoperative and postoperative factors were much more significant determinants of delayed extubation than preoperative factors.
- ICU LOS: The median ICU LOS was 24 h (range 16-868 h); 232 (18.6%) patients had ICU LOS > 48 h. Univariate analyses showed 14 predictors of prolonged ICU LOS. Multiple logistic regression analysis revealed eight independent predictors of prolonged ICU LOS: *Preoperative*: emergency surgery, grade 4/4 left ventricular function, female gender and renal insufficiency. *Postoperative*: myocardial infarction, stroke, atrial arrhythmia and renal insufficiency.
- Mortality: Univariate analyses showed 10 predictors of mortality. *Preoperative*: gender, repeat surgery, emergency surgery, myocardial infarct < 1 week, left ventricle grade 4/4, renal insufficiency or failure, stroke and inotrope use. *Intraoperative*: myocardial ischemia and CPB duration. Multiple logistic regression analysis revealed four independent predictors of mortality: preoperative inotrope usage, CPB > 120 min, female gender and myocardial infarction < 1 week.

It is the intraoperative and postoperative morbidity which ultimately determine the feasibility of early extubation and ICU LOS. Therefore, every patient should be a candidate for early extubation (Table 2.1).

Although it is possible to extubate patients on the operating table, the early risks of hypothermia, bleeding and cardiorespiratory instability outweigh the potential cost saving and other benefits, and is not recommended by most centers. One must not forget the delay and the costs of operating room (OR) time to fully awaken and extubate patients in the OR.

MEDICAL AND ECONOMIC IMPLICATIONS

We have compared the morbidity and mortality outcome of early and late extubation in a prospective randomized trial:

- Cardiovascular: There is no significant difference in the incidence of postoperative myocardial ischemia and ischemia burden between the

early and late extubation patients; more importantly, there was no increase in CK-MB levels or myocardial infarction rate (Fig. 2.1).

- Sympathoadrenal stress: Early extubation anesthetic management adequately suppresses the perioperative catecholamines (norepinephrine, epinephrine, and cortisol) stress response.
- Respiratory: The first hour postextubation is most crucial in respiratory care as reflected by the apnea index in either early or late extubation (Table 2.2). The tidal volume and central respiratory drive also progressively improved. Postextubation oxygen (O_2) saturation, respiratory

Table 2.1. Independent predictive factors of delayed extubation, prolonged ICU LOS and hospital mortality in cardiac surgical patients post early extubation anesthesia.

	Preoperative	**Intraoperative**	**Postoperative**
Delayed extubation > 10 h	Age	Inotropes IABP	Atrial arrhythmia
ICU LOS > 48 h	Emergency LVF 4/4 IABP Female	IABP Inotropes	MI CVA Renal dysfunction
Hospital Mortality	Inotropes Female MI < 1 week	Inotropes IABP CPB > 120 min	

Abbreviations: IABP, intra-aortic balloon pump; MI, myocardial infarction; h, hour; LVF, left ventricular function; CVA, cerebral vascular accident

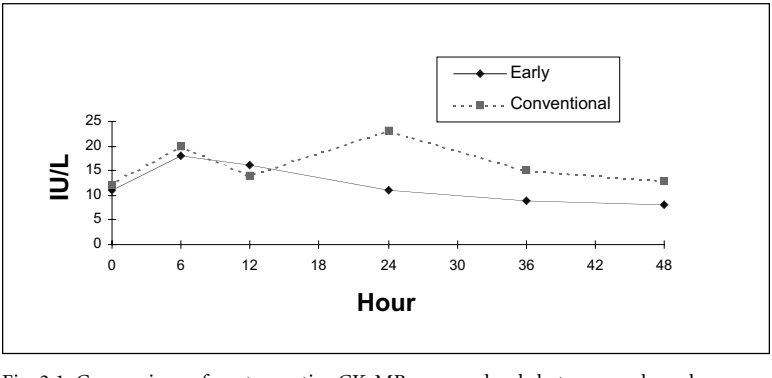

Fig. 2.1. Comparison of postoperative CK-MB enzyme levels between early and conventional extubation patients. (Reprinted with permission from: J Thorac Cardiovasc Surg 1996; 112:755-764. ©1996 by Mosby-Year Book, Inc.)

Table 2.2. Postextubation apnea

Extubation	Early	Conventional
Incidence	27.5% (14/51)	33.3% (17/51)
Duration (second)	17.7 ± 23.0	15.7 ± 28.6
Index (rate/h)		
1 h	13	15
2 h	4	8
3 h	2	9
4 h	2	8

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rate, labored breath index and apnea characteristics were similar between both groups. In addition, early extubation improves intrapulmonary shunt fraction by 30-40% post-CABG surgery.

- Neurological: Patients extubated early performed better and returned to baseline level earlier than patients extubated late in a mini-mental state test. This allows earlier mobilization, chest tube removal and oral intake of food, resulting in reduced LOS in ICU and hospital.
- Early extubation does not increase postoperative mortality rate.

It has been shown that resource use measured as LOS and cost are strongly influenced by severity of illness of patients, postoperative complications and efficiency of the nursing unit. The process of postoperative care must be modified to complement early tracheal extubation for maximum cost efficiency. Fast track cardiac anesthesia provides the opportunity for the paradigm shift in postoperative care of cardiac surgical patients.

Early extubation anesthesia drugs do not incur an increase in perioperative anesthetic drug costs. It reduces ICU LOS and encourages earlier mobilization leading to earlier hospital discharge. Thus it allows cost shifting with earlier discharge of patients from the high costs of the ICU to the lower costs of the ward. Including all complications in the cost analysis, early extubation anesthesia and management further reduced ICU costs (52.7%) and total CABG costs (25%) when compared with late extubation in our program.

Additional cost savings can be achieved by fewer cardiac surgery cancellations due to backlog of ICU beds leading to a loss in operating room time and associated staffing and hospital costs.

FAST TRACK CARDIAC SURGERY TEAM

Our fast track cardiac program team approach includes:

- preoperative patient education
- same day admission surgery
- expeditious and meticulous surgical management
- early extubation anesthetic technique
- horizontal integration of ICU and step-down unit as postcardiac surgical unit
- dedicated medical coverage
- flexibility in nursing practices and supporting services

Communication among members of the cardiac patient management teams (cardiovascular surgeon, cardiac anesthesiologist, ICU staff, nurses, respiratory therapists, physiotherapists and social workers) is vital to the success of the early extubation and a fast track surgical cardiac program. Nursing management needs to support single-day ICU stay and transfer, changes to analgesia and sedation practice and accelerated weaning and tracheal extubation of patients. Early postoperative mobilization of patients and walking on postoperative day 1 is recommended. Early resumption of oral intake and chest tube removal, with planned hospital discharge on postoperative days 4-6 is facilitated.

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Preoperative Assessment

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Most patients referred for heart surgery have been evaluated by their family physician and cardiologist. The cardiac surgeon must, however, review the medical history to avoid surprises in the operating room or during the postoperative period. In addition to a careful review of the cardiovascular system, all other systems must be evaluated to determine the patient's ability to withstand the operation.

BASIC EVALUATION

Medical History

The initial interview is important because it allows the heart surgeon to obtain the medical history and to establish a patient-doctor relationship. The general inquiry should include the following symptoms of cardiac disease: chest pain, dyspnea, fatigue, hemoptysis, syncope, palpitation, peripheral edema and cyanosis.

The degree of physical disability that cardiac symptoms causes is expressed by the New York heart association functional classification:

Class I—No symptoms on ordinary physical activities.
Class II—No symptoms at rest; ordinary activities provoke symptoms.
Class III—No symptoms at rest; mild activities provoke symptoms.
Class IV—Symptoms at rest or during any activity.

The severity of angina pectoris is graded by the Canadian cardiovascular society classification:

Class I—Only very strenuous physical activity causes angina.
Class II—Moderate physical activity causes angina. Examples: walking more than two blocks on the level or climbing more than one flight of stairs at normal pace provoke pain.

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Class III—Mild physical activity causes angina. Examples: walking one block on the level or one flight of stairs at normal pace provokes pain.
Class IV—Any physical activity causes pain.

Past History

The following items summarize the important aspects of the past medical history.

Previous operations

Thoracotomy, saphenous vein stripping/ligation, peripheral vascular surgery including carotid endarterectomy

Allergies

Drugs and other agents

Medications

Anticoagulants, antiarrhythmics, antiplatelet agents, ACE-inhibitors, diuretics

Review of systems

- Cardiovascular system—Anatomical and functional assessment, past cardiac procedures, risk factors, and investigation.
- Central nervous system—Transient ischemic attacks (TIAs) or previous stroke requires a full neurologic work-up.
- Respiratory system—If chronic obstructive lung disease is suspected, spirometry, pulse oxymetry and arterial blood gases should be obtained prior to surgery.
- Endocrine system—Diabetes and its complications should be recorded.
- Hematologic system—All patients should be asked about bleeding disorders. In addition, those of African heritage should have sickle cell screening.
- Renal system—Impaired renal function and renal dialysis increase the risk of perioperative complications. Renal transplant recipients seem to do well if renal function is normal but the Renal Transplant Service should manage the drugs and follow the patient perioperatively.
- Gastrointestinal system—Active peptic ulcer disease, active hepatitis, cirrhosis, and other gastrointestinal problems can seriously affect the outcome of cardiac surgery and must be carefully evaluated.
- Peripheral vascular system—Venous and arterial disease should be noted. Aorto-iliac occlusive disease may prevent the insertion of intra-aortic balloon pump through a femoral artery.
- Genitourinary system—It may be difficult to insert a Foley catheter in patients with prostate problems.
- Musculoskeletal system—Major skeletal deformities or active arthritic conditions may interfere with airway management, ambulating and recovery.

Family History

Family history of CAD, congenital heart disease, Marfan syndrome, malignant hyperthermia and other hereditary disorders should be recorded.

Social History

Marital status and living conditions should be considered in the discharge planning.

Physical Examination

Height, weight, and vital signs must be obtained on admission and recorded. Examination of the eyes, airway, neck, chest and abdomen should be performed and all findings recorded. Pelvic and rectal examination do not have to be done unless indicated. All peripheral pulses must be checked. The carotid and subclavian arteries should be auscultated for bruits. In patients for coronary artery bypass, the Allen's test must be performed in case a radial artery is used as a bypass conduit.

See appendix 1 for the history and physical examination form used at Division of Cardiovascular Surgery of Toronto General Hospital.

PREADMISSION TESTS

Blood Tests

Complete blood count (CBC), PT, PTT, INR, electrolytes, glucose, BUN, and creatinine should be obtained in all patients. Specific blood tests may also be appropriate for those with co-morbid conditions.

Urinalysis

Urinalysis is a simple method to detect urinary tract and renal disease and should be routinely obtained.

Chest X-ray

A posterior-anterior and lateral views chest x-ray should also be routinely obtained and displayed in the operating room, particularly in re-operations because the lateral view can give an idea where the heart and the ascending aorta are in relation to the sternum.

Electrocardiogram

This is an indispensable test to diagnose heart rhythm abnormalities and myocardial ischemia. However, a normal electrocardiogram does not rule out severe underlying cardiac problem.

ASSESSING THE RISK AND BENEFIT OF SURGERY

The indications and contraindications to treat heart diseases with surgery continue to evolve and a full discussion is beyond the scope of this handbook. How-

ever, certain principles and basic rules will be discussed. Surgeons must know the natural history of heart diseases and how therapeutics can modify them. Surgery is a form of therapy which is always associated with morbidity and, unfortunately, mortality. These factors must be weighed against the natural history of the disease and/or medical treatment. The influence of co-morbid conditions must also be weighed in the evaluation of the natural history and outcomes of medical and surgical therapy.

Ventricular Function

Ventricular function is the most important determinant of outcome of all heart diseases. Patients with severe LV dysfunction usually have a poor prognosis but surgery can sometimes dramatically change it. LV dysfunction due to CAD is among the most difficult to assess and to determine the effect of revascularization. A simple rule is that patients who have severe left ventricular dysfunction but good arteries to bypass usually do very well at least from the operative risk viewpoint. Those with bad ventricles and marginally graftable coronary arteries are usually poor surgical candidates. There are numerous tests to determine viability of a non-functioning segment of myocardium, none of which are completely reliable. These are thallium and sestamibi myocardial imaging, positron-emission tomography (PET) scanning, and magnetic resonance imaging (MRI).

Volume overloading conditions such as aortic and mitral insufficiency may irreversibly damage the myocardium. Surgical correction of these lesions may not change the prognosis but can improve the symptomatology. LV pressure overload caused by aortic stenosis ultimately depresses ventricular function and may cause myocardial fibrosis. Relief of the obstruction is almost always followed by improvement in ventricular function.

Coronary Artery Disease

Isolated proximal disease in large coronary arteries is ideal for bypass surgery and probably confers the best clinical outcomes. Small, diffusely diseased coronary arteries are not suitable for bypass surgery. Unfortunately, a large proportion of patients lie in between these two extremes, and the decision to operate is based more on the symptomatology or on the fact they have multi-vessel disease than in the probability of long-term patency of the grafts. Only experience can teach which of these vessels are graftable and which grafts stay patent.

Heart Valve Disease

Repair of heart valves is preferable to replacement because the ideal artificial heart valve has not yet been developed. However, repair is not feasible in all patients with heart valve disease. Thus, the selection of type of heart valve prosthesis (tissue or mechanical) and size (effective valve area) are important aspects of the surgical treatment.

Other Heart Diseases

A variety of other cardiac diseases can be surgically treated. For each one, the rules of assessing risks/benefits are the same and must always be considered before surgery is offered.

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INFORMED CONSENT

The risks and benefits of surgery must be clearly outlined to the patient, and with patient's permission, also to his/her family. Potential complications should be explained in a friendly and caring way. Although one should not dwell on complications that are inconsequential, details of serious ones such as death, stroke, myocardial infarction, infection, and others pertinent to the particular type of surgery ought to be discussed. In case of language barrier, the interpreter must also witness the consent by signing it.

THE PREADMISSION CLINIC

Over 70% of all operations are performed in our hospital on same-day admit (SDA) patients. This "one time shopping" plan of preoperative evaluation must be done in a very predictable manner in order that patients do not get canceled on the day of surgery and to ensure patient satisfaction and optimal medical outcomes.

A quality assurance program should be in place to:

- assess patient satisfaction
- evaluate surgical cancellation rates
- allow for recommendations for program improvement including maximizing efficiency

The objectives of the SDA program include:

- patient preoperative evaluation and optimization (including patient teaching)
- teaching (undergraduate, postgraduate)
- procurement of patients for clinical research studies

Many health care personnel are involved with an SDA program: clerk, laboratory technicians, nurses, nurse practitioners, house staff and physicians (surgical, medical and anesthesia).

PREOPERATIVE MEDICATION ORDERS

Cardiac Medications

All regular cardiac medications should be continued to the morning of surgery. This includes nitroglycerin patches. Exceptions to this rule include:

• **Amiodarone**: This drug should be stopped 4 weeks preoperation (half life 35 days). However, if it is required for significant ventricular dysrhythmia treatment, it is important to discuss this with the cardiovascular surgeon and cardiologist. Potential intraoperative problems include resistant bradycardia, hypotension, low systemic vascular resistance, and possible oxygen toxicity.

• **ACE inhibitors**: Stopping these drugs 24 hours prior to surgery is controversial. There is a small risk of hypotension perioperatively with these drugs.

Anticoagulants

• **Warfarin**: Warfarin should be stopped 4-5 days prior to surgery. If the patient is at high risk of thrombosis he/she must be admitted and started on intravenous heparin.

• **Heparin**: Intravenous heparin should be stopped 2-3 hours preoperation (unless the patient is on IABP support).

• **ASA/Ticlopidine/NSAIDS**: These should be stopped 7-10 days preoperation if possible.

Psychotropic Drugs

• **MAO inhibitors**: Long acting—discontinue 2 weeks preop; short acting—discontinue 1 week preop.

Others

• Steroids and anti-rejection drugs (transplant patients) must be continued.

Preoperative Sedation for Same Day Admission

• A standard form is given to the patient with specific written instruction on cardiac medication and preoperative sedation for the same day admission. This is ordered by the anesthesiologist to be given in the hospital on day of surgery.

• Lorazepam: 1-3 mg sublingually.

• Narcotics: usually not required, IM narcotics may be given in appropriate holding areas. These are usually given with a major sedative (i.e., perphenazine).

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Early Extubation Anesthesia

Davy C.H. Cheng

Early extubation anesthesia is the key to success for a fast track cardiac program. An important challenge for anesthesiologists is participation in development and implementation of the fast track cardiac program reflecting our knowledge and management skill in perioperative care.

PREOPERATIVE

Preoperative patient education of the perioperative course is important to reduce anxiety and to establish the patients' expectations. A preadmission clinic and same day admission program will reduce the hospital length of stay (LOS). Furthermore, it will reduce surgery cancellation or delay from last minute abnormal blood tests or suboptimal clinical condition of patients.

INTRAOPERATIVE

Early extubation anesthesia (EEA): our anesthetic regimen is a balanced anesthesia technique with low dose narcotic, propofol and inhalational agents.

1. Premedication: lorazepam 1-3 mg sublingual, 1 h preoperation.
2. Prophylactic antifibrinolytic treatment with tranexamic acid 50-100 mg/kg i.v. intraoperatively.
3. Induction: propofol (0.5 mg/kg) or thiopental (1 mg/kg), low dose narcotic (fentanyl total 10-15 μg/kg), pancuronium (0.15 mg/kg), midazolam (1-3 mg).
4. Precardiopulmonary bypass (CPB): isoflurane (0.5-2%), midazolam (total 0.07-0.1 mg/kg).
5. CPB: propofol infusion 2-6 mg/kg/h.
6. Post-CPB: postoperative analgesia (indomethacin or diclofenac 50-100 mg PR, if not contraindicated) is essential and sedation (propofol) are titrated to allow for tracheal extubation within 1-6 h.
7. Tight fluid balance and aggressive arrhythmia control.

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Other ultra-short acting narcotics, such as remifentanil, are currently being evaluated for their safety and cost-effectiveness.

The potential benefit of intrathecal morphine or intravenous methylprednisone to facilitate fast tracking in cardiac surgery is contradictory. It has been found that intrathecal morphine actually delays extubation time post-CABG surgery due to prolonged ventilatory depression.

A low incidence of intraoperative recall in fast track cardiac anesthesia (0.3%) may relate to the use of a balanced anesthetic technique coupled with a propofol infusion during CPB which gives better control of anesthetic delivery and more reliable anesthesia at this critical time. It is crucial to treat hypotension during CPB with adjustment of pump flow, inotropes or vasopressors rather than reducing or discontinuing the propofol or inhalational anesthetic.

POSTOPERATIVE

Although preoperative high-risk patients are more likely to experience postoperative complications, it is the intraoperative and postoperative morbidity which ultimately determine the feasibility of early extubation and ICU LOS. Therefore, expeditious and meticulous surgery is vital to avoid prolonged CPB, coagulopathy, inadequate myocardial revascularization/protection, low cardiac output syndrome and stroke.

Nursing management should support single-day ICU stay and transfer, changes to analgesia and sedation practice, and accelerated weaning and tracheal extubation of patients. This allows for earlier chest tube removal, mobilization, and oral intake of food on postoperative day 1, facilitating ICU and early hospital discharge. Postoperative management plans should be set up for continuous improvement in quality of care and cost savings. These include the ventilation weaning protocol, extubation guideline (Table 4.1), arrhythmia management, postoperative anticoagulation protocols, ICU and hospital discharge guidelines which will be discussed in detail in later chapters and summarized in the appendices.

Table 4.1. Tracheal extubation guideline

CNS: Responsiveness and cooperation
CVS: CI > 2.0, absence of uncontrolled arrhythmia
RESP: VC > 10 cc/kg, NIF > –20 cm H_20; pH > 7.30, PaO_2 > 80 mm Hg on FiO_2 < 0.5
BLEEDING: Chest tube drainage < 100 cc/h
RENAL: Urine output > 0.5 cc/kg/h
TEMP: > 36.0° C

CNS = central nervous system; CVS = cardiovascular system
CI = cardiac index; RESP = respiratory system; VC = vital capacity
NIF = negative inspiratory force; TEMP = core temperature

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Perioperative Monitoring and Intraoperative Echocardiography

Corey Sawchuk, Harry Rakowski

The goals of perioperative monitoring for the patient having cardiac surgery include the assessment of cardiopulmonary function and detection of reversible disease processes in the perioperative period that influence myocardial performance and tissue perfusion. The most basic and important monitor of the patient is the primary caregiver from surgery, anesthesia, intensive care and nursing. Advanced technologies available to the cardiac care team optimize perioperative management of cardiac surgery patients.

STANDARD MONITORS

Basic perioperative monitoring includes:

- five-lead electrocardiogram (ECG) monitoring with ST segment analyses (Leads II, V)
- noninvasive blood pressure measurement
- direct arterial pressure
- pulse oximetry
- end tidal gas analysis
- central venous pressure

CENTRAL VENOUS MONITORING (CVP)

- CVP measurements assess the filling of the right ventricle and are particularly important on cardiopulmonary bypass (CPB) to monitor venous drainage.
- Central venous cannulation usually occurs post induction of anesthesia except in select patients with hemodynamic compromise or poor venous

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access. Insertion via internal jugular venous or subclavian venous approach, is under aseptic conditions using a Seldinger technique. The cordis line is capped for CVP monitoring or if needed, a Swan-Ganz catheter can be inserted.

- Attention to avoid puncture of the carotid artery and accidental sheath insertion is mandatory. Clinical assessment of catheter pressure prior to sheath insertion is recommended.

SWAN-GANZ CATHETERIZATION

Swan-Ganz catheters remain the controversial gold standard of invasive perioperative hemodynamic monitoring of high-risk cardiac surgical cases. Popularity of this technique remains high due to its ease of utilization in the perioperative period, continuous monitoring capabilities, and low morbidity and mortality.

The majority of information crucial to the management of the cardiac surgical patient is obtained post-CPB, following chest closure and into the postoperative period. Besides absolute data measured from the catheters, it is important to recognize trends in hemodynamic parameters that may indicate cardiopulmonary compromise. Early diagnosis and intervention of hypovolemia and low cardiac output states can prevent early onset of multi-organ system dysfunction that result in prolonged hospital stay.

Information routinely accessed from the catheters include:

- continuous assessment of ventricular filling use central venous pressure (CVP) and pulmonary artery pressure (PAP)
- pulmonary capillary wedge pressure (PCWP)
- intermittent cardiac output (CO) determination
- mixed venous gas measurements if indicated

Indications for Swan-Ganz catheters include**:**

- left ventricular dysfunction (ejection fraction < 40%)
- high risk of poor revascularization secondary to poor distal vessels
- preoperative hemodynamic instability
- anticipated prolonged CPB time
- significant co-existing medical disease (pulmonary, cerebral, renal)

Avoid complications from over wedging the catheter by:

- use of 1.5 cc syringe for balloon inflation
- Swan-Ganz catheter distance < 50 cm
- pulling catheter back 5 cm on CPB
- limit wedge measurements if anticoagulated
- use pulmonary artery (PA) diastolic measurements as an indicator of left ventricular filling

Patients with heart failure, tricuspid and pulmonary valve regurgitation, and pulmonary hypertension may be difficult for Swan-Ganz catheter insertions. Reverse Trendelenburg, tilting the patient right side up, and brief cessation of posi-

tive pressure sometimes aid in the passage of the catheter. Consider fluoroscopic or echocardiographic guidance in difficult cases.

Complications of central venous cannulation and Swan-Ganz catheters include:

- pneumothorax
- cardiac trauma
- dysrhythmias
- knotting of Swan-Ganz catheter
- infection
- thrombus formation
- pulmonary artery rupture
- pulmonary infarction

Common Swan-Ganz pitfalls:

- rapid infusion of intravenous fluids during CO determination giving false low measurements
- wrong volume or temperature of injectate
- improper transducer calibration or drift

TRANSESOPHAGEAL ECHOCARDIOGRAPHY (TEE)

Intraoperative TEE is administrated by the Echocardiography laboratory within the Division of Echocardiography at Toronto General Hospital. High quality assurance of this technology is vital and only experienced and certified clinicians perform examinations. Interpretation of all intraoperative TEEs are performed by senior echocardiography fellows and cardiologists. A direct line linking the operating room to the echocardiography laboratory is available for on-line interpretation and consultation for intraoperative examinations.

Current practice of intraoperative TEE at Toronto General Hospital reflects well-recognized indications for TEE as a diagnostic tool for complex cardiac surgery. Approximately 15% of all cardiac procedures have an intraoperative exam. This low number of examinations represents a cost effective approach of performing examinations in selected patients. We soon expect the numbers of exams to increase to include monitoring of ventricular function and volume and epivascular aortic imaging for high risk cardiac surgical cases. The postoperative TEE is an important diagnostic tool to assess indications to return to the operating room secondary to bleeding, ischemia, or cardiac structural abnormalities.

Indications for intraoperative TEE:

- mitral valve repair
- aortic valve repair and aortic valve sparing procedure
- aortic valve replacement with stentless biological valve
- adult congenital heart disease
- ventricular myectomy in IHSS
- ventricular remodeling procedures
- endocarditis

- cardiac tumor resection
- heart transplant
- perioperative cardiovascular instability or difficult CPB separation

Contraindications to intraoperative TEE:

- lack of informed consent (relatives)
- esophageal pathology (stricture, cancer, diverticulae)
- c-spine instability

Significant complications of perioperative TEE are uncommon but include:

- failure to pass the TEE probe (< 2% of patients)
- pharyngeal injury and bleeding
- dental trauma
- dysrhythmias
- significant esophageal injury requiring surgical correction (rare)

Multiplane TEE probes are used in the majority of patients. We use pediatric biplane probes for smaller patients with previous difficulty passing the larger multiplane probes. We focus all intraoperative exams to assess the primary indication for the exam and guide further surgical and medical therapy. After the initial focused assessment we do a complete structural and functional exam of the heart and major vasculature.

MITRAL VALVE REPAIR

The preoperative evaluation of the mitral valve assesses the etiology of mitral regurgitation and associated valve lesions to predict prospects for successful repair (Table 5.1 and Table 5.2).

Success of mitral valve repair is high with good patient selection. In cases where residual mitral regurgitation is more than mild, multiple factors including patient tolerability of an extended cross-clamp duration and CPB run influences the decision to re-repair, replace or accept the current repair.

Postoperative TEE assessment of mitral valve repair includes:

- optimization of loading conditions
- doppler determination of residual mitral regurgitation from multiple views
- pulmonary venous flow patterns and quantification
- site of residual mitral regurgitation and potential for re-repair
- re-assessment for alternative etiologies of residual mitral regurgitation (left ventricular dysfunction, left ventricular outflow tract obstruction and systolic anterior motion of the mitral valve leaflet)

Table 5.1. Mitral and aortic valve pathology

Valve	Etiology	% Reparable	Factors Limiting Repair
Mitral	Myxomatous	85-90%	Barlows valve, ant. leaflet involved, multiple valve segments
	Rheumatic	50%	Leaflet calcification, fusion, and immobility, chordal shortening
	Ischemic	35%	Irreversible wall motion abn., papillary muscle rupture, unable to remodel LV surgically or with revascularization.
	Congenital	75%	Inadequate leaflet tissue
	endocarditis	25%	Degree of leaflet destruction, annular involvement, calcification
Aortic	Bicuspid valve	20%	Age, raphe calcification, degree of prolapse or bileaflet prolapse
	Non-rheumatic valve	80%	Number of prolapsed leaflets, calcification, tears or perforation, annular dilatation
	Non-rheumatic valve with aortic root disease	80%	Number of prolapsed leaflets, calcification, tears or perforations, sinotubular junction size

Table 5.2. Quantification of regurgitant valve lesions

Valve	Method	Mild	Moderate	Severe
Mitral	Color jet area	$< 4\ cm^2$	$4.0\text{-}8.0\ cm^2$	$> 8.0\ cm^2$
	Pulmonary venous flow	Normal	Systolic blunting	Systolic reversal
	Regurgitant orifice area	$< 0.1\ cm^2$	$0.1\text{-}0.5\ cm^2$	$> 0.5\ cm^2$
Aortic	Aortic insufficiency jet/LVOT	< 0.24	0.25-0.49	> 0.50
	Aortic reversal	none	significant	= forward flow
	Aortic insufficiency press. $t^1/_2$	> 600 msec	300-600 msec	< 300 msec

AORTIC VALVE REPAIR

Current surgical options for the management of aortic valve insufficiency are expanding to include valve repair. Intraoperative TEE assessment of the aortic valve provides additional critical information for the appropriate surgical management of aortic valve disease. Morphology and pathology of the aortic valve and aortic root help to predict reparability and further surgical management.

Decisions to continue with repair or alternative surgical procedures are complex and are similar to the previous discussion regarding mitral valve disease. Postoperative assessment of aortic valve repair includes an assessment of LV function and the degree of aortic valve insufficiency.

The detection of persistent new wall motion abnormality and difficult separation from bypass should prompt exploration of the coronary arteries for a surgically correctable cause.

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Weaning from Cardiopulmonary Bypass

Annette Vegas

Successful weaning from cardiopulmonary bypass (CPB) requires a team approach between the surgeon, anesthesiologist and perfusionist. Anticipating which patient might be difficult to wean is at best inaccurate, but experience supports age, preoperative cardiac function and duration of CPB and aortic crossclamp as major risk factors. In anticipating a difficult wean the choice of prophylactic pharmacologic agents is variable. The early and timely introduction of mechanical support in the form of an intra-aortic balloon pump (IABP) is preferred over excessive pharmacologic agents.

WEANING TECHNIQUES

Weaning from Bypass

- Core temperature greater then 37.5°C.
- Reperfusion time at least 8 minutes to replenish myocardial ATP stores.
- Adequate metabolic milieu, hematocrit (HCT) > 0.20, potassium (K^+) < 6.0 mmol/L, bicarbonate (HCO_3^-) > 20 mmol/L.
- Critical to have stable heart rate and rhythm, preferably sinus at 70-100 beats per minute:
  - if HR too slow pace ventricular or atrial-ventricular or atrial, if cannot pace check leads, cable, pacer box, K^+ too high
  - if HR too fast can slow down with atrial filling, consider β block or cardioversion
  - if ventricular irritability: increase perfusion pressure (MAP), consider adding to the pump the following drugs: magnesium sulfate (1-2 grams), lidocaine (1-1.5 mg/kg) or amiodarone (150 mg), defibrillate with 5-20 J if needed
- Ventilator on to deliver 100% O_2 at adequate minute ventilation and lungs fully re-expanded.

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- Monitors reset and re-zeroed, if arterial line discrepancy consider (a) non-invasive blood pressure (NIBP), (b) aortic root line, (c) femoral line.
- Watch filling pressures from pulmonary artery diastolic pressure (PA_D) or central venous pressure (CVP) and observe the right heart filling. Try not to overfill the heart even if the systemic blood pressure appears low as overdistension impairs ventricular contractility.
- Check the cardiac output (CO) when (1) off CPB, (2) finished transfusing through the aortic cannula, (3) atrial cannula removed, and (4) heart in neutral position. The measured CO and calculated systemic vascular resistance (SVR) give further information on whether to manipulate afterload or contractility if the blood pressure is low and the filling pressures adequate.
- Most patients can tolerate a relatively low BP (SBP 60-80 mm Hg) for 3-5 minutes and often it is a short time before the patient's heart begins to function well without inotropes.
- If the patient is doing poorly remember the simplest and fastest solution is to go back on CPB and consider the options (Table 6.1). Try to differentiate whether the problem is technical (graft, valve, or rhythm) or myocardial dysfunction (either right ventricular, left ventricular or biventricular).
- Other options if fail to wean:
  - further reperfusion on CPB
  - further hemodynamic manipulation with inotropes, vasopressors, afterload reducers
  - improve preload, pacing
  - IABP
  - further surgical manipulation (redoing graft, replace rather then repair valve)
  - further investigations: lab (HCT, K^+, HCO_3^-), transesophageal echocardiogram (TEE)
  - nitric oxide to selectively reduce pulmonary vascular resistance (PVR)
  - left ventricular assist device (LVAD)

Table 6.1. Management options for weaning from CPB

Right ventricular failure	Left ventricular failure	Bi-ventricular failure
milrinone	dopamine	dopamine
norepinephrine ± NTG	epinephrine	epinephrine
nitric oxide	dobutamine	dobutamine
isoproterenol	milrinone ± norepinephrine	milrinone
PGE_1 (Alprostadil)	IABP	norepinephrine
nifedipine sublingual		IABP
avoid N_2O, acidosis, low PO_2		

FACTORS FOR DIFFICULTY IN WEAN FROM CPB

- duration of CPB (> 120 minutes)
- incomplete repair (graft failure, prosthetic valve, native repair)
- inadequate myocardial protection (left ventricular distention, inadequate cooling, inadequate cardioplegia, ventriculotomy, prolonged ventricular fibrillation, air or debris in coronaries, surgical trauma)
- reperfusion injury
- preload inadequate or overload (ventricular distention)

CONSEQUENCES OF PROLONGED WEAN

- myocardial distention and damage
- prolong pump time
- potential systemic hypotension and organ damage
- need for multiple inotropes
- use of mechanical assistance
- increase potential need for blood products

THEORY AND PRACTICE

RECOVERY PATTERNS AND POSTOPERATIVE INOTROPIC SUPPORT

Myocardial dysfunction after coronary artery bypass graft (CABG) surgery is a common problem with predictable recovery patterns during the first 24 h postoperation.

Myocardial function initially improves during the first few minutes post-CPB, but then begins to deteriorate until 4-6 h after surgery, when gradual improvement in function begins and complete recovery occurs within 24 h. Poor preoperative myocardial function may require a longer recovery time (Fig. 6.1).

Analysis by logistic regression of various intraoperative and preoperative factors associated with the use of inotropes are: low ejection fraction, prolonged CPB, older age, prolonged aortic cross-clamp, cardiac enlargement, and female sex.

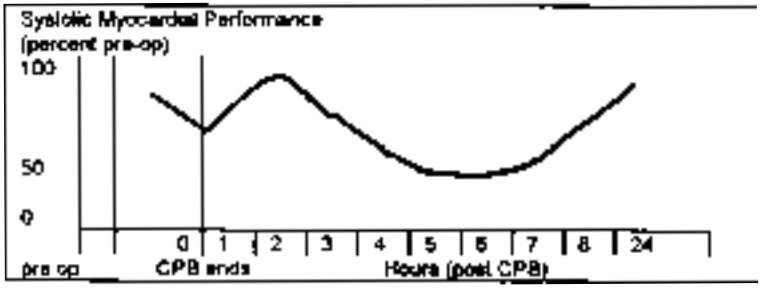

Fig. 6.1. Myocardial recovery post-CPB.

Prophylactic Use of Inotropes

In everyday clinical practice, hemodynamic data available during separation from CPB is limited, so filling pressures and visual evaluation of contractility are to be relied upon to guide therapy. Despite pre-existing myocardial dysfunction, some patients will not require support to separate from CPB. It is uncertain at present whether prolonged inotropic stimulation of the stunned myocardium has deleterious short term or long term effects on humans.

The short term need for inotropic drug support appears to be an indicator of postoperative ventricular dysfunction only and the use of inotropes per se does not influence long-term outcome in the surgical population.

Choice of Inotropic Drug

It depends on pathophysiology of the patient's pre-existing heart disease and reperfusion injury.

Patients in congestive heart failure (CHF) have ventricular dilatation which causes an increase in ventricular wall mass and results in higher energy demands. This may require increased coronary blood flow and cause a decrease in blood pressure which results in higher circulating catecholamines and activation of the renin/angiotensin system. This in turn causes:

- depletion of myocardial norepinephrine stores so that indirect acting agents are less effective (dopamine, dopexamine)
- decrease β1 receptor density (β2 receptors not affected) so the that ratio β1: β2 changes from the normal β1: β2 (80:20) to β1: β2 (60:40) in the failing heart
- β1 desensitization (down-regulation) so that β1 receptor agonists used alone may have a plateau effect

Reperfusion injury results in "stunned myocardium" that can be defined as failure of myocardial function in normal or ischemic areas to return to baseline after periods of hypoxic injury or reperfusion. This may be due to:

- reduced high energy phosphate levels
- intracellular calcium overload
- superoxide radicals
- abnormalities of micro-circulatory flow

Inotropes will not reverse actively ischemic dysfunctional myocardium. Stunned myocardium will respond to inotropes indicating that adequate ATP stores can be recruited to restore proper ventricular function.

Ideal Inotrope

The following are desired characteristics of the ideal inotrope.

- not cause tachycardia or arrhythmias
- avoid raising diastolic filling pressures
- maintain diastolic coronary perfusion pressure
- increase O_2 delivery
- increase myocardial blood flow

- rapid onset and termination
- devoid of myocardial toxicity

There is *no* ideal inotrope at present.

Classes of Inotropic Drugs

- cAMP dependent: adrenergic, dopaminergic, PDE inhibitors, thyroid hormone, glucagon
- cAMP independent: digoxin, calcium, α adrenergic

INDIVIDUAL AGENTS

Calcium

In vivo studies show calcium chloride ($CaCl_2$) significantly increases MAP and blood ionized calcium concentration, but have no effect on cardiac index (CI).

At Toronto General Hospital, 1 g $CaCl_2$ is given by the perfusionist into the pump when separating from CPB. Patients who have hypertrophic myocardium, high MAP or a short CPB run $CaCl_2$ may be omitted. Intraoperative ionized calcium levels are not done.

Adrenergic Agonists

Typically act on β1 (increase HR and inotrope), β2 (relax smooth muscle), α1 (postsynaptic vasoconstrict), α2 (presynaptic vasoconstrictive) receptors. Adrenergic catecholamine receptor activity is summarized in Table 6.2.

Most clinical studies have shown minimal hemodynamic differences between dopamine and dobutamine in surgical patients. Dopexamine offers little advantage over dobutamine. Epinephrine produced greater increase in stroke volume comparable to dobutamine without as great an increase in HR. Little evidence that β adrenergic receptor antagonists (esmolol) can be administered to selectively inhibit tachycardia without also inhibiting the ability of an adrenergic agonist to increase stroke volume.

At Toronto General Hospital dopamine is frequently run as a background infusion (1-5 μg/kg/min) during CPB (a) to maintain MAP with propofol infusion, (b) to protect renal function, (c) to facilitate weaning from CPB. Low dose epinephrine is a reasonable second choice, though consider patients who develop high dose requirements for early mechanical support (IABP) or additional

Table 6.2. Adrenergic catecholamine receptor activity

Agent	α 1	α 2	β 1	β 2	dopamine
Dopamine	++	+	+	+	+++
Dobutamine	∅	∅	+++	++	∅
Epinephrine	+++	+++	++	++	∅
Norepinephrine	+++	+++	+	∅	∅

pharmacologic therapy (milrinone/norepinephrine). Remember adrenaline also squeezes the venous system so filling pressures may increase and require the addition of a small venodilating dose of nitroglycerin.

Phoshpodiesterase (PDE) Inhibitors

PDE inhibitors are noncatecholamine, nonadrenergic inotropes that do not act on β1 adrenergic receptor, but inhibit intracellular PDE III which results in an increase in intracellular cAMP.

Advantages

- increase contractility and CO
- decrease PCWP, SVR, PVR (require higher doses to decrease PVR)
- no change in HR, MvO_2, MAP
- proarrhythmogenicity is uncommon
- potentiate action of β1 agonists
- prevent myocardial ischemia by: dilate arterial conduits and inhibit thrombus formation (platelets), perfusion injury due to white blood cell derived oxygen free radicals

Disadvantages

- systemic vasodilation so often need to add a vasopressor

Amrinone: loading dose 1.5 mg/kg followed by infusion of 10 μg/kg/min. Amrinone appears to be as effective as epinephrine, but more prone to produce vasodilatation. Amrinone may be especially useful when used in combination with β adrenergic receptor agonists for patients with left or right ventricular dysfunction.

Milrinone: European Multi-center Trial Group evaluated efficacy post-CPB. Showed improved cardiac outputs at doses of 50 μg/kg i.v. bolus and infusion 0.50 μg/kg/min ($T_{1/2}$ 50 minutes). Higher doses further reduced BP and were associated with more arrhythmias.

Enoximone (imidazolam): Third generation is under trial in the U.S. and not yet available in Canada.

At Toronto General Hospital, milrinone has gained favor as the first line drug of choice for patients with right ventricular failure or high pulmonary artery pressures. A loading dose of 50 μg/kg is given into the pump prior to separation and if necessary, an infusion is started in CVICU. Low dose norepinephrine is the vasopressor of choice to maintain an adequate MAP.

Thyroid hormone

In euthyroid, hypothermic patients measured free T3 increases with heparin and falls to lower limit of normal on CPB, then continues to fall for up to 24 h postoperatively. Thyroid hormone in animals has been shown to increase myocardial contractility by consuming less energy substrate than normal. Dose of T_3 0.8 μg/kg i.v. at release of aortic cross-clamp, followed by 0.8 μg/kg over 6 h, then taper.

At Toronto General Hospital, despite measured low serum T3 levels on CPB and the benefit of T3 on myocardial contractility, thyroid hormone has not been used in patients who are warm, euthyroid and unable to wean from CPB.

Glucose Insulin Potassium (GIK)

Dose of D50W 1 g/kg + Insulin 1.5 units/kg + KCl 10 mmol.

GIK has positive inotropic effects though the cellular mechanisms are not clear. It has been advocated for management of low cardiac output in septic shock; not proven effective in weaning patients from CPB.

Steroids

Steroids have been briefly advocated in management of septic shock, but lost favor when failed to document any improved outcome in two multi-center trials. It has been claimed to decrease lung dysfunction post CPB, but actually increases the time to extubation.

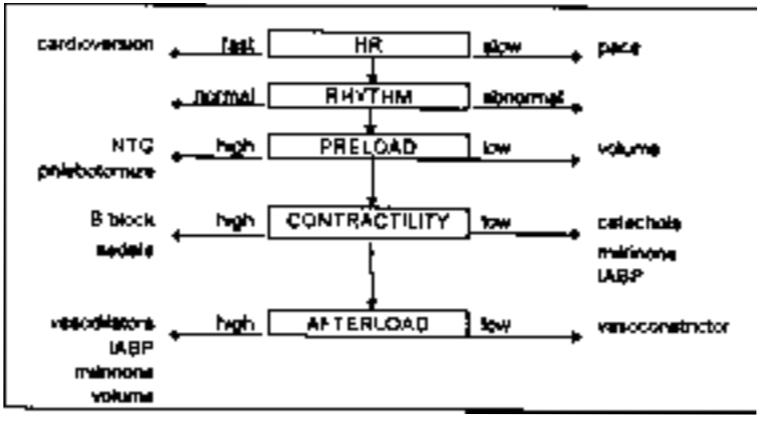

Fig. 6.2. Weaning from CPB: parameters and management.

Table 6.3. Summary of hemodynamic effects and doses of drugs

	CO	SVR	BP	PVR	CVP	HR	PCWP	Infusion in D5W	rate μg/kg/min
dopamine	↑	↑	↑	↔	↑	↑	↑	200 mg/250 ml	3-20
dobutamine	↑	↓	↑	↓	↓	↑/↓	↓	250 mg/250 ml	3-20
milrinone	↑	↓	↔	↓	↓	Ø/↑	↓	20mg/100ml	0.375-0.75
epinephrine	↑	↑	↑	↑	↑	↑	↑	4 mg/250 ml	0.01-0.03
calcium	↑/Ø	↑	↑	Ø/↑	Ø	Ø	Ø	1 gm bolus	———
norepinephrine	↑	↑	↑	↑	↑	↔	↑	4 mg/250 ml	0.01-0.03
phenylephrine	↓	↑	↑	↑	↑	↓	↑	40-80 ug bolus	0.3-0.7
nitroglycerin	↔/↑	↔/↑	↓	↔/↓	↓	↔/↑	↓	100 mg/250 ml	to effect
nitroprusside	≠	Ø	Ø	Ø	Ø	≠	Ø	50 mg/250 ml	to effect

SUMMARY

The key to successful weaning from CPB is communication, a little patience, checking and rechecking the parameters summarized in Figure 6.2 and choosing the best drug as summarized in Table 6.3.

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Blood Conservation and Transfusion Requirement

Jacek M. Karski

INTRODUCTION

Depending on surgical technique, length of operation and preexisting diseases, usually a small proportion of patients bleed excessively and require re-exploration of the mediastinum after elective cardiac surgery. Roughly one-half of these patients have coagulopathies and the other half have surgically correctable bleeding.

POST-CPB COAGULOPATHY

Postoperative coagulopathy results from an activation of fibrinolysis and platelet dysfunction. Fibrinolysis is due to thrombin generation, fibrin production and activation of factor XII. Platelet dysfunction is due to fibrinolysis and plasmin formation with redistribution of platelet receptors.

Although there is no consensus as to the nature of damage inflicted on platelets by CPB the end point remains the same: platelet damage causes excessive blood loss after surgery performed under CPB.

PREDICTORS OF POSTOPERATIVE BLEEDING

Predictors of patients with excessive bleeding (> 750 ml in 6 h) are indicated in Table 7.1. Preoperative coagulation results do not predict patients at high risk for bleeding.

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Table 7.1 Predictors of excessive bleeding

Septic patients
Preop liver dysfuntion; CHF
Acute aortic dissection
Complicated re-operations for congenital surgery
Coagulopathies (pre-operative)
Prolonged CPB time (over 120 min.)
Renal failure (?)
Aspirin therapy (?)

Abnormal postoperative coagulation results confirm ongoing coagulopathy from fibrinolysis and platelet dysfunction. These changes in the coagulation system start immediately following sternotomy and persist for up to 24 h after surgery.

Coagulopathy can occur in patients with routine operations and normal preoperative coagulation profiles.

PREVENTION OF POSTOPERATIVE BLOOD LOSS

There are mechanical and pharmacological ways to reduce blood loss after cardiac surgery. Some methods are proven effective while others are of questionable value.

Methods of Mechanical Blood Loss Reduction

- Suctioning of blood back to CPB circuit from the operating field which requires early heparinization.
- Retransfusion of all blood from CPB after surgery.
- Autotransfusion after cardiac surgery. The effectiveness of autotransfusion in reducing the need for blood transfusion is well documented.
- Presurgical blood donation with or without erythropoietin is effective. There is limited application in cardiac surgery because of the urgency of the surgery, degree of illness, and cost of autologous predonation program.
- Use of cell saver during pre CPB period is unpopular due to expense.
- Hemodilution with whole blood collection and reinfusion after CPB.
- Platelet plasmapheresis followed by reinfusion of autologous platelet concentrate.

Pharmacological Methods

- Pretreatment with antifibrinolytic agents before surgery.
- Use of antifibrinolytics after surgery.
- Use of desmopressin in patients with suspected platelet dysfunction.
- Increase hemoglobin level preoperatively by giving erythropoietin.

ANTIFIBRINOLYTICS

Antifibrinolytic drugs reduce blood loss after cardiac surgery when given to the patient *before* surgery. Less defined is the role of antifibrinolytics in the treatment of postoperative bleeding.

Synthetic antifibrinolytic agents (ε-aminocaproic acid and tranexamic acid) bind to lysine sites on plasminogen. They prevent the interaction of plasminogen with fibrin and inhibit its activation to the active protease form, plasmin. They also bind to plasmin and prevent its binding to fibrin and hence its destruction. Tranexamic acid (TA) blocks plasmin-induced platelet activation during CPB, thus preserving platelet function and promoting hemostasis after surgery.

Aprotinin, a serine proteinase inhibitor, inhibits plasmin, complement activation, plasma and tissue kallikreins. Laboratory studies and in vitro experiments show that aprotinin can protect platelet function during CPB. Preservation of platelet receptors in patients receiving aprotinin correlates to a reduction of postoperative bleeding.

Aprotinin (Trasylol)

Aprotinin is a single chain polypeptide isolated from cow lung which expresses activity in kallikrein inhibitor units (KIU). It is a potent antifibrinolytic that inhibits kallikrein and also modifies complement activation.

A number of clinical studies prove the effectiveness of giving aprotinin before and during CPB in reducing blood loss (by 36%) and blood transfusion requirements (by 50%).

The high dose of aprotinin used in these studies was calculated on the basis of the need for complete inhibition of kallikrein in vivo, at a blood level of 200 KIU/ml. The effectiveness of a low dose aprotinin infusion (half-dose regime) has not been proven to reduce bleeding or decrease the need for postoperative transfusion. A different regime of aprotinin dose based on its pharmacokinetics in cardiac surgical patients is outlined in Table 7.2.

Tranexamic Acid (Cyklokapron)

Antifibrinolytic effect through:

- formation of a reversible complex with a modified plasminogen
- competitively inhibits the activation of enterokinase and non-competitively inhibits its proteolytic activity
- weak effect on thrombin
- even weaker inhibitive effect on plasmin

The elimination half-life of TA is 80 minutes and about 90% of the dose is recovered from the urine after 24 h. Direct comparison of potencies indicates that TA is 6 to 10 times more potent than ε-aminocaproic acid and sustains greater antifibrinolytic activity in rat tissue.

Data from our earlier studies indicate that a single i.v. dose (10 g or 125 mg/kg) of TA given to patients before sternotomy:

Table 7.2. Dose regime of Aprotinin (KIU)

Type of regime	Loading dose	CPB dose	Maintenance	Blood Level
High	2 x 10^6	2 x 10^6	0.5 x 10^6 / h	200 KIU/ml
Half-high	1 x 10^6	1 x 10^6	0.25 x 10^6 / h	50 KIU/ml
Low	2 x 10^6	0	0	n/a
(Levy) high	52,000/min x 30 min	26,000/min x 60 min 500,000 in CPB	10,400 / min	200 KIU/ml
(Levy) low	divide the Levy high dose by a factor of 5			50 KIU/ml

- reduces the number of patients who bleed excessively (> 750 ml in 6 h) from 18-2%
- reduces blood loss in the postoperative period by 50% over the first 6 h and by 35% over the first 24 h following surgery when compared to placebo
- reduces the need for PRBC transfusion when comparing excessively bleeding and non-bleeding patients

We currently recommend 100 mg/kg of TA for patients operated on under moderate to deep hypothermia (< 29°C) and/or high risk patients. Patients with a low risk for bleeding and who are operated under mild hypothermia (> 33°C) will need only 50 mg/kg of TA before surgical incision. We use a single postanesthesia induction dose of TA to prevent fibrinolysis and protect platelets.

ε-aminocaproic acid (Amicar)

Pretreatment reduces blood loss by 30% and blood transfusion requirements in the postoperative period.

Range of doses varies from 5 g given i.v. before surgery followed by a continuous i.v. infusion of 1 g/h for 6 h to a dose of 15 g given as a single bolus before surgery.

In our retrospective comparison of the effectiveness of ε-aminocaproic acid (15 g) to TA (10 g), there was no significant difference in reduction of blood loss. However, TA 10 g was more effective in reducing the occurrence of excessive bleeders and in reducing the blood transfusion requirement.

Comparison of Effectiveness of Aprotinin vs. Tranexamic Acid

Direct comparison of the effectiveness of aprotinin vs. TA suggests that either drug given preoperatively reduces blood loss to the same extent in low risk patients.

Meta-analysis does not show any significant differences in the reduction of blood loss between patients pretreated with either aprotinin or synthetic antifibrinolytic. Patients receiving aprotinin had a significantly reduced postoperative blood transfusion requirement when compared to placebo.

There has been no study to date that compares the effectiveness of these drugs on blood loss and transfusion requirements in high risk patients.

Advantage of aprotinin vs. tranexamic acid:

- possibly more effective in the reduction of blood loss and transfusion requirements in high risk patients

Disadvantages of aprotinin vs. tranexamic acid:

- cost (5-8 times) greater
- adverse effect on renal function (1%)
- allergic reaction with first time use (0.6%)
- increased risk of allergic reaction with repeated use
- affects ACT readings

Antifibrinolytics and Risk of Thrombosis

There is a possible risk of an increased thrombotic tendency during treatment with any fibrinolytic inhibitor. To date there is no published report of thrombotic complications in cardiac patients receiving antifibrinolytics perioperatively.

Recent studies addressed the issue of early graft occlusion in patients receiving preoperative aprotinin therapy and showed no difference in graft patency among treatment groups. There has been no comparable study performed with respect to graft patency following synthetic antifibrinolytics.

Desmopressin (DDAVP)

It increases the level of the von Willebrand factor and improves platelet function. In only 4 of 13 trials, there was a significant reduction in blood loss when DDAVP was given to prevent postoperative bleeding in cardiac surgery. The role of DDAVP in reducing blood loss during cardiac surgery is limited to its postoperative use in bleeding patients with uremia or preoperative ASA therapy.

BLOOD PRODUCT ADMINISTRATION

Transfuse blood and blood product only with a strict medical indication. The prophylactic use of blood products is not indicated in modern cardiac surgery. Our algorithm for management of microvascular bleeding is presented in Appendix 8.

Packed red blood cells (PRBC) are given only if hematocrit is 18% or less during CPB or less than 20% on two consecutive measurements while in CVICU following surgery.

Treatment of Bleeding

- Platelets (5 units) transfused if the platelet count < 50 x 10^9L^{-1}
- Fresh frozen plasma (2 units) transfused if INR > 1.5 times the normal value.
- Cryoprecipitate 5 units given if fibrinogen is below 2.0 g/L.
- Protamine sulphate (50-100 mg) diluted in 50 ml of normal saline and infused over 30 minutes is given in CVICU if the ACT exceeds the baseline value by more than 10%.
- Patients who bleed more than 200 ml per hour for 2 consecutive hours or more than 400 ml in 1 h are treated with repeated primary dose of TA given over 30 minutes.
- Desmopressin (16-20 μg) diluted in 50 ml of normal saline and infused intravenously over 30 minutes are given to bleeding patients with renal failure and patients on ASA.

SUMMARY

The combination of both mechanical and pharmacological means of reducing blood loss and transfusion requirements in cardiac surgery seems to be the most logical way of preventing the need for blood transfusion. Table 7.3 summarizes the recommendation to minimize the risk of blood and blood product transfusion in cardiac surgery.

Table 7.3. Blood transfusion prevention in cardiac surgery

- Patient hemoglobin preoperatively > 120 g/L (iron supplement)
- TA 100 mg/kg i.v. before sternotomy for high risk patients
- TA 50 mg/kg i.v. for all routine patients
- Reinfusion of pump blood at the end of the procedure
- Autotransfusion of shed mediastinal blood up to 6 h postoperatively
- TA (repeat primary dose over 30 minutes) for patients bleeding > 400 ml in the first hour or 200 ml for 2 consecutive hours
- Desmopressin 16-20 μg i.v. for bleeding patients with renal failure or recent ASA intake
- Strict adherence to blood and blood product transfusion protocol

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Anesthesia for Special Cardiac Surgical Procedures

Annette Vegas

ANESTHESIA FOR MINIMALLY INVASIVE CORONARY ARTERY BYPASS

Introduction

Represents a recent trend in cardiac surgery, with two major advantages:

- Avoid problems of cardiopulmonary bypass (CPB) including problems with hemostasis, electrolytes, fluid overload, stroke, neuropsychiatric disturbances.
- Favorable economics as ideal for early extubation, may decrease length of hospitalization and may permit early return to work.

Universal term to describe a variety of procedures and approaches which may or may not involve CPB:

- A series of small holes or ports in the chest and CPB using the femoral vessels. Referred to as port access coronary artery bypass or PACAB or portCAB.
- A combination of ports and small incision directly over the coronary artery to be bypassed without using CPB. Referred to minimally invasive direct coronary artery bypass (MIDCAB), minimally invasive surgery (MIS), keyhole or left anterior small thoracotomy (LAST).
- Harvest of vascular conduits may include the saphenous vein or left internal thoracic artery (LITA) using thoracoscopy or multilevel dissection.

These procedures present unique challenges to both the surgeon and anesthesiologist, combining aspects of cardiac and thoracic surgery.

Anesthesia

Goals

- safe continuous general anesthetic
- short reversible anesthetic

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- maintain hemodynamic stability (adequate systemic and myocardial perfusion pressure)
- optimal surgical field (one lung down, slow heart)
- monitor for myocardial ischemia particularly during ischemia preconditioning

Preparation

Patient should have a complete anesthetic assessment as per routine CABG procedure, frequently in an outpatient clinic to facilitate same day admission. Continuation of medications to the time of surgery should occur and a sedative premedication ordered as per institutional preference (Lorazepam 1-2 mg sublingual). Room and monitor setup for full CPB as if through median sternotomy, major differences are:

- Plan for reversible anesthetic so tend to use shorter acting agents (muscle relaxant) and less narcotic. Some centers use a thoracic epidural for intraoperative anesthesia and postoperative analgesia and permit extubation in the operating room.
- CPB machine setup dry outside of the operating room to avoid patient contamination.
- Usual cardiac resuscitation drugs including vasodilators, pressors and inotropes. In addition have drugs available to induce bradycardia during coronary anastomosis.
- Ability to monitor and treat induced myocardial ischemia.
- Cell saver for bleeding.

Monitoring consists of standard CABG monitors with these additional considerations:

- Additional invasive monitors include an arterial line, central venous pressure (CVP) or Swan-Ganz to measure cardiac outputs (CO) and pulmonary artery (PA) pressures and transesophageal echocardiography (TEE) to monitor for myocardial ischemia.
- It is important to maintain normothermia by increasing room temperature, using warm prep solution, and actively warming the warm patient.
- ECG monitoring with V2 and V5 (surgeon places lead anteriorly in second intercostal space in the midclavicular line) is important during ischemia preconditioning. Obtain pre-incision ECG tracings for comparison against any changes during ischemia preconditioning or post coronary anastomosis.

Induction

Induction consists of a modified early extubation protocol with less narcotic and shorter acting muscle relaxant. Fentanyl 5-10 μg/kg, midazolam 1-2 mg, rocuronium 50 mg, ± sodium thiopental 1-2 mg/kg. Consider avoiding pancuronium with associated tachycardia.

- May use combined general and regional anesthesia technique.
- Ventilation may require one lung (double lumen tube, bronchial blocker or single lumen endotracheal tube (ETT)) depending on approach to the heart and LITA harvest.

Maintenance

- Any combination of inhalational agents, narcotics, propofol and muscle relaxation that allows manipulation of hemodynamic and possibly extubation in the operating room
- Heparin 5000 units i.v., no antifibrinolytic medication required unless on full CPB
- Prophylactic nitroglycerin drip not used routinely here
- Resuscitation drugs: phenylephrine, calcium, dopamine
- Drugs to slow the heart: beta blockers (esmolol, metoprolol), diltiazem, adenosine as summarized in Table 8.1

Surgical Procedure

Detailed description in chapter 25.

Hypotension usually occurs when pericardial sutures are placed for exposure and responds to volume or pressors. The heart may require ischemic preconditioning to tolerate subsequent safe occlusion of the vessel during anastomosis. Myocardial ischemia should be monitored for:

- ST segment elevation
- increase pulmonary artery pressures
- V wave in PA trace
- decrease CO
- wall motion abnormalities on TEE

There is a need for bradycardia during coronary anastomosis, aiming for less than 50 beats per minute. It is preferable to preoperative β blockade as it eliminates intraoperative maneuvers, decreases cost, increases ease, decreases frustration and increases safety. There is unpredictable drug response with the agents used in Table 8.1.

Cardiopulmonary Bypass

A perfusionist and CPB machine should be readily available. At TTH, we assemble a dry pump and keep it outside the room to avoid equipment contamination. There is usually sufficient time to prime the pump, give full dose heparin and antifibrinolytic for CPB if necessary.

Postoperative Management

- By avoiding CPB there is frequently:
  - no coagulopathy and less chest tube losses
  - less fluid and electrolyte imbalance
  - accurate and low postoperative fluid balance, without need for diuresis
  - no need for K^+ replacement
- Early extubation in operating room or post cardiac surgery unit.
- Monitor for ischemia with ECG and enzymes.
- Pacemaker wires may not be inserted so avoid postoperative bradycardia.

Table 8.1. Drugs to slow heart rate

Drug	Bolus Dose	Infusion
Esmolol (Brevibloc)	5-20 mg	2.5 g/250D5W titrate to effect
Metoprolol	1-5 mg	————
Diltiazem (Cardiazem)	5-10 mg	15 mg/h
Adenosine (Adenocard)	6-12 mg	————
Neostigmine	2.5-5.0 mg	————

- Chest tube removed the following day
- Postoperative chest x-ray to ensure the lung is re-expanded
- LITA spasm treated with nifedipine 10-20 mg sublingual or intranasal q4h, nitroglycerin infusion
- Pain is often more than standard CABG therefore, patient may require supplemental analgesia through intercostal blocks and/or patient controlled analgesia (PCA).

ANESTHESIA FOR PARTIAL LEFT VENTRICULECTOMY (PLV)

End Stage Heart Failure

Clinical syndrome defined by the inability of the heart to meet metabolic needs of the body. Manifested by signs and symptoms of both systolic (forward) and diastolic (backward) flow. Common etiology:

- Ischemic (60-70%), valvular, HBP, idiopathic cardiomyopathy (20%).
- Incidence: 1.0% of US population (2 million), 400,00 patients (20%) died, 40,000 (2%) heart transplant candidates, 2,300 (0.11%) receive heart transplants.
  Goal of treatment is to improve symptoms, reduce complications and improve survival.

Medical management includes:

- nonpharmacological: salt restriction (2-3 g/day), regular exercise, limit alcohol (< 2 ounces/day)
- pharmacological: diuretics, vasodilators, ACE inhibitors, beta blockers, anticoagulation

Surgical options include:

- cardiomyoplasty: poor long term results, ventricular tachycardia
- artificial heart: rarely used
- mechanical assist devices: frequently used bridge to transplantation
- PLV
- heart transplantation: limited resource due to availability of donors

Anesthesia

Goals

- safe continuous general anesthetic
- maintain hemodynamic stability (adequate systemic and myocardial perfusion pressure)
- full cardiopulmonary bypass
- monitor for myocardial ischemia, heart failure, arrhythmia

Preparation

Patient should have a complete anesthetic assessment as per routine CABG procedure. These are sick patients with poor ventricular function, cardiac cachexia and other end organ damage. They frequently have arrhythmias, decreased intravascular volume and are afterload reduced from medical therapy.

Continuation of medications to the time of surgery should occur. They tolerate preoperative sedation poorly, therefore decrease dose or eliminate entirely.

Room and monitor setup for full CPB as through median sternotomy.

Monitoring consists of standard CABG monitors. Additional invasive monitors include an arterial line, central venous pressure(CVP) or Swan-Ganz to measure cardiac outputs (CO) and pulmonary artery (PA) pressures. Transesophageal echocardiography (TEE) to assess degree of mitral regurgitation and ventricular dimensions.

Technique

Carefully titrated anesthesia induction. Patients are not candidates for early extubation and may benefit from a higher dose narcotic technique. Maintenance of anesthesia should be with a combination of narcotics, benzodiazepines and inhalational agents as tolerated by the patient.

Surgical Procedure

Surgical details of the procedure are found in Chapter 26.

Despite a relatively short CPB run a major challenge is in weaning the patient. This frequently requires a combination of inotropes, pressors and mechanical support including an LVAD.

Avoidance of hypertension and excessive left ventricular stimulation is mandatory to prevent excessive bleeding and potential rupture of the ventricle. Monitor and treat arrhythmias aggressively.

Full reversal of heparin with protamine. May require additional blood products if pre-existing coagulopathy. Tranexamic acid 100 mg/kg i.v. at induction.

Postoperative Management

Initially patients may be sicker than preoperation. They require stabilization of hemodynamics to avoid extremes of hypotension (end organ ischemia) and hypertension (ventricular rupture and bleeding). Ventilate and wean from the ventilator over hours when hemodynamics stable. Patients are prone to arrhythmias, commonly ventricular, that should be aggressively treated. Amiodarone i.v. is our preference. Perioperative complications include renal failure (22%), arrhythmia

(15%), respiratory failure (10%), bleeding (10%) and infection (2%) Patients will need to continue on heart failure treatments prior to hospital discharge. They may require prophylactic treatment for arrhythmias and anticoagulation to prevent clot formation on the ventricular suture line.

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Anesthetic Management of Diseases Affecting Cardiopulmonary Bypass

Ramiro Arellano

DIABETES MELLITUS

The most common endocrinopathy affecting up to 7% of the population. It manifests as elevated serum glucose levels due to relative or absolute insulin deficiency.

Presents in two clinical forms:

- Type I diabetics all require exogenous insulin to control blood glucose levels and avoid diabetic ketoacidosis.
- Type II diabetics are initially treated by diet control and weight loss. With progression of disease patients require oral hypoglycemics and/or insulin.

Multi-system end-organ damage may occur. Preoperative assessment of end-organ damage guides intraoperative therapeutic intervention (Table 9.1).

Note the extent of glucose control (fasting blood glucose levels, hemoglobin A_{1C}) and use of hypoglycemic medications by the patient.

Perioperative glucose control is best managed with a sliding scale infusion of D5W and Regular insulin. Tight control of blood glucose requires frequent monitoring of blood sugar to maintain serum glucose between 10-12 mmol/l.

RENAL INSUFFICIENCY

Patients with renal dysfunction present particular challenges to the anesthesiologist during cardiopulmonary bypass (CPB) surgery. The incidence of perioperative renal dysfunction is 7.7%, whereas oliguria renal failure occurs in 1.4% of CPB patients.

End stage renal disease from various etiologies itself causes multi-system effects including: hypertension, anemia, abnormal coagulation and alterations in intravascular volume, electrolytes and acid-base status.

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Table 9.1. Complications of Diabetes Mellitus

Organs Affected	Preoperative Assessment	Anesthetic implications
Cardiovascular System		
1. Hypertension	Baseline BP. Type and dose of antihypertensive medication	Aim to keep pre- and post-bypass blood pressure within ± 20% of preoperative values
2. Reduced cerebral perfusion due to atherosclerosis	Investigate history of TIA/stroke or carotid bruits with Doppler ultrasound +/- cerebral angiograms	Although of controversial benefit, high risk patients may undergo carotid endarterectomy followed by cardiac surgery
3. Reduced renal perfusion due to atherosclerosis	Serum creatinine level	Avoid nephrotoxic drugs perioperatively (e.g., NSAIDS). Depending on level of renal impairment, may need to avoid or reduce dosage of drugs which are renally excreted. May require hemoconcentration or dialysis on bypass
Nervous System		
Autonomic dysfunction	History of delayed gastric emptying and esophageal reflux	Rapid sequence induction or modified rapid sequence induction with cricoid pressure
Connective Tissue		
"Stiff man syndrome"	Careful airway assessment since conventional laryngoscopy may be difficult in up to 40% of type I diabetics	Plan for possible difficult intubation by having airway adjuncts and trained personnel available at induction

Optimize chronically dialyzed patients by timely preoperative peritoneal or hemodialysis. Take care to protect patients not yet on dialysis from further deterioration in perioperative renal function. Consider intraoperative and postoperative dialysis for refractory management of volume and electrolyte disturbances.

Preoperative Assessment

- etiology and duration of renal dysfunction, type and frequency of dialysis
- examination for dry weight, baseline blood pressure, dialysis line or fistula
- review of laboratory data: hemoglobin, platelet count, BUN, creatinine, calcium, magnesium, phosphate, prothrombin time and partial thromboplastin time
- review of medications
- consider use of erythropoietin therapy in anemic patients
- consult nephrologist for perioperative management of diuretics, dialysis and immunosuppressive medication in renal transplant patients

Perioperative Management

- Continue usual medications to time of surgery.
- Patients treated by hemodialysis should receive hemodialysis the day before surgery to optimize fluid, electrolyte and metabolic status and improve uremic platelet dysfunction.
- For elective cases, if renal function deteriorates following cardiac catheterization postpone surgery until recovery occurs.

Intraoperative Management

- During the pre- and postbypass phase, monitor volume status and cardiac output with pulmonary artery catheter or transesophageal echocardiography. Adjust volume status to maximize cardiac output and perfusion of vital organs.
- Aim to keep pre- and postbypass systemic blood pressure within ± 15% of preoperative values. Keep MAP 60-70 mm Hg during CPB.
- Modify dose of drugs to account for renal metabolism or excretion and altered volume of distribution or protein binding.
- Reduce risk of acute renal failure by increasing renal blood flow and urine output (especially if oliguria exists) using:
  - mannitol
  - low dose dopamine (3-5 μg/kg/min)
  - furosemide
  - prostaglandin E_1
- Avoid high K^+ cardioplegia solutions.

Postoperative Management

- Treat hyperkalemia with calcium chloride ($CaCl_2$), insulin and dextrose, sodium bicarbonate ($NaHCO_3$), diuretics or dialysis.
- Avoid nephrotoxic drugs (e.g., NSAIDS) and contrast dyes perioperatively.
- Transfuse RBCs to maintain Hb ≥ 80 g/dl (preferentially use RBCs ≤ 7 days old).
- Consider use of desmopressin acetate (DDAVP) after CPB to improve uremic platelet dysfunction.
- Transfuse platelets and FFP guided by clinical condition of patient and laboratory results.

SICKLE CELL TRAIT AND DISEASE

Sickle cell hemoglobinopathy is a recessive abnormality which produces hemoglobin S. Hemoglobin S accounts for 20-45% of the total hemoglobin in patients with sickle cell trait (heterozygous form) and for 80-98% of the total hemoglobin of patients with sickle cell disease (homozygous form).

Patients with sickle cell trait have few clinical symptoms and rarely experience sickle cell crises. Patients with the homozygous defect experience sickle cell crises from vaso-occlusive episodes that occur when sickled RBCs aggregate resulting in:

- severe hemolytic anemia
- organ ischemia/infarction affecting the heart, lungs, brain, kidneys, spleen and bone

Patients with sickle cell trait or disease are at high risk of potentially fatal thromboses during CPB surgery. Since all RBCs contain some hemoglobin S they can all sickle if sufficiently severe conditions present.

The deoxyhemoglobin state of hemoglobin S leads to RBC sickling. The tendency to sickling is exacerbated by:

- hypoxemia
- acidosis
- increased concentrations of 2,3-DPG
- infection
- hypothermia
- capillary stagnation

Perioperative management is designed to prevent sickling which causes hemolysis or vaso-occlusive phenomena (Table 9.2).

- Screen all at risk patients for hemoglobin S preoperatively as sickle cell trait may be completely asymptomatic. Patients with a positive screen should have a hemoglobin electrophoresis to determine the percentage of each type of hemoglobin present.
- Consult a hematologist preoperatively for sickle cell trait and sickle cell disease patients.
- Completely assess: cardiovascular system (evidence of myocardial ischemia, pulmonary hypertension, CHF), renal function, anemia, analgesic use (tolerance to narcotics), hydration state.

CRYOPROTEINS

Cold agglutinins are serum antibodies that are activated by decreased blood temperature to produce RBC agglutination or hemolysis. Hemolysis results from compliment activation.

Table 9.2. Management of patients at risk for sickle cell disease

- Reduce risk of perioperative sickle cell crisis by preoperative partial exchange transfusion with Hb-A donor blood to dilute Hb-S RBCs
- The ideal ratio of Hb-S to Hb-A has not been established. However, recommendations range from reducing the proportion of Hb-S containing RBCs from less than 5% to less than 33%
- Avoid arterial or venous hypoxemia, acidosis, dehydration, hypothermia and hyperosmolarity
- Treat hypotension with volume administration and avoid vasopressors if possible

If unrecognized, RBC agglutination during hypothermic bypass may lead to multiorgan ischemia from prolonged vascular occlusion.

Cold agglutinins may be caused by:

- Monoclonal antibodies associated with lymphoreticular neoplasms which are generally irreversible.
- Polyclonal variants associated with acute infectious disease (e.g., mycoplasma, infectious mononucleosis, cytomegalovirus) which may remit spontaneously in weeks.

Preoperatively screen the blood of all patients undergoing hypothermic cardiopulmonary bypass for cold agglutinins. A positive screen at 4°C requires further determination of the thermal amplitude and antibody titer. The thermal amplitude defines the temperature below which the antibodies become active. Activation of antibodies increases exponentially as temperature decreases below the thermal amplitude. The higher the titer of cold agglutinins the greater the clinical significance. Consult the hematology service preoperatively if cold agglutinins are identified in routine preoperative screening of patients' blood.

Evidence of agglutination during hypothermic bypass includes:

- agglutination within vessels in the surgical field
- agglutination in the blood cardioplegia reservoir as blood from the patient is mixed with cold cardioplegia solution
- hemolysis with hemoglobinuria
- agglutination of blood in a syringe during phlebotomy

If cold agglutination occurs, it may or may not be reversed by rewarming the patient to temperatures above the thermal amplitude.

Perioperative management involves prevention of complement activation and resultant problems of agglutination and hemolysis:

- If cold agglutinins have been caused by an acute infection, postpone elective hypothermic cardiac surgery for several weeks until antibody has cleared.
- Use warm cardioplegia for myocardial protection and maintain normothermic or mildly hypothermic systemic temperatures to ensure that blood temperature remains above the thermal amplitude.
- If cold cardioplegia is used, wash out blood from the coronary circulation with warm cardioplegia at the onset of bypass and rewarm the heart with warm cardioplegia before myocardial reperfusion.
- Low systemic flows during bypass may reduce noncoronary collateral flow and subsequent cooling of this blood.
- Vent the left ventricle to avoid cooling and stagnation of blood.
- Use crystalloid cardioplegia to avoid agglutination of the cells in the solution when delivered at low temperature.
- Use a septal temperature probe to monitor myocardial temperature.
- Warm cold intravenous fluids, blood, and plasma administered during surgery.
- Consider plasmapheresis or total exchange transfusions in patients with high titer, high thermal amplitude cold agglutinins who must undergo hypothermic bypass.

Treatment of suspected cold agglutination during hypothermic bypass:

- Confirm satisfactory ACT (i.e., rule out inadequate anticoagulation).
- Verify with blood bank that cold agglutination is present rather than an unrecognized alloantibody.
- Use crystalloid cardioplegia to dilute the antibody in the coronary circulation.
- Rewarm patient to systemic temperatures greater than 28-30°C.
- Inspect extracorporeal circuit and oxygenator carefully for cell aggregates.

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Malignant Hyperthermia

Jane E. Heggie

INTRODUCTION

Malignant hyperthermia (MH) is a potentially fatal genetic myopathy inherited in an autosomal dominant fashion. It is asymptomatic until triggered by either a non-depolarizing muscle relaxant and/or potent inhalational vapors used in anesthesia. The incidence of an MH reaction varies from 1: 6,000 to 1: 40,000 anesthetics. In the past, a fulminant MH crisis was virtually fatal; however now mortality is less than 5%. The decrease in mortality is due to improved education of patients and operating room personnel, improvements in anaesthetic monitoring and the availability of dantrolene.

MALIGNANT HYPERTHERMIA CRISIS

An MH crisis is the result of massive release of calcium from the sarcoplasmic reticulum into the skeletal muscle cell. This is triggered by the muscle relaxant succinylcholine or the use of potent inhalational vapors before, during or after CPB.

There may be a significant delay between the exposure to the triggering agent and the onset of the crisis. Most cases present intraoperatively, though there are case reports of patients only being diagnosed and treated postoperatively in the intensive care unit (ICU).

MH is a hypermetabolic state that is characterized by the following:

- muscle rigidity
- tachycardia
- rising end tidal CO_2
- hypoxemia
- hyperkalemia
- fever

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- evidence of rhabdomyolysis with elevated CK and myoglobinuria

During cardiac surgery the diagnosis of MH may be difficult as CPB may mask many of the signs. Persistent acidosis and an elevated pCO_2 despite adequate ventilation on CPB should arouse suspicion. Fever is usually a late sign. Myoglobinuria may be difficult to distinguish from CPB related hemolysis.

Once an MH episode is suspected the anesthesiologist or ICU physician should call for assistance and dantrolene.

Prompt treatment is necessary to prevent morbidity:

- Discontinue all volatile inhalational agents. Hyperventilate with 100% oxygen at high gas flows of at least 10 liters per minute. If possible use a clean circuit though do not delay other treatments to change circuits.
- Delegate an assistant to mix dantrolene. Rapidly administer dantrolene, the initial dose as 2-3 mg/kg with supplemental increments up to 10 mg/kg. Use a central venous line as dantrolene may cause venous thrombosis. Absence of a central line should not delay therapy, administer the drug peripherally until central line is obtained. Each bottle of dantrolene contains 20 mg of dantrolene and 3 g of mannitol and reconstitutes with 60 cc of sterile water. Continue to administer dantrolene until signs of MH (e.g., hypercarbia, rigidity, tachycardia and fever) are controlled. Doses in excess of 10 mg/kg have been used. Dantrolene can cause profound weakness and will delay the patient's time to extubation.
- Administer bicarbonate to correct the metabolic acidosis as guided by blood gas analysis.
- Treat hyperkalemia with correction of the acidosis as well as insulin with dextrose, and hyperventilation.
- Arrhythmias usually result from hyperkalemia and acidosis. Treat life-threatening arrhythmias from hyperkalemia with intravenous calcium chloride ($CaCl_2$). If they persist, despite correction of the underlying cause treat with anti-arrhythmics. Use appropriate anti-arrhythmics except for calcium channel blockers that may cause a severe negative inotropic response in the myocardium in the presence of dantrolene.
- Anticipate renal failure secondary to rhabdomyolysis. Maintain a diuresis of greater than 2 ml/kg/hr. This is facilitated by the mannitol in the dantrolene vial and in the CPB prime.
- Although the patient is having a potentially fatal anesthetic reaction, you will still have to maintain an anaesthetic with intravenous techniques which may include propofol, benzodiazepines and narcotics.

POSTCRISIS MANAGEMENT

Manage the patient in an ICU setting for at least another 24-48 h. Recrudescence of an MH crisis can occur:

- Administer boluses of dantrolene 1 mg/kg intravenously every 6 h for 48 h. Use oral dantrolene in extubated patients.

- Perform serial arterial blood gases (ABG), serum electrolytes, CK, urine myoglobin and clotting studies and therapy titrated accordingly. We follow CK q 4-6 hourly and check urine for myoglobin daily.
- The differential diagnosis of MH includes sepsis, thyroid storm and catecholamine secreting tumors. Once admitted to the ICU draw cultures and bloodwork for endocrinopathies. Occasionally patients labeled as having an MH crisis have been found to have a pheochromocytoma.

Counseling of the family through the department of anesthesia or regional MH diagnostic center should occur. Both the Malignant Hyperthermia Association of Canada and in the U.S. provide educational literature in the management of MH patients.

ELECTIVE MANAGEMENT OF MALIGNANT HYPERTHERMIA SUSCEPTIBLE PATIENTS

The most common presentation of an MH susceptible patient is a family history of MH (Table 10.1) rather than a personal history of an MH Crisis. There are several diagnostic test centers for MH in North America and Europe that provide the Caffeine Halothane Contracture test. Most patients have not undergone formal investigation. The absence of an MH crisis in the patient's own past anesthetic exposures does not imply a nonsusceptible state. Some patients have had two or three anesthetics before their first reaction.

All Departments of Anesthesia should have a policy and procedures manual for the management of MH cases.

PREOPERATIVE

Premedication is optional. Preoperative dantrolene is rarely needed and not given at our institution. Select indications may include patients with a previous life-threatening reaction or rhabdomyolysis requiring dialysis. A baseline CK is helpful. Many patients with MH have elevated resting CK and this may be confused as evidence of a crisis or perioperative myocardial infarction.

If a dedicated MH anaesthetic machine is not available then use vapor free tubing, bellows and soda lime. Flush the machine with high gas (oxygen or air)

Table 10.1. Family history and MH susceptibility

Degree of Relative	Chance of MH Susceptibility
1st–Parent, sibling, child	50%
2nd–Aunt, uncle, niece, nephew	25%
3rd–cousin	12.5%

flows for at least 20 minutes before use. The likelihood of a crisis occurring during the use of a trigger free technique is virtually zero. Nevertheless, dantrolene should be immediately available.

INTRAOPERATIVE

Monitor temperature continuously in both the axilla and nasopharynx. Avoid succinylcholine and potent inhalational vapors. Remind the perfusionist to avoid inhalational vapor exposure during CPB. In the past there has been discussion of stress or catecholamine induced MH reactions. Vasopressors cause hypermetabolism in a pig model. We are unaware of an MH crisis in humans that occurred during cardiac surgery where trigger-free techniques have been employed.

POSTOPERATIVE

Vital sign monitoring including temperature is continuously monitored for at least 4 h.

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Combined Cardiac and Thoracic Surgery

Peter Slinger

CONTROVERSIES

There is no complete agreement about the surgical management of patients found to have both cardiac and thoracic surgical lesions.

The One-stage Combined Procedure

- avoids a second anesthetic and surgical incision
- may reduce hospital stay and have economic benefits

The Two-stage Procedure

- may be associated with less blood loss than with pulmonary resection in heparinized patients
- may allow better operative exposure and staging of mediastinal nodes for malignant lung lesions
- may be associated with better long term survival due to the immunologic consequences of CPB

Suggested Management

- Preferred management at this institution is a one-stage combined procedure because of the documented comparable long-term survival with a low incidence of short-term morbidity and mortality.
- Most cardiac operations can be combined with thoracic procedures for either malignant or benign disease.

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PRESENTATION

Patients with combined surgical lesions present in one of three patterns:

1. An asymptomatic lung lesion found during evaluation for cardiac surgery.
2. A patient being investigated for lung pathology is found to have significant cardiac disease.
3. A previously undetected lung lesion is found intraoperatively after sternotomy.

In scenarios 1 or 2, adequate pulmonary assessment can be arranged preoperatively to guide perioperative anesthetic management. In scenario 3, anesthetic management will be more ad hoc. However, most of these "surprise" lesions are benign (granulomas, bullae) and require only simple wedge resection without intraoperative lung isolation or loss of postoperative pulmonary function.

PREOPERATIVE EVALUATION

Assess respiratory function in three related areas: lung mechanics, pulmonary parenchymal function and cardiorespiratory reserve.

Respiratory Function

1. Mechanics

- The most valid test of lung mechanics is spirometry, specifically use of the forced expiratory volume in one second as a percentage of normal ($FEV_{1\%}$) to derive the predicted postoperative (ppo) $FEV_{1\%}$ based on the amount of functioning lung resected. A ppo-FEV_1 < 40% is associated with increased respiratory complications; ppo-FEV_1 < 30% may require prolonged weaning from mechanical ventilation.

2. Cardio-Respiratory Reserve

- The most valid tests: maximal oxygen consumption (VO_2max), exercise tolerance and oxymetry will not be applicable in most cardiac patients.
- Useful indirect tests are the echocardiogram or cardiac catheterization to show left and right ventricular function.

3. Lung Parenchyma

- The diffusing capacity of the lung for carbon monoxide (DLCO) is a noninvasive test performed at the time of spirometry. A ppo-DLCO < 40% increases risk.
- Arterial blood gases PaO_2 < 60 mm Hg, $PaCO_2$ > 45 mm Hg also increases risk.

Chronic Obstructive Pulmonary Disease (COPD)

A large proportion of the adult thoracic surgical population has COPD particularly those with > 20 pack-year history of smoking. Severity of COPD is graded

according to the degree of airflow obstruction: severe = FEV_1 < 35%, moderate = 35-60%, mild = 60-80%. Anesthetic considerations in COPD include:

- Bullae/blebs. The potential exists for rupture during positive pressure ventilation resulting in a pneumothorax or bronchopleural fistula. Consider lung isolation and avoid nitrous oxide.
- Hypercapnia. Moderate or severe COPD patients may be "CO_2-retainers" who will develop an increased respiratory dead space when supplemental oxygen is administered and subsequently retain more CO_2 during spontaneous ventilation. F_iO_2 must be titrated carefully postoperatively keeping the oxyhemoglogin saturation as low as clinically acceptable to aid weaning from mechanical ventilation.
- Bronchodilators must be continued perioperatively.

Lung Cancer

Preanesthetic assessment of patients with lung cancer should consider the "4 Ms":

- Mass effects: obstructive pneumonitis, SVC syndrome, Pancoast syndrome, etc.
- Metabolic effects: Eaton-Lambert syndrome, SIADH, hypercalcemia, etc.
- Metastases: specifically brain, bone, liver, adrenal
- Medications: chemotherapy bleomycin, adriamycin, corticosteroids, etc.

Smoking

- Stopping smoking for < 8 weeks preoperatively has not been shown to benefit cardiac surgical patients.
- Stopping for any period preoperatively decreases the risk of respiratory complications in thoracic surgical patients.
- Patients should be advised to stop smoking.

Chest Radiography and CT Scan

The majority of abnormalities of the tracheobronchial tree which cause problems for lung isolation can be appreciated from assessment of the preoperative chest x-ray and CT scan. These abnormalities will often not be described in a written or verbal report. The anesthesiologist must examine the radiograph prior to induction.

Mediastinoscopy

- Due to the difficulty assessing subcarinal nodes for staging via a sternotomy, all known lung cancer patients should have a mediastinoscopy as the first step of a combined procedure.
- Because of the risk of brachiocephalic artery compression during mediastinoscopy, monitor the circulation in the right arm (e.g., right-sided radial arterial line or pulse oximeter).
- Allow for surgical access to the suprasternal notch after central line placement.

ANESTHETIC TECHNIQUE

To decrease the risk of bronchospasm, intubate COPD patients with reactive airway disease during deep general anesthesia. Consider the possibility of surgical clamping of an in situ PA catheter during lung resection. Placement of a transesophageal echo (TEE) probe with an in situ double-lumen tube can be done safely.

LUNG ISOLATION TECHNIQUES

- Three basic options: double-lumen endobronchial tubes, single-lumen endobronchial tube or bronchial blockers (Fig. 11.1).
- Left-sided double lumen tubes are the optimal choice because they offer continuous access to both mainstem bronchi for suction, fiberoscopy, application of continuous positive airway pressure (CPAP) and they are more stable intraoperatively than bronchial blockers.
- Due to their limited versatility, double lumen tubes are often not practical in cases with abnormal tracheobronchial anatomy.
- In cases with abnormal anatomy or when the need for lung isolation was not foreseen, pass a bronchial blocker intraluminally through an in situ single-lumen tube for one-lung isolation.
- An 8 Fr Fogarty venous embolectomy catheter with a 10 cc balloon is useful as a bronchial blocker. It can be passed together with a 4 mm fiberoptic bronchoscope through a single lumen tube > 7 mm ID.
- Confirm position of all endobronchial tubes and blockers with fiberoptic bronchoscopy.

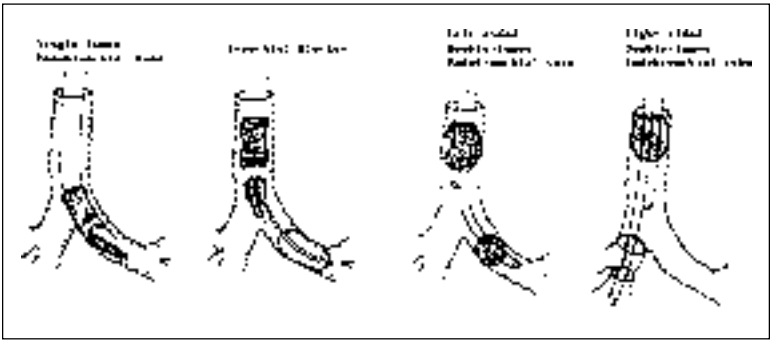

Fig. 11.1. Methods of lung isolation.

Hypoxemia During One-lung Ventilation (OLV)

- Hypoxemia occurs in a minority of patients during OLV. This hypoxemia is largely due to intrapulmonary shunt in the nonventilated lung which may not be adequately compensated by hypoxic pulmonary vasoconstriction.
- Patients more likely to develop hypoxemia during OLV are those with increased alveolar-arterial oxygen gradients during two-lung ventilation, good preoperative spirometry, right-sided surgery and those receiving vasodilators.
- Maneuvers to prevent hypoxemia during OLV are the use of high F_iO_2 and a sustained vital capacity inflation of the ventilated lung to recruit atelectatic areas just prior to OLV.
- Treatment of hypoxemia: CPAP with 2-3 cm H_2O of oxygen after reinflation of the nonventilated lung is the most useful therapy.
- Other treatments of hypoxemia: application of positive end expiratory pressure (PEEP) to the ventilated lung is of benefit in a small and unpredictable minority of patients. Similarly, individual patients may benefit from a larger (14 ml/kg) or smaller (7 ml/kg) than usual (10 ml/kg) tidal volume during OLV.

COMBINED PROCEDURES

Cardiac Valvular Surgery

- Due to the risk of contamination of the operative field from an open bronchus it is recommended to complete the cardiac procedure, wean from CPB and close the pericardium before the pulmonary resection.
- Lung isolation and OLV are necessary for lung resection in these cases.
- A double-lumen endobronchial tube placed at induction is optimal airway management.

Aorto-coronary Bypass (and other cardiac procedures)

- Can be performed during CPB, lung isolation and OLV may not be necessary.
- Optimal to perform the pulmonary resection at the end of CPB after aortic cross-clamp removed.
- For cases where difficult weaning from CPB is anticipated-optimal management is to wean from CPB and stabilize then perform the pulmonary resection. Lung isolation will aid in these cases.
- Since problems weaning from CPB are not always predictable, a double-lumen tube should be used.

Cardiopulmonary Bypass Management

- Due to the increased incidence of phrenic nerve injury and diaphragmatic paralysis associated with topical cooling (slush) of the heart during CPB, topical cooling is not advised during combined cardiac-thoracic procedures.

POSTOPERATIVE ANALGESIA

Thoracic epidural infusion analgesia with a combination of local anesthetic and opioid provides better postoperative pulmonary function in high-risk thoracic surgical patients.

Because of concerns about the risk of epidural hematoma formation with anticoagulation and the generally improved pulmonary function and pain control after sternotomy vs. thoracotomy incisions, epidural analgesia is not widely used for cardiac surgery in North America.

For selected patients having combined cardiac-thoracic procedures with poor respiratory function, placement of an epidural catheter 12-24 h preoperatively is a useful option.

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Anesthesia for Intrathoracic Transplantation

Karen M. McRae

INTRODUCTION

Successful management of the patient undergoing heart, lung or heart-lung transplantation requires planning, skill and collaboration between members of the perioperative team. The greatest variation of practice between centers is in the domain of lung transplantation (single vs. double transplant, the use of CPB).

Anesthesia

Clear communication about the *timing of the induction* of anesthesia of the transplant patient is vital. The predicted cross-clamp time of the donor aorta, the estimated duration of travel of the allograft from the donor site and the difficulty of the removal of the failing organ should be considered in the choice of induction time. Clinically, duration of organ preservation of < 6 h is sought in donor hearts and lungs.

Prior thoracic surgery, adhesions or congenital anomalies may make dissection difficult. The entire team must be present: surgeon, anesthesiologist, perfusionist and nurses, the induction period of these critically ill patients is frequently eventful and may require emergent response. The immunosuppressed state of transplant recipients requires strict adherence to sterile technique during the placement of catheters.

Donor Management

Management of the multiorgan donor may be complicated by the competing concerns for different organs. The desire for increased hydration to maintain urine output via donor kidney may need to be tempered by the need to avoid excessive extravascular volume which will increase neurogenic pulmonary edema in the

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lung. Many patients will exhibit diabetes insipidus after brain death, desmopressin (DDAVP) 4 μg i.v. is routinely required.

Although hypotension and autonomic instability occurs in > 80% of donors, the use of inotrope is tempered by the avoidance of tachycardia. The inotrope of choice is dopamine, preferably at < 10 μg/kg/min.

Methylprednisolone 2 g i.v. (Solu-Cortef) is given to lung donors, which is associated with increased successful lung donor recovery. The lungs are flushed with 50 ml/kg of low K^+ Dextran solution supplemented with prostaglandin E_1, while inflated with 50% F_iO_2, maintained at 4°C.

Donor hearts are flushed with 2 liters of crystalloid cardioplegia.

LUNG TRANSPLANTATION

Etiology of End Stage Lung Failure

Airways disease

Chronic obstructive pulmonary disease (4% at TTH), emphysema (including alpha-1-antitrypsin deficiency, 33%), bronchiolitis obliterans (2%), cystic fibrosis (26%), bronchiectasis (4%). These patients:

- have hyperinflation and may have bronchospasm
- at risk for life-threatening gas trapping with positive pressure ventilation
- often have right heart dysfunction

Pulmonary vascular disease

Primary pulmonary hypertension (6%), pulmonary hypertension secondary to congenital heart disease, in combination with cardiac repair (Eisenmenger's syndrome). These patients have:

- near systemic PA pressures at the time of transplant, poorly responsive PVR
- severe right heart failure
- all patients require CPB
- little tolerance for myocardial depression or increased PVR: anesthetics, hypercarbia, hypoxemia may produce cardiovascular collapse
- may be anticoagulated, requiring reversal prior to transplant

Diseases destructive of pulmonary parenchyma

Idiopathic pulmonary fibrosis, fibrosis secondary to inhalation, radiation, drug injury (all fibrosis 18%), collagen vascular disease, sarcoidosis (1%), lymphangioleiomatosis, eosinophilic granulomatosis. These patients have:

- reduced gas exchange apparatus
- a small chest cavity making surgical technique difficult, surgical manipulation may be poorly tolerated hemodynamically

Preoperative Recipient Patient Management

Patients with copious secretions should have chest physiotherapy (cystic fibrosis) and bronchodilators prior to the OR.

Patients on anti-arrhythmic, systemic anti-hypertensive or pulmonary vasodilator medication should continue to take these medications. Of note, patients on any of the new continuous pulmonary vasodilator therapy such as prostaglandin by i.v. infusion (Flolan) or nitric oxide by mask should be maintained on their therapy until on CPB to avoid catastrophic increases in PA pressures.

Many patients with chronic lung disease are taking H_2 blockers and gastric prepulsant medication for gastrointestinal reflux; these should be continued. Most lung transplant patients have full stomachs.

No routine premedication, judicious sedation in the OR.

Monitor/Lines

Five lead ECG, noninvasive BP cuff, pulse oximeter, naso-pharyngeal temperature probe, urinary catheter.

An arterial line and pulmonary artery catheter. The PA catheter is left in place throughout the procedure whenever possible, prior to clamping and cutting the PA on each side the surgeons should be reminded to palpate the artery for the presence of the Swan-Ganz.

A forced air warming blanket is used.

Anesthetic Technique

A modified rapid sequence induction may be required. Induction with pentothal, narcotic, midazolam, and ketamine have been used successfully. Fentanyl 15-50 μg/kg, midazolam 5-15 mg, pancuronium are typically used.

Patients are ventilated with oxygen and air, never nitrous oxide which will increase PVR and will expand intravascular gas emboli. Inhaled isoflurane may be used initially but is poorly tolerated as patients become more hemodynamically unstable.

To ensure amnesia supplement anesthesia with an infusion of propofol at 50 μg/kg/min, younger patients with cystic fibrosis are particularly at risk of intraoperative awareness.

The Role of Cardiopulmonary Bypass (CPB)

At TTH CPB is used during lung transplantation only as indicated for hemodynamic instability or inability to oxygenate/ventilate. Approximately 30% of cases require CPB: 10% anticipated (pulmonary hypertension), 20% unanticipated.

When CPB is used the patient is anticoagulated with heparin 300-400 units/kg, for ACT > 460. During CPB for cardiac repair (ASD,VSD) the heart is arrested, and moderately cooled. During CPB without cardiac surgery, the heart remains warm and beating.

Advantages of CPB

- right heart is unloaded (decreased afterload)
- provides greater hemodynamic stability avoiding interruptions to venous return by surgical manipulation

Disadvantages of CPB

- obligate infusion of crystalloid
- heparinization with resultant bleeding from raw surfaces, coagulopathy
- red blood cell trauma (hemolysis)
- platelet dysfunction, activation of complement, neutrophil activation and systemic inflammatory response may contribute to increased reperfusion injury in transplant allograft

Ventilation

Separate ventilation of each lung is required in almost all cases.

Patient with severe **obstructive airway disease** require:

- high peak airway pressures to maintain gas exchange (ICU ventilator frequently required to maintain adequate ventilator gas flow)
- increased expiratory phase, to minimize gas trapping
- treatment of bronchospasm
- constant suctioning of secretions (cystic fibrosis, bronchiectasis)
- patients may start with severe but compensated hypercapnia, pH is a better determinant of inability to ventilate than $PaCO_2$.
- progressive hypercarbia/acidosis (pH persistently < 7.20), or hypoxia on OLV may require CPB

Patients with **pulmonary vascular disease** require:

- avoidance of hypoxemic or hypercapnic episodes, in severe pulmonary hypertension one lung ventilation is not attempted
- avoidance of atelectasis and excessive pulmonary distention (both increase pulmonary vascular resistance)

Significant Intraoperative Events

- Hypoxia is common during OLV (intrapulmonary shunt in the non-ventilated lung), improves when the contralateral PA is clamped.
- A period of particular risk occurs immediately after reperfusion of the first transplant lung: ventilation may go preferentially to the compliant allograft, perfusion is predominately to the diseased native lung. Clamping the native PA may avoid CPB.
- The entire cardiac output forced through the first allograft, may worsen reperfusion syndrome requiring CPB.
- The newly transplanted lung has no bronchial circulation, thus it is warm and ischemic. During CPB, it is essential to allow some cardiac ejection by decreasing CPB flow. The first allograft is then ventilated at low tidal volume and perfused via the PA.

Ischemia-Reperfusion Injury

- A common cause of early allograft dysfunction, manifest as impaired gas exchange, decreased pulmonary compliance, noncardiogenic pulmonary edema. Radiologic findings include reticular interstitial disease and air space disease. Frequently asymmetric, the first transplanted lung having worse disease. Treatment is supportive, mechanical ventilation with PEEP 5-15 cm H_2O.
- At TTH 20% of patients are extubated within 24 h postoperatively, 30% will have a severe reperfusion injury requiring several days of ventilatory support. The remaining 50% will have a short course of postoperative ventilation.
- Poor perfusion of the lung may contribute to gas exchange abnormalities at any time during a lung transplant, or in the postoperative period, if hypoxia occurs in the presence of hypotension. Improving the hemodynamic profile may result in improved gas exchange.

Hemodynamic Management

Specific treatment modalities

Inotropes for hypotension

- norepinephrine (4 mg/250NS) start at 0.01 μg/kg/min titrate up, for treatment of low SVR, RV failure
- dopamine (200 mg/250NS) 2.0-20 μg/kg/min for low CO, or when low SVR without increasing HR

Pulmonary vasodilators for ↑ PAP

- nitroglycerin (100 mg/250D5W) dosage to effect
- nitroprusside (50 mg/250D5W) dosage to effect
- nitric oxide (NO) 10-80 ppm by inhalation, also used for postreperfusion hypoxia
- PGE_1 (Prostin) 0.01-0.1 μg/kg/min, rarely used since introduction of NO

Inotropes for RV failure, ↑ PAP

- milrinone load 50 μg/kg over 15 minutes then 0.5 μg/kg/min
- isoproterenol (1 mg/250D5W) 1-5 μg/min

Arrhythmias are common, onset of atrial fibrillation during surgical manipulation is usually poorly tolerated, requiring cardioversion. Non-sustained ventricular tachycardia is frequently observed, sustained ventricular arrhythmias are usually related to acute electrolyte abnormalities which should be treated aggressively.

Fluid and Electrolytes

In principle it is desirable to keep the thoracic surgical patient "dry" to minimize fluid overload and pulmonary edema. In lung transplantation fluid underreplacement contributes significantly to hemodynamic instability during mediastinal manipulation. There appears to be a significant third space loss likely into the interstitial space (systemic inflammatory response).

There is **potassium** released during reperfusion of transplant lungs, and small patients with obstructive airways disease (and tendency to hypercapnia) with an insidious rise in potassium are at particular risk for hyperkalemic cardiac arrest at the time of second lung reperfusion. Treatment of potassium (K^+) > 5.0 with insulin and glucose infusion is warranted.

Patients typically arrive in the ICU depleted of calcium and magnesium. Calcium 1-2 g in small bolus doses and magnesium infusion of 2-4 g may avoid some arrhythmias.

Hematologic Considerations

Blood loss is variable, but requirement for 2-4 units PRBC during a bilateral lung transplant is common. Massive bleeding can occur. Optimal hematocrit during lung transplantation is approximately 0.25.

Synthetic antifibrinolytic agents in the absence or after termination of CPB may promote thrombosis. Intravascular thrombosis is likely a component of acute graft failure. At TTH, tranexamic acid 100 mg/kg is administered only to patients requiring CPB and those with significant ongoing coagulopathy and bleeding. Elevated INR and PTT are common postoperatively, but may or may not be associated with clinical bleeding.

Antibiotics

Patients with chronic bacterial colonization of their respiratory tract (cystic fibrosis, bronchiectasis) receive a predetermined antibiotic regimen to cover their known pathogens.

Patients who are not colonized with antibiotic-resistant organisms receive a second generation cephalosporin (cefotaxime 1.5 g) at the time of transplant.

Immunosuppression

Methylprednisolone 0.5-1.0 g is given i.v. on induction of anesthesia, followed by 0.5 mg/kg/day x 3 days. Intravenous cyclosporine 2-3 mg/kg/day and azathioprine 1.5 mg/kg/day are started as intravenous infusions on arrival in the ICU.

HEART TRANSPLANT

Etiology of End Stage Heart Failure

- ischemic cardiomyopathy (60% at TTH)
- idiopathic cardiomyopathy (25% at TTH)
- congenital heart disease (valvular or complex congenital, may have had corrective or palliative surgery in the past)

Preoperative Recipient Patient Management

- Continue ongoing therapy with digoxin, diuretics, vasodilators (ACE I, hydralazine and beta blocker as tolerated).

- Most patients will have elevated pulmonary pressures. While no absolute rules exist, general guidelines for maximally acceptable PA pressures include: i) systolic PA pressure < 45 mm Hg, ii) systolic PA pressure which are one-half systemic (provided systemic pressure > 80 mm Hg), iii) calculated pulmonary vascular resistance < 6 woods unit, iv) transpulmonary gradient = mean PAP -mean PCWP < 10-15 mm Hg. Irreversible pulmonary hypertension may preclude transplantation.
- Many patients will have experienced life-threatening arrhythmias related to their end stage heart disease, take particular note of chronic amiodarone use and prior insertion of automatic defibrillators.
- There is no routine premedication. Antacid prophylaxis, H_2 blockers and gastric prepulsants may be indicated for a full stomach.

Monitors/Lines

- Five lead ECG, noninvasive BP cuff, pulse oximeter, nasopharyngeal temperature probe, urinary catheter.
- Radial arterial line and PA catheter placed in the left internal jugular as the right internal jugular is routinely used for postoperative myocardial surveillance biopsies.
- Although transesophageal echocardiography is not routinely used during heart transplantation at TTH, it is available as needed for any diagnostic dilemma.

Anesthetic Technique

- A modified rapid sequence induction for full stomach. A variety of regimens have been used successfully for induction of anesthesia: fentanyl, sufentanil, etomidate, ketamine and midazolam have been described.
- At TTH, the typical induction regimen for early extubation (detailed in chapter 4) is used with dosing modified to the patient's condition. Nitrous oxide is not used, as it may increase pulmonary vascular resistance and will tend to increase the size of any gas emboli entrapped in the graft.
- To maintain adequate perfusion pressure to organs, inotropes and vasopressors (dopamine, epinephrine, norepinephrine) are frequently required. Heart transplant patients have usually been carefully diuresed preoperatively; hypotensive episodes prior to CPB are treated with *inotropes and not fluid.* Excess infused fluid will contribute to pulmonary hypertension and right heart failure.

Cardiopulmonary Bypass (CPB)

- Anticoagulation and anesthetic maintenance as for routine bypass.
- Prior to sternotomy discuss with surgeons the need for fem-fem bypass (possibly pre-induction) for the patient at particular risk of bleeding or vascular damage at sternotomy.

- Anticipate a prolonged bypass run as there (i) may be difficulty removing the native heart, (ii) may be a delay in arrival of donor heart, (iii) may be difficulty anastomosis and (iv) may be difficulty weaning from CPB. Consider ultrafiltration/hemoconcentration on CPB, particularly when patients are volume overloaded.

The Physiology of the Transplanted heart (Acute Effects)

There is loss of efferent sympathetic and parasympathetic innervation to the transplanted heart.

- The vagus will no longer have influence on the sinus and AV nodes.
- The sinus node may have an increased refractory period, atrial conduction may be prolonged resulting in first degree AV block.
- Indirect sympathomimetic agents will have no effect on the transplanted heart (Table 12.1).

There is loss of chemosensitive and mechanosensitive afferents from the left ventricle. The denervated heart retains its intrinsic control mechanisms, including a normal Frank-Starling effect and intact alpha and beta adrenoreceptor responses to circulating catecholamines. In the absence of innervation the heart lacks the ability to respond acutely to hypovolemia and hypotension with reflex tachycardia or increased contractility. Cardiac output is maintained through an increase in stroke volume; the transplanted heart is therefore *preload dependent.*

Weaning from CPB

After aortic unclamping, isoproterenol (1 mg/250 D5W) is administered by infusion at a rate titrated to maintain a HR of 90-110 beats/min.

Potential complications

- RV dysfunction: caused by incomplete myocardial protection, worsened by increased recipient pulmonary vascular resistance. Transient dysfunction is common, treatment with inotropes and afterload reduction of PA pressures.
- LV dysfunction: may be caused by prolonged ischemic time, poor myocardial preservation.

Table 12.1. Response to drugs of the transplanted heart

Drugs	action	normal		denervated	
		HR	BP	HR	BP
anticholinergics	indirect	↑	∅	∅	∅
anticholinesterases	indirect	↓	∅	∅	∅
propranolol	direct	↓	∅	↓	∅
verapamil	direct	↓	↓	↓	↓
ephedrine	both	↑	↑	±↑	↑
pancuronium	indirect	↑	∅	∅	∅
phenylephrine	direct	↓	↑	∅	↑

- Decreased SVR (vasodilatory hypotension): this is worsened by long-term amiodarone exposure which can result in a vasoplegia, or low SVR responsive only to extremely high doses of exogenous catecholamines.

Specific treatment modalities for weaning from CPB may include:

1) Inotropes for RV failure: (milrinone, dobutamine, dopamine, epinephrine, norepinephrine).
2) Vasodilators for pulmonary hypertension: (nitroglycerin, nitroprusside, nitric oxide (NO) 10-80 ppm by inhalation, prostaglandin E1 30-50 μg, infusion 0.05 ug/kg/min). Nitroprusside and prostaglandin will cause significant systemic vasodilatation and are used infrequently with the availability of nitric oxide.
3) Pacing or isoproterenol to maintain HR > 90 beats/min.
4) Norepinephrine infusion to maintain SVR, very high doses may be required.

Patient management must be individualized, however dopamine and isoproterenol are routinely used at TTH followed by milrinone and norepinephrine as second line therapy. Early introduction of inhaled nitric oxide and mechanical support with an IABP is initiated if this pharmacological approach is inadequate.

Fluid management continues to require careful attention: the LV output is preload dependent yet the RV is easily overloaded, a CVP and PCWP < 10 may improve RV function. Sparing use of crystalloid, hemofiltration of excessive intravascular volume on CPB may permit infusion of blood products frequently required to treat coagulopathy.

Hematology

Tranexamic acid 100 mg/kg is given prebypass.

Heart transplant patients may be at risk for coagulopathy due to preoperative anticoagulation (if on warfarin), long CPB duration, perioperative liver dysfunction secondary to right heart failure. Early and aggressive intervention is warranted as surgical re-exploration for bleeding is often poorly tolerated. Extra tranexamic acid, desmopressin (16-20 ug), FFP, platelets and cryoprecipitate may be required. After separation from CPB, all infused fluids should be warmed to avoid patient cooling and the attendant coagulopathy.

Immunosuppression and Antibiotics

Induction immunosuppression with:

- RATS 0.15 cc/kg/24hrs by infusion
- azathioprine 1 mg/kg po preoperatively
- methylprednisolone (Solu-Medrol) 0.5-1.0 mg i.v. on induction of anesthesia
- Cefazolin 1 g prior to sternotomy and q8hrs for 24 h.

HEART-LUNG TRANSPLANT

This procedure will be offered to patients with both end stage cardiac and pulmonary disease, usually complex congenital heart disease who have secondary pulmonary hypertension.

The care of patients undergoing heart-lung transplant will incorporate many of the considerations of heart transplantation (the denervated heart), and lung transplantation (reperfusion syndrome). Technical issues include ventilation via a single lumen endotracheal tube which may need to be pulled back to accommodate the tracheal anastomosis.

NITRIC OXIDE

In transplantation nitric oxide (NO) is predicted to have particular benefit for the treatment of postoperative right heart failure (heart and lung Tx), hypoxemia due to severe reperfusion syndrome (lung and heart-lung Tx), and endothelial dysfunction in transplant allografts.

NO induces vasodilation in vascular endothelium. NO traverses to vascular smooth muscle cells and results in activation of guanylate cyclase which increases cGMP, causing smooth muscle relaxation.

NO is delivered by inhalation, directly via pulmonary alveoli to the pulmonary circulation, resulting in decreased PVR. NO also improves V/Q matching as it is delivered to areas with ventilation and will improve perfusion to those areas. NO is inactivated locally by hemoglobin, hence there is virtually no systemic effect.

Start NO at 10-80 ppm and monitor PVR, RV function and oxygenation.

If therapeutically beneficial, NO is continued, typically for several days, weaned slowly with assessment of respiratory parameters after each change. Patients with severe right heart dysfunction or severe V/Q mismatch are decreased by 10 ppm every 4 h to avoid rebound pulmonary hypertension.

During administration of NO, toxic metabolites form and should be monitored:

1) nitrogen dioxide: $NO + O_2 \Rightarrow NO_2$ (has pulmonary toxicity, acceptable limit < 5 ppm)
2) methemoglobin: NO + Hgb ⇒ intermediate ⇒ methemoglobin, serum level monitored once daily, acceptable upper limit < 5%

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Heparin Associated Thrombocytopenia and Alternatives to Heparin

Patricia McNama, Annette Vegas

INTRODUCTION

Heparin is a commonly used easily reversible anticoagulant in clinical practice. Despite its long relatively safe record of use, complications can occur. The past 30 years has heightened awareness of the serious complication of immune complex mediated Heparin Associated Thrombocytopenia (HAT) without or with Thrombosis (HATT). As a result a burgeoning field for alternatives to heparin for anticoagulation has evolved. Their use in cardiopulmonary bypass (CPB) has been anecdotally described in the literature.

HEPARIN

Is a water soluble heterogenous mucopolysaccharide organic acid with molecular weight 3,000-30,000 daltons. It is endogenously present in mast cells in the liver and lung. Commercial preparations are from bovine and porcine sources.

Heparin acts by reversibly binding to antithrombin III (ATIII) accelerating the ability of ATIII to neutralize thrombin (IIa) and Xa. The heparin/ATIII complex also inactivates IXa, XIa, XIIa and plasmin but to lesser degrees.

Heparin is administered intravenously or subcutaneously. The duration of action depends on temperature, dose, liver and renal function. The average $T_{1/2}$ is 1.2-2 h and after 4-6 h there is no therapeutic effect. Monitoring of effect occurs with PTT, ACT and heparin levels.

Platelet factor 4 (PF4) easily neutralizes the effects of heparin by binding in a 1:1 complex which is inactive and cleared. Blood products do not reverse heparin.

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

Common side effects include hemorrhage, allergic reactions, thrombocytopenia, altered protein binding, hypotension, and a decrease in ATIII levels.

HEPARIN ASSOCIATED THROMBOCYTOPENIA

Pathophysiology

Due to a lack of standard diagnostic criteria and lab tests, the incidence of HAT has been variably reported in the literature (1.1-30%). More recent estimates puts the incidence at 3.0%. HAT is increased with prior exposure to heparin and the type of heparin.

Platelet Factor 4 (PF4) is a heparin binding protein stored in platelets and released into plasma following platelet activation. Heparin/PF4 complexes normally do not have adverse sequelae. Specific antibodies to Heparin/PF4 complexes generate IgG complexes (Heparin associated Platelet Aggregating Factor) that activate platelets. Platelets are activated by the Fc portion of IgG and Heparin/PF4 in the immune complex may bind to adjacent platelets or to the same platelet. These activated platelets release procoagulant microparticles that provide a phospholipid surface to accelerate thrombin generation. This leads to platelet activation and thrombosis or their removal by the spleen and thrombocytopenia.

There are two distinct types of HAT distinguishable clinically, but not with diagnostic tests (Table 13.1).

The diagnosis of HAT is based on:

- thrombocytopenia during heparin therapy (> 50% decrease)
- absence of other causes of thrombocytopenia
- resolution of thrombocytopenia after discontinuation of heparin
- confirmation of heparin dependent platelet antibody by in vitro testing

Heparin Associated Thrombocytopenia and Thrombosis (HATT) presents clinically as

- Arterial thromboses, most often involving major vessels of extremities, less frequently cerebral and mesenteric vasculature and implicated in myocardial, kidney and adrenal infarction.

Table 13.1. Comparison HAT I and HAT II

	HAT I	HAT II
Thrombocytopenia	mild seldom < 100,000	severe, often < 60,000
Onset after heparin	1-2 days	4-14 days (sooner if repeat exposure)
Symptoms	none, mild	may lead to thrombosis, bleeding uncommon
Heparin continues	recovery of platelet count	nonrecovery of platelet count
Heparin stopped	recovery of platelet count	recovery of platelet count 5-7 days
Treatment	no specific	stop heparin

- Venous thrombosis including deep venous thrombosis (DVT), pulmonary embolism (PE) and warfarin associated venous limb gangrene.
- Skin lesions at heparin injection sites.
- Overall 30% mortality and 20% risk of limb amputation.

DIAGNOSIS

Usually suspected by a decrease in platelet count though thrombosis may be the presenting complaint.

Laboratory diagnosis involves assays for HAT antibodies which are either:

1. functional assays: measure heparin dependent platelet activation
   - ^{14}C Serotonin Release Assay
   - platelet aggregation test
   - heparin induced platelet activation test (HIPA)
2. antigen assays (ELISA technique): measure IgG, IgM, IgA antibodies

Note the following:

- HIPA assay, ^{14}C Serotonin Release Assay, PF4/Heparin ELISA are sensitive assays for the detection of HAT II antibodies.
- The clinical relevance of HIT II antibodies without clinical symptoms is unresolved. Screening of patients for HAT II antibodies cannot be recommended currently.
- With clinical symptoms and decreased platelet count the detection of HAT II antibody is important for diagnosis and future management.

MANAGEMENT

Strategies depend on whether indication for heparin is surgical or nonsurgical. In all instances *stop heparin.*

NONSURGICAL PROBLEM (DVT TREATMENT/PROPHYLAXIS)

- alternate anticoagulant considered (LMWH, warfarin, danaproid, ancrod)
- antiplatelets (ASA, dipyridamole, ticlid) though results have proven disappointing
- massive PE/severe DVT: consider a fibrinolytic
- surgery: inferior venocaval filter, arterial embolectomy

SURGICAL PROBLEM (VASCULAR, CARDIAC)

1. Decrease antibodies:
   - delay surgery to decrease total circulating antibody load
   - plasmapheresis to eliminate antibodies

2. Platelet inhibitors and heparin: combination of antiplatelet agents and heparin with negative in vitro platelet aggregation test. Disappointing results have not proven reliable.
3. Alternate anticoagulants (Table 13.2).

ALTERNATIVES TO HEPARIN FOR CPB

Low Molecular Weight Heparins (LMWHS)

A new class of anticoagulants widely used in Europe with few approved in North America. They are fragments of heparin produced by chemical or enzymatic depolymerization of standard heparin.

Unlike standard heparin, LMWHs are unable to bind thrombin (IIa) and antithrombin III (ATIII) simultaneously so they cannot accelerate the inactivation of IIa, but do inhibit Xa activity. The anti-Xa/anti-IIa ratio of LMWHs ranges from 2:1-4:1 and are based on in vitro testing that may not reflect in vivo anticoagulant activity.

Largely due to LMWH's inability to bind to plasma proteins or endothelial cells, these molecules have excellent bioavailability, increased biological half life and can be administered 1-2 times per day. Current approved indications are DVT prophylaxis/treatment and hemodialysis.

Monitoring may be difficult as at prophylactic doses there is no effect on standard clotting tests and anti-factor Xa levels do not necessarily correlate with thera-

Table 13.2. Alternative anticoagulants to heparin

Drug	Source	Chemical Structure	Mechanisms of action	Cross-reactivity with heparin antibodies
LMWH	porcine and bovine mucosa extract; synthetic	glycosaminoglycans: mean MW 4-6.5 kDa	inhibits factor Xa > thrombin	yes : 90%
Heparinoid	porcine mucosa extract	mixture of heparan, dermatan and chondroitin sulfates	inhibits factor Xa > thrombin	yes: 10%
Ancrod	Malayan pit viper venom extract	"thrombin like" enzyme	proteolysis of fibrinogen	no
Argatroban	synthetic	arginine analog	thrombin active site inhibitor	no
Hirudin	leech salivary gland extract	66 amino acid polypeptide	thrombin active site inhibitor	no
Hirulog	synthetic	hirudin derived peptide	thrombin active site inhibitor	no
Iloprost	synthetic	prostacyclin analog	adenylyl cyclase activator	no
Aspirin	synthetic	acetylsalicylic acid	cyclo-oxygenase inhibitor	no

peutic efficacy. Protamine will neutralize anti-IIa effect (90%) but only 60-70% of Xa activity. Clotting factors are of no use.

There is a > 90% cross reactivity with heparin induced antibody. While there are a number of different manufacturers of LMWHs, there is little evidence that their clinical efficacy is significantly different if dosed appropriately.

A patient for AVR was administered **Enoxaparin** 20 mg i.v. into the prime, 150 mg postaortic/atrial cannulation and 70 mg i.v. bolus 60 minutes later. CPB was initiated 15 minutes after the 150 mg i.v. bolus and lasted 110 minutes. Factor Xa levels intraoperatively were between 0.84-0.92. Postoperative blood loss totaled 2500 cc and the patient received additional protamine and desmopressin. At re-exploration oozing from the aortic suture line was noted and treated with local Tissel. The patient received a total of 5 units PRBC, 4 units FFP and 7 units platelets.

A patient undergoing a redo MVR was administered Tedelparin. The patient received 10,000 IU Sc q12h preop (Xa levels 0.2-0.4 IU/ml) then 10,000 IU i.v. intraoperatively with an additional 5,000 IU in the prime. The CPB time was 90 minutes and uneventful. The patient received protamine 50 mg i.v. and no blood products.

Danaproid Sodium (Orgaran)

Is a mixture of low molecular weight (6000 daltons) sulfated glycosaminoglycans derived from porcine intestinal mucosa but devoid of heparin and heparin fragments.

Consists of (a) heparin sulfate with low affinity for ATIII (80%), (b) heparin sulfate with high affinity for ATIII (4%), (c) dermatan sulfate (8-16%) (d) chondroitin sulfate (< 8.5%).

Acts by inhibiting factor Xa and to a lesser extent IIa (ratio 20:1) using ATIII. There is minimal or no effect on platelets. The elimination $T_{1/2}$ of anti-Xa activity is 25 h but IIa activity is 7 h. It is predominantly excreted as inactive Xa/ATIII complex by the kidneys and is not removed by dialysis. No simple way to reverse its action except time as it is only partially neutralized by protamine. There is < 10% cross reactivity with heparin induced antibody.

Table 13.3. Commercially available LMWHs (in Canada)*

Generic	Trade	Producer	Antifactor Xa/IIa ratio	Mean MW (range)	Saccharide units	Plasma half life (min)
Enoxaparin*	Lovenox	Rhone Poulenc	2.7:1	4500 (3k-8k)	10-27	129-180
Dalteparin*	Fragmin	Kabi Pharm.	2.0:1	5000 (2k-9k)	7-30	119-139
Nadroparin	Fraxiparin	Sanofi-Winthrop	3.2:1	4500 (2k-8k)	7-27	132-162
Tinzaparin*	Logiparin	Novopharm	1.9:1	4500 (3k-6k)	10-20	111
Ardeparin	Normoflo	Wyeth-Ayerst	2.0:1	6000 (2k-15k)	7-50	200
ORG 10172	Lomoparin	Organon***	20:1	6500		1100

*** not LMWH but Danaproid

Dosing recommendations for cardiac surgery are as follows:

1. 125 U/kg iv bolus post thoracotomy
2. 2 U/ml in priming fluid of pump
3. 7 U/kg/hr iv infusion on CPB
4. if clotting noted additional bolus 1250 U

A report on 230 patients treated with Orgaran briefly mentioned 10 patients who underwent ACB. Their regimen included a bolus of 8750 units and adding 7500 U into the pump as well as running an infusion of 1500 U/hr during the CPB run. Antifactor Xa levels were monitored. The only comment is higher then normal bleeding.

A report on 47 patients receiving various dosing regimens showed 45 of 47 successfully completing CPB while 2 were abandoned intraoperatively. Complications included 18 of 45 (40%) patients demonstrating intraoperative clotting requiring additional booster injections of Orgaran, and increased postoperative bleeding which required exploration in 17 of 45 (37%) patients.

Ancrod (Arvin)

Thrombin like enzyme (proteinase) obtained from the venom of the Malayan pit viper that is highly specific for fibrinogen, producing anticoagulation by defibrinogenation. Enzymatically cleaves fibrinogen to split off A fibrinopeptides resulting in small fibrin polymers which are unstable and do not cross link to form thrombin. They are markedly susceptible to digestion by plasmin. It stimulates release of plasminogen activator from endothelium. There are no direct platelet effects but FDP inhibits platelet aggregation.

Require initial loading dose (1-2 U/kg) over 8-12 h followed by maintenance (0.5 -1.0 U/kg/24 h). Rapid defibrinogenation may overwhelm the RES and paradoxically cause a hypercoagulable state.

There is a changing rate of metabolism that depends on removal by the RES. Initially rapid elimination ($T_{1/2}$ 3-5 h for first few hours) but longer $T_{1/2}$ as concentration decreases (90% of Ancrod is cleared by 4 days). Monitor with fibrinogen levels: therapeutic 0.2-0.7 g/L. Fibrinogen levels usually return to normal within 24 h of stopping drug. Reversal with cryoprecipitate, but long $T_{1/2}$ of drug.

Ancrod was used in 20 patients for CABG. Patients received an infusion of 8.4 U/hr x 12 h with fibrinogen levels checked q 4 h. Once the target fibrinogen level of 0.4-0.8 g/L was reached the infusion was stopped, if fibrinogen > 0.8 gm/L the infusion was restarted at 2.1 U/h. The average total Ancrod dose requirement was 1.65 ± 0.55 U/kg. Patients had uneventful CPB runs, with an average time of 92 minutes. Perioperative blood loss and blood product requirements were higher compared with a control group of 20 patients.

Though successful, drawbacks exist as ancrod has to be administered over at least 6 h and serial fibrinogen levels followed. The duration of action is difficult to predict and is prolonged in patients with renal dysfunction. Despite an antidote being commercially available, ancrod is not easily reversible though cryoprecipitate will increase fibrinogen levels. Postoperative bleeding is likely to be higher and the role of autotransfusion is poorly defined. Finally and most disturbing is

that despite an adequately defibrinogenated patient (level 0.2-0.4 g/L) one patient receiving ancrod at this institution clotted off the pump.

Hirudin

Anticoagulant from medicinal leeches that acts as a direct anti-thrombin. Currently available as recombinant DNA (disulphatohirudins) or synthetic hirudin analog peptides (hirulogs). Unlike heparin, acts by binding tightly to formed thrombin independent of AT III cofactor. Prevents fibrinogen clotting and activation of V, VIII, XIII and platelets thus minimizing further thrombin formation. Renal elimination with short $T_{1/2}$ therefore no need to reverse. There is no antidote though it is hemofiltered.

Trialled extensively in animal models. One patient for AVR with HIT received a bolus of 0.2 mg/kg followed by an infusion of 0.1 mg/kg to maintain a PTT between 60-80 sec (hirudin level 1-1.5 ug/ml). The patient was bolused 9 mg 10 minutes prior to CPB and 5 mg was added to the prime. Additional boluses were given through the pump run of 83 minutes. Chest tube losses were 240 cc and no thrombotic sequelae were noted.

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Cardiopulmonary Bypass Technique

TTH Perfusionists

INTRODUCTION

The major abnormalities produced by CPB are alterations of pulsatility and blood flow patterns, exposure of blood to foreign surfaces and shear stresses, and exaggerated stress responses. This is exacerbated by the addition of varying degrees of hypothermia and hypotension.

CIRCUIT

To provide a still bloodless heart by diverting blood into a heart-lung machine which can perform the functions of (a) respiration (ventilation, oxygenation), (b) circulation and (c) temperature regulation.

Overall design of circuit depends in large part on the type of oxygenator used:

- Bubble oxygenator: Blood flows by gravity through the oxygenator and then is pumped into the patient through small bore polyvinyl chloride tubing
- Membrane oxygenator: Gravity drains venous blood to reservoir. Oxygenator is positioned after pump because high resistance to flow across membrane oxygenator

There are two basic types of pumps: roller and centrifugal.

Oxygenators function as lungs for oxygen (O_2) and carbon dioxide exchange.

COMPONENTS

These include the basic components of reservoir (venous, cardiotomy, sump), membrane oxygenator, cardioplegia delivery set, and roller pumphead (arterial, vent, cardioplegia).

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MONITORING AND SAFETY DEVICES

- ultrasonic bubble detectors on arterial tubing: audible alarm and will shut pump down
- level detectors: probe on venous reservoir: audible alarm only if low
- high pressure alarms: audible alarm at 400 mm Hg, pump shuts down at 450 mm Hg
- computer controlled perfusion: can index cardiac output (pump flow), trend flows and pressures, control cardioplegia delivery and provide pulsatile flow
- venous O_2 saturation meter (normal 70-80%)
- emergency hand cranks

PRIME SOLUTION

Takes into consideration (1) osmolality, (2) electrolytes, (3) volume of circuit and (4) hemodilution:

- Ringers lactate 2 L (isotonic balanced electrolytes), heparin 5000 units (prevent dilution), $NaHCO_3$ 50 meq (buffer), Mannitol 25 g (osmolality)
- blood added to prime only if initial hematocrit very low
- can minimize prime volume if notified in advance: smaller oxygenator, shorter tubing
- goal is to wet and de-bubble circuit completely, takes about 30 minutes to set up or prime circuit

CIRCULATORY CHANGES DURING CPB

Maintenance of circulation is no longer dependent on "homeostatic" mechanisms but "cardiac output" (CO) is now the pump flow rate. In normothermic adults, generally accepted pump flows are in the range of 2.2-2.5 L/min/m^2. In hypothermic patients lower flow rates 1.8 L/min/m^2 may be acceptable.

MAP = CO (pump flow) x SVR

Going on bypass usually results in a drop in MAP, at adequate pump flows which is a result of lowered SVR (phenomenon A) due to:

- ↓ blood viscosity (2° hemodilution)
- ↓ vascular tone (diluted catecholamines, ↓ PO_2)
- complement activation, prostanoids, neutrophil degranulation, histamine release

*Note: Phenomenon A is not seen with blood prime, only as an quinous prime. It usually lasts 5-10 minutes and management consists of either ↑ pump flows or using vasopressors.

During maintenance of CPB usually have a gradual ↑ in MAP (relative hypertension) as a result of an increased SVR (phenomenon B) due to:

- ↓ in total vascular cross-sectional area secondary to closure of portions of microvasculature
- constriction of vascular tone (↓ temp, ↑ circulating catecholamine)

- ↑ blood viscosity (third spacing, diuresis and hypothermia)
- ↓ generation C3a, C4a

Rewarming CPB/unclamping, have ↓ MAP usually due to ↓ SVR from:

- recirculation of cardiac metabolites (adenosine)
- temperature related vasodilatation

METHODS OF ADJUSTING BP

Maintenance of cardiovascular stability requires interplay of machine (pump) function and patient factors such as SVR and venous compliance. Both of these factors are relatively easily manipulated by pump flow control and/or drugs.

Varying pump flow rate:
- ↑ flow ⇒ ↑ trauma to blood cells
- ↓ flow ⇒ tissue perfusion will suffer (acidosis)

*Note: Hypertensive states are seldom treated with reduced flow rates. Low flow states as requested by the surgeon are usually because of flooding at the surgical site; are timed by the perfusionist and announced to the surgeon on a minute to minute basis.

Primary means to raise BP: ↑ SVR

- phenylephrine (Neo-Synephrine) 100-200 μg bolus
- norepinephrine (Levophed) 4 mg/250D5W as infusion

Primary means to lower BP: ↓ SVR

- anesthetics: propofol, vapor, narcotics, benzodiazepines
- direct agents: sodium nitroprusside (nipride) 50 mg/250D5W as infusion

Risks of high MAP include:

- more rapid rewarming of ischemic heart (↑ flow through non-coronary collaterals)
- risk of cerebral hemorrhage in anticoagulated patient
- risk of pump tubing disconnection

PUMP FLOW VS. PRESSURE (MAP)

Although acceptable flow rates are fairly well established, there is considerable controversy about acceptable arterial pressures during CPB. At any given flow rate there is marked variability in arterial pressure from patient to patient. The overriding concern with low arterial pressures is adequacy of organ perfusion, which depends on O_2 consumption, blood flow distribution and intrinsic autoregulatory capability of various vascular beds.

- Effective pump flow is flow from the oxygenator that results in tissue perfusion. It may be considerably less than the "set" blood flow due to losses from physiological shunts (bronchial blood flow) and vent suction.
- Perfusion pressure is determined by the interaction of blood flow and overall arterial impedance which is primarily a function of vasomotor tone and blood viscosity.

Autoregulation of blood flow to various vascular beds (brain, kidney, coronary) refers to the ability of organ vasculature to regulate local resistance in order to maintain constant flow despite significant changes in perfusion pressure. Autoregulation is preserved in some organ vascular beds during CPB despite a nonpulsatile flow pattern, hemodilution and hypothermia. This has been well documented for cerebral blood flow (CBF) on CPB which is maintained in the face of hypothermia (20 ° C) and MAP 30-50 mm Hg. The effects of autoregulation in other organs is less well documented. At normothermia though cerebral autoregulation begins to fail at perfusion pressures < 50 mm Hg.

At TTH perfusion runs calculated flows and aims to maintain MAP 50-60 mm Hg.

PULSATILE VS. NONPULSATILE FLOW

Nonpulsatile flow is the usual standard as it is easy to use and compatible with patient survival. Some evidence suggests that pulsatile flow during CPB provides a more physiologic milieu and probably better tissue perfusion.

There are conflicting studies on the subject, likely because of the difficulty of isolating pulse contour from many other operant factors during CPB. No studies have shown improved survival and there are problems associated with pulsatile bypass. The arterial inflow cannula has a cross-sectional area 8-10 times smaller than the aorta. During the pressure pulse, the transcannula pressure gradient across the cannula must be sharply increased to at least 400 mm Hg over that seen during nonpulsatile perfusion if the same total flow is to be maintained to benefit renal function. This is difficult to obtain with either the roller or centrifugal pump heads without increasing the likelihood of various jet related effects including hemolysis, shearing of aortic atherosclerotic plaques, aortic dissections, cannula site leaks and dislocations.

Despite the lack of outcome studies, pulsatile flow may be advantageous in the following groups of patients: (1) CAD: at risk for ischemia/infarct with ↑ L vent work, (2) significant arterial disease, (3) chronic arterial hypertension, (4) chronic end organ insufficiency (renal, hepatic).

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HYPOTHERMIA VS. NORMOTHERMIA BYPASS

Originally systemic hypothermia was touted as a means to providing better organ protection. Other effects include:

- slows biochemical reactions
- ↑ blood viscosity
- changes in blood gases:
  a) changes in O_2/Hgb dissociation curve: ↑ binding O_2 with ↓ temp (left shift)

b) changes in solubility of O_2/CO_2: ↓ temp ⇒ ↑ gas solubility in liquid, therefore partial pressure of gas will ↓. So at skin temp 27 °C pH = 7.56 and pCO_2 = 25 mm Hg, while at 37 °C pH = 7.40 and pCO_2 = 40 mm Hg

Effect of normothermic bypass on end organ function is summarized in Table 14.1.

Table 14.1. Effects of normothermic bypass on organ function

1. **heart/myocardium**
   ◊ chemical cardiac arrest ↓ MvO_2 by 90%
   ◊ further reduction of MvO_2 to 97% under hypothermia (22°C)
   ◊ during normothermia further cardioplegia is needed to meet metabolic requirements include:

	continuous warm cardioplegia	intermittent cold cardioplegia
advantages	aerobic metabolism during arrest ? less postop low CO ? less enzymatic infarctions	cold protection ? less periop infarctions ? less IABP
disadvantages	technical difficulties because of continuous blood in field risk of warm ischemia	no myocardial benefit at temp < 20°C

2. **neurologic**
   ◊ stroke leading cause of postop mordidity

◊ 2 patterns:	focal neurologic	neuropsychiatric
	◊ most likely embolic ◊ unlikely to benefit from hypothermia as occur during normothermic parts of CPB	◊ global perfusion ◊ mechanism unclear but similar high incidence in normothermic and hypothermic CPB

3. **metabolic** (It is important to run adequate flows of 2.4 to 2.5 L/min/m² on normothermic CPB)	normothermic large A-V O_2 differences low venous O_2 sat ↑ lactate acidosis	hypothermic less A-V O_2 differences low O_2 extraction
4. **splanchnic**	no difference	no difference
5. **renal**	overall CPB ↓RBF and u/o	?↑transient dysfunction
6. **hemostasis**	↓bleeding, ↓platelet dysfunction	↑beeding, ↑platelet dysfunction
7. **hemodynamic**	↓↓SVR post-CPB (↑vasoactive cytokines) ?low afterload facilitates wean from CPB ↑circulating catecholamines	less↑circulating catecholamines less vasoctive cytokines ?less need for vasopressors

ABG ANALYSIS ALPHA (α) VS. PH STAT

Cerebral blood flow (CBF) is autoregulated over a range of mean arterial pressures (MAP) 60-150 mm Hg. This is accomplished by maintaining tight regional/metabolism coupling:

↓ temp ⇒ ↓metabolism ⇒ ↓CBF

Uncoupling of the autoregulation mechanism occurs with intracerebral pathology and CO_2.

1. **pH stat**: exogenous CO_2 is added ⇒ passive cerebral vasodilation occurs overriding the usual autoregulation mechanism.

(+) CBF can be maintained at lower perfusion pressure, luxury cerebral perfusion can occur

(-) steal from stenosed cerebral arteries, ↑ delivery of emboli to cerebral circulation

2. **α stat**: as temperature falls → pCO_2 ↓ → cerebral blood flow ↓ but maintain cerebral autoregulation and flow/metabolism coupling and enables brain to adjust CBF in response to metabolic demands

(+) may be more physiologic, lessen RBC hemolysis

(-) may maintain adequate regional CBF, despite overall ↓ CBF

Theories aside and in the absence of overwhelming evidence (outcome studies) the practicality is that in the U.S. there is still a 50:50 split between either management strategy. At TTH, αstat is the predominant ABG management.

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Circulatory Arrest and Neuroprotection

Christopher M.S. Feindel, Ludwig Fedorko

INTRODUCTION

Circulatory arrest is necessary for operations on the distal ascending aorta and aortic arch. It is a highly effective technique that allows the surgeon to operate in this area with relative ease and without the constraints of vascular clamps and a blood obscured surgical field. The obvious limiting factor is the safe period of time that the brain can survive without a blood supply.

At normal body temperature the human brain rapidly ceases to function within seconds of losing its blood supply and within 3-5 minutes permanent damage will occur. Even the simplest operation of the aortic arch cannot be performed within this time. If the patient is cooled to 20°C, circulation to the brain can be arrested safely for periods up to 60 minutes or possibly longer. This allows the surgeon sufficient time to perform even the most complex operations on the aortic arch and ascending aorta.

INDICATIONS

Circulatory arrest falls into two broad categories, planned and unplanned.

1. Planned operations on the distal ascending aorta or aortic arch include:
   - true and false aneurysms of the ascending aorta and aortic arch or dissections involving the proximal aorta
   - complex reoperative cardiac surgery where the heart and/or aorta may be dangerously adherent to the sternum

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2. Unplanned operations requiring circulatory arrest include:
   - replacement of badly diseased ascending aorta in the process of performing an otherwise routine cardiac operation
   - iatrogenic aortic dissection occurring at a cannulation site
   - treatment of massive air embolism

TECHNIQUE OF CIRCULATORY ARREST

The technique of induced circulatory arrest is straight forward and can be easily implemented by any experienced cardiac surgeon. Excellent communication and attention to detail are critical to avoid a catastrophe. It is therefore worthwhile for the surgeon to first review with other members of the team exactly what is going to happen and this can normally be done as the patient is being cooled. All necessary items such as tubing, cannulae etc. should be organized. Time and attention to detail are of the essence and there is little margin for error.

The specific circumstances of the patient determines the location of cannulation sites. Arterial inflow is established via either the aortic arch, ascending aorta or the femoral artery. Venous drainage is preferred via the right atrium but via the femoral vein or veins if necessary. For example femoral or axillary artery cannulation must be used if the patient has an aortic dissection.

The patient is cooled to 20-25°C rectally depending on the anticipated duration of circulatory arrest. Both nasopharyngeal and rectal temperature probes are used. A bladder catheter with a temperature probe is very useful if available. The heart will spontaneously fibrillate as it cools and requires insertion of a sump via the right superior pulmonary vein to decompress the left ventricle. If the patient has an aortic dissection with an incompetent aortic valve, manual compression of a fibrillating heart may be required as the sump may not maintain sufficient decompression of the heart. Avoid cross clamping the aorta at this stage.

Once the patient's systemic temperature has been lowered to the desired level the cardiopulmonary bypass (CPB) machine is turned off. The perfusionist drains the venous blood out of the patient but intermittently clamps the venous line to prevent excessive drainage from the patient. The patient is positioned head down, the ascending aorta is opened and the pathology is inspected. At this stage depending upon the operation that is required, retrograde cerebral perfusion is commenced. (See below) Valuable time during the period of circulatory arrest should not be wasted on establishing cardiac protection at this time. The heart is already cool and can tolerate much longer periods of ischemia than the brain.

After completing the distal prosthetic graft to distal ascending aorta and/or aortic arch the arterial inflow cannula is positioned through the graft and secured. If the arterial cannula was initially placed in the femoral artery it is repositioned into the aortic graft at this time. Resumption of femoral retrograde perfusion is not recommended and is absolutely contraindicated if the patient has an aortic dissection where there is a possibility of a second reentry tear in the distal descending aorta.

The pump is turned on slowly to fill the aorta with blood and once filled the open end of the prosthetic graft is clamped. Full bypass is resumed and the remainder of the operation is completed. Cardioplegia is given at this time and rewarming is begun which in most cases allows sufficient time to reach normothermia by the time the rest of the operation is completed.

RETROGRADE CEREBRAL PERFUSION

Retrograde cerebral perfusion has been introduced as an adjunct to cerebral cooling as a means to protect the brain during periods of circulatory arrest. In this technique the patients body and brain temperature are cooled to 20-25°C by CPB.

After the heart lung machine is turned off a cannula is placed directly into the superior vena cava and blood at 10°C is perfused in a retrograde fashion at a rate of 300-500 ml/min. By observing dark blood emanating from the origins of the cerebral vessels in the aortic arch the surgeon confirms the presence of retrograde flow through the brain.

Retrograde cerebral perfusion allows the surgeon an even greater safe period of circulatory arrest at a specific temperature compared to not using retrograde perfusion. Alternatively it may reduce the need for cooling the patient to low temperatures to achieve the same safe period of circulatory arrest without retrograde perfusion. Cooling the patient to temperatures of 25-28°C rather than to less than 20°C may help to reduce the negative affects of hypothermia such as coagulopathy and cerebral edema.

Although retrograde cerebral perfusion does not provide blood flow to all areas of the brain it has the important advantage of ensuring that air and atherosclerotic debris are kept out of the cerebral vessels by maintaining a constant retrograde flow of blood in them.

During the period of circulatory arrest the anesthesiologist may choose to encase the patient's head in ice packs in order to promote cerebral cooling. In addition steroids may be given to help reduce cerebral swelling although there is little evidence that steroids are beneficial.

PHARMACEUTICAL AND OTHER NEUROPROTECTIVE ADJUNCTS

15

There is little clinical evidence of effective pharmacotherapy to limit the extent of neurological injury resulting from hypothermic circulatory arrest. However, there is a considerable amount of data to suggest that appropriate CPB management may help to minimize the negative neurological and cognitive outcomes after cardiac surgery.

ACID/BASE CONTROL

The αstat method allows drift of blood pH towards more alkaline values as the patient's temperature drops. The pH-stat method involves elevating and maintaining pCO_2 at 40 mm Hg to compensate for reduced activity of $[H^+]$ ion in lower temperatures.

The αstat method preserves cerebral autoregulation, results in reducing cerebral blood flow (CBF) at lower temperature. By contrast, with the pH-stat method, cerebral autoregulation is lost and this results in a passive increase or decrease of cerebral flow in response to perfusion pressure changes. Although animal experiments confirm better uniformity and faster cooling of the brain when pH-stat mode is used, there is a risk of cerebral hyperperfusion and subsequent edema with high perfusion pressure being used.

Clinical studies suggest that patients undergoing CABG surgery have better neurological and cognitive outcomes when α stat of blood pH control is used compared to the pH stat method. Therefore, αstat is preferred for adult patients undergoing profound hypothermia and circulatory arrest. However, in the pediatric population, pH stat is preferred over the αstat method.

GLUCOSE CONTROL

Severe hyperglycemia in the setting of global or focal brain ischemia has been demonstrated to be harmful. Glucose levels are often elevated in patients undergoing CPB intraoperatively and postoperatively. While it is not known what levels of glucose are acceptable it is important to treat hyperglycemia particularly if it occurs prior to circulatory arrest. Hyperglycemia during global ischemia is known to exacerbate metabolic acidosis of the brain.

TEMPERATURE MONITORING AND REWARMING PHASE

Hypothermia is the most powerful neuroprotective agent known to date. However, there is less room for error during hyperthermia. Even short periods of brain temperature over 40-41°C may result in brain injury particularly in brains subjected to previous ischemic insult. Therefore, it is important to limit the rate of systemic rewarming in order to avoid cerebral hyperthermia. A technical point one must remember is that blood temperature may exceed the measured nasopharyngeal temperature by as much as 3°C during this phase.

PHARMACOTHERAPY

There are no clinical trials demonstrating the efficacy of neuroprotective drugs in reducing cerebral injury during circulatroy arrest. Potential anesthetics such as

propofol, thiopental, isoflurane display neuroprotective propterties in animal experiments, but none has been shown to be effective in patients undergoing circulatory arrest.

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Hemoconcentration and Hemodialysis During CPB

Teraze McDougall, Mary Scholz

The use of hemoconcentration (ultra-filtration) and hemodialysis during CPB should be discussed with both the surgeon and anesthesiologist. This chapter will review the techniques and the equipment required.

HEMOCONCENTRATION (ULTRA-FILTRATION)

This process involves selective separation of plasma water and low molecular weight solutes from the intravascular cellular components and plasma proteins of blood using a semipermeable membrane filter. The driving force for movement across the membrane is convective transport. This is the hydrostatic pressure differential occurring across the membrane.

Hemoconcentrator/Hemodialyzer Specifics

Polysulphone hollow fibers; Priming Volume – 120 ml; Pressure Drop – 30 mm Hg; Max blood flow – 600 ml/min; Molecular Cutoff – 60,000 Daltons; Surface Area – 1.8 m^2

Factors affecting hemoconcentration rate is shown in Table 16.1.

Hemoconcentration Technique

The hemoconcentrator can be connected into the recirculation line coming off the oxygenator. Blood flow from the recirculation line will be directed into the inlet of the unit and the outlet would be connected back into the cardiotomy reservoir. The hemoconcentrator can also be connected off the manifold. The device's inlet would receive blood flow from the arterial side of the manifold and the outlet would again be connected to the cardiotomy reservoir (Fig. 16.1).

Indications and Benefits

- Reduce the effects of hemodilution that occurs during CPB from pump prime dilution, crystalloid cardioplegia and high circulating blood volumes.
- Elevate hematocrits in order to improve the oxygen carrying capacity of the blood.

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Table 16.1. Factors affecting hemoconcentration rate

Increase Rate	Decrease Rate
Increase surface area	Increase hematocrit
Increase pore size	Decrease temperature
Increase flow	Increase serum proteins
Increase transmembrane pressure	

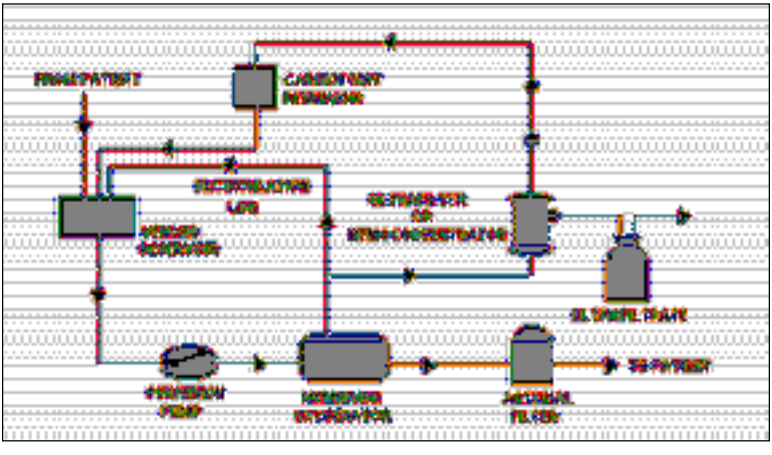

Fig. 16.1 Circuit for hemoconcentration or dialysis.

• Increase plasma protein concentration which may increase plasma oncotic pressure promoting reduced tissue edema.

• Decrease plasma potassium levels. Done by removing plasma water then replacing this volume with a crystalloid solution containing no potassium.

• Preserve intravascular coagulation factors and platelets.

Contraindications and Considerations

While no absolute contraindications exist for the use of hemoconcentration, consider the following:

• Filtrate volumes exceeding 3-5 L may produce rebound hyperkalemia, so monitor serum potassium.

• Consider carefully in sickle cell patients due to the fragility of the patient's RBCs. Use lower transmembrane pressures to minimize hemolysis.

• Heparin may be filtered off during the process of hemoconcentration. Monitor ACTs closely.

• If the hemoconcentrator is cut into the recirculation line, the perfusionist must remember that this is an added shunt and that there may be a decrease in MAP and that flows should be adjusted accordingly to compensate.

• Consider the biocompatibility of the hemoconcentrator unit. Membranes made out of polyacrylonitrile and polysulfone have been shown to trigger less of an immune response (i.e., complement activation). There is some information to suggest that the use of hemoconcentration can reduce levels of certain inflammatory response mediators. These mediators have been found in the filtrate as well as adhering to the hemoconcentrator itself.

• Hemoconcentration for more than 1-2 h may lower bicarbonate levels due to preferential movement of the anion. Monitor blood gases regularly.

• Observe the recommended blood flow through the unit. Limit the transmembrane pressure to prevent hemolysis.

• Maintain a minimum blood flow through the device to avoid stagnation and/or thrombus formation.

HEMODIALYSIS

This process involves movement of solute and water through a semi-permeable membrane. The driving forces behind this movement are the hydrostatic pressure like that of hemoconcentration and the concentration gradient created by the use of dialysate.

Dialysis uses diffusion and hydrostatic pressure to drive solutes across a semipermeable membrane. Concentration gradients are created by flushing dialysate solution counter current to blood flow on the effluent side of the device. Alternating 2000 ml of 0.5% DIANEAL with 1000 ml of normal saline with 50 meq sodium bicarbonate added helps to maintain a normal pH (Fig. 16.2).

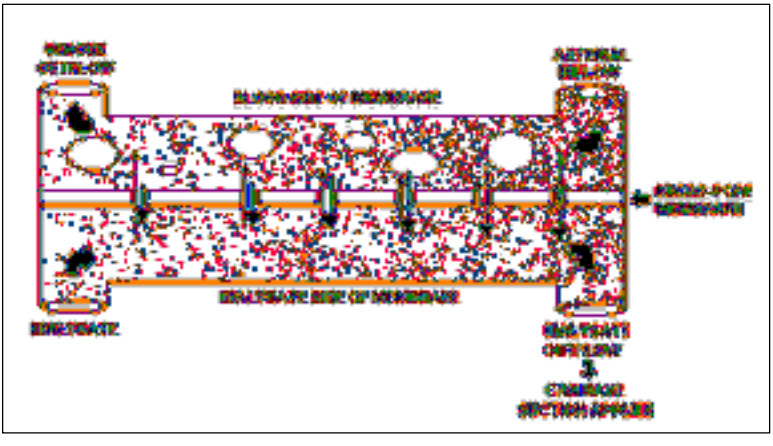

Fig. 16.2. Membrane for hemodialysis.

By using the recirculation line as the arterial inlet to dialyzer, high volume blood flow can be achieved. The venous side or outlet side is connected to a filtered port of the cardiotomy reservoir (Fig. 16.1).

Indications and Benefits

- Intra-op hemodialysis delays immediate post-op hemodialysis and associated complications at an unstable time.
- Solute levels (i.e., K^+) and blood volume can be adjusted to promote cardiac efficiency.
- Also see benefits of hemoconcentration.

Perfusion Considerations

- Maintain adequate heparinization and other drug levels.
- Maximize renal function intraoperatively; consider use of low-dose dopamine and/or pulsatile flow during aortic cross-clamp time.
- Maintain a minimum blood flow through the device to avoid stagnation or thrombosis.
- Optimal blood flow through device is 300-500 cc/min.
- Can add suction to the effluent side to promote fluid removal.

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Intra-Aortic Balloon Pump

Arthur Melo, Mindy Madonik, Annette Vegas

MECHANISM OF ACTION AND ROLE

The Intra-Aortic Balloon Pump (IABP) only augments existing cardiac function without increasing oxygen (O_2) demand. Inflation during diastole augments coronary perfusion and O_2 delivery. Deflation during systole reduces afterload against which the heart must pump, thereby decreasing demand. IABP is a useful temporary mechanical support for reversibly failing myocardium.

Indications

- postcardiotomy cardiogenic shock (inability to wean from cardiopulmonary bypass)
- cardiogenic shock unresponsive to medical therapy:
  - postmyocardial infarction (MI)
  - primary myocardial dysfunction
  - ventricular septal defect
  - acute mitral regurgitation
- unstable angina; pre-/post-MI, failed angioplasty
- ventricular tachyarrhythmias caused by ischemia
- bridge to transplantation
- intraoperative pulsatile flow generation

Contraindications

- aortic regurgitation
- aortic dissection
- thoracic aneurysm or abdominal aortic aneurysm (for femoral approach)
- severe peripheral vascular disease (during cardiac surgery consider transthoracic route)
- severe blood dyscrasias

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MONITORING

Following insertion of an IABP check a chest x-ray (CXR) to ensure optimal placement. The tip of the balloon should be in the descending aorta below the origin of the left subclavian and above the renal arteries. On CXR the balloon marker is roughly at the left second intercostal space.

Always put the balloon on standby prior to flushing the arterial line. Avoid sampling blood through the arterial port. Never turn off the IABP in situ except for removal.

If excessive catheter whip (distorted arterial line), consider a bubble trap to dampen the arterial trace. Place just distal to the arterial transducer.

Vigilance is required to avoid IABP complications listed in Table 17.1. Monitor distal leg perfusion, urine output and daily platelet counts. Prompt removal of the IABP catheter should occur with ipsilateral limb ischemia, visceral ischemia or balloon rupture.

It might be difficult to diagnose cardiac arrest in a patient who is paced and has an IABP. Turn off the IABP and look at the arterial or pulmonary artery trace and if flat, chances are the patient has no cardiac output (CO). Start chest compression and put the IABP on pressure setting until establishment of effective cardiac output, then resume 1:1 rate with ECG trigger.

The balloon inflates with helium and has a nonthrombogenic surface hence does not require systemic heparinization. Patients returning from the OR with an IABP are not systemically heparinized unless more then 48 h postoperation.

WEANING AND REMOVAL

The decision to wean from IABP is usually made when the patient is hemodynamically stable. Slight variations in this process occur but generally decrease the IABP frequency to 1:2 or 1:3, or decrease the amount of augmentation over several hours. Check appropriate hemodynamic parameters (BP, CO, urine output) during weaning. Stable patients may be extubated prior to IABP removal.

Standard removal technique involves:

- Inspect the insertion site to ensure a routine percutaneous insertion. Some femoral cut down balloon catheters and all transthoracic balloon catheters require operative surgical removal.

Table 17.1 Complications of IABP

- Ipsilateral limb ischemia
- Bleeding
- Thrombocytopenia
- Infection
- Aortic dissection or perforation
- Visceral ischemia (renal, gut, spinal cord)
- Balloon rupture
- Emboli

- Always check and correct coagulation status prior to removal of the IABP.
- Avoid uncontrolled hypertension during the removal by sedating the patient or using vasodilators (norepinephrine) to prevent excessive bleeding.
- Clean the insertion site with Betadine and remove the stay sutures. Turn the console off and ensure the balloon is deflated. Determine where the percutaneous insertion point is and aim to apply pressure just cephalad and directly over the artery. Pull smoothly and ensure tip is intact and back bleeding occurs to remove any clot. Compress manually with sterile gauze for 20-30 minutes. Compression should be forceful enough to prevent bleeding but not to occlude the vessel entirely. After removal, patients are on bed rest for 4 h with a sandbag over the removal site.

BALLOON OPERATION

Three parameters can be adjusted during operation of the IABP:

- **Trigger modes:** Trigger is the signal that the system uses to identify the beginning of the cardiac cycle and starts IABP inflation. The triggers include ECG, pacing, internal or pressure.
- **Timing:** Inflation must occur just after aortic valve closure for proper diastolic augmentation, and deflation must be complete just as the aortic valve opens for proper afterload reduction. Optimal timing can be determined by reference to the aortic pressure waveform and adjustments to the inflation and deflation timing dials.
- **Amount of augmentation:** can be varied by the ratio of heart rate to inflation frequency (1:2, 1:1) and degree to which the balloon is inflated each time (percent augment).

CONVENTIONAL TIMING

Inflation

Rapid inflation of the balloon just after the aortic valve has closed (dicrotic notch) markedly elevates and augments diastolic pressure and displaces blood volume. The further into diastole that inflation begins the lower the diastolic augmentation pressure, and the less hemodynamic advantage.

Effects:

- Increase coronary blood flow
- Augment perfusion to the aortic arch and distal systemic circulation
- Increase coronary collateral circulation

Deflation

Balloon deflation occurs just before the aortic valve opens or during isovolumetric contraction. This reduces aortic end diastolic pressure (AoEDP), thereby lowering the resistance against which the left ventricle must eject in systole. Deflation has two rules:

1. Balloon AoEDP should be lower than patient AoEDP.
2. Balloon assisted systole should be lower than patient systole.

Effects:

- Reduction of AoEDP achieved by the balloon's deflation just before the next systole enables the left ventricle to eject against a lower resistance. (afterload reduction). Aim for 8-10 mm Hg (max. 15 mm Hg).
- The systole following balloon inflation should be lower than unassisted systole. The decreased afterload reduces the maximum tension required in systole.
- Myocardial oxygen consumption (MVO_2) is decreased because cardiac work during isovolumetric contraction is reduced.
- Cardiac output is increased.
- Reduction in peak left ventricular pressure generated during systole reduces left to right shunting secondary to ventricular septal defects and reduces the amount of regurgitation in mitral insufficiency.

Proper IABP timing (Fig. 17.1):

- Increasing coronary perfusion from inflation of the balloon during diastole. (See ❶)
- Decreasing afterload (AoEDP) from deflation of the balloon prior to systole. (See ❷)

Components of the Datascope IABP Console

- ON/OFF SWITCH: turns console on or off
- ZERO BUTTON: use to zero the arterial line. Depress and hold until zeroed.
- AUTOFILL BUTTON: fills empty balloon. Depress until autofill message appears
- TRIGGERS: either choose ECG, pacing, internal or pressure
- IABP BALLOON FREQUENCY: 1:1, 1:2 or 1:3
- AUGMENTATION: minimum to maximum inflation of balloon

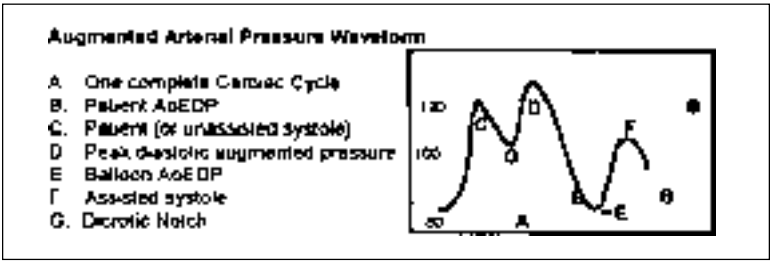

Fig. 17.1. Proper IABP timing.

- INFLATION/DEFLATION SLIDE CONTROLS: for setting optimal beginning and end of balloon inflation
- STANDBY: temporarily stops balloon from inflating (i.e. to flush balloon, deal with artifact) or starts the balloon
- ECG: select different leads and adjust size (look for largest R wave)
- REFERENCE LINE: moves to help set timing and compare pressures
- PRESET: highlights part of arterial trace balloon is inflated during

TROUBLE SHOOTING IABP (FIG. 17.2)

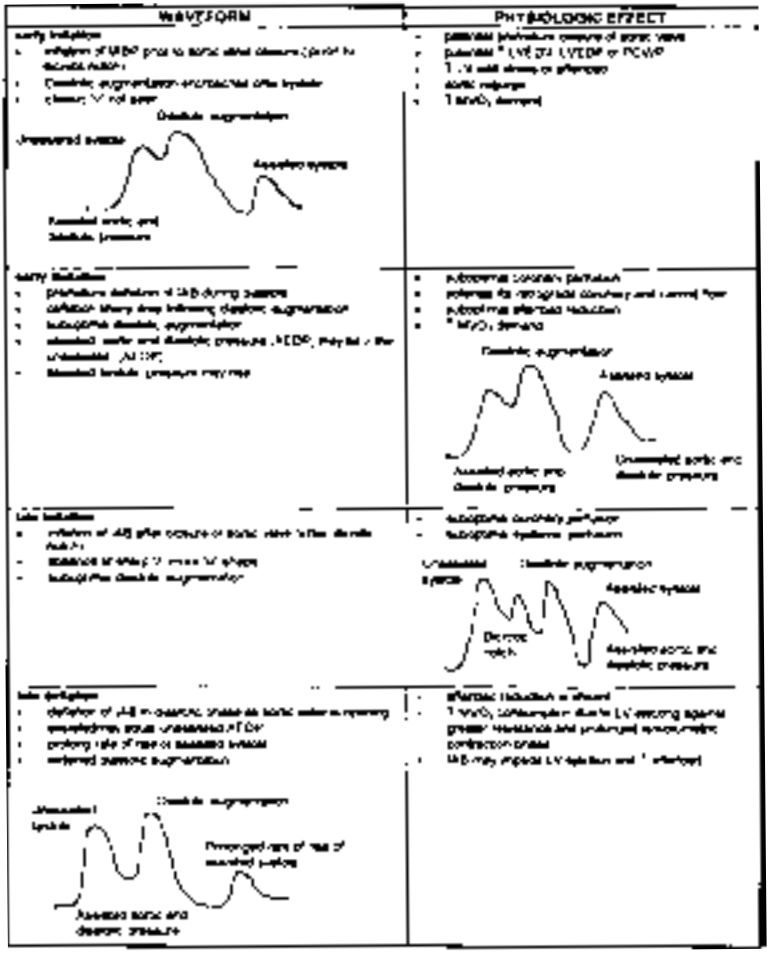

Fig. 17.2. Troubleshooting IABP.

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Aortocoronary Bypass: Surgical Technique Including Cannulation Techniques

Anthony Ralph-Edwards

AORTOCORONARY BYPASS SURGERY

Indications for Surgery

The objective of the coronary artery bypass graft (CABG) is complete revascularization of the myocardium. This is accomplished by bypassing all arteries with severe stenoses (≥ 50% diameter reduction), except those of small caliber (less than 1 mm in diameter). This operation is performed for one or more of the following reasons:

1. Symptoms
   - class 3 or 4 chronic stable angina
   - unstable angina refractory to medical management
     - ◊ positive stress tests post-MI have 15-20% one-year mortality
     - ◊ 40-50% non-Q MI and 15% Q-wave MI develop early postinfarction angina
     - ◊ 6 month mortality of post-MI angina 56%
2. Acute ischemia or hemodynamic instability postpercutaneous transluminal coronary angioplasty (PTCA)
   - emergency surgery post-PTCA is needed in 3-4% of patients
   - 30-40% incidence of Q-wave MI
   - 5-6% operative mortality
3. Acute evolving infarction within 4-6 h of onset
4. Markedly positive stress test before major intra-abdominal or vascular surgery
5. Ischemic pulmonary edema
   - usually left main or triple vessel disease, impaired LV function
   - 70% 2-year mortality managed medically
6. To improve survival in patients with
   - left main stenosis > 50% (annual mortality 10-15%)
   - three vessel disease with EF < 50%

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- three vessel disease with EF > 50% and significant inducible ischemia
- one and two vessel disease with extensive myocardium at risk, lesions not amenable to PTCA

7. Other
   - significant coronary lesions accompanying other cardiac lesions requiring surgical correction
   - congenital coronary anomalies associated with increased risk of sudden death
     - origin of left main from the right aortic sinus
     - origin of the right coronary artery (RCA) from the left aortic sinus, proximal RCA runs between the pulmonary artery and aorta

CONDUITS FOR CORONARY BYPASS SURGERY

Saphenous vein grafts:

- at 10 years, approximately 50% occluded, 25% stenotic, 25% appear angiographically normal. These results may be improved with cholesterol lowering agents.
- use short saphenous if greater saphenous not available (similar patency).
- segments from leg match size of the arteries better than segments from thigh (no significant difference in long term patency).

Internal thoracic artery (ITA), when anastomosed to left anterior descending (LAD):

- 90-95% patency at 10 years
- increased 10-year patient survival
- improved event-free survival (angina, MI)
- no increase in operative risk
- it can be used as free graft
- if graft has adequate caliber (> 1.5 mm diameter) and bleeds freely considered adequate
- contraindications:
  - inadequate mammary blood flow
  - chest wall irradiation
  - avoid bilateral internal thoracic arteries in diabetics and obese patients

Right gastroepiploic artery:

- usually 1.25-1.5 mm at distal end
- long enough to be used to bypass any of the three major vessels
- atherosclerosis rare
- good long term patency
- concerns:
  - increased morbidity from laparotomy incision
  - repeat upper abdominal surgery may damage graft origin
  - do not use as free graft
  - arterial grafts are vasoreactive postoperatively

Radial artery:

- time consuming to harvest
- no proven improvement in long term results
- prone to severe spasm postoperatively, usual protocol requires i.v. nitroglycerin given postoperatively for 24 h followed by nifedipine XL or amlodipine
- harvest technique:
  - no touch technique
  - on exposure irrigated with papaverine solution
  - stored in solution of 50% heparinized saline with papaverine and 50% blood

Patency rates with arm veins, cryopreserved or fresh homologous saphenous, Gore-Tex graft are very poor. These grafts may be useful in desperate situations.

Revascularization strategies:

- All vein grafts
  - hemodynamic instability
  - use of thoracic artery contraindicated or unsuitable (poor flow)
  - advanced age
- Left internal thoracic artery to left anterior descending artery, remaining grafts veins
  - standard operation
  - demonstrated improved freedom from subsequent cardiac events
  - survival advantage
  - no increase in perioperative morbidity or mortality
- All arterial revascularization
  - theoretic improvement in long term graft patency (no prospective randomized trials)
  - patients faster to ambulate without leg incisions
  - may be useful in young patients with hyperlipidemia
  - increased time required for conduit harvest
  - possible increased perioperative complication rate sternal infection, graft spasm
  - patients require treatment with calcium channel antagonists postoperatively

Endarterectomy:

- Avoid whenever possible, as may it increase perioperative myocardial infarction rate.
- Usually more prudent to graft posterior interventricular if possible rather than perform endarterectomy on distal RCA.
- Most frequently performed when distal RCA is large and occluded and not amenable to direct bypass.

Distal anastomosis: Single vs. Sequential

- Theoretical advantage is higher flow rates throughout most of the vein, possibly resulting in higher patency rate.
- Useful if limited vein or artery.

- More susceptible to technical error; all anastomoses at risk if problem with proximal.
- Single anastomosis method of choice.
- On long term follow up, more adverse events occur in patients receiving sequential grafts.

Redo bypass grafting:

- Operative mortality 2-3 time higher than first operation.
- 10% perioperative myocardial infarction rate.
- Redo sternotomy increased risk, adhesions may result in laceration of aorta, right ventricle or bypass grafts.
- Can assess risk from review of old operative note (closure of pericardium, use of LITA), old discharge summary (postoperative bleeding, infection) and chest x-ray (lateral view demonstrates proximity of right ventricle to posterior of sternum (look for clips on mammary artery pedicle to outline path of vessel and relation to sternum). Rarely CT or MRI scans of the chest may be employed.
- If repeat sternotomy appears risky, initial exposure of femoral vessels or actual femoral cannulation and bypass (decompresses right ventricle, reduced likelihood of injury) may be safer. Femoral cannulation if bleeding problems during sternotomy.
- Handling of the heart minimized to prevent arrhythmia, embolization of debris from old grafts. Initial dissection to gain exposure of right atrium and aorta to permit cannulation. Retrograde cardioplegia delivery usually used to prevent embolization from patent but diseased grafts provide protection to ischemic areas and territory of LITA. Old grafts usually removed or ligated early to prevent embolization.
- Meticulous hemostasis required to avoid postoperative bleeding secondary to large area of dissection required.
- ITA may be insufficient to revascularized area supplied by stenotic but patent vein graft.

CANNULATION TECHNIQUES

Arterial cannulation

- Distal ascending aorta is the preferred site for arterial cannulation. Alternative sites: transverse arch, femoral artery and axillary artery.
- Table 18.1 summarizes preoperative patient features identifying increased risk for cannulation complications.
- Table 18.2 lists some common complications of cannulation and cardiopulmonary bypass.
- Contraindications to aortic cannulation include aortic aneurysm, aortic dissection or severe atherosclerotic disease. Table 18.3 outlines options for the management of patients with a calcified or diseased aorta requiring cannulation.

Table 18.1. Preoperative risk factors for aortic cannulation complications

Peripheral vascular disease
History of stroke or documented cerebrovascular disease
Abdominal aortic aneurysm
Osteal stenosis of RCA or left main
Advanced age
Renal failure
Aortic calcification seen on CXR

Table 18.2. Complications of aortic cannulation

Bleeding	
Dissection:	0.02-0.2% with aortic cannulation
	0.2-3.0% with cannulation of femoral artery
	4.0% incidence arterial complications to leg
Atheroembolism	
Intramural placement	
Malposition	
Air embolism	

Table 18.3. Management options for patient with calcified aorta

Careful assessment of risk vs. benefits of procedure
Pedicle mammary artery grafts with fibrillatory arrest
Alternate sites of proximal anastomosis of venous conduits (ITA or innominate artery)
Replacement of ascending aorta using hypothermic circulatory arrest
Off pump bypass with pedicle arterial grafts

- Aids to determine site of cannulation in the aorta include palpation under controlled hypotension, epiaortic ECHO and the type of operation.
- Prior to commencing CPB check:
  - no air in aortic lines
  - visual pulsation in aortic line
  - carotid pulses present
  - activated clotting time (ACT) over 475 seconds
- If aortic line pressure is high during bypass:
  - tip of cannula malpositioned (selective cannulation of carotid/innominate arteries, cannula is against aortic wall)
  - occlusion by cross clamp
  - aortic dissection
  - kink in tubing
  - thrombosed arterial line filter
  - line clamp
  - cannula too small for attempted flow rate

Aortic Dissection Related to Cannulation and CPB

- differentiate from hematoma which is localized and superficial
- intraoperative dissection carries a high mortality rate. Key to successful treatment is early recognition.
- management:
  - femoral cannulation
  - hypothermic circulatory arrest
  - resect primary tear and replace aorta with graft
  - re-establish antegrade arterial perfusion
  - do not place cross clamp on dissected proximal aorta

Venous Cannulation

- gravity drainage system
  - no air
  - venous reservoir kept below patient level
- Determinants of flow:
  - central venous pressure
  - height difference between patient and reservoir bag
  - resistance to flow (cannula size)
- Venous cannulation techniques:
  - bicaval caval occlusion – Right heart operations
  - bicaval no occlusion – Mitral procedures
  - cavoatrial – Coronary bypass, aortic valve surgery

Trouble Shooting

1. Inadequate venous return

- must distinguish between continuous problem versus only with cardiac displacement.
- causes:
  - patient/reservoir bag height difference < 18 inches
  - inadequate volume
  - venodilators
  - cannula too small
  - kinked venous line
  - air lock
  - obstructed cannula
  - malpositioned cannula
- maneuvers to increase venous return:
  - reposition cannulae
  - additional atrial/femoral cannula
  - use of hard shell venous reservoir with suction
  - use of Biomedicus pump in venous return line

- complications of venous cannulation:
  arrhythmias
  poor venous return to the heart
  laceration and bleeding of the atrium
  laceration and bleeding from the vena cava
  tie tape related bleeding
  capture of monitoring lines
  displacement of pacemaker leads

2. Persistent left superior vena cava (LSVC)
   - 2.0–4.0% of patients with coronary artery disease
   - LSVC usually drains into coronary sinus
   - if adequate innominate vein may occlude LSVC during procedure
   - if small or absent innominate vein cannulate coronary sinus or LSVC directly
3. Unable to decompress right heart
   - ruptured sinus of Valsalva aneurysm (intra-cardiac left to right shunt)
   - atrio-ventricular septal defect (Gerbode's defect)
   - persistent LSVC
   - anomalous pulmonary venous drainage (pulmonary veins draining to right atrium)

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19 Myocardial Protection

Michael A. Borger, Richard D. Weisel

INTRODUCTION

Myocardial protection has contributed to the improved results of cardiac surgery. Recent reviews have suggested that modern methods of blood cardioplegia with integrated delivery systems have contributed independently to lower rates of morbidity and mortality.

Adequate myoprotection is an important aspect of the fast-tracking process for cardiac surgery patients and a good working knowledge of various cardioplegic techniques is essential for the practicing cardiac surgeon. Inadequate myoprotection increases the risk of postoperative low cardiac output syndrome, which in turn is an important cause of increased morbidity and mortality. Low cardiac output syndrome nullifies the beneficial effects of fast-tracking, with increased ICU and hospital lengths of stay and increased resource utilization.

This chapter will briefly examine important issues in myocardial protection including cardioplegia composition, temperature management, delivery techniques and future directions of research.

CARDIOPLEGIA COMPOSITION

Since 1984, we have used only blood-based cardioplegia at Toronto General Hospital. Blood cardioplegia improves postoperative ventricular function by increasing oxygen delivery and oxygen consumption, and by preserving myocardial high-energy phosphate stores when compared to crystalloid cardioplegia.

Initially the crystalloid component of our cardioplegia contained buffering agents (e.g., THAM, CPD), mixed at a blood to crystalloid ratio of 1:1 or 2:1. As we use higher blood to crystalloid ratios, however, we no longer feel these agents are necessary because of blood's potent intrinsic buffering mechanisms. Increased blood to crystalloid ratios may also decrease intraoperative volume administration, thereby minimizing end-organ edema and dysfunction.

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Our current cardioplegia solution is a blood-crystalloid composition mixed at a ratio of 8:1, with a high potassium solution for cardiac arrest and a low potassium solution for maintenance. The crystalloid component of our cardioplegia has been extensively simplified over time, now consisting of only potassium, magnesium and dextrose dissolved in sterile water (Table 19.1).

CARDIOPLEGIA TEMPERATURE

Cardioplegia temperature management has been extensively analyzed at our institution with initial work focusing primarily on hypothermia. Hypothermia decreases myocardial metabolism and is therefore useful when prolonged myocardial ischemia is anticipated. Decreased myocardial metabolism allows for prolonged cardioplegic interruptions thereby facilitating surgical exposure (Table 19.2). However, myocardial hypothermia delays the recovery of cardiomyocyte metabolism and function. In addition, deep hypothermia with topical cooling may impair other organs, contributing to postoperative atelectasis or phrenic nerve injury. For these reasons, we avoid deep myocardial hypothermia and do not use topical cooling.

The development of near continuous cardioplegia delivery, along with the known deleterious effects of hypothermia, led to the advent of warm cardioplegia. If warm cardioplegia were given continuously during the cross-clamp period, then the temperature-dependent mitochondrial enzymatic function could be preserved during cardioplegic arrest. After cross-clamp removal immediate recovery

Table 19.1. Toronto General Hospital cardioplegia solution composition

Component	High K Solution	Low K Solution
KCl	30 meq/L	8 meq/L
$MgSO_4$	6 meq/L	6 meq/L
Dextrose	50 mmol/L	50 mmol/L

Table 19.2. Advantages and disadvantages of warm vs. cold cardioplegia

Cardioplegia Type	Advantages	Disadvantages
Cold	↓ myocardial metabolism, enables cardioplegic interruptions for complex surgical procedures	delays cardiomyocyte recovery, impairment of local tissue function (topical cooling)
Warm	rapid recovery of myocardial metabolism, metabolic enhancement for patients with active ischemia	↑ risk of neurologic injury, requires near continuous delivery
Tepid	optimal temperature?	

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of normal ventricular metabolism and function would be anticipated. Preservation of normal myocardial metabolism during cardioplegia is particularly useful for patients who are actively ischemic upon presentation to the operating room in order to permit cellular repair while the heart is arrested but perfused.

We compared normothermic blood cardioplegia to hypothermic blood cardioplegia in a randomized trial of 1700 patients undergoing coronary bypass surgery and demonstrated a decreased incidence of postoperative low cardiac output syndrome in the patients receiving warm cardioplegia. However, investigators at Emory University in Atlanta found an increased risk of stroke and other neurologic events during warm cardioplegia and normothermic cardiopulmonary bypass. These investigators employed systemic warming to 37°C and a partial occluding clamp for proximal anastomoses, and the combination of hyperthermic cerebral perfusion and cerebral emboli may have resulted in the high incidence of postoperative neurologic events.

Recently we have evaluated "tepid" cardioplegia, which produces a myocardial temperature near 29°C and found that this technique resulted in more rapid recovery of myocardial metabolism and ventricular function than warm (37°C) or cold (4°C) cardioplegia (producing cardiac temperatures of 37°C and 18°C, respectively). Currently, cardiac surgeons at our institution employ mild to moderate hypothermic cardioplegia (myocardial temperature 18-29°C) in combination with mild hypothermic cardiopulmonary bypass (systemic temperature 34°C). We feel this strategy simultaneously optimizes cerebral and myocardial protection.

CARDIOPLEGIA DELIVERY

Cardiac surgeons have several options for cardioplegia delivery including antegrade (via the aortic root or coronary ostia), retrograde (via the coronary sinus), and combined cardioplegia (antegrade via saphenous vein grafts plus retrograde). The optimal delivery technique should be tailored to each patient and more complicated operative procedures often require more complex myoprotective strategies.

Antegrade cardioplegic delivery via the aortic root is the most common technique employed by cardiac surgeons. It is used to arrest the heart immediately after cross-clamp application (with high potassium solution) in the majority of patients at TTH. We administer 500-1000 ml of antegrade cardioplegia (more if left ventricular hypertrophy is present) at maximum achievable flow with an aortic root pressure of 70 mm Hg. The left ventricle is continuously observed in order to ensure dilatation, secondary to aortic valve incompetence, does not occur. Direct cannulation of the coronary ostia is frequently used for aortic valvular procedures particularly if aortic incompetence is present. After the initial arresting dose of antegrade cardioplegia, low potassium cardioplegia (300 ml) is administered intermittently throughout the procedure in order to maintain cardiac arrest. For coronary bypass surgery, maintenance doses are given after each anastomosis. For valvular surgery, maintenance doses are given every 15-20 minutes.

The cardioplegia cannula is used as a vent (attached to atmospheric drainage only) between maintenance doses. Careful de-airing of the aortic root is employed before each maintenance dose.

The main advantage of antegrade cardioplegia is its ease of administration (Table 19.3). However, antegrade cardioplegia is contraindicated in the presence of severe aortic insufficiency and is relatively contraindicated in reoperative coronary bypass surgery (because of the risk of saphenous vein graft embolization during dissection of the heart). In addition, antegrade cardioplegia may be technically difficult to deliver during aortic valve surgery and may result in suboptimal myocardial perfusion distal to occlusive coronary artery disease. Retrograde cardioplegia via the coronary sinus was therefore developed as an alternative to antegrade delivery. Retrograde cardioplegia allows for near continuous delivery and may be ideally suited for warm heart surgery. Visualization of coronary artery anastomoses is better than during antegrade delivery, and therefore cardioplegic interruptions are minimized. However, retrograde cardioplegia results in decreased perfusion of the right ventricle and posterior interventricular septum when compared to antegrade delivery. In addition, retroplegia may result in a large amount of nonnutritive flow through Thebesian channels with subsequent inhomogeneous cardioplegic administration.

The technique of retrograde cardioplegia involves insertion of a catheter into the coronary sinus either prior to initiation of cardiopulmonary bypass or with partial interruption of venous return during cardiopulmonary bypass (in order to distend the right atrium and facilitate cannulation of the coronary sinus). Cardioplegia is administered throughout the procedure at 200 ml/minute, ensuring that the coronary sinus pressure does not exceed 40 mm Hg (excessive pressure may result in endothelial damage or coronary sinus rupture). The aortic root is simultaneously vented during cardioplegic delivery. De-airing of the coronary sinus between cardioplegic doses is not necessary.

Table 19.3. Advantages and disadvantages of various cardioplegia delivery techniques

Method of Delivery	Advantages	Disadvantages
Antegrade	simple to use	contraindicated in severe AI, risk of coronary embolization in redo CABG, requires frequent interruptions, difficult during AV surgery, ↓ perfusion distal to coronary occlusions
Retrograde	near continuous delivery	↓ perfusion to RV and posterior septum, nonnutritive Thebesian flow
Combined	maximizes myocardial perfusion	relatively complex delivery system

The advantages of both antegrade and retrograde delivery can be optimized during coronary bypass surgery by using combined cardioplegia. Combination cardioplegia results in better myocardial perfusion compared to antegrade or retrograde delivery alone, but has the disadvantage of requiring a relatively complex delivery system (Table 19.3). We use a single pump head to deliver 200 ml/min of cardioplegia simultaneously to the coronary sinus and to completed saphenous vein grafts. Care is taken to ensure that the coronary sinus pressure does not exceed 40 mm Hg. Venting of the aortic root is performed throughout the procedure. We found that flow rates less than 200 ml/min produced inadequate perfusion and induced anaerobic lactate production. Higher flow rates did not increase nutritive myocardial perfusion, but rather increased perfusion into the left ventricular cavity through Thebesian channels.

Near continuous cardioplegic delivery often results in the administration of large volumes of cardioplegia, which in turn may cause hyperkalemia and postbypass pacemaker dependence. At TTH, we minimize hyperkalemia by using the following stepwise approach:

- serum $K^+ > 5.0$ mmol/L—remove crystalloid component of cardioplegia
- serum $K^+ > 5.5$ mmol/L—furosemide 40 mg i.v. and/or insulin 10 units i.v.
- serum $K^+ > 6.0$ mmol/L—consider ultrafiltration.

FUTURE DIRECTIONS

Future research in myocardial protection will most likely focus on high risk patients where operative procedures continue to result in significant cardiac morbidity and mortality. Ischemic preconditioning is the most powerful endogenously-mediated form of myocardial protection, but is difficult to apply clinically. Pharmacologic additives, such as adenosine, nicorandil, or nitric oxide precursors may be able to reproduce the effects of ischemic preconditioning. Restoration of aerobic myocardial metabolism through agents such as insulin and dichloroacetic acid offers another potential mechanism of myoprotection. These agents are currently undergoing rigorous evaluation at Toronto General Hospital and other investigative centers.

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Surgery of Heart Valve, the Aortic Root, Ascending Thoracic Aorta and Transverse Arch

Tirone E. David

Heart valve disease is the second most common indication for open-heart surgery in adults. Calcific aortic stenosis and mitral regurgitation due to myxomatous disease of the mitral valve are now the most common causes of heart valve disease in North America. A large number of patients with rheumatic heart disease are still seen, particularly among immigrants from developing countries.

Aneurysms of the proximal thoracic aorta are common. Degenerative disease of the media is the principal cause of these aneurysms. They may present as chronic enlargement of the proximal aorta or as an acute or chronic type A aortic dissection. Bicuspid aortic valve is found in approximately 15% of patients with proximal aortic aneurysms. Surgery is recommended when the transverse diameter of the ascending aorta reaches 60 mm. In patients with the Marfan syndrome, operation should be performed when the diameter of the aortic root reaches 55 mm. If the aortic valve can be repaired in patients with aortic root aneurysms, we now recommend surgery when the transverse diameter reaches 50 mm. Patients with the Marfan syndrome and family history of acute aortic dissection, replacement of the ascending aorta and proximal arch should be performed when the diameter exceeds 40 mm. Some patients with degenerative disease of the aorta may develop aneurysm of the entire thoracic and sometimes abdominal aorta. This condition is referred to as "mega-aorta syndrome".

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AORTIC VALVE REPAIR

Aortic Stenosis

- Decalcification of senile aortic valve disease should be performed only in elderly patients with limited life span and with mild or moderate aortic stenosis (AS) in whom the primary indication for surgery is coronary artery disease.
- Percutaneous balloon valvotomy is sometimes performed in adult patients with advanced AS to improve their general condition in preparation for surgery.

Aortic Regurgitation

- Aortic valve repair is possible in at least one-half of the patients with aortic insufficiency (AI) caused by dilatation of the aortic root who have a tricuspid aortic valve, including patients with the Marfan syndrome.
- Congenitally bicuspid aortic valve with AI due to prolapse of the anterior leaflet (the one attached to the septum) can also be repaired in children and young adults.
- AI due to subaortic ventricular septal defect can also be corrected by means of valve repair.

Operative Technique

- Leaflet prolapse is repaired by a triangular resection of the central portion of the leaflet. If the prolapsing leaflet is thinned and overstretched, it can be reinforced with a double layer of 6-0 Gore-Tex suture along its free margin.
- AI due to dilatation of the aortic root is repaired by aortic root remodeling or reimplantation of the aortic valve.

AORTIC VALVE REPLACEMENT

- Practically all adult patients with severe AS and those with AI caused by diseased leaflets require aortic valve replacement (AVR).
- There are two types of valves for AVR: mechanical and biological.
- Mechanical valves are durable but require permanent anticoagulation with warfarin sodium. The INR should be maintained at 2-3. In carefully anticoagulated patients the risk of hemorrhage is of 1-2% per year, and the risk of thromboembolic events is also 1-3% per year.
- Biological valves are less durable than mechanical valve but do not require anticoagulation and the risk of thromboembolic events is 1-2% per year for stented and even lower for stentless biological valves.
- Matching the patient to the type of artificial heart valve is an important clinical decision. Patients should be well informed of the pros and cons

of each type of valve. The patient's aortic pathology, associated diseases, age, education, life style and living conditions must be considered. Because biological valves have a limited durability, the risk of reoperation must be considered in patients who may out-live their valves. Thus, the risk of life-long anticoagulation with mechanical valves should be weighed against the risk of reoperation with biological valves. Biological valves should be used in patients who cannot be anticoagulated, those who do not wish to be anticoagulated, and in those whose life span is shorter than the life span of the biological valve.

- Another important aspect in matching the patient to the valve is the hemodynamic result of AVR. Most prosthetic aortic valves are obstructive and mean transvalvular systolic gradients of 15-20 mm Hg are common after AVR. When prosthetic valves with small effective orifices are implanted in large patients, unacceptably high gradients may result and adversely effect the clinical outcome of the operation The effective orifice index of prosthetic aortic valves should be at least 0.9 cm^2/m^2.
- After AVR all patients are at risk of developing infective endocarditis which is relatively high (1-2%) in the first year but decreases thereafter (0.2-0.5% per year).

Operative Technique

- Incision: full median sternotomy or upper-half median sternotomy (minimal access surgery)
- Cannulation of ascending aorta and right atrium for cardiopulmonary bypass. Mild systemic hypothermia (we allow the temperature to fall to 34°C). We prefer a transverse aortotomy for all cases of AVR. Both coronary artery orifices are cannulated and continuous cold blood cardioplegia is given at 150-200 ml/min depending on left ventricular mass. In patients with concomitant coronary artery disease, retrograde coronary sinus cardioplegia can be used.
- The diseased aortic valve is completely excised. All calcific deposits should be removed from the annulus and anterior leaflet of the mitral valve. The annulus is sized with a specific sizer made by the valve manufacturers. If the annulus is small (less than 23 mm), consideration should be given to patch enlarge the aortic annulus or to use a different type of valve (stentless biological valves have much greater effective orifice than stented ones for any given aortic annulus). If coronary artery bypass is needed, the grafts are done before implantation of the aortic valve.
- Mechanical valves should be secured with multiple interrupted sutures of 2-0 polyester and stented bioprosthetic valves with multiple horizontal mattress sutures with small pledgets.
- The aortotomy is closed in single or double layer with polypropylene sutures. Vein grafts are anastomosed to the ascending aorta. The left side of the heart is carefully deaired and the aorta unclamped.

Cardiopulmonary bypass is discontinued within 5-10 minutes if continuous blood cardioplegia was used.

Aortic Valve Replacement with Stentless Biological Valves

AVR with stentless biological valves (pulmonary autograft, aortic homograft and aortic xenograft) is a more complex operative procedure than AVR with stented valves. These valves can be implanted in the sub-coronary position or as a root replacement (root inclusion or full root). A sound knowledge of the functional anatomy of the aortic root is necessary to implant stentless valves in the sub-coronary position. The diameter of the aortic annulus and the diameter of the sinotubular junction are important geometric features of the normal aortic root and must be considered during these operations.

Pulmonary Autograft

- AVR with pulmonary autograft (Ross procedure) consists of replacing the diseased aortic valve with the patient's own pulmonary valve, and in implanting a semilunar valve homograft (e.g., pulmonary valve homograft) in the pulmonary position. The pulmonary autograft can be implanted in the subcoronary position or as a root inclusion or as a root replacement. The sub-coronary implantation technique should be used only in patients in whom the geometry of the aortic root is similar to the geometry of the pulmonary root. We believe that the root inclusion technique is better than full root replacement because of uncertainties regarding the fate of the pulmonary sinuses in the systemic circulation (which may dilate excessively and cause AI).
- Pulmonary autograft failure is rare in carefully selected patients. Failures are usually due to technical problems or wrong patient selection.
- The Ross procedure is a technically demanding operation.
- It is ideal for children and young adults to avoid anticoagulation.
- It should not be used in patients with annuloaortic ectasia or dilated aortic root. The pulmonary root has the same embryologic origin as the aortic root and may dilate when transferred to the systemic circulation because of increased stress.
- A pulmonary homograft is better than an aortic homograft for reconstruction of the right side during this operation. Freedom from pulmonary homograft stenosis varies with recipient's age (the younger the worse); in young adults approximately 20% will require reoperation at 10 years because of stenosis.

Aortic Homograft

- AVR with aortic valve homograft is performed using the same techniques as for any stentless biological valves (sub-coronary implantation, root inclusion or root replacement). Most surgeons favor the root replacement because it is the simplest and gives the most predictable functional results.

- Durability of aortic valve homograft is limited; at 10 years approximately 20% of the patients will require reoperation. The failure occurs earlier in younger patients.
- Aortic valve homograft is particularly useful in patients with aortic root abscess.
- Procurement is a problem. Valves from donors older than 40 years of age are often not good.

Stentless Xenograft Aortic Valves

There are numerous stentless xenograft valves available for AVR but only the Medtronic Freestyle (Medtronic, Minneapolis, MN) and the Toronto SPV (St. Jude Medical, St. Paul, MN) are approved for clinical use in North America. Both are porcine valves: the Freestyle is a root and the Toronto SPV is a fully scalloped valve. The Medtronic Freestyle can be implanted as a root or can be trimmed and used in the subcoronary position. The Toronto SPV can be implanted only in the subcoronary position.

- The hemodynamic features of stentless porcine aortic valves are excellent and comparable to those of aortic homograft. The mean systolic transvalvular gradients are usually lower than 10 mm Hg regardless of the size of valve implanted.
- The superior hemodynamic features of these valves may have a beneficial effect in patients' long term survival.
- The clinical outcomes have been comparable to those of other bioprosthetic valves.
- It is unknown whether these stentless valves are more durable than stented porcine valves because they have been used for less than a decade.

AVR in Patients with Infective Endocarditis

Patients with infective endocarditis may need urgent or emergent AVR because of acute AI, large vegetations, aortic root abscess and/or false aneurysms and fistulas. The single most important aspect of surgery for active infective endocarditis is complete resection of all infected tissues and implantation of a new valve in a clean area. If a paravalvular abscess is present, it is necessary to radically excise the abscess and adjacent tissues to eradicate the infection. The left ventricular outflow tract and surrounding structures can be reconstructed with fresh autologous pericardium or glutaraldehyde fixed bovine pericardium. Although not indispensable for a satisfactory clinical outcome, aortic valve homografts are ideal for these patients.

Postoperative Care

- Routine ICU care as for any patient undergoing open-heart surgery.
- We do not anticoagulate patients with biological aortic valves; they are given aspirin. Patients with mechanical valves are anticoagulated with warfarin sodium starting on the 1st POD. We give heparin only if they cannot take po medications (see appendix 9).

- Approximately 5% of patients require permanent transvenous pacemaker.
- An echocardiogram is performed on the 5th or 6th POD before discharge to rule out valve malfunction, pericardial effusion, and to assess ventricular function.

MITRAL VALVE REPAIR

Mitral Stenosis

- Rheumatic fever is practically the only cause of mitral stenosis (MS). Percutaneous balloon valvotomy provides excellent functional results in young rheumatic patients with pure MS and pliable leaflets. Open valvuloplasty is reserved for patients with associated valve disease such as aortic and/or tricuspid. It consists of commissurotomy, resection of thickened chordae tendineae, and splitting of papillary muscles to increase leaflet mobility.

Mitral Regurgitation

- A variety of pathologic processes can cause mitral regurgitation (MR). The most common are myxomatous diseases of the mitral valve, coronary artery disease and rheumatic fever. Myxomatous disease of the mitral valve is the most common cause of MR in North America. Over 75% of all patients with MR due to myxomatous disease of the mitral valve are suitable for mitral valve repair and the long-term results are excellent. The physiopathology of MR caused by coronary artery disease is not well understood, and consequently, repair gives less than satisfactory results. Rheumatic MR can be satisfactorily repaired when the leaflets are thin and pliable. The ideal pathology for mitral valve repair is myxomatous degeneration of the mitral valve.
- Prolapse of the posterior leaflet is usually corrected by quadrangular or rectangular resection of the prolapsing segment. The annulus is plicated and the posterior leaflet is repaired. Plication of the annulus in a single area should be avoided in patients with dominant circumflex artery and in those in whom more than 2 cm of leaflet is resected. For those, 2-3 cm of nonprolapsing posterior leaflet is detached from the annulus and a sliding plasty is performed.
- Prolapse of the anterior leaflet is corrected by chordal transfer or chordal replacement with 5-0 Gore-Tex sutures.
- A ring annuloplasty is often necessary during repair of the mitral valve for long-standing MR. We believe that posterior annuloplasty with a Dacron ring is sufficient for patients with myxomatous disease of the mitral valve.

MITRAL VALVE REPLACEMENT

- Most patients with MR due to ischemic heart disease, MS or MR due to rheumatic heart disease and advanced myxomatous disease need mitral valve replacement (MVR).
- In patients with rheumatic valves and those with advanced myxomatous changes in both leaflets, the mitral valve should be completely excised and each major papillary muscle trunk should be resuspended with a double armed 4-0 Gore-Tex suture. This suture is secured to the fibrous portion of the papillary muscle first and then passed through the mitral annulus and tied together. It should be approximately 1.0-1.5 cm long. After implanting the prosthetic mitral valve, the suture is passed through the sewing ring of the valve and the ends tied together again.
- In patients with ischemic MR, the sub-aortic portion of the anterior leaflet should be excised and the commissural areas of the anterior and the entire posterior leaflet are left intact.
- 12-14 sutures of 2-0 polyester with pledgets are passed through the mitral annulus and the sewing ring of the prosthetic valve. The retained native leaflets are incorporated in these sutures. The pledgets should be left on the atrial side when part of the leaflets are retained and can be left in either side of the annulus when the leaflets are completely excised.
- Either a mechanical or a bioprosthetic valve can be used for MVR. The risk of thromboembolic complication is similar with both types of valves but the first requires anticoagulation with warfarin sodium and the second does not. Bioprosthetic valves in the mitral position are not as durable as in the aortic position.
- Preservation of the native chordae tendineae and leaflets or resuspension of both papillary muscles with Gore-Tex sutures prevent the dreadful complication of posterior wall rupture after MVR.
- The risk of hemorrhagic and thromboembolic complications after MVR is higher than after AVR.

Complex Mitral Valve Surgery

Mitral valve surgery in patients with heavily calcified mitral annulus, mitral annulus abscess due to infective endocarditis, or destroyed mitral annulus because of previous MVR can be technically very demanding and associated with high operative mortality and morbidity. We believe that the best approach is to completely excise the diseased mitral annulus (calcium bar, abscess, or scar with old pledgets) and reconstruct it with either autologous pericardium or glutaraldehyde fixed bovine pericardium. When the entire mitral annulus needs reconstruction, a strip of pericardium 2 cm wide and 10-12 cm long is sutured to the endocardium of the left ventricle posteriorly and to the intervalvular fibrous body superiorly. The other end of the pericardial patch is sutured to left atrial wall (care

must be taken to avoid damage to the circumflex artery which is close to the left atrial wall). If only a portion of the annulus is destroyed, a semi-circular patch is used to repair the annulus. The prosthetic valve is secured to the reconstructed mitral annulus.

Postoperative Care

- Routine ICU care as for any patient undergoing open-heart surgery.
- We anticoagulate all patients who had mitral valve surgery with warfarin sodium which is started on the 1st POD. They are also started on i.v. heparin on the 1st POD. Patients with bioprosthesis and after mitral valve repair are anticoagulated for 3 months if they are in sinus rhythm and the INR is maintained between 2 and 3. Patients with mechanical valves are anticoagulated permanently and the INR is maintained between 3.0 and 3.5 (see appendix 9).
- An echocardiogram is obtained on the 5th or 6th POD to rule valve malfunction, pericardial effusion, and to assess ventricular function.

TRICUSPID VALVE REPAIR

Isolated tricuspid valve disease is uncommon. It is usually associated with mitral valve disease, particularly in patients with rheumatic heart disease and pulmonary hypertension. Rheumatic involvement of the tricuspid valve is uncommon; it can produce stenosis or a mixed lesion. The most common tricuspid valve lesion is regurgitation due to right ventricular dilatation due to pulmonary hypertension or right ventricular dysfunction; it can also occur after MVR in patients with normal pulmonary artery pressure, and it is believed to be due to distortion of the tricuspid annulus caused by the prosthetic mitral valve. Patients who need MVR and have more than mild tricuspid regurgitation should have concomitant tricuspid annuloplasty. Various techniques can be used to reduce the tricuspid annulus area to increase the coaptation of the leaflets. The following is the one we have used for years and the results have been satisfactory.

- A double layer of a 2-0 polypropylene suture is passed in and out of the anterior and posterior portions of the tricuspid annulus. Small teflon felt pledgets are used to buttress this suture in multiple places to prevent it from cutting through the annulus. The tricuspid annulus is reduced to an area approximately equal to the area of the anterior leaflet of the tricuspid valve.

TRICUSPID VALVE REPLACEMENT

The tricuspid valve should be repaired whenever possible because the results of tricuspid valve replacement (TVR) are less than satisfactory in most patients. Both mechanical and bioprosthetic valves can be used in the tricuspid position.

- The anterior and posterior leaflets are excised but the septal leaflet should be left in if possible. The prosthetic valve is secured to the tricuspid annulus and septal leaflet with multiple horizontal mattress sutures of 2-0 polyester. If pledgets are used they can be left in either side of the annulus.
- A permanent pacemaker lead should be implanted on the right ventricular epicardium after TVR in case of heart block or atrial fibrillation with slow ventricular rate.

MAZE PROCEDURE FOR ATRIAL FIBRILLATION

The Maze III procedure consists in making certain incisions in the right and left atria to reduce the number of reentrant circuits available to maintain atrial fibrillation. This operation can be performed in isolation or combined with other operative procedures. We believe it should be combined with mitral valve repair and MVR with bioprosthetic valves to avoid long term anticoagulation in patients with long-standing atrial fibrillation. Atrial pacing is often needed soon after the maze procedure; it takes 5-10 days for most patients to regain sinus rhythm. Approximately 20% of patients require a permanent pacemaker because of sinus node dysfunction. These patients often retain fluid postoperatively and need diuretics during the first month. We anticoagulate our patients during the first 3 postoperative months.

SURGERY FOR CHRONIC ASCENDING AORTIC ANEURYSM

- We cannulate the distal aortic arch for arterial return and the right atrium for venous drainage for CPB.
- If the aneurysm is limited to the proximal one-half of the ascending aorta, resection and replacement can be performed with a clamp on the distal ascending aorta. If the aneurysm extends to the origin of the innominate artery, resection and replacement is performed under hypothermic circulatory arrest and retrograde cerebral perfusion (see chapter 15).
- Continuous antegrade cold blood cardioplegia is used for myocardial protection.
- The aortic valve is often dysfunctional in patients with aortic root/ascending aortic aneurysms. If bicuspid, AVR should be performed unless the aortic root and the aortic valve are normal which is unusual.
- If the aortic root is dilated but the three aortic valve leaflets are fairly normal, an aortic valve sparing operation can be performed. Otherwise, composite replacement of the aortic valve and ascending aorta with reimplantation of the coronary arteries is performed.

- If the aortic root is normal, simple supracoronary replacement of the ascending aorta is performed with a tubular dacron graft.
- If the aortic sinuses are not dilated and the aortic valve is incompetent in spite of fairly normal leaflets, dilation of the sinotubular junction is probably the cause of valve dysfunction. Simple adjustment of the sinotubular junction to a diameter slightly smaller than the length of the free margins of the leaflets corrects the aortic valve incompetence. This can be accomplished as the graft is sutured to the sinotubular junction of the aortic root. If the aortic valve leaflets are abnormal, AVR and supracoronary replacement of the ascending aorta is performed.

SURGERY FOR MEGA-AORTA SYNDROME

- We prefer to cannulate the aneurysm directly for arterial return for CPB. This is best done with echocardiographic guidance to avoid a calcified plaque or laminated thrombi.
- The patient is cooled to 20°C and the circulation is arrested. Retrograde cerebral perfusion is used.
- Retrograde or antegrade blood cardioplegia is used for myocardial protection.
- The transverse arch is open widely. The brachiocephalic arteries are separated from the aneurysm leaving 1 cm of arterial wall around the orifices. A 30 cm long tubular dacron graft is used. The diameter of the graft should be from 26-32 mm depending on the size of the patient. Grafts of small diameter may increase afterload and cause heart failure. One of the ends of the graft is inverted inside of itself to reduce its length to approximately one-half. The graft is placed in the descending thoracic aorta aneurysm and the folded ended is sutured to the proximal descending thoracic aorta using a continuous 3-0 or 2-0 polypropylene suture. Large bites are used but they should be close together to prevent anastomotic leakage. The graft is then everted and an opening is made in its superior aspect to reimplant the brachio-cephalic arteries with a continuous 4-0 or 3-0 polypropylene suture. An opening is made in the graft and an arterial cannula inserted for antegrade perfusion. This technique of a loose end of the graft inside the descending thoracic aneurysm is called "elephant trunk".
- The proximal ascending aorta and aortic root are dealt as described above.

SURGERY FOR ACUTE TYPE A AORTIC DISSECTION

- Operation should be performed as soon as the diagnosis is established because delaying it increased operative mortality and morbidity.

- A femoral artery and the right atrium are cannulated for CPB. If there is any evidence of malperfusion when bypass is commenced, an axillary artery should be cannulated for arterial return.
- The ascending aorta should be not be clamped. The patient is cooled to approximately 24°C and the circulation is arrested. Retrograde cerebral perfusion is used.
- The ascending aorta should be transected 0.5 cm from the orifice of the innominate artery and the transverse arch is explored. If it contains no intimal tears, only the ascending aorta is replaced. If the arch contains a tear, it also should be replaced.
- A dacron graft of appropriate diameter is sutured to the inside of the artery using a fine cardiovascular needle and extreme care is exercised to avoid intimal tears as the needle is passed through the dissected aorta.
- The femoral or axillary cannula is removed and inserted into the graft used to replace the ascending aorta to establish antegrade arterial perfusion.
- The aortic root is repaired or replaced depending on the extensiveness of the dissection and aortic valve pathology.

Postoperative Care

- Bleeding is a major problem in these patients and meticulous intraoperative hemostasis is indispensable. In patients who underwent hypothermic circulatory arrest and in those with acute aortic dissections, transfusion of platelets and cryoprecipate is often necessary for normal hemostasis.
- Hypertension should be treated promptly early after surgery, particularly in patients with acute type A aortic dissection.
- Beta-blocker therapy should be given to patients with aortic dissections and in those with the Marfan syndrome.
- These patients should be followed with annual CT or MR scans.

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Heart Transplantation

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21

The development of experimental organ transplantation began in 1905 when Drs. Carrel and Guthrie (France) performed an extrathoracic heterotopic canine transplant. Limited by a lack of cardiopulmonary bypass research it was not until the 1960s that Shumway (US) perfected the technique for intrathoracic canine orthotopic heart transplantation. Hardy (US) in 1964 performed a xenotransplant of a chimpanzee's heart into a human. In December 1967 Dr. C.N. Barnard (South Africa) opened the early era of heart transplantation by performing the first successful human orthotopic heart transplant. Despite a flurry of early heart transplants and some favorable long-term results, rejection and infection became insurmountable obstacles and the procedure lost popularity. The introduction of Cyclosporine in the 70s revolutionized transplantation to what it is today and ushered in the second era of heart transplantation. In Toronto heart transplants have been performed in both eras.

RECIPIENT

The majority of patients referred to Toronto General Hospital heart failure team have ischemic (60%) or idiopathic (25%) cardiomyopathy with the minority having valvular or congenital problems.

Currently accepted indications and contraindications for heart transplantation are presented in Table 21.1.

Potential candidates for heart transplantation undergo a thorough evaluation (Table 21.2) and are assessed by a multidisciplinary committee to allow for the equitable distribution of scarce donor resources. The average time to transplant at TTH is 165 days. Across Canada there is a priority staging system for status based on clinical condition (5 status categories).

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

Table 21.1. Indications and contraindications

Indications
1. end stage heart disease refractory to other surgical or medical management
2. NYHA III–IV symptoms with maximal medical therapy and prognosis for 1 year survival < 75%
3. no other major organ or system disease
4. emotionally stable with social support
5. medically compliant and motivated
Contraindications
1. incurable malignancy
2. major system illness
3. irreversible major organ disease
4. active systemic infection
5. emotional instability
6. ? age
7. obesity
8. irreversible pulmonary hypertension

Table 21.2. TTH precardiac transplant assessment protocol

Consultation	**Investigation**
cardiology	right and left heart catherizations
cardiovascular surgery	two-dimensional echocardiogram
respiratory	electrocardiogram
transplant immunology	pulmonary function tests
psychiatry	arterial blood gases
psychology	chest x-ray
dental surgery	abdominal ultrasound
social work	antibody screen HBV, HCV, HIV
chaplaincy	HLA typing, anti-HLA antibodies
transplant co-ordinator	digoxin levels
	antibody titres CMV, herpes simplex, Epstein Barr, toxoplasmosis
Laboratory	
ABO blood type and screen	
CBC, ESR, smear, reticulate	
PT, PTT, bleeding time	
lytes, BUN, creatinine, uric acid	
glucose (fasting, 2hr PC)	
cholesterol, triglycerides	
liver function tests	
protein electrophoresis	
thyroid function tests	
stools (parasites, c/s, blood)	
urinalysis, 24hr Cr clearance and protein	

Patients are optimized medically with digoxin, diuretics and vasodilators (ACEI, hydralazine and β-blockers). Outpatient dobutamine is not used as the results have been less than promising. Decompensated patients are admitted to hospital and managed with IV inotropes or vasodilators. At TTH mechanical support is limited to endotracheal intubation and the IABP as ventricular assist devices are not used as a bridge to transplantation. More recently, in carefully selected patients, salvage surgery for ischemia, valves and left ventricular volume reduction has been performed.

The recipient is admitted to hospital when an appropriate donor is identified. Most patients have elevated pulmonary artery pressures, which may be easy to control if treatment is commenced a few hours prior to transplantation. Although a patient may be on the list, recent uncontrolled pulmonary hypertension will preclude a transplant. While no absolute rules exist, general guidelines for maximally acceptable pulmonary artery pressure (PAP) include: a) absolute systolic PAP > 45 mm Hg, b) PAP which are 1/2 systemic (provided systemic > 80 mm Hg), c) calculated pulmonary vascular resistance < 6 woods, d) transpulmonary gradient = mean PAP - mean PCWP < 10-15 mm Hg.

Because the number one cause of pulmonary hypertension in these patients is an increase in total body water, most patients will receive an intravenous diuretic. It is imperative that this practice of minimal fluid is maintained in the operating room prior to surgery. Any hypotensive episodes should be treated with inotropes and not fluids.

DONORS

The availability of donor organs remains the major limiting factor to widespread heart transplantation. This has led to a plateau of the number of annual heart transplants at 4000 cases worldwide. To address this issue newer broader donor eligibility criteria have been suggested.

At TTH we consider donor hearts from patients up to 50-55 years of age although the risk of atherosclerosis increases with age. In Canada allocation of donor organs is made through the M.O.R.E. (Multiple Organ Retrieval and Exchange) program. A potential heart donor undergoes a three stage screening process which includes:

- M.O.R.E.: age, gender, height/weight, ABO, cause of death, CMV, HBV, HIV, HCV status
- cardiologists: age < 55, hemodynamic stability (dopamine < 20 ug/kg/hr) ECG, CXR, ABG, Echo, ± angiogram, absence of severe hypotension, prolonged cardiac arrest, septicemia
- surgeons: intraoperative inspection by harvest team

Matching is according to blood type and body size and weight. It is mandatory to ABO match to avoid hyperacute rejection. The donor body weight should be within 25% of the recipient's. Recipients with high pulmonary pressures are considered high-risk recipients and may benefit from a larger heart. A HLA screen of

antibodies (Panel Reactive Antibodies = PRA) is performed to minimize incompatibility but due to time constraints full HLA tissue matching is not usually available at the time of transplant.

SURGICAL TECHNIQUE FOR DONOR HARVEST

At TTH the harvest team consisting of 1-2 cardiac surgery fellows is sent to the recipient hospital to examine the donor heart. If there is any evidence of coronary disease or of graft (heart) dysfunction, the heart is generally not used for transplantation but is used for homograft valves. In a 2-year period, only 38% of the hearts offered for donation were actually used for transplantation. Of those rejected hearts, 66% were used for homograft valves.

Recently, our transplant team moved to the surgical implantation technique of bicaval anastomosis. This has affected organ procurement as now both superior and inferior vena cava are transported to the recipient site. The superior vena cava (SVC) should be excised superior to the azygous vein and, if possible, the brachiocephalic (innominate) vein. As much pulmonary artery as possible should be obtained. This is obviously difficult when lungs are being harvested at the same time. For some recipients with congenital anomalies, concomitant donor lung harvesting is a contraindication as these patients may need more pulmonary artery. If the lungs are not used, then the pulmonary artery should be taken at the pulmonary hila.

Once all of the harvesting teams have adequately prepared their organs, the cardiac team determines when the aortic cross-clamp goes on and cardioplegic solution is administered to the heart and any other organs. Myocardial protection is afforded by 2-3 liters of cold crystalloid cardioplegia. Very little is done clinically to enhance this cardioplegic solution. The heart is then excised and placed in cold saline and double bagged. Cold remains the cornerstone of organ preservation so the donor heart is transported in an ice cooler at 4-6°C. Donor ischemic time is defined as the time from aortic cross-clamp at harvest to cross-clamp release following implantation and should be less then 4-6 h.

SURGICAL TECHNIQUE FOR ORTHOTOPIC HEART TRANSPLANT

The transplant recipient is approached through a standard median sternotomy. Venous drainage is established by directly cannulating both vena cavae with right angle cannulae which facilitates the cava-to-cava transplant technique. Arterial return is via the ascending aorta or aortic arch.

Total cardiopulmonary bypass is established and the heart is excised. Initially the right atrium and interatrial septum are incised, then the left atrial free wall and finally the aorta and pulmonary artery are divided. The posterior wall of both the left and right atria are left intact.

A sump is placed through the right superior pulmonary vein of the recipient in order to drain collateral blood flow from the lungs.

The donor heart is given 1000 cc of recipient blood cardioplegia into the aortic root. It is given initially at room temperature and then cooled to 10-15°C after the first 300-500 cc infusion. Subsequent doses of 300-500 cc are given after each of the major anastomoses.

The donor heart is prepared by incising between the pulmonary vein openings and removing any excess tissue.

The first suture line is started at the left atrial appendage of the donor heart taking care to align it with the remnant of the recipient left atrial appendage. The free wall of the left atrium is sewn and then the interatrial septum. The sump is repositioned into the left ventricle of the donor heart prior to completing the suture line of the left atrial roof.

The right atrium of the donor heart is connected to the recipient using either the atrial-to-atrial connection or the direct vena cava to vena cava connection. In the direct atrium to atrium connection the right atrium of the donor heart is incised from the inferior vena cava (IVC) to the atrial appendage. The opened atrium is sewn to the remnant of the recipient right atrium. In the cava-to-cava technique the recipient inferior vena cava is sewn to the IVC opening of the donor right atrium and the superior vena cava of the donor and recipient are connected. The cava-to-cava technique is preferred. It results in better right atrial function and fewer episodes of bradyarrythmias post-transplantation.

The pulmonary artery catheter is manually directed through the right ventricle and into the right pulmonary artery at this time. The recipient and donor pulmonary arteries are connected followed by anastomosis of the recipient and donor aorta.

Once the cross-clamp is removed, air is carefully removed from the heart and the sump is removed. After adequate reperfusion an appropriate inotropic agent, usually isoproterenol is started to help wean the heart from bypass. Other inotropic agents are introduced if necessary. The surgeon must carefully observe the heart as it is being weaned from bypass. Right ventricular function is often the limiting factor in coming off bypass and this is best assessed by direct observation. The normal right ventricle of a donor heart is particularly sensitive to high afterload and distention must be avoided. Right ventricular function is also adversely affected by small air emboli that are entrapped in the right coronary circulation. Further measures must be taken to minimize pulmonary vasoconstriction such as avoiding acidosis or hypercarbia. Communication amongst surgeon, anesthesiologist and perfusionist is critical at this time.

If pulmonary hypertension is a concern, nitric oxide is administered via the endotracheal tube. This has the advantage of directly dilating pulmonary vessels without producing peripheral vasodilatation.

The patient is decannulated, and protamine is carefully given to reverse the heparin. Pulmonary pressures usually rise at the same time as blood begins to clot but this is transient in most cases. Occasionally small doses of intra-aortic adrena-

line are useful to overcome transient right ventricular failure and may be preferred to going back on bypass.

After securing homeostasis the pericardium and sternum are closed and the patient is transferred to the intensive care unit.

IMMEDIATE POSTOPERATIVE MANAGEMENT

This is often more varied and specific to the patient than the actual intraoperative anesthetic technique. The principles are as follows:

- Keep heart rate fast (may need Isuprel and/or pacing).
- Keep dry; diurese to keep central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) ≤ 10. This improves right ventricular function. Remember to treat the patient and not a specific number (i.e., CVP or fluid balance).
- Treat high pulmonary artery pressures with NO, nitroglycerin, milrinone, diuresis, nipride.
- Support the right ventricle with inotropes and afterload reducers for high pulmonary artery pressure (e.g., dobutamine, dopamine, levophed, milrinone and adrenaline).
- Support the left ventricle if necessary, that may warrant an intra-aortic balloon pump (IABP) as well as inotropes and fluid loading. Wean inotropes as soon as possible starting with the most potent (e.g., adrenaline).
- If the cardiac index is poor and continues to be so check mixed venous oxygen saturations and echocardiography to rule out tamponade and assess heart function.
- Continue to treat coagulopathy aggressively.
- Monitor for, prevent and treat other organ system failure (e.g., renal, respiratory, hepatic and neurological).

Premature extubation of heart transplants may lead to further morbidity as they are less likely to tolerate hypoxemia and hypercarbia which may raise pulmonary artery pressures and contribute to right ventricular dysfunction. A quick ventilator wean prior to extubation with assurance that the patient's hemodynamics and urine output is maintained is critical.

A straight-forward heart transplant patient may be a candidate for early extubation and should be off most inotropes within 24-48 h with the possible exception of renal dopamine and isuprel. Some may be discharged to the floor by the 2-4 postoperative day.

IMMUNOSUPPRESSION

The recipient's host defenses recognize HLA on allograft cells as foreign. The goal of immunosuppression is selective modulation of the recipient's immune

response to prevent rejection while maintaining defenses against infection and neoplasia and minimizing toxicity. This may consist of an early intense induction phase (cytolytic) followed by long-term maintenance therapy.

Current TTH heart transplant immunosuppression protocols are summarized in Table 21.3.

COMPLICATIONS

The majority of transplant patients experience some form of rejection though less then 5% have hemodynamic compromise. The presence of hemodynamic compromise and the time out from transplant are key determining factors as to whether the patient requires treatment. Unless unstable, the minority of patients require treatment. Risk factors for rejection include: female recipient, HLA mismatch and female donors. There is poor correlation between clinical characteristics which include: low grade fever, malaise, pericardial friction rub, SVT, low cardiac output and CHF and degree of rejection.

There are no noninvasive tests to detect rejection and the gold standard continues to be endomyocardial biopsies. At TTH, surveillance biopsies are typically performed q weekly x 1 month then q2 weeks x 2 months then q monthly until 6 months then at 3-6 month intervals for 1-2 years. Rejection is mediated by cellular reaction, and histologically the pattern and density of lymphocyte infiltration in addition to the presence or absence of myocyte necrosis determines the severity grade of cellular rejection.

Table 21.3 TTH heart transplant immunosuppression protocols

Induction Phase
RATS⇒ 0.15 cc/kg/24 h if RATS + then OKT3 5 mg i.v./d Azathioprine 1 mg/kg preop Solu-Medrol 0.5-1.0 gm i.v. preop
Maintenance Phase
Cyclosporine 5 mg/kg PO bid or 1-2 mg/kg i.v. q12 h Azathioprine 2-4 mg/kg/d **or** mycophenolate mofetil 1 gm PO bid Solu-Medrol 0.25 mg/kg q6h x 48 h, then prednisone 0.5 mg/kg/d
Acute rejection
Solu-Medrol 1 gm daily x 3d RATS OKT 3

Recent follow-up shows that only 34% of patients work full-time 2 years posttransplant while 76% do not require hospitalization. Associated morbidity posttransplant includes: hypertension, renal dysfunction, hyperlipidemia, diabetes and malignancy. World-wide overall 1 year survival is 79% with an annual mortality rate of 4%. Of note 1-year survival has not improved over the last 10 years of transplantation since 1986. Our 1-year survival is 79% and our 5-year survival is approximately 60%.

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Adult Congenital Heart Surgery

Nancy C. Poirier, Glen S. Van Arsdell, William G. Williams

This chapter describes the more common abnormalities that arise in the adult congenital heart population, outlining the anatomy, pathophysiology, indication for surgical intervention, surgical procedures and perioperative management at Toronto General Hospital (Table 22.1).

Table 22.1. Toronto Congenital Cardiac Centre for Adults (TCCCA) surgical procedures 1973-1998

Pathology	Number of Operations	%
HOCM	238	21
Miscellaneous	230	21
ASD secundum	117*	11
Tetralogy of Fallot	114	9
VSD	95	9
Univentricular physiology	76	7
Subaortic stenosis	48	4
ASD primum	44	4
AV defect	43	4
PAPVD/ASD	37	3
Coarctation	35	3
TGA	22	2
Other ASD	10	1
TOTAL:	1109	

* represents approximately 10% of patients undergoing repair of ASD at Univerity of Toronto

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ATRIAL SEPTAL DEFECT (ASD)

Anatomy

Most common congenital lesion in adult population; 4 types: secundum, sinus venosus (SVC and IVC type; with or without PAPVD), coronary sinus, primum.

The anatomy should always be confirmed intraoperatively, regardless of the congenital lesion. In the presence of an ASD, the pulmonary venous return should be evaluated. Pulmonary valve should be examined intraoperatively if a gradient of 30 mm Hg or more has been measured preoperatively. In most instances the gradient is due to increased pulmonary flow without valvular stenosis.

Intraoperative evaluation after repair should include:

- RV and PA pressures to rule out RVOTO
- Oxygen saturation of the RA, PA and aorta to eliminate the possibility of a residual intracardiac shunt
- TEE

Pathophysiology

Shunting across the ASD depends on the size of the defect, the compliance of the LV (decreases with age) with respect to that of the RV and to the PVR. The left to right shunt results in RV volume loading, dilation and dysfunction in addition to pulmonary congestion, atrial arrhythmia and in a few patients, pulmonary vascular disease (10-15% of patients). Closure before the age of 24 years is associated with a normal life expectancy. Atrial arrhythmia is common after the age of 40 years and may persist after ASD repair.

Surgical Indications

ASD with hemodynamic repercussions

a. RV volume and/or pressure overload and failure
b. Lower exercise tolerance
c. Atrial arrhythmia
d. Paradoxical embolism resulting in TIA or CVA

Asymptomatic with Qp:Qs > 2:1

Surgical Contraindications

Pulmonary vascular disease: (see Table 22.2)

Table 22.2. Relative contraindications to surgery due to pulmonary vascular disease

PAP > 2/3 SAP
PVR > 2/3 SVR without pulmonary reactivity to O_2 or NO
Qp:Qs < 1.5:1
Lung biopsy with Heath Edwards ≥ II

Surgical Procedure

Sternotomy, right anterior thoracotomy (4th intercostal space), or mini-incision

Ostium secundum:

- Suture closure for the smaller defects and autologous pericardial patch is used in larger defects. The majority of adults require a patch closure.
- Endovascular devices placed in the catheterization laboratory are feasible for lesions with a 5 mm rim and a defect between 10 and 20 mm in diameter (depending on the type of device and expertise).

Sinus venosus:

Superior type has no upper septal border

- Usually associated with PAPVD. Upper and middle pulmonary veins (inferior pulmonary veins infrequently) drain into the SVC or the SVC-RA junction.
- The anomalous pulmonary venous drainage is tunneled by the ASD patch into the LA.
- To avoid obstruction of the SVC-RA junction, augmentation may be achieved with a SVC-RA patch or SVC division and reimplantation onto the RA appendage.

Inferior type has an absent lower margin

- Special attention must be directed to close the defect by suturing the lower margin of the patch in the LA to prevent the IVC draining into the RA. If patient is in chronic atrial fibrillation, consider concommitant right-sided Maze procedure.

Postoperative Management

General recommendations

- Swan-Ganz optional (if associated lesions), PA line if pulmonary vascular disease. CO reliably estimated by monitoring peripheral perfusion and hemodynamic parameters.
- Atrial and ventricular temporary pacemaker wires for arrhythmic diagnosis and therapy.
- Extubation a few hours after surgery when postoperative bleeding decreases and discharge from the ICU Postoperative day 1.
- Diuretics based on preoperative weight.
- Early mobilization and discharge 3-4 days postoperatively.

Hypotension

- Early postoperative low CO after routine ASD closure is often related to hypovolemia or tamponade.
- Inotropic support and pulmonary vasodilation may be required in the presence of high PVR and RV dysfunction (see Table 22.3).

Atrial fibrillation

- Standard medical therapy (see appendix 10).

Table 22.3. Treatment of RV dysfunction and /or pulmonary hypertension

Adequate sedation and paralysis
PH > 7.45: Hyperventilation for target PCO_2 = 25-30 mm Hg, Bicarbonate IV
Arterial O_2 saturation approximately 100%
Adequate RV volume loading with CVP = 15-18 mm Hg
Prevent and treat lung disease/lesions: atelectasis, pneumonia, pulmonary edema
Pulmonary vasodilators: nitroprusside, nitroglycerin, amrinone/milrinone, PGE_1, NO
Inotropic support: dobutamine, amrinone/milrinone

Follow-up

Pre-existing atrial fibrillation often persists and is treated with standard medical therapy. Late atrial fibrillation can occur in up to one-third of patients.

Pericardial effusions and tamponade can occur up to several weeks after surgery. Surgical drainage may be necessary. Pericarditis is treated with ASA 325-650 mg po q6 h and if refractory, prednisone 50-100 mg po daily.

Endocarditis prophylaxis for 6 months after surgery.

VENTRICULAR SEPTAL DEFECTS (VSD)

Anatomy

Pathological classification: perimembranous (most frequent in adults), inlet, muscular, outlet and multiple

May be isolated but more frequently is associated with other congenital lesions (coarctation, RVOTO, AI or fibromuscular subaortic stenosis).

Pathophysiology

Small restrictive VSDs are more frequently seen, resulting from incomplete spontaneous closure or residual leaks from previous patch closures.

Left to right shunting causes:

- Increased pulmonary blood flow with progressive RV hypertrophy and dilation, frequently leading to RVOTO
- LV volume overload also resulting in hypertrophy and dilation
- reduction in LV forward flow and decreased perfusion.
- Pulmonary vascular disease (Eisenmenger syndrome) may develop in 50% of adult patients with a large VSD and no RVOTO

Surgical Indications

The major indications for surgery in adults with a VSD are the presence of complications of the VSD, namely:

- RVOTO
- Aortic valve prolapse causing AI
- Ruptured sinus of valsalva aneurysm

- Endocarditis
- Subaortic fibromuscular stenosis

In adult patients, only a few will require surgery for an isolated persistent high flow shunt. (Qp:Qs > 1.5)

Surgical Contraindications

Pulmonary vascular disease: (see Table 22.2)

Surgical Procedure

Prebypass the site of VSD can be confirmed by palpation over the surface of the heart. Associated lesions are evaluated and managed appropriately before closing the VSD.

Dacron patch closure used in the vast majority of patients

- A right atriotomy is used to close perimembranous and inlet VSDs in addition to mid-muscular and inlet septal muscular VSDs.
- Other muscular VSDs can be approached first by a right atriotomy. If unsuccessful, a right ventriculotomy close to the site of the defect provides an adequate access.
- Left ventriculotomy is an alternative for apical VSDs, however this approach is associated with morbidity. Intraoperative device and percutaneous device closure are very promising options for muscular VSDs especially when there are multiple defects.
- Outlet defects are repaired through the PA. Associated aortic valve prolapse and secondary regurgitation is addressed by an aortic valvoplasty through the aortic root.

Postoperative Management

General recommendations

- Swan-Ganz optional (if associated lesions). PA line if reactive pulmonary vascular disease. PA gases to monitor CO and to detect an intracardiac shunt.
- CO is estimated by monitoring peripheral perfusion i.e., diuresis, skin temperature, hemodynamic parameters, acid-base status.

Low cardiac output

- Eliminate the possibility of a residual intracardiac shunt with echocardiographic imaging or by calculating the Qp:Qs from the blood samples of the SVC and PA.
- RV failure and/or high PVR (see Table 22.3)
- Hypovolemia: LAP < 12 → Volume
- LV failure: LAP > 12 → Inotropic support (dobutamine, amrinone/milrinone)
- Afterload reduction if normal BP

Arrhythmia

- Heart block and atrial fibrillation

Follow-up

Pericardial effusions and tamponade can occur up to several weeks after surgery.

Endocarditis prophylaxis for 6 months after surgery.

Aortic valve incompetence can continue to progress or reoccur after corrective surgery; therefore monitor periodically by echocardiography.

Trivial suture line leaks are common and tend to resolve over time.

PATENT DUCTUS ARTERIOSUS (PDA)

Pathophysiology

Shunting across the PDA depends on the relative PVR and SVR. Left-to-right shunting increases pulmonary blood flow which results in LA and LV dilation with progressive LV failure. Pulmonary hypertension causes secondary RV hypertrophy and may lead to RV dysfunction. PDA can become calcified and aneurysmal resulting in compressive symptoms (upper respiratory obstruction or dysphagia).

Surgical Indications

Presence of PDA

Endarteritis

Surgical Contraindications

Pulmonary vascular disease (see Table 22.2)

Surgical Management

If not calcified, patients < 40 years: Left posterolateral thoracotomy through the 4th or 5th intercostal space allows for adequate exposure for ligation or division and suturing of an uncomplicated PDA.

If calcified, patients > 40 years: Median sternotomy and an open technique is used in patients with a short, heavily calcified, and brittle PDA or in the presence of degenerative aortic disease which renders clamping of the aorta difficult and is associated with a high embolic risk. The open technique consists of patch closure of the PDA through the main PA. To facilitate the repair, the flow through the ductus arteriosus can be reduced by compressing the PDA, by occluding it, using a 16F foley catheter and/or lowering CPB flow transiently. Trendelenburg position should be used to avoid cerebral air embolus.

Postoperative Complications

RV dysfunction and /or pulmonary hypertension (Table 22.3)

Recurrent laryngeal nerve palsy

Gastric distention

- vagus nerve trauma may require gastric decompression for few days

Chylothorax

- a pleural effusion characterized by the presence of chylomicrons and 90% of the cell count consisting of lymphocytes.

COARCTATION

22

Anatomy

Congenital narrowing of the descending aorta usually between the left subclavian artery and ductus arteriosus, rarely at the level of the abdominal aorta.

Often associated with other lesions for example bicuspid aortic valve (85% of patients), VSD and mitral valve abnormalities.

Pathophysiology

In the second and third decade, patients usually have upper body hypertension with normal mean pressures distal to the coarctation.

Very few adult patients are exempt from complications of coarctation, aortic, intracranial and intercostal artery aneurysms with potential dissection and rupture; premature CAD, LV hypertrophy and dysfunction, endocarditis.

Collateral flow originates from branches of the subclavian arteries. If femoral pulses are present, some flow through the distal thoracic aorta is preserved and collateral flow is underdeveloped. These patients are considered at a higher risk for postoperative paraplegia.

Surgical Indications

All patients with coarctation or recoarctation with narrowing of the aorta > 50% with respect to the native aorta, in the presence of a gradient > 20-30 mm Hg at rest and/or exercise measured at cardiac catheterization.

Dilation and stenting of coarctation in adults is now being used not only for restenosis after a prior coarctation repair but also has become accepted for primary interventions. A surgical approach will be favored if vascular access is difficult, is also at the level of the arch obstruction, if there are other indications for surgery (post-stenotic dilation or aneurysm), failure to completely relieve the obstruction endovascularly, and in the presence of important atherosclerosis of the distal aorta.

Surgical Procedures

Left posterolateral thoracotomy through the 4th intercostal space.

If femoral pulses are palpable (collateral flow under-developed) or repair complicated (reoperation), repair can be performed at 34°C, using left atrial-femoral bypass, maintaining distal perfusion above 70 mm Hg and a proximal pressure of 100-140 mm Hg.

If femoral pulses are not palpable (highly developed collateral system) with a distal pressure of greater than 70 mm Hg during aortic cross-clamp, correction can be done without CPB at 34°C.

The repair consists of resection of the coarctation and end-to-end anastomosis if the aorta can be mobilized to provide a tension-free anastomosis. However, some adults will require an interposition tube graft. If the aorta cannot be liberated adequately (i.e., reoperation), a Dacron or Gore-Tex patch aortoplasty is the procedure of choice.

In adults with coarctation and a hypoplastic distal aortic arch, especially with recoarctation, repair is performed through a median sternotomy, with hypothermic CPB and circulatory arrest. A patch aortoplasty is usually possible although an interposition graft may be required.

Postoperative Complications

Residual effusions and pneumothoraces are drained with extreme caution or avoided because of potential trauma to aneurysmal intercostal arteries.

Paraplegia

- 1% of patients especially in the presence of poorly developed collateral circulation.
- Routine use of invasive monitoring, for example spinal pressure catheter, is not without complications therefore avoided.
- Paraplegia and paresis can be prevented or treated by:
  - maintaining adequate distal aortic perfusion pressures
  - keeping the CVP < 10 mm Hg to control the CSF pressure
  - spinal tap and drainage to maintain CSF pressure at 10 cm H_2O
- Perioperative spinal cord preservation techniques is crucial in preventing spinal cord damage.

Paradoxical hypertension

- Treat aggressively for systolic BP = 100-120 mm Hg to avoid excessive postoperative bleeding, aortic rupture or dissection, postcoarctation syndrome and cerebral complications. Treatment includes adequate analgesia and sedation and β blockade (Esmolol 0.25-0.50 mg/kg i.v. loading dose followed by 50-200 μg/kg/min i.v. infusion; Labetalol 1-4 mg/min i.v. infusion) Alternatives to β blockade include nifedipine, hydralazine and ACE inhibitors.

Postcoarctation Syndrome

- It has been reported that approximately 5% of patients have postoperative abdominal pain, ileus with possible bleeding and perforation due to a mesenteric arteritis. However, with modern anesthesia and postoperative control of systemic blood pressure, these events are seldom seen.
- Conservative management is usually all that is necessary which includes 1) periodical evaluation to rule out mesenteric ischemia and/or perforation (physical examination, WBC, ABG, abdominal x-ray) and 2) adequate BP control, avoiding potential damage to mesenteric arteries that are suddenly subjected to high BP. 3) Patients are kept NPO and with an NG tube for at least 24 h or until bowel sounds are heard.

Chylothorax, phrenic palsy, recurrent laryngeal palsy, complications of groin cannulation.

Follow-up

Hypertension (50% of adult patients operated), heart failure and cerebrovascular disease are treated aggressively.

Endocarditis prophylaxis for 6 months.

Following any aortic reconstruction particularly after a patch aortoplasty, a CxR in addition to an annual thoracic MRI or CT scan should be performed to document any aneurysm or obstruction at the level of the repair or native aorta.

TETRALOGY OF FALLOT

Anatomy

The most common cyanotic lesion in adults.

Anterocephalad displacement of the infundibular septum results in four classical lesions: RVOTO, over-riding aorta, malalignment VSD and RV hypertrophy.

Pulmonary atresia with VSD is an extreme form of TOF. There is no communication between the right ventricle and the pulmonary arteries. Pulmonary blood flow originates from either a PDA or major aortopulmonary collateral arteries (MAPCA). Palliative procedures including systemic-pulmonary shunts may have been performed. MAPCA are classified as communicating, if they connect with the central PA, or noncommunicating if they do not. They are also defined as dominant if they supply most of the lung and nondominant if the PA perfuse the lung.

Pathophysiology

RVOTO causes right-to-left shunting through the unrestrictive VSD resulting in cyanosis.

Surgical Indication

Unrepaired TOF

- Surgical decision-making requires an assessment of the degree and location of RVOTO, the size of the PA, the course of the coronary arteries, identification of the VSDs and the presence of systemic-pulmonary collaterals.

10% of patients corrected at young age will need reoperation 20 years after complete repair. For the great majority of patients, reoperation is performed at the level of the RVOT for pulmonary valve insufficiency and/or stenosis.

Surgical indications include:

- Residual PS with RV pressure > 2/3 LV pressure
- Free PI associated with residual VSD and/or PS, progressive RV enlargement, progressive TR, arrhythmias, RVOT aneurysm or symptoms with documented deteriorating exercise tolerance.

- Residual VSD with shunt > 1.5:1 (< 5% of patients)
- Severe AI or mild to moderate AI with symptoms and/or LV dilation and systolic dysfunction.
- Aortic root > 55 mm
- Development of severe atrial (mostly flutter or fibrillation) or ventricular arrhythmias which accompany deteriorating RV function.

Surgical Procedures

Definitive repair requires ligation of any systemic-pulmonary shunt, upon establishing CPB. Repair is performed through a right atriotomy with a concomitant incision in the main PA. The infundibular muscle bundles are resected, the VSD is closed using a Dacron patch, a pulmonary valve commissurotomy is performed and the PA are augmented with pericardial patches. A right infundibulotomy is often required to alleviate RVOTO and to facilitate the VSD closure. Transannular patch repairs are avoided if possible since postoperative PI is not well tolerated in adult patients.

Pulmonary valve regurgitation is a common indication for surgery in adults who underwent repair in childhood. Free PI results in gradual RV dilation, progressive TR, and associated arrhythmias. The pulmonary valve is replaced with a large porcine bioprosthesis (29-33 mm diameter) placed at the level of the annulus, angled to the direction of the main PA and oriented such that the struts do not obstruct the branch PA. The RVOT is augmented with bovine pericardium to accommodate the oversized prosthesis. The procedure can be performed without arresting the heart if there is no intracardiac shunting.

Definitive correction of pulmonary atresia with VSD can be accomplished in one procedure if the true PA are dominant and of adequate size. The VSD is closed with a Dacron patch, any noncommunicating, accessible MAPCA are connected to the PAs (unifocalization) and the RV to PA continuity is established with a valved conduit. If the collaterals are dominant, and/or central PA absent or hypoplastic: a) the noncommunicating MAPCA are detached from their origin, b) they are anastomosed directly with a short interposed Gore-Tex graft or with a pericardial tube to lobar arteries, c) communicating MAPCA are occluded at their origins and d) the central PA are reconstructed with Gore-Tex tube grafts. The lung with the least flow is addressed first; the contralateral side is unifocalized 6 months later. During this second procedure, the VSD is closed and the RV-PA continuity is established if adequate pulmonary flow has successfully been established to more than 10 bronchopulmonary segments.

Postoperative Management

RV dysfunction

- Due to prior RV dysfunction, right ventriculotomy, residual lesions (VSD, RVOTO or PI), or inadequate myocardial protection.
- Low CVP (< 15-20 mm Hg) usually indicates hypovolemia and requires adequate volume replacements; and control mediastinal bleeding.

- High CVP (> 20 mm Hg) reflects possible RV dysfunction. CO can be effectively increased with inotropic support (dopamine, dobutamine amrinone/milrinone) and by increasing the HR through atrial pacing especially in the presence of a small noncompliant hypertrophied RV.

Respiratory distress

- Sudden pulmonary reperfusion and the use of CPB may produce pulmonary edema due to increased pulmonary capillary permeability.
- Pulmonary atresia and less frequently PS are associated with systemic-pulmonary collaterals. A left-to-right shunt at the pulmonary level can result in desaturation that can be improved with coil occlusion of collateral flow. During unifocalization procedures, manipulation of the lung parenchyma while on CPB, fully heparinized, can result in pulmonary hemorrhage and secondary desaturation.
- Following complete repair of TOF in adults, intrapulmonary ventilation/perfusion mismatch is common resulting in decreased O_2 saturations for up to 7-10 days postoperative.

Arrhythmia and AV block

FOLLOW-UP

Periodical echocardiographic control of the repair to detect further RV dysfunction and recurrent RVOTO, notably prosthetic valve deterioration and stenosis.

Endocarditis prophylaxis for life.

ASA 80 mg po OD following a bioprosthetic pulmonary valve replacement.

SINGLE VENTRICLE (FONTAN OPERATION)

ANATOMY/ PATHOPHYSIOLOGY

There are a number of morphologic variants that result in a single ventricle supporting the systemic and pulmonary circulations.

Arterial saturation depends on pulmonary blood flow, the degree of mixing and intracardiac streaming. An increase in pulmonary blood flow relative to the systemic blood flow increases arterial saturation at the expense of a decrease in systemic CO.

Single ventricle physiology is presently treated through bypassing the right side of the heart with a direct connection of systemic veins to the PA using either a bicavo-pulmonary shunt (BCPS) (partial shunt of 35% of the systemic return) or the Fontan operation (complete bypass of the RV). These procedures are now done in childhood and only a few adult patients have a balanced circulation that allows long-term survival without intervention.

Biventricular repair with a BCPS is performed in patients with RV dysfunction and/or hypoplasia not severe enough to warrant a univentricular repair and is termed "1 1/2 ventricular repair."

Indications for Reoperation in Fontan Patients

Complications following Fontan procedure: obstruction in the Fontan circuit, arteriovenous malformations, incapacitating atrial arrhythmia, protein losing enteropathy and pulmonary venous obstruction.

Residual ASD with right-to-left shunting, symptoms or cyanosis

Residual shunt secondary to a previous palliative surgical shunt or residual ventricle to PA connection

Subaortic stenosis

Significant AV valve regurgitation

Table 22.4. Single ventricle patient classification and procedure selection

Parameter	Surgical Risk		
	Low	Intermediate	High
Pulmonary vascular resistance (Woods)	< 2	2-3	> 3
Mean pulmonary artery pressure (mm Hg)	< 15	15-20	> 20
Trans-pulmonary pressure (mm Hg)	< 7	7-12	> 12
Ventricular end-diastolic pressure (mm Hg)	< 6	6-12	> 12
Ventricular ejection fraction (%)	> 50	40-50	< 40
Ventricular outflow tract gradient (mm Hg)	< 20	20-30	> 30
A-V valve regurgitation	none	mild	≥ mod
Surgical option	Fontan	BCPS-SPS* ± late Fontan	BCPS-SPS + correct anomalies

BCPS-SPS = Bicavo-pulmonary shunt associated with a systemico-pulmonary shunt if arterial saturation is inadequate.

Surgical Procedure

The BCPS consists of a terminolateral anastomosis of the SVC to the anterior wall of the right PA. The junction of the SVC and RA is oversewn.

The lateral tunnel Fontan operation consists of tunneling the IVC and SVC within the RA directly to the PA confluence. The main PA is transected and oversewn. To alleviate potentially high right-sided pressures, a fenestration (a 4-5 mm communication between the tunnel and the left atrium) is made in higher risk patients, which includes most of the adult population.

There are a number of other modifications of the Fontan. An extracardiac Fontan, the interposition of a 20-22 mm diameter Gore-Tex graft between a BCPS and the IVC, can be performed without cardioplegic arrest and minimal atrial manipulation.

Perioperative Consideration

CO is dependent on the right-sided preload. Pulmonary blood flow is passive.

Pulmonary blood flow is dependent on the transpulmonary gradient = CVP–LAP. The transpulmonary gradient must be approximately 7 mm Hg.

The CVP must be supranormal (14-20 mm Hg) for adequate pulmonary blood flow and LV filling.

Decreases in pulmonary blood flow are due to hypovolemia, increase in PVR, distortion of the PA or the pulmonary veins, any other obstruction along the Fontan circuit, AV valve regurgitation and systolic or diastolic ventricular dysfunction.

Postoperative Management

General recommendations

- CVP, PA and LA pressure, and TEE monitoring identify potential causes and guide the treatment of low CO.
- Ascites, hepatomegaly, large pleural effusions and protein-losing enteropathy are common. Diuretics can limit these complications and together with pleural drainage allow for adequate pulmonary function.
- Early extubation is highly encouraged since it restores a negative intrathoracic pressure, which promotes flow through the pulmonary circulation.

Low cardiac output

- Hypovolemia: colloid solution including blood products (targeted Hb = 120-130 g/L) are administered to maintain CVP 14-20 mm Hg.
- Obstruction along the circuit with gradients > 2-3 mm Hg can be clinically important and should be confirmed by echocardiography and /or cardiac catheterization. Reintervention in the form of dilation, stenting or surgery depends on the severity of hemodynamic repercussions.

Elevated PVR:

- Hyperventilation and bicarbonate to maintain PH > 7.50 with arterial O_2 saturation of 100% are crucial noninvasive means of decreasing PVR.
- Appropriate sedation and paralysis.
- Pulmonary vasodilators: Amrinone, nitrates, PGE_1, NO.
- PVR increases with PEEP > 3, however atelectasis must also be avoided. Larger tidal volumes with short inspiratory times are beneficial.
- LV dysfunction with increased LA pressures: Inotropic support (amrinone/milrinone) increases contractility and/or diastolic compliance therefore decreases filling pressures. Afterload reduction immediately postoperation (nitroprusside) and long-term (ACE inhibitors) can also decrease filling pressures and improve LV function especially in the presence of any systemic AVvalve regurgitation.
- Loss of sinus rhythm and AV asynchrony causes AV valve regurgitation and increases LA pressures and should be treated aggressively.

Hypoxia

- A fenestration allows reduction in the in CVP, however resulting in desaturation that is proportional to the size of the fenestration. Shunting can be reduced by increasing CO, reducing the CVP and optimizing pulmonary blood flow.
- Lung disease, e.g., atelectasis, pneumonia and pulmonary edema.

- Abnormal systemic venous connections.
- Low CO especially when the $S_VO_2 < 50\%$.

Follow-up

Semiannual or annual echocardiographic evaluation of the Fontan circuit and ventricular function.

Warfarin: Indicated in fenestrated Fontan, classic Fontan with dilated RA, and following thrombotic complications. Targeted INR = 2-3.

ACE inhibitors: particularly with AV valve regurgitation.

Arrhythmia

Fenestration closure is performed percutaneously 6-12 months postoperatively especially if persistent right-to-left shunting and arterial desaturation. A TEE must be performed before catheterization to identify thrombus in the Fontan circuit that could potentially embolize.

HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY (HOCM)

Anatomy/Histology

Abnormal unexplained segmental myocardial hypertrophy usually marked at the septum, immediately below the aortic valve. Genetic predisposition, 55% of patients have autosomal dominant transmission.

Histologically, the septal myocardium is characterized by disarray of the alignment of the myocytes. This is associated with abnormal intimal and medial thickening of intramyocardial coronaries.

Pathophysiology

Subvalvar dynamic obstruction is due to septal hypertrophy and narrowing of the outflow tract of the LV (and rarely in the RV) resulting in flow acceleration and a pressure gradient. The flow acceleration pulls the mitral valve (usually the anterior leaflet) toward the septum in late systole (systolic anterior motion, SAM) thereby aggravating the obstruction and producing MR. MR develops in late systole as the anterior mitral leaflet moves toward the ventricular septum. Following myectomy, the MR is minimal unless presence of concomitant mitral valve disease.

Increases in contractility, reduction in preload and vasodilation decreases ventricular volume and promotes SAM.

Markedly reduced diastolic compliance with high filling pressures.

LV function is usually supranormal with EF > 80%. LV failure occurs late in the course of the disease and is a result of an MI and/or severe MR.

Surgical Indication

Patients intolerant to medical treatment or with incapacitating symptoms despite optimal treatment with an LVOT gradient > 50 mm Hg under resting

conditions or > 80 mm Hg provoked with amyl nitrate, isoproterenol or postextrasystolic beat with a septal thickness greater than 18 mm.

Young patients with atrial fibrillation and patients thought to be high risk of sudden death can also benefit from myectomy. Risk factors for sudden death include markedly increased LV wall thickness and mass, "malignant" family history, syncope, ischemia, survivors of a sudden death episode, slow bursts of asymptomatic ventricular tachycardia on Holter and inducible sustained ventricular arrhythmia on electrophysiologic testing.

Surgical Procedure

Preoperative TEE is performed to determine the depth and length of the septal muscle resection needed to completely alleviate the obstruction.

A generous transverse aortotomy and delicate retraction of the aortic valve allows the hypertrophic septum to be visualized beneath the right coronary cusp.

The first incision of the septal myectomy is 2-3 mm to right of the center of the right coronary cusp and directed towards the ventricular apex. The second incision is made 2 mm from the lateral fibrous trigone, below the commissure between the right and left coronary cusp and runs parallel to the first incision. The third incision is placed approximately 2-3 mm below the aortic annulus and joins the other two incisions. The depth of the incisions is calculated such that the resulting septal thickness is approximately 8 mm thick.

Postoperative TEE can precisely measure the septal thickness, identify any residual LVOT obstruction, SAM and MR or detect iatrogenic AI or VSD.

Postoperative Management

General Recommendations

- Monitoring: Swan-Ganz catheter
- Maintain preload: diastolic PAP or wedge > 18 mm Hg: colloid solution or blood products (targeted Hb = 100 g/L)
- Avoid peripheral vasodilation: e.g., nitrates
- Avoid inotropes. If hypotensive despite adequate preload, administer a vasopressor, e.g., phenylephrine.
- Tachycardia and hypertension are treated with β-blockers

Arrhythmia

- Postoperative LBBB in 45% of patients. 1-2% requires permanent pacemaker.
- Rapid atrial fibrillation should be treated with metoprolol 5 mg i.v. given over 5-10 min, repeated twice at 15 min intervals if needed. Specific antiarrhythmic medications should be used cautiously (see Table 22.5).
- If unstable, electrical cardioversion should be attempted immediately.
- If atrial fibrillation persists more than 24 h, begin a continuous heparin infusion and attempt electrical cardioversion once anticoagulated. If cardioversion is successful, begin sotolol 40-80 mg po BID. If unsuccessful, warfarin is begun.

Table 22.5. Postoperative antiarrhythmic medications—HOCM patients

Acceptable	**Controversial/ avoided**
β-Blockers	Verapamil
Disopyramide	Digitalis
Amiodarone	

- Prophylaxis: rapid atrial fibrillation during the immediate postoperative period is common therefore postoperative prophylaxis is recommended using sotolol 40 mg po BID the morning following surgery which may be increased to 80 mg TID if HR > 80 beats/min at rest.

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Ventricular Aneurysm Resection

Lynda L. Mickleborough

LEFT VENTRICULAR ANEURYSM

Diagnosis of Left ventricular aneurysm

Area of akinesis or dyskinesis on angiogram.

Calcification may be present.

Final diagnosis depends on intraoperative assessment of scarring and thinning in the nonfunctioning area of myocardium.

Indications for Surgery

Angina.

Congestive heart failure.

Ventricular arrhythmias.

Mitral regurgitation related to increasing ventricular size/papillary muscle distortion.

To improve prognosis (triple vessel disease or left main stenosis).

Operative Technique

Assessment of aneurysm and repair is best performed on empty beating heart.

Incision in scarred area and palpation of surrounding wall to assess regional wall motion.

Removal of any endocardial clot.

Extent of resection tailored to restore left ventricular size and shape towards normal as much as possible.

Insertion of left ventricular vent through right superior pulmonary vein after removal of endocardial clot.

If area of the septum is thinned, patch septoplasty is performed using glutaraldehyde fixed bovine pericardium.

In most cases, incision is closed with a modified linear closure using 2-0 polypropylene mattress sutures over felt strips followed by a second layer of a continuous suture for hemostasis.

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

In cases with fresh infarction and friable tissues, endoaneurysmorrhaphy with a patch sutured to the endocardium with continuous 3-0 polypropylene sutures. Some surgeons prefer this method for all cases of ventricular aneurysms.

VENTRICULAR ARRHYTHMIAS, MAPPING, VENTRICULAR TACHYCARDIA ABLATION

Indications

Clinical and inducible ventricular tachycardia

Operative Technique

Mapping procedure carried out on cardiopulmonary bypass at normothermia using an epicardial sock and endocardial balloon array of electrodes.

Induction of VT, mapping of activation sequence and target area identified on electrode array.

The left ventricle is opened through area of scarring and thinning to locate the arrhythmia focus.

Excision of endocardial scar in arrhythmogenic area ± cryoablation at the periphery of excision if target area is on the septum.

Postoperative Considerations

Initially patients post aneurysm resection will need a rapid heart rate and increased filling pressures to maintain adequate cardiac output.

As the heart adapts to the repair procedure, diuresis can be initiated to get rid of extra fluid without compromising output.

Watch for ventricular arrhythmias and treat them with intravenous amiodarone.

On the 2nd or 3rd postoperative day start an ACE inhibitor and monitor renal function.

Postoperative echocardiogram to assess mitral valve function and rule out thrombus associated with area of repair. If thrombus is present, anticoagulate for 3 months.

Patients on preoperative amiodarone are at increased risk for ARDS; keep on lowest F_i0_2 to maintain adequate oxygenation; vigorous diuresis as soon as possible; aggressive treatment for suspected pulmonary infection.

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Combined Cardiac and Vascular Surgery

Charles M. Peniston

CAROTID ARTERY SURGERY AND CARDIAC SURGERY

Patients may present for surgery with both significant cerebrovascular (CVD) and cardiac disease. The incidence of significant coronary disease in patients with symptomatic carotid stenosis is 25-30%. The incidence of significant carotid stenosis in patients undergoing coronary artery bypass graft (CABG) is 6-8%. Only when the indications for both procedures are present should a combined procedure be considered. The primary reason for simultaneous procedures is to prevent a perioperative stroke and/or perioperative myocardial infarction.

Diagnosis of Carotid Disease

Symptoms: transient ischemic attacks (TIA); previous stroke
Physical examination: neck bruit
Noninvasive testing:

- Duplex scanning combines Doppler scanning and ultrasonography and is the preferred method.
- Oculoplethysmography
- CT scan or MRI should be done if there is a history of stroke in the past

Invasive testing:

- Carotid angiography should be done when there is concern about the distal vessels or disease in the aortic arch.

Indications for Carotid Endarterectomy

Symptomatic

- Previous TIA or stroke with 75-99% stenosis of the ipsilateral internal carotid artery

Asymptomatic

- This is controversial. The ACAS study showed that patients with more than 60% stenosis who underwent endarterectomy had a lower incidence of subsequent ipsilateral CVA than those who were treated medically.

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

- Our practice has been to perform a combined procedure when there is severe compromise of the cerebral circulation, e.g., complete occlusion of one carotid artery and >75% stenosis of the other carotid artery.

Operative Technique

- Simultaneous exposure of the heart and carotid artery.
- Endarterectomy while harvesting the saphenous vein.
- Optional use of carotid shunt while doing endarterectomy.
- Saphenous vein patch of the endarterectomised carotid artery.
- Proceed with the CABG.

The carotid endarterectomy can also be performed during cardiopulmonary bypass. Moderate or even deep hypothermia may provide additional cerebral protection but it may prolong the operation.

Intraoperative Concerns

Diseased aorta suggested by palpable plaques, scarred adventitia and immobile adventitia. Avoid cannulation through a plaque:

- Consider cannulation of the arch, beyond the origin of the carotid arteries to avoid proximal embolization.
- Epiaortic scan to look for plaques.
- If there is extensive disease in the ascending aorta, consider replacement of the ascending aorta and possibly the proximal arch under deep hypothermic circulatory arrest.

Postoperative Care

Maintain adequate perfusion pressure, oxygenation and cardiac output. Watch for bleeding/hematoma in neck incision.

Attempt to wake the patient early after arrival in the ICU to assess neurological status. After awakening with or without extubation the patient may be:

- Awake and oriented: Routine postoperative management
- Confused without localizing signs: Rule out other causes for confusion and consider imaging with Doppler if concerned about patency of the carotid artery.
- Hemiplegic or hemiparetic: Image with angiogram and then possibly return to OR for re-exploration of the carotid artery

COMBINED CORONARY ARTERY BYPASS WITH ABDOMINAL AORTIC ANEURYSM REPAIR

Some patients present with symptomatic coronary disease and have a large (> 5 cm) abdominal aortic aneurysm (AAA). Simultaneous repair of the AAA with coronary artery bypass graft (CABG) should be considered. Potential advantages of this include the ability to scavenge blood shed while the aneurysm is being

repaired. It is also possible to put the abdominal aortic clamp in place with very little pressure on the aorta simply by temporarily turning down the pump flow.

Diagnosis of AAA

- Symptoms: back pain, abdominal pain
- Physical examination: palpation of the abdomen, complete vascular exam
- Imaging: ultrasound, CT scan, MRI scan, angiography
- CT scan is probably the most useful study because it demonstrates the size and location of the AAA (i.e. infrarenal). Consider angiography when there is concern about the renal arteries or the distal vascular system.

Indications for Combined AAA Repair and CABG

- Otherwise healthy patient with a low perioperative risk.
- AAA >5 cm which can be repaired with either a straight tube graft or aortobi-iliac replacement.
- Significant coronary artery disease requiring surgical intervention.

Operative Technique

- Perform CABG first.
- With patient on cardiopulmonary bypass, the abdomen is open and aneurysm repaired.

Postoperative Care

- Maintain adequate blood pressure but avoid pressures above 120 mm Hg for the first 6 h, unless there is evidence of decreased renal perfusion.
- Leave nasogastric tube in place for 2 or 3 days or until there is evidence of normal gastrointestinal peristalsis.
- Optimize fluid administration as significant intra-abdominal third spacing occurs.
- Observe respiratory function closely after extubation as there may be more compromise because of more intra-abdominal swelling.
- Maintain adequate urine output.
- Beware of excessive fluid requirements or falling hemoglobin, which might suggest occult intra-abdominal bleeding.

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Minimally Invasive Cardiac Surgery

Robert J. Cusimano

Minimally invasive cardiac surgery (MICS) is a "catch all" phrase for many operations performed in cardiac surgery. Most are procedures performed on the heart to treat coronary artery disease, aortic and mitral valvular disease, atrial septal defects, pericardial windows, epicardial pacemaker placement and cardiac biopsies. Most procedures utilize the heart lung machine, with cannulae placed in the usual location in the great vessels or via the femoral vessels. It also denotes removal of the greater or lesser saphenous veins via scopes of various sorts. All methods, either centrally or peripherally try to avoid large incisions and/or the heart lung machine. Presumably by decreasing tissue trauma, the inflammatory response of cardiopulmonary bypass, perioperative complications, and blood transfusion requirements, length of hospital stay and time to fully recover can be reduced. However intuitive this is, it remains to be proven. This chapter will concentrate on MICS- coronary bypass (MICS-CABG), as the other operations are performed through smaller incisions but in the same manner.

INDICATIONS

- Single or double vessel disease
- Proximal coronary lesion reachable by a minimally invasive approach
- Failed or recurrent angioplasty or stent
- High risk for open procedure
  - Ascending aortic atheroma or calcified aorta
  - Multiple other patent grafts
  - Co-morbid conditions making a conventional operation risky

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

OPERATIVE TECHNIQUE

General Preparation:

Maintain normothermia in patient by raising the room temperature. Use warm prep solution to decrease heat loss.

Patient position as for standard CABG. Prep for standard bypass, with wide lateral prep but medial to the V2 chest lead.

ECG lead placement for usual bypass includes a V6 chest lead. Although adequate for right coronary artery (RCA) bypass, require V2 lead placement for any anterior operation (i.e. LAD). Place ECG dot in the second intercostal space in the midclavicular line. In this position, will be able to pick up anterior wall changes (and missed by V6) during occlusion of the LAD. Remind the anesthesiologist to make a pre-incision tracing of the V2 lead for comparison to the postoperative (anastomosis) V2. This modified chest lead placement is very important.

Incisions:

Left Anterior Descending (LAD)

Curvilinear fourth left intercostal space, with a medial vertical and lateral horizontal component. The vertical portion allows tissue traction superiorly and lengthening of the incision should the need arise.

Begin dissection laterally in the interspace (midclavicular line is sufficient) and the pericardium open in an oblique-medial superior manner (i.e., parallel to the LAD). If unable to find the LAD (it lies in approximately the midclavicular line) due to its intramyocardial or sub fat position, abandon the thoracotomy approach for a sternotomy approach. Search for the LAD before left internal thoracic artery (LITA) dissection in order to save time and potential damage to the LITA, should the LAD not be found easily by this approach.

After locating the LAD, identify the LITA. Exercise great care here, as the LITA is usually found deep to fat. Once found, identify the vessel in subsequent intercostal spaces by mentally tracing its course deep to the rib. Dissect it to the first and fifth intercostal spaces. If need to displace a rib, fracture it on one side (usually laterally) and reflect rather than remove it. Despite good initial results, patients with resected ribs occasionally develop chest wall depression that is both unsightly and potentially dangerous, as it overlies the heart.

Right Coronary Artery (RCA)

Use a midline subxiphoid incision to reach the RCA on the inferior wall. The incision runs from the xiphisternum (which is excised) to half way to the umbilicus. Divide the diaphragm anteriorly to reach the heart. The peritoneum can be reached for the gastro-epiploic artery as can the right internal thoracic artery. Suture the diaphragm to the fascia or skin anteriorly. The right coronary is very easily reached. The posterior descending is harder to reach. The posterolateral is almost impossible.

Sternotomy

Perform a full or partial sternotomy when more than one vessel is in need of bypass or as a standard approach. If a partial sternotomy is chosen, do not hand off the sternal saw until skin closure, in case a full sternotomy is required.

Elevate the heart by placing one or two sponges posterior to the heart. Blood pressure initially falls with this maneuver but eventually becomes adequate. Do this maneuver early to allow cardiac adaptation. With full sternotomy and LAD bypass (the easiest), tack the left (but not right) pericardium to the pectoralis fascia thus rotating the heart and LAD closer to the midline. Blood pressure again will fall at this point and may require additional volume to improve.

Anastomosis and Vessel Occlusion

Preconditioning is not required on totally occluded vessels. However, in patients with large territory vessels, a 5 minute preconditioning (if tolerated) allows subsequent safe occlusion of the vessel. Usually, these three 5 minute periods will precondition the heart adequately. If not tolerated, use open methods of cardiopulmonary bypass.

Place 4-0 monofilament suture deeply around the vessel and snare with a tourniquet both proximally and distally. Initially only the proximal snare is made snug by the surgeon, who applies enough pressure to allow compression of the vessel. Overzealous snaring may lead to vessel damage as can shallow placement of suture around the vessel. Once the incision in the vessel is made, the distal snare can be made snug if there is significant back bleeding. Clearly the pressure for the second snare is less and in some, gentle pulling versus snaring is all that is required. An adequate but not excessive distance must be left between proximal and distal snares to allow for anastomosis and needle clearance.

STABILIZATION OF THE HEART

Fancy and costly devices such as the octopus can be used. Good stabilization can be achieved by using sharp towel clips between the chest wall and the tourniquet which have been displaced parallel to the vessel. If further stabilization is required, a second assistant can use a Kelly clamp to stabilize one of the tourniquets at the level of the heart.

HEART RATE CONTROL

Despite the use of stabilizers and the above measures, heart rate control (i.e. low) is helpful. If the patient has not been beta-blocked preoperatively, Metoprolol (versus Esmolol, which is expensive) is well tolerated. Intravenous Diltiazem in increasing doses and titrating as a continuous drip of 3 mg/hr is also effective. Clonidine 0.2-0.3 mg given 2-3 h preoperatively allows very good heart rate control. Avoid drugs known to cause tachycardia (e.g., pancuronium). Blood pressure

control to 85-90 mm Hg is adequate, especially during snaring. Artificially raising it will cause an elevated heart rate and MVO_2, obviously not required during coronary occlusion.

POSTOPERATIVE CARE

Perioperative anesthetic management is detailed in chapter 8.

Attempt to extubate patients in the operating room. This will be easier if there has been communication with the anesthesiologist preoperatively and the patient has been kept warm. Consider removal of the patient's Foley catheter prior to leaving the operating room.

Intensive Care Unit nurses and house staff are accustomed to high urine outputs of patients on bypass. Avoid administering excessive fluids to manage borderline urine outputs. Make every effort to mobilize these patients on the same day of surgery.

Pericarditis is common in these patients.

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Partial Left Ventriculectomy (PLV)

Robert J. Cusimano

The gold standard operation for dilated cardiomyopathy with poor ventricular function has been and remains heart transplantation. Dr. Randas Batista, a heart surgeon in Brazil, developed a new operative strategy based upon his theory that a basic problem with dilated hearts is the lack (or lag) of concomitant muscle hypertrophy which should accompany the increase in diameter. Through examination of many hearts of different sizes and species, both healthy and diseased, he came to realize that there was a constant relationship between size (radius) of the ventricle and muscle mass, regardless of species (mass = 4 x radius^3). He also found that although total muscle mass increased with dilated hearts, it did not increase in the same ratio as "normal" hearts, thus leading to a relative lack of muscle mass for that size (radius) of the heart. By sacrificing some muscle, thereby reducing the radius of the heart, the amount of muscle required for that particular radius more closely approximates that actually present, thus improving the muscle mass shortcomings and improving efficiency of the dilated heart.

INDICATIONS

The role of this operation in the management of dilated cardiomyopathies remains unknown.

Dr. Batista claims that the key to the operation is large diameter (radius) of the left ventricle, the larger the better. Minimum cut off is 70 mm (end diastolic) dimension as measured by echocardiogram. All patients should have a cardiac catheterization to define the presence of coronary disease. Valvular abnormalities need to be corrected.

Since the procedure is new, patients should have failed other forms of therapy. Most patients require mitral valve repair/replacement and/or coronary artery bypass. Tricuspid regurgitation is also very common and may get worse postoperatively. The best candidates are those with a large LV and preserved right ventricular size and function.

This procedure is largely experimental and should be performed only in centers with capability for left ventricular assist devices.

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

OPERATIVE TECHNIQUE

Intraoperative transesophageal echocardiography (TEE) is mandatory, as mitral valve repair is a frequent additional operation. Ventricular remodeling may distort mitral valve anatomy as well as tricuspid valve.

Partial left ventriculotomy

- Incision starts at the apex. Obviously, scarred tissue is excised. If there is no scar tissue, the incision is carried parallel to both the LAD and posterior descending branches.
- Upon entering the LV, the papillary muscles are identified. The incision is carried between the two papillary muscle heads. There is often an (or more than one) accessory papillary muscle which we have preserved.
- The incision is carried to approximately 2-3 cm from the AV groove to avoid damage to the circumflex coronary artery. The incision is beveled with more endocardium being excised than epicardium. This beveling is very important as it prevents undue tension and, thus, postoperative bleeding. If the papillary muscles are excised, a valve is inserted from the ventricular aspect.
- A 2-0 monofilament suture on a large needle is used to close the ventriculotomy from the base to the apex in simple over and over stitches without pledgets. The tension on each bite is important, otherwise it will cut through the muscle. A second layer is used. The first is for strength and a second layer is for hemostasis. If the muscle has been beveled, bleeding will be minimized.

Mitral valve surgery

- Mitral valve regurgitation is so common in these patients that a comment about repair is warranted. We have used a technique described by Alfieri wherein the mid-portion of the anterior leaflet is sutured to the mid-portion of the posterior leaflet, creating a bi-orifice mitral valve. It has been very effective in our patients. If replacement is needed, it is easily performed through the ventriculotomy.

Tricuspid valve regurgitation of moderate or greater severity should be repaired .

POSTOPERATIVE CARE

The patients are very sick, although better postoperatively than preoperatively. Atrial and ventricular wires are placed and intra-aortic balloon pump pump used if required.

Early postoperative care is improved by volume expansion and afterload reduction.

A predischarge echocardiogram to examine both ventricular function and, especially, valvular function.

Patients are given amiodarone for 6 months to prevent ventricular dysrhythmias.

Perioperative care of these patients is further discussed in chapter 8.

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AICD and Pacemakers Insertion

Charles M. Peniston

AUTOMATIC IMPLANTABLE CARDIOVERTER DEFIBRILLATOR (AICD)

An automatic implantable cardioverter defibrillator (AICD) is a totally implantable electronic device capable of recognizing and treating ventricular tachyarrhythmias and bradycardia. It consists of a generator that contains a battery, computer logic and telemetry antenna. One or more leads connects the generator to the heart. Initially implants were performed by a left thoracotomy and all leads were placed on the surface of the heart. Presently, most devices are placed transvenously with a single coaxial lead. This lead has a dual function of sensing the cardiac rhythm as well as acting as an electrode with the generator being the other electrode. Biphasic depolarizations are used because of reduced defibrillation thresholds. In addition, AV AICDs are available that can pace both the atrium and the ventricle after cardioversion.

Indications for AICD Insertion

Arrhythmia not controlled by drug therapy
Intolerance of antiarrhythmic drugs
Cardiac arrest (but absence of a myocardial infarction) in a patient who is not a candidate for other surgical or medical treatment

Contraindications

Short life expectancy (less than 6 months)
Uncontrolled ventricular arrhythmias
Terminal illness

Sites of Implantation

Subpectoral, transvenous (usually in the left side)
Subxiphoid
Subcostal
Median sternotomy
Anterolateral left thoracotomy

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

Operative Technique

The proper functioning of this device is usually more important than the actual implantation which is relatively easy and therefore one may require the assistance of skilled personnel from the Electrophysiology Department.

General anesthetic is preferred.

Place R2 defibrillator pad on the patient's chest, one over the sternum, one between the shoulder blades.

For transvenous route, cannulate the cephalic vein to limit the possibility of pneumothorax and reduce the incidence of lead crush factors. If necessary the subclavian vein can be cannulated. Avoid blood clotting on the stylet of the leads. Use fluoroscopy to ensure proper lead placement, at the apex of the right ventricle.

Measure defibrillator thresholds (DFTs). The threshold should be less than 20 J. If inadequate, try changing the polarity of the defibrillation shock. Consider placement of an SVC or innominate lead.

Place generator subpectorally, after successful defibrillation. Administer perioperative antibiotics. Irrigate the incision with bacitracin antibiotic solution. Always secure the lead over the suture sleeves.

Postoperative Care

These patients rarely require use of ICU beds. Most patients go to the recovery room and then to the ward. They will require telemetry. A magnet should be nearby to inhibit the device if it is inappropriately shocking the patient. Follow-up is in the EP Clinic. An EP test is done 24 to 48 h after implantation.

Potential Complications

Infection

Hemorrhage, pneumothorax

Inadvertent discharge (oversensing)

Undersensing

Conversion of VT to VF

Crosstalk with previously inserted pacemaker. Avoid this by placing the AICD lead high on the interventricular septum.

Always obtain PA and lateral chest x-ray after implantation.

Long Term Complications

Battery depletion

Lead dysfunction

Exit block

Lead fracture

PACEMAKERS

A pacemaker consists of a generator that connects leads to the right side of the heart. Transvenous leads are placed through the subclavian vein into the right

ventricle. Epicardial leads are tunneled either to the infraclavicular fossa or the subcostal position. All patients undergoing cardiac surgery have temporary epicardial pacemaker leads placed on the heart. These exit at the lower end of the sternotomy incision and can connect by cables to an external generator.

Indications for Permanent Pacemaker

Symptomatic patients with

- AV block
  3°
  2° Mobitz I, Mobitz II
  Incomplete with 2:1 or 3:1 block
- Sinus node dysfunction
  Sinus bradycardia
  Sino-atrial block
  Sinus arrest
  Tachy-brady syndrome

Attempt to maintain AV synchrony when inserting a pacemaker even if the patient has intermittent atrial fibrillation. The atria can contribute up to 20% of the total cardiac output which is especially important in patients with heart failure and reduced ventricular compliance.

Pacemaker syndrome, defined as pounding in the chest and feelings of weakness, may occur when using VVI pacing in patients with atria that are still contracting. This is due to retrograde VA conduction, contraction of atria against closed AV valves and reduced cardiac output.

Temporary Pacemakers

Every patient after cardiac surgery will have temporary epicardial pacemaker wires placed on the right ventricle and usually also on the right atrium. Their purpose is two-fold (i) temporary pacing, (ii) diagnostic electrograms. They are removed by continuous traction 4-5 days postoperatively. The patient should remain flat for 15-30 minutes after removal of the wires.

Operative Technique

At Toronto General Hospital almost all pacemakers are inserted under local anesthesia with an anesthesiologist standing by to provide intravenous analgesia and sedation.

The generator is placed in a subcutaneous pocket in the infraclavicular fossa on either the right or the left side. Ideally, use the cephalic vein. If this is too small then cannulate the subclavian vein.

Under fluoroscopy the lead is guided into position in the right ventricle and the right atrium if necessary. With dual chamber pacing insert the ventricular lead first followed by the atrial lead.

Obtain threshold measurements. When inserting the device it is important to have capable personnel who are skilled in programming the generator.

After inserting the pacemaker leads it is important to have the patient take a deep breath and cough to ensure that the lead is fixed in position.

Irrigate the incision with bacitracin antibiotic.

For generator replacement when the battery is reaching the end of its life, one must consider that the patient may be fully pacemaker dependent. One must be able to rapidly connect the pacing systems analyzer to the lead once it has been disconnected from the old generator.

Potential Complications

Lead displacement (< 1.5%)
Lead entrapment
Infection
Battery depletion
Generator dysfunction
Under sensing/over sensing
Exit block (rising pacing threshold)
Lead fracture
Pneumothorax
Hemothorax
Innominate vein thrombosis

Crosstalk. This is sensing of an atrial stimulus in the ventricular channel. This can result in suppression of the ventricular stimulus.

Pacemaker mediated tachycardia. An arrhythmia in which the dual chamber pacemaker acts as the antegrade limb tachycardia and the natural pathway acts as the retrograde limb, setting up a re-entrant cycle. Avoid this by having upper rate limits set on the pacemaker and by adjusting the ventricular atrial refractory period.

Special Considerations

Electrocautery. This may be sensed and interpreted as increased heart rate and therefore suppress the pacemaker. It may reprogram the pacemaker to its backup mode (VOO). It may cause complete and permanent loss of pacing. If possible use bipolar cautery.

Magnetic mode. When a magnet is placed over the pacemaker it usually converts it to VOO mode preventing inhibition by cautery. However, different pacemakers respond in different ways.

Electromagnetic interference. Potential sources include electrocautery, cellular telephone, MRI scanners, microwaves and powerful radio and radar transmitters. None of these are usually problematic. If the patient is dependent on the pacemaker, then it is prudent to avoid all of these sources. Hold the cellular telephone at least 6 inches away from a pacemaker. Pacemakers can be affected by lithotripsy, mechanical trauma and damaged by radiotherapy if not properly shielded.

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Ventricular Assist Devices (VAD)

Gideon I. Cohen

COMMONLY UTILIZED VENTRICULAR ASSIST DEVICES (VAD)

- Pneumatic: Abiomed
  Thoratec
  Heartmate
  Jarvik-7 Total Artificial Heart (TAH)
- Electrical: Heartmate
  Novacor
- Centrifugal: Biomedicus
- Rotary: Hemopump

INDICATIONS FOR LEFT VENTRICULAR ASSIST DEVICE (LVAD) IMPLANTATION

Patient selection is the key to success with LVAD use. Due to the rapidly evolving nature of LVAD technology along with the increasing pool of experimental data, indications for use are constantly changing. Indeed, due to the invasive nature and associated complications of mechanical devices, LVAD support has traditionally been reserved for the most critically ill patients. However, limited success in patients with severe end-organ damage along with advances in perioperative management have prompted a recent trend towards pre-emptive rather than end-stage application of support.

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

Patient Population

- Patients who cannot be weaned from CPB (although semi-implantable/pericorporeal devices may be more effective for patient stabilization prior to LVAD placement).
- Transplant candidates too ill to survive waiting period for cardiac transplantation (patients with blood type O or positive reactive antibody titer).
- Heart failure patients with acute decompensation.
- Nontransplant candidates (i.e., > 65 years of age or relative contraindications to transplantation such as amyloidosis or severe diabetes).

Hemodynamic Criteria

- Requirement for inotropic/IABP support
- Pulmonary capillary wedge pressure > 20 mm Hg
- Systolic blood pressure < 80 mm Hg
- Cardiac index < 2 L/min/m^2

ABSOLUTE AND RELATIVE CONTRAINDICATIONS TO LVAD IMPLANTATION

Cardiac Factors

- Right ventricular failure (RV ejection fraction < 10%; although LVAD not contraindicated if used in combination with RVAD).
- Uncorrected aortic insufficiency (may prevent optimal systemic perfusion).
- Severe aortic stenosis (repair may preclude requirement for LVAD support).
- Uncorrected severe mitral stenosis (may limit LVAD filling).
- Uncorrected tricuspid insufficiency (right ventricular function must be optimized).
- Coronary artery disease (angina and ischemic myocardial injury may persist following LVAD placement).
- Atrial arrhythmias (may compromise right ventricular function; if refractory, long term anticoagulation should be implemented to prevent thrombus formation regardless of device).
- Ventricular arrhythmias (relatively well tolerated; however, may decrease LVAD flow by 20% and systemic blood pressure by <20%. Aggressive attempts at early electrical or pharmacological cardioversion are indicated to prevent thrombus formation and further injury to ventricles).
- Atrial and ventricular septal defects (should be repaired at the time of LVAD insertion to prevent right-to-left shunting and desaturation secondary to reduction of left ventricular filling pressures).

Extracardiac Factors:

- Age > 70 years (relative contraindication)
- Severe neurological injury or psychiatric disorder
- Extracranial cerebrovascular disease
- Severe obstructive or restrictive pulmonary disease (FEV1 < 30% predicted)
- Recent pulmonary embolism with infarction
- Fixed pulmonary hypertension (pulmonary vascular resistance > 8 Woods units)
- Renal failure
- Severe liver disease/insufficiency or coagulopathy
- Severe gastrointestinal malabsorption
- Active ulcer disease or intestinal infection
- Severe nonreconstructable peripheral vascular disease precluding peripheral cannulation
- Sepsis
- Miscellaneous:
  - blood dyscrasias
  - long term steroid therapy
  - unresolved malignancy
  - metastatic cancer
  - positive HIV test
  - body surface areas < 1.5 m^2

BIVENTRICULAR VAD INDICATIONS

- Severe pulmonary hypertension
- Right ventricular ejection fraction < 15%
- Giant cell myocarditis
- Re-transplant candidates
- Severe pulmonary edema

SEMI-IMPLANTABLE / PERICORPOREAL VAD

Abiomed

- Pneumatic pump
- Univentricular or biventricular capability
- Central cannulation:
  - Inflow: right or left atrium
  - Outflow: aorta or pulmonary artery
- Inserted off cardiopulmonary bypass
- Spares left ventricular apex

- Minimal training required
- Patient is non-ambulatory
- Full anticoagulation required

THORATEC

- Pneumatic pump
- Univentricular or biventricular capability
- Central cannulation:
  - Inflow: right atrium, left atrium, or left ventricle
  - Outflow: aorta or pulmonary artery
- Cardiopulmonary bypass necessary for insertion
- Reduced left ventricular pressure with apical cannulation
- Heparin bonded circuit enables postponement of anticoagulation
- Low thromboembolic rate
- Patient semi-ambulatory

BIOMEDICUS

- Centrifugal pump
- Univentricular or biventricular capability
- Oxygenation capability for ECMO purposes
- Peripheral or central cannulation:
  - Inflow: right atrium via femoral vein or directly
  - Outflow: femoral artery or ascending aorta
- Veno-arterial mode for cardiac or cardiorespiratory support
- Veno-venous mode for pulmonary support
- Inserted off cardiopulmonary bypass
- Heparin bonded circuit enables postponement of anticoagulation
- Patient non-ambulatory

HEMOPUMP

- Rotary pump
- Delivers 4.5 L/min (25,000 rpm)
- Univentricular capability
- Percutaneous insertion via femoral artery
- Partial anticoagulation
- May induce hemolysis
- Patient non-ambulatory
- Improved results in pediatric population
- Not currently available in North America

PERIOPERATIVE CONSIDERATIONS

- Large ventricular aneurysms are plicated and excised to prevent occlusion of inflow cannula.

Table 28.1. Implantable LVAD

	Heartmate	Novacor
Insertion	cardiopulmonary bypass	cardiopulmonary bypass
Location	preperitoneal	preperitoneal
Inflow	left ventricular apex	left ventricular apex
Outflow	ascending aorta	ascending aorta
Valves	25 mm porcine Hancock	25 mm bovine pericardial
Mode	fixed or automatic	fixed or automatic
Stroke Volume	85 mL	70 mL
Pump Index	11 L/minute	10 L/minute
Manual Pump	Yes	No
Surface	Textured (pseudo-neointima formation)	Polyurethane
Anticoagulation	Antiplatelet agents	Complete
Portability	Complete	Complete

- Patent grafts from previous coronary bypass procedures are preserved; myocardial regions at risk may be bypassed at time of device implantation to prevent symptoms and reduce the risk of right-sided failure.
- Tricuspid annuloplasty may be necessary at the time of implantation if significant insufficiency exists.
- Transesophageal echocardiography is useful for de-airing and for detection of a patent foramen ovale (which must be closed prior to pump insertion) or left ventricular apical thrombus.
- Blood products, if necessary due to bleeding during VAD placement, are administered via leukocyte filters to reduce the risk of antigen exposure which may complicate subsequent transplantation.
- Left atrial pressure monitoring useful for early detection of three common perioperative problems: tamponade, right heart dysfunction and hypovolemia.
- Pulmonary hypertension and right ventricular failure secondary to cardiopulmonary bypass-induced release of thromboxane A2 and transfusion-induced cytokine activation may complicate LVAD placement in patients with chronic left ventricular failure. Changes in right ventricular contractility due to ventricular interdependence may also contribute to right ventricular failure following LVAD insertion and may be predictable via low preoperative right ventricular stroke work indices. Although such failure is often amenable to volume loading, pharmacological inotropic support and/or inhaled nitric oxide (NO) therapy, BIVAD capability should be readily accessible for those cases with refractory right heart failure.
- Prosthetic membranes may be utilized to facilitate abdominal wall closure in smaller patients.
- Aprotinin may be utilized intraoperatively to reduce bleeding as well as transfusion-related complications.

- Arginine vasopressin in low doses (0.04 units/minute) is a useful pressor agent in those patients with VAD-related, catecholamine-resistant vasodilatory hypotension.

LVAD COMPLICATIONS

LVAD-Related

- Mechanical failure
- Cannula obstruction
- Thromboembolism
- Hemolysis
- Bleeding
- Infection; endocarditis in bioprosthetic valves
- Valve failure
- Worn cams/rollers
- Gastrointestinal problems

Cardiac-Related

- Left ventricular dysfunction
- Right ventricular failure
- Arrhythmias
- Tamponade
- Valvular insufficiency
- Myocardial ischemia
- Endocarditis in native valves

Systemic-Related

- Hypo/hypervolemia
- Pulmonary vasospasm/hypertension
- Multi-organ failure
- Infection

Although the above complications can occur at any time postoperatively, bleeding and pulmonary hypertension are most common < 48 h postoperatively, infection is most common < 30 days postoperatively, and device failures are most common > 30 days postoperatively.

MYOCARDIAL RECOVERY AFTER LVAD PLACEMENT

- Normalization of cardiothoracic ratio
- Decrease in left ventricular end-diastolic dimension
- Improvement in ejection fraction and cardiac index

- Decrease in pulmonary capillary wedge pressure and pulmonary vascular resistance
- Reduction in myocytolysis
- Calcium uptake and calcium-binding rates of isolated sarcoplasmic reticulum vesicles normalized
- Myocardial tissue lipid levels normalized
- Plasma norepinephrine levels normalized
- Disorganization of mitochondrial cristae reversible after LVAD placement

TOTAL ARTIFICIAL HEART (JARVIK-7)

- Pneumatically driven
- Biventricular support
- Four mechanical valves
- Implanted in pericardium after native heart explanted
- Implanted medially or in left lateral position
- Anastomoses to both atria, aorta and pulmonary artery
- Pneumatic drivelines exit skin below left costal margin
- Patients fully anticoagulated +/- antiplatelet agents
- External console or portable pneumatic drive

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Lung and Heart-Lung Transplantation

Shaf H. Keshavjee

INTRODUCTION

The surgical options for the treatment of end stage cardiopulmonary failure have evolved considerably since the initial success of heart-lung and lung transplantation. The first successful single lung transplant and the first successful bilateral lung transplant were performed in Toronto in 1983 and 1986 respectively. The first successful heart-lung transplant was performed at Stanford in 1981.

Combined heart-lung transplantation is reserved for combined irreversible cardiac and pulmonary failure. Lung volume reduction (LVR) is a procedure that may be used in selected cases of end stage emphysema, as an alternative to transplant, as a bridge to transplant or in combination with transplant. The current algorithm for the selection of lung and heart-lung transplant procedures at the University of Toronto is illustrated in Figure 29.1.

Donor Lung Protection

Methylprednisolone 2 g i.v. to donor
PA flush with LPD (Low Potassium Dextran) solution, 50 ml/kg, 4°C
PGE_1 500 mg direct injection into pulmonary artery
PGE_1 500 mg in flush solution
Lungs are stored at 4°C in the inflated state ($FiO_2 > 0.5$) for transport

Intraoperative Lung Protection

Keep lung cool using a cooling jacket during implantation
Clear blood and secretions and *gently* reinflate (25 cm H_20) prior to reperfusion
Gradually release PA clamp over 10 min (modified reperfusion technique)
Wash out flush solution and de-air through atrial anastomosis

SURGICAL TECHNIQUE

The lung transplant operation requires cooperation and precise communication between the surgeon and the anesthesiologist. A single lung transplant is per-

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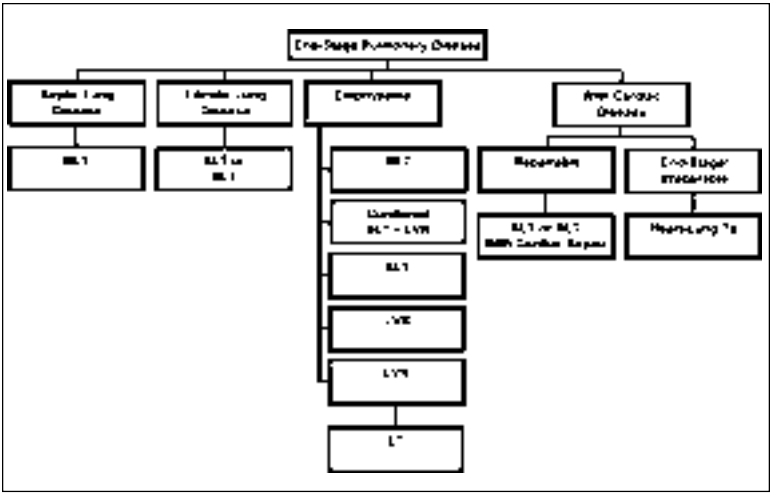

Fig. 29.1. The University of Toronto algorithm for lung and heart-lung transplantation. (SLT = single lung transplant, BLT = bilateral lung transplant, Tx = transplant, LVR = lung volume reduction).

formed through a standard posterolateral thoracotomy and a bilateral lung transplant is performed through a transverse fourth intercostal space thoracosternotomy.

Bilateral Lung Transplant Procedure

A pneumonectomy is performed on the side receiving the least blood flow (as determined by a preoperative quantitative V/Q scan). The bronchus, pulmonary artery and left atrium/ pulmonary veins are prepared for anastomosis. The donor lung is trimmed on the back table and then placed in the recipient's chest on a cooling jacket. The patient is given Solumedrol 500 mg i.v. and 2000 U heparin.

The bronchial anastomosis is performed first, with a running 4-0 Maxon suture on the membranous wall and interrupted mattress 4-0 Prolene sutures on the cartilaginous wall. The bronchus is intussuscepted for one ring. The anastomosis is further buttressed with sutures through peribronchial tissues. The bronchial tree is then suctioned clear by the anesthesiologist.

A vascular clamp is placed on the proximal PA and the vessel is appropriately trimmed. The pulmonary arterial anastomosis is performed in an end-to-end fashion using a running 5-0 Prolene suture interrupted in two places.

The pericardium surrounding the pulmonary vein stumps is incised and an atrial clamp is placed on the lateral wall of the left atrium. The vein stumps are opened and their orifices joined to form a common atrial cuff. The atrial anastomosis is then performed using a running 4-0 Prolene suture interrupted in two places. This is done with a horizontal mattress suture in a *completely everting* technique so that there is perfect endothelium-to-endothelium apposition. The atrial anastomosis is left untied at this point for later de-airing.

The cooling jacket is removed and the lung is gently re-inflated to a maximum pressure of 25 cm H_2O. Once the lung is inflated, ventilation is initiated with an FiO_2 of 0.5, maximum peak pressure 15-25 cm, PEEP 2-5 cm. The pulmonary artery clamp is *gradually* removed over a period of 10 min to provide gradual reflow (re-warming and distension) of the pulmonary vasculature. The flush solution and blood is allowed to drain out of the atrial anastomosis, and once the lung is de-aired, the atrial clamp is removed and the atrial suture is secured.

The patient is allowed to stabilize for a period of 10-15 min. The pneumonectomy and implantation procedure is then similarly performed on the contralateral side in the case of a bilateral lung transplant. The patient is supported by the native lung while the first lung is implanted and by the newly transplanted lung while the second lung is implanted. Cardiopulmonary bypass therefore is not routinely used for lung transplantation. In approximately 30% of cases, CPB is necessary. This is usually for cases where CPB is clearly needed—heart-lung transplant, combined lung transplant and cardiac repair and severe pulmonary hypertension ——or in cases where oxygenation or ventilation become impossible or the patient becomes hemodynamically unstable. The indications for CPB are listed below.

Cardiopulmonary Bypass for Lung transplantation

- **Indications**

SLT: Pulmonary hypertension (primary or secondary)
Inadequate gas exchange
Hemodynamic instability

BLT: Pulmonary hypertension
Dysfunction of first graft
Progressive increase in PAP / hypotension
Pulmonary edema / hypoxia

Separate lung ventilation impossible

- **Cannulation**

Standard: Aortic root and right atrium (2 stage venous cannula)
Right SLT: Right femoral artery and right atrium
Left SLT: Descending aorta or femoral artery, femoral vein or proximal PA

- **Special Considerations**

Do not persist unduly before resorting to CPB if problems with hypoxia or hypotension.
Decrease to 1/2 to 3/4 full CPB flow at reperfusion—to gently reperfuse at low pressure.
Maintain ventilation of first lung implanted once reperfused.
Maintain low-pressure reperfusion of first lung (3/4 full flow) to prevent warm ischemia (note: the transplanted lung has no bronchial blood flow).

Heart-Lung Transplant Procedure

The heart-lung transplant procedure is also carried out through a transverse thoracotomy incision. The exposure of the posterior mediastinum and pleural spaces that is afforded by this approach has significantly decreased the incidence

of postoperative hemorrhage (compared to a median sternotomy). The technique for heart-lung transplantation, which is briefly described below, is adapted from the original description of the procedure by the Stanford group.

The patient is cannulated for CPB in the standard fashion (aorta/bi-caval). The aorta is cross-clamped and the heart is excised by transecting the aorta and PA and incising the right and left atria. The pericardium is incised anterior and parallel to the phrenic nerves bilaterally. The posterior wall of the left atrium is divided in the midline and the pulmonary vein/left atrial mobilization is completed extrapericardially, posterior to the phrenic nerve pedicles. The PA is divided at the bifurcation and a button of tissue is left on the ductus arteriosus to preserve the recurrent nerve. The PAs are dissected laterally. Both main bronchi are divided in standard fashion. The carina is then excised taking care to minimally disrupt the blood supply to the distal trachea.

The heart-lung graft is then placed in the chest. The right lung is passed under the right atrium and right phrenic nerve pedicle and the left lung is passed under the left nerve pedicle. The trachea is anastomosed in an end-to-end fashion using a running 4-0 Maxon suture on the membranous wall and interrupted 3-0 Prolene sutures on the cartilaginous wall. The anterior pretracheal donor and recipient tissues are approximated to buttress the anastomosis. Gentle ventilation with an FiO_2 of 0.5 is then initiated.

The donor right atrium is opened from the IVC to the base of the atrial appendage, and the right atrial anastomosis is performed using a running 3-0 Prolene suture. A bi-caval anastomotic technique may also be used. The aortic anastomosis is then performed using a running 4-0 Prolene suture. The heart is then carefully de-aired and the cross-clamp is released.

POSTOPERATIVE CARE

The postoperative care of the lung (LTx) or heart-lung (HLTx) transplant patient begins in the operating room. After the completion of the implantation procedure, meticulous attention must be paid to the hemodynamic optimization of the patient. Cardiac output, filling pressures and SVR should be measured. These patients often have a low SVR syndrome which requires institution of an infusion of an alpha agent (norepinephrine) and judicious volume replacement. While volume overload is not desirable, volume depletion is also undesirable in that it contributes to hemodynamic lability – essentially one should aim for an euvolemic state.

After the completion of the operation, the double lumen endotracheal tube is exchanged for a single lumen tube and a bronchoscopic examination is performed to inspect the bronchial anastomosis and to clear the airway of blood and secretions.

The patient is transferred to the intensive care unit. The CVP and PCWP are kept as low as possible to maintain a mean BP > 65 mm Hg with adequate urine output. Often the low SVR state persists for 12-24 h and a continuous infusion of levophed ± dopamine is required. Patients with elevated pulmonary artery

pressures preoperatively tend to be more hemodynamically labile and more difficult to manage.

Ventilatory Support

A baseline arterial blood gas is obtained on an FiO_2 of 1.0, PEEP 5 cm on arrival in the ICU. Conventional ventilation techniques are used with PEEP—aim to keep the plateau pressure < 35 cm H_2O. In ~20% of cases, reperfusion injury is severe and life threatening. If conventional ventilation is inadequate, in cases of severe ischemia-reperfusion injury, nitric oxide is used to improve oxygenation and to decrease pulmonary artery pressure. A PGE_1 infusion can also be beneficial in reperfusion injury. In severe cases extracorporeal membrane oxygenation (ECMO) is used to support the patient until the acute lung dysfunction recovers. In general, at least 50% of patients will be extubated within 24-48 h of surgery.

Immunosuppression

- Solumedrol 500 mg i.v. before reperfusion
- Solumedrol 0.5 mg/kg/day x 3 days
- Prednisone 0.5 mg/kg/day from day 4-14, then taper 5 mg/week to 20 mg/day
- Cyclosporine 2-3 mg/kg/day i.v., change to Neoral po 5 mg/kg bid (target level 250-350 until 6 months).
- Azathioprine 1.5 mg/kg/day i.v./po

Anticoagulation

Desirable in that bronchial anastomotic healing depends on microcirculatory blood flow through pulmonary–bronchial collaterals and DVT/pulmonary embolism prophylaxis is also necessary. Start immediately postop (hold if bleeding a concern).

Heparin 100 U/hr i.v. x 7 days, then 5000 U sc q 12h until discharge

Rheomacrodex (5% Dextran 40) 500 ml i.v. over 24h x 7 days

Infection Prophylaxis

Bacterial: Perioperative antibiotic prophylaxis: Cefuroxime 1.5 g i.v. 30 min preoperatively, and q8 h x 48 h. Adjust according to culture results of donor and recipient. Cystic fibrosis patients: Ceftazidime 2g i.v. + Tobramycin 5 mg/kg i.v. (or according to known organisms/sensitivities in the patient)

CMV: All CMV positive (donor, recipient or both): Gancyclovir 5 mg/kg bid x 14 days, then q M W F for 12 weeks. Negative donor and recipient: Acyclovir 400 mg po tid x 3 mo (Herpes prophylaxis). Positive donor and negative recipient: CMV hyperimmune globulin immediately and q 2 wk x 16 weeks.

Pneumocystis carinii*:* Septra od q M,W,F—starting on day 14

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Routine CVICU Care: Early Extubation to Discharge

Annette Vegas, Davy C. H. Cheng

EARLY TRACHEAL EXTUBATION AND PROCESS OF CARE

The process of postoperative care must be modified to complement early tracheal extubation for maximum cost efficiency. Fast track cardiac anesthesia provides the opportunity for the paradigm shift in postoperative care of cardiac surgical patients.

The concept of providing graded levels of care in the cardiovascular intensive care unit (CVICU) can be categorized by patient flow and postoperative recovery:

1) Conventional Model: This is the conventional flow of patients from operating room (OR) to ICU, then to a free standing intermediate care unit and ward.
2) Parallel Model: This is a free standing intermediate care unit which directly admits postoperative cardiac patients and operates in parallel to an independent ICU.
3) Integrated Model: This is a fully integrated intermediate care unit with ICU. Patients are admitted directly from OR and recover with flexible nursing ratio for different acuity level of care.

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CVICU—A VARIABLE CARE UNIT

Structure

Our CVICU contains 28 beds and admits approximately 2,700 cardiac surgical patients annually. For maximum flexibility, each bed provides direct and indirect patient observation and can function either as an ICU bed or a less intensively monitored bed with privacy for the patient and family. Sliding glass partitions allow for 1:2 to 1:3 nursing care as the patient progresses through intermediate levels of care. The major advantage is the continuity of patient care with the provision of appropriate levels of care individualized to the patient without transfer to another hospital site.

Staffing/ Support Services

A medical director (cardiac anesthesiologist) and a nursing manager are responsible for the finance, workload and quality of care. This is a semi-closed unit with the admissions under the staff surgeon. Routine daily medical issues are managed by a cardiac anesthesiologist with a cardiovascular surgical anesthesia fellow. This allows continuity of medical care and implementation of clinical pathways for cost-effective care. There is overnight in house fellow coverage.

The recommended nurse/patient ratios vary through a shift depending on the acuity of patients and case-mix. The aim is to minimize transfer or transport of patients between units, but to match the nursing need to the acuity of the patients. In order to achieve the most efficient utilization with flexibility, this unit has a pool of part-time nurses available to compliment the scheduled nurses for variation in workload during each nursing shift. This lowers the service workload cost per patient.

Respiratory therapist and point of care laboratory test is available at all times. Other supporting professionals such as pharmacists, physiotherapists, nutritionist, and social workers are available as required.

Resource Utilization

Flexible staffing patterns allow for cost savings during periods of low utilization of the service and low patient acuity. This type of CVICU would balance patient needs with appropriate staffing and monitoring resources. Proposed advantages include: decreased cost, improved ICU utilization and avoidance of ICU readmission. The routine care of patients after open-heart surgery is detailed in the following chapters.

ADMISSIONS

There should be a smooth transition of care from the operating room team to the Cardiovascular Intensive Care Unit (CVICU) personnel.

Anesthesiologist's Report

- surgical procedure and brief history
- other diseases: hypertension, diabetes, renal, etc.
- allergies
- technical problems: difficult intubation, line insertion
- anesthetic regimen: propofol, fentanyl, muscle relaxant
- prebypass stability: blood pressure (BP), rhythm, bleeding
- full CPB or no CPB: duration, circulatory arrest, difficulty arresting heart
- weaning off bypass: number of attempts, pacing, drugs, intra-aortic balloon pump (IABP), hemodynamics (BP, CO, PAD)
- metabolic: potassium (K^+), glucose, urine output, Hb (PRBC), A-a oxygen gradient
- bleeding: pharmacotherapy (protamine, tranexamic acid, desmopressin, ε-aminocaproic acid), blood products

Surgeon's Report

- CABG: native circulation (quality of bypasses and distal vessels)
  number of bypasses, location and quality
  current left ventricular function
  endarterectomy, vein patch, radial artery graft, other arterial grafts
  need for nitroglycerin, calcium channel blockers or antiplatelet agents
- Valve: repair or replacement, tissue vs. mechanical, anticoagulation plan
- Congenital: which way does the blood flow?, what lines?, hemodynamic goals?
- blood pressure guidelines (low if bleeding or friable aorta, high if cerebral or renal disease)

Admission Procedure

The nurse, surgeon, anesthesiologist and CVICU fellow work together to admit and stabilize the patient. This includes the following:

- Transduce and zero the pressure lines
- Put patient on the ventilator, auscultate lungs and check endotracheal tube (ETT) placement
- ECG leads by nurses
- Scan quickly heart rate (HR), rhythm, BP and filling pressures
- Check Swan-Ganz position, should be inserted < 55 cm and trace to ensure not in right ventricle or wedged, assess cardiac output (CO)
- Check blood loss from chest tubes, urine output and general tissue perfusion
- Ensure pacemaker box is attached and functioning

Standard admission order sheets (Appendix 2) and clinical forms (Appendix 4) are filled out. Remember to order continuing operative drugs (e.g., inotropes, dipyridamole and preoperative bronchodilators, steroids, anticonvulsants, etc.).

Routine labs drawn within 30 minutes of admission include complete blood count, prothrombin time, partial thromboplastin time, serum electrolytes, creatinine, glucose and echocardiogram. Order additional tests if indicated: calcium

(Ca^{2+}), magnesium (Mg^{2+}), liver function tests, fibrinogen, cardiac enzymes and CXR (Appendix 7).

ROUTINE POSTOPERATIVE COURSE

Patients are continuously monitored with bedside ECG, oxygen saturation (SaO_2), transduced arterial line and CVP (or PA pressure) and hourly urine outputs. Cardiac outputs are done every 6 h or as clinically indicated and the diastolic pulmonary artery pressure (PAD) is followed rather than repeated pulmonary capillary wedge pressure (PCWP). Test results (ABG, K^+, glucose and Hct) are rapidly available as needed. Additional labs including CBC, PT/PTT, Cr, Ca^{2+}, Mg^{2+} are usually done daily.

The majority of patients regardless of the procedure return to the CVICU sedated on a propofol infusion 2-6 mg/kg/h for the first 1-2 h while the following is assessed:

- Temperature: if < 36.0°C use warming blanket and warm to at least 36.5°C
- Ventilation: reasonable A-a gradient, CxR (if needed)
- Hemodynamics: systolic BP 85-120 mm Hg, C.I. > 2, stable HR and rhythm
- Nasogastric tube is present
- Urine output adequate (> 1 cc/kg/hr)
- Minimal bleeding (< 50-75 cc/hr)
- Initial labs checked

Discontinue the propofol infusion when the patient meets the above criteria for early extubation. Reverse the patient's residual neuromuscular blockade (neostigmine 2.5 mg and either atropine 1.2 mg or glycopyrrolate 0.4 mg i.v.) and allow the patient to awaken. Patients may become bradycardic post reversal so ensure the patient has a pacemaker box attached and checked. Standard post extubation orders are filled out (Appendix 2).

COMMON MANAGEMENT STRATEGIES

Shivering

Tends to increase arterial PCO_2, decrease arterial PO_2 and may interfere with ventilation. Treat with meperidine 25-50 mg i.v., increase the propofol infusion or pancuronium 2 mg i.v. if needed. The patient should be warm and not shivering before reversal of neuromuscular blockade.

Fluids

Most patients require additional volume to maintain optimal filling pressures and adequate urine output. During the first 24 h, avoid large volumes of crystalloids

in favor of colloids either as pentaspan 500 cc (max. 2000 cc/24 hr) or 5% albumin 500 cc i.v. After 24 h, patients who are extravascularly volume overloaded may benefit from 25% albumin 100 cc i.v., to help mobilize fluid intravascularly. If patients are intravascularly volume depleted with low filling pressures, low urine output and adequate arterial PO_2, give a 500 cc volume challenge of normal saline, pentaspan or 5% albumin.

Autotransfusion

Patients are autotransfused their shed mediastinal blood for up to 6 h postoperatively. Exceptions include patients with actively infected bacterial endocarditis, contaminated circuits or unexplained air leaks in the circuit. The reinfused products represent unwashed red cells, activated tissue factors and any residual circulating heparin. This may exacerbate ongoing bleeding but does allow for maintenance of Hb.

Blood Products

Hemoglobin is initially maintained through autotransfusion and packed red blood cells (PRBC) are given for Hb < 7 g/dL. Sick, elderly or patients with impaired ventricular function (grade 4) may benefit from an Hb closer to 10 g/dL, particularly in the setting of borderline hemodynamics. Healthy patients with a low initial Hb, in the absence of ongoing bleeding or instability, will likely hemoconcentrate over the following 24 h and are unlikely to need transfusions. The decision to transfuse requires individual patient assessment. Patients who pre-donated should receive their own whole blood back postoperatively if they are anemic. Administer additional blood products in the face of ongoing bleeding and documented abnormalities (Appendix 8).

Bleeding

Expect some amount of postoperative bleeding, though the rate and total volume lost elicit different responses from individual surgeons. Each surgeon has a different threshold of wanting to be kept informed and the final decision to re-explore. Consider the following abnormal rates of bleeding and inform the staff surgeon:

- bleeding > 400 cc/hr for first hour
- bleeding > 200 cc/hr for first 2 h
- bleeding > 100 cc/hr (after first 4 h, max. 1000 cc)

Extubation

Manage early extubation patients according to the CVICU weaning protocol (Appendix 6). Consider T-piecing or an endotracheal ventilation catheter (ETVC) in patients who are known difficult intubation.

Postoperative Pain

In order to facilitate early extubation, patients receive less intraoperative narcotics than in the past. Patients without renal dysfunction, ulcer disease, diabetes

or age > 70 receive indomethacin or diclofenac 50-100 mg rectally before extubation. Pre- and postextubation, patients receive intermittent i.v. boluses of either morphine 2-4 mg or demerol 10-25 mg for additional pain relief. Consider patients who are young or demonstrate high narcotic requirements for patient controlled analgesia (PCA). Within 24 h of extubation most patients are managed by oral analgesics Tylenol #3 or equivalent.

SEDATION

Patients who are not early extubation candidates are sedated postoperatively with an infusion of narcotics and or benzodiazepines:

- narcotics: morphine at 1-4 mg/h ***or*** Fentanyl at 100-200 ug/h
- benzodiazepine: midazolam at 1-3 mg/h

UNSTABLE PATIENTS AND CHEST RE-OPENINGS

While the majority of patients follow a stable postoperative course and are discharged from CVICU within 12-24 h, some are critically ill. These patients frequently have an IABP, multiple inotropes, low urine output, unstable hemodynamic or even an open chest. These patients require continuous intensive management and frequent attention to their clinical status. Strive for 12-24 h of relative stability prior to gradually weaning inotropes or IABP. Abruptly discontinuing support may lead to an acute deterioration in the patient's condition.

Chest re-opening is a potentially life saving intervention. Indications vary but are usually related to an acute failure to thrive (arrest or pre arrest), to relieve tamponade or facilitate open cardiac massage and resuscitation. Patients may require further operative interventions (see chapter 33).

MEDICAL ROUNDS

During morning rounds the patient's course in CVICU is presented by the assigned nurse and management decisions made regarding:

- need for antihypertensives, anticoagulation
- review of current ECG and laboratory results (CBC, Cr, ABG)
- urine output, fluid balance and need for volume or diuresis
- chest tube removal
- adequacy of pain control
- suitability for transfer out of CVICU

In complicated patients additional time is spent on:

- review of drugs and hemodynamics
- ventilation requirements and plan for weaning or extubation
- further investigations or consultations

DISCHARGES

Multidisciplinary (surgeon, anesthesiologist, fellow, RT and charge nurse) round from 7:15-8:15 every morning for discharge decisions. Should a patient's condition change making them not suitable for discharge, they remain in CVICU and are managed appropriately with medical attention and nursing staff ratio. Standard CVICU discharge clinical form (Appendix 5) and discharge order sheet (Appendix 3) are filled out.

Points to remember:

- Examine the patient and check morning hemodynamics, urine output, ECG and bloodwork.
- Check CXR post chest tube removal to rule out a pneumothorax.
- Order either ECASA or appropriate anticoagulation.
- Reorder the preop medications including non-cardiac medications. Do not resume anti-anginals (unless periop MI) but continue anticholesterolemia, anti-arrhythmic and afterload reduction. Continue antihypertensives though initial dose may need to be adjustment based on current BP.
- Fluid restriction and furosemide orders. Check filling pressures (CVP, PAD) prior to central line removal, cumulative fluid balances and current weight before filling out orders. Remember to fluid (1000-1500 cc) and salt (1 g salt diet) restrict patients with poor ventricular function, renal or hepatic dysfunction and if extravascular volume overload.

DELEGATED ACTS AND PROCEDURES

Nurses in the CVICU are usually assigned based on the acuity of the patient. New admissions and high acuity patients are nursed 1:1, while less acute patients are nursed 1:2 or 1:3. The nurses are able to perform the following delegated medical acts:

- extubation according to protocol (Appendix 6)
- remove cordis or central line, Swan-Ganz catheter
- remove arterial line from radial and femoral sites
- remove chest tubes
- start peripheral intravenous line
- defibrillate in an emergency
- setup IABP console and assist with insertion
- help time and trouble shoot IABP problems
- draw mixed venous gases from pulmonary artery catheter

Currently physicians are responsible for removing IABP and extubation in prior difficult intubation.

The respiratory technicians (RT) can:

- wean oxygen and ventilation according to protocol (Appendix 6)
- test lung mechanics prior to extubation (vital capacity and negative inspiratory force)
- assist in determining the optimal mode of ventilation
- assist during bronchoscopies
- transport and accompany a stable ventilated patient to radiology
- manage the patient's airway during an arrest

When indicated consult subspecialty services which include neurology, nephrology, infectious disease, electrophysiologic service (EPS) and follow-up echocardiograms by cardiology department.

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Analgesia Techniques

Gerald O'Leary, Alan N. Sandler

INTRODUCTION

With high dose opioid anesthesia and overnight ventilation for cardiac surgery, perioperative pain management was not previously a major issue. Now with extubation 0-4 h following admission to CVICU, a balance has to be found between good analgesia for patient comfort/incentive spirometry and over sedation, in order to avoid cardio-respiratory compromise.

Anatomy

Chest wall somatic pain transmitted centrally via intercostal nerves

Cardiac visceral pain transmitted via vagus, cervical sympathetic or upper five thoracic sympathetic ganglia

Pain Sites

Pain is usually from the chest tube rather than the median sternotomy incision (exceptions include rib fracture/excision, costochondral, minithoracotomy pain).

Chronic back pain exacerbation usually responds better to mobilization than bed rest.

Post-thoracotomy pain syndromes (neurogenic/muscular) will likely be more frequent with the change from sternotomy to thoracotomy.

Surgery does not exclude angina recurring postoperatively.

MODALITIES

Nurse Administered Opioid Analgesics

Excellent technique allowing individualized doses as needed, cost effective, no new equipment, minimal patient education

Less effective if nurse to patient ratio exceeds 1:2, other patient labor intensive, narcotic not readily available, still have dose related narcotic side effect; nausea, ileus, sedation, confusion, etc.

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Our bias is morphine 1-10 mg/h i.v., followed by 1-2 tablets of Tylenol #3/Percocet PO.

NSAID

Excellent supplemental agents to treat musculoskeletal thoracic pain, reduce narcotic intake and side effects. Mode of action, analgesia and contraindications are similar for all NSAID.

We avoid in true ASA hypersensitivity, GI bleeds, renal dysfunction, diabetes and age > 70.

Our bias is indomethain or Diclofenac 50-100 mg pr q12 hr x 2 doses.

Intrapleural Local Anesthetic Analgesia

Unilateral thoracic analgesia, with bolus doses; duration of action variable, < 4 h with chest tubes, ineffective for chest tube removal

May be of use with mini-thoracotomy

Intercostal Blocks

Can use for fractured ribs/costochondral disarticulation, mini-thoracotomy

NSIADs have similar efficacy

PCA Opioids

Advantages are return of control, self titrate analgesia to effect.

Disadvantages are risk of confusion and over sedation during the patient's initial learning curve, conversion to PO meds in < 12 h, need for education, equipment, etc. Pain scores and sedation are not less with PCA vs nurse administered narcotics.

We use PCA in < 1% patients after cardiac surgery.

Intrathecal Opioids

Morphine 0.5-1.0 mg intrathecally prior to CPB results in reliable analgesia and reduced narcotic intake postoperatively, but delays early extubation (time to extubation was > 10 h from arrival in ICU with intrathecal morphine 10 μg/kg).

It is not our routine practice to use this technique.

Thoracic Epidural Opioids/Local Anesthetic Agents

Advantages include:

- reliable intense perioperative analgesia
- reliably attentuates the stress response of CPB
- induces thoracic cardiac sympathectomy and decreases coronary vascular resistance
- decreases heart rate and use of β-blockers after CPB and increases myocardial oxygen supply
- successfully used in medically and surgically refractory angina
- decreases myocardial infarct size in animals

Disadvantages include:

- delays tracheal extubation
- risk of epidural hematoma unknown, perhaps as high as 0.35%; increased with heparinization for CPB; insertion (blood tap delay OR 24 h, cost in lost OR time, problems with same day admit patients), possible hematoma with catheter removal (if coagulation abnormal, valve surgery, etc.)
- opioid: pruritis, urinary retention, nausea, sedation, respiratory depression
- local anesthetic: block height, etc. hypotension and therefore decreased coronary perfusion

Studies:

- have small numbers (total about 2000 patients)
- no prospective double blind trials
- none use clinical outcome as primary end point

Therefore although stimulating, we do not currently use this technique for patients undergoing CPB. It may be useful in patients not undergoing CPB, such as MIDCAB procedure.

COMMON PROBLEMS

Opioid Allergies

Side effects (nausea, vomiting, constipation, pruritis, sedation, confusion, respiratory depression) are in general dose related rather than drug specific.

Codeine allergies are usually nausea and constipation. Simple treatment alternatives include Tylenol or Percocet plus laxative.

Opioid Tolerant Patients

MS Contin: maintain on opioid to prevent withdrawal, convert daily PO dose x 1/3 for daily i.v. baseline dose, expect 50-300% additional narcotic for an attempt at analgesia

Methadone same for MS Contin patients, conversion methadone dose x 1.5 for morphine dose, enroll experienced help early. Numerous issues need to be addressed.

Alcohol Abuse

Individualized opioid dose, balance increase opioid needs/risk of confusion against agitated patient in pain.

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Common Problems in the CVICU

Annette Vegas

CENTRAL NERVOUS SYSTEM (CNS)

Seizures

Postoperative seizures can sometimes be difficult to differentiate from residual muscle relaxant, rigors or shivering.

Classification and pharmacological management of seizure is summarized in Table 32.1.

In addition to documenting location, type and frequency of seizures, serum electrolytes (calcium, magnesium, sodium) and glucose are checked.

Seizures are usually transitory due to emboli (air, debris) but if persistent may require an electroencephalogram (EEG) and neurology assessment.

Coma

Comatose patients are unusual after cardiac surgery. Metabolic, drug or a primary CNS event need to be ruled out. An early CT scan if the patient is hemodynamically stable will help document structural neurological damage.

Consider CNS protection by normalizing arterial oxygenation (PaO_2), carbon dioxide ($PaCO_2$), serum glucose and maintain an adequate cerebral perfusion pressure. There is no current role for mannitol, steroids or calcium channel blockers.

Alcohol Withdrawal

Alcohol abuse is an infrequent problem with preoperative patients but should be suspected if there is a history of high alcohol intake. Alcohol withdrawal is characterized by the following three phases:

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Table 32.1. Classification and management of seizures

Partial seizures (maybe secondarily generalized)	**Generalized seizures**
i) simple (conscious) ii) complex (impaired conscious)	i) petit mal, ii) myoclonic iii) grand mal (tonic-clonic)

- $1^{st}/2^{nd}$ ⇒ benzodiazepines: Diazepam 2-5 mg i.v., Lorazepam 1-2 mg i.v.
- 3^{rd} ⇒ Phenytoin 10-15 mg/kg i.v. (max. 1.0 gm) load, maintain 3-5 mg/kg/day
- 4^{th} ⇒ Phenobarbital 10-15 mg/kg i.v. (max. 1.0 gm) load, maintain 2-4 mg/kg/day
- status ⇒ Thiopental 1 mg/kg/h (500 mg/ 250 D5W)
  Midazolam 0.2 mg/kg bolus then 0.05-0.2 mg/kg/h

- alcohol tremulousness (6 h): tremor, diaphoresis, nausea, insomnia, disorientation, hallucinations
- seizures or rum fits (10-30 h): usually 1-2 seizures which stop in 24 h
- delirium tremens (48 h): severe disorientation, auditory or visual hallucinations, extreme agitation, increased autonomic activity (fever, tachycardia, diaphoresis, tachypnea, cardiac instability)

Treatment: Thiamine 100 mg i.v. daily for 3 days (order prophylactic in high risk patients). Multi-vitamins 1 ampoule in 1 liter normal saline over 24 h daily if poorly nourished, haloperidol 2 mg-10 mg i.v. q 1 h prn, alcohol by NG tube (30 cc beer or whiskey q 6h standing).

RESPIRATORY

Arterial-alveolar (A-a) Gradient

Differential diagnosis includes pre-existing chronic obstructive pulmonary disease (COPD), endotracheal tube (ETT) misplacement, bronchospasm, pneumothorax, effusion, airspace disease and post-CPB adult respiratory distress syndrome (ARDS).

Examine the patient and check a chest x-ray (CXR) for correctable causes.

Management involves:

- if intubated: add PEEP (7.5-10 cm H_20), increase inspired oxygen (F_iO_2), diurese, consider bronchoscopy with lavage for sputum, bronchodilators
- if extubated: pain control, chest physiotherapy, diuresis, increase F_iO_2, facial continuous positive airway pressure (CPAP), bronchodilators
- if prior exposure to amiodarone, may wish to minimize F_iO_2
- if pneumonia: sputum culture and gram stain, bronchoscopy, consider antibiotics early if prosthetic materials in heart

Chest Tube Removal

usually removed by nurses 12-24 h postoperatively based on following:

- minimal drainage < 50 cc/4 h
- no air leak
- stable respiratory status
- surgeons' permission

A CXR is performed 1 h later to rule out pneumothorax.

If airleak, cross-clamp chest tubes as proximal to patient as possible to determine if the patient or system is leaking. If persistent bubbling despite clamping then likely system that is the problem. Conversely, if bubbling stops then most likely patient. If unsure then clamp chest tubes and check clinical status and CXR prior to removal.

Pneumothorax

If hemodynamically and respiratory status adequate, confirm by CXR. If > 50% pneumothorax, patient symptomatic, poor gas exchange on arterial blood gases (ABG) or tension pneumothorax then insert a chest tube. Repeat CXR to confirm tube placement and lung re-expansion. Alternatively consider needle thoracostomy to aspirate air.

CARDIOVASCULAR

Hypertension

There are many drugs available to treat postoperative hypertension. The choice depends on desired hemodynamic parameters and the presence of renal dysfunction or myocardial ischemia (Table 32.2).

In the early postoperative period use short acting easily titratable i.v. drugs to adapt to changing hemodynamic parameters and ensure adequate absorption. If intubated may need to increase sedation and give more pain relief. Patients who have pre-existing hypertension should resume their preoperative medications, at adjusted doses, when tolerated in the postoperative period.

- Nitroglycerin (NTG) 100 mg in 250cc D5W at 5-50 cc/h (good for ischemia or high filling pressures)

Table 32.2. Antihypertensive drugs

	preload	afterload	HR	contractility	renal	ischemia
Vasodilator	↓	↓	↑	↑	ok	good
Calcium channel blocker	↔	↓	↓	↓	ok	good
ACE inhibitor	?↓	↓↓	∅	↑	no	no
β-Blockers	↑	↑	↓	↓	ok	good

- Sodium nitroprusside 50 mg in 250 cc D5W at 5-50 cc/h (good to reduce afterload)
- β-Blockers:
  - ⇒ Esmolol 10-20 mg i.v. bolus and infusion if needed
  - ⇒ Metoprolol 1-5 mg i.v. bolus
  - ⇒ Labetalol 5-20 mg i.v. bolus
- Calcium Channel Blockers:
  - ⇒ Nifedipine 10 mg sublingual (good for arterial spasm)
  - ⇒ Diltiazem 5-10 mg i.v. q 1 h as infusion 100 mg/100cc
- Hydralazine 5-10 mg i.v. bolus q 4h
- Chlorpromazine 2.5-10 mg i.v. slowly

Resume oral medications when tolerated:

- β-Block if fast HR, good left ventricular function (LVF)
- Angiotensin Converting Enzyme (ACE) Inhibitor if poor LVF and good renal function
- Hydralazine if poor LVF and poor renal function
- Calcium channel blockers, Amlodipine if arterial conduit

Hypotension

If the BP is dropping fast, quickly put the patient in Trendelenburg, stop sedation and vasodilators. Giving volume and $CaCl_2$ 1 g i.v. are useful temporizing measures until a more definitive diagnosis is reached.

There is a wide differential and management strategies (Table 32.3).

Bradycardia

Ventricular and or atrial epicardial leads are placed intraoperatively. Manage slow rhythms by pacing preferably atrial > atrioventricular > ventricular alone. Pace patients in asystole, third degree heart block or bradycardia and if the condition persists more than 48 h the patient may need a permanent pacemaker. At Toronto General Hospital (TTH), patients cannot be transferred to the ward if totally pacer dependent without any safe underlying rhythm. Patient may go to ward on telemetry with pacer on (pacing or sensing) provided there is a slow but adequate underlying rhythm.

Sinus bradycardia may respond to low dose dopamine, isoproterenol, or epinephrine and usually recovers. (see Appendix 11a)

Tachycardia

Treatment depends on whether atrial or ventricular origin and hemodynamic stability of patient.

- ventricular fibrillation or tachycardia is managed by ACLS protocol (Appendix 11d).
- atrial dysrhythmias should be further differentiated as supraventricular tachycardia (SVT) or atrial fibrillation (AF) (Appendix 10).

Table 32.3. Diagnosis and management of hypotension

Diagnosis	Treatment
1. ↓ SVR • vasodilation (drugs, histamine, fever, ↑ CO_2, acidosis, sepsis) • ↓ blood viscosity	1. ↑ SVR • eliminate vasodilating factors • administer vasoconstrictor • correct anemia
2. ↓ preload (CVP, PA_D) • hypovolemia, venodilation • ↓ venous return (IPPV, tamponade) • loss of atrial kick • RV failure (↓ LV preload)	2. ↑ preload (CVP, PA_D) • position patient to ↑ venous return (Trendelenburg) • fluids: colloid, blood • relieve tamponade, ↓ airway pressure • restore atrial kick (AV pacing, cardiovert) • Rx: RV failure (↑ CVP, ↓PVR, inotrope)
3. ↑ afterload (↑ SVR) • hypertension, pain • vascular obstruction • ↑ airway pressure (affects RV) • hypovolemia	3. ↓ afterload (↓ SVR) • consider volume • administer vasodilator (Nipride) • consider inodilator (milrinone) • IABP
4. dysrhythmias • ↑ HR, ↓ HR • atrial • ventricular	4. treatment • correct etiology (↑/↓ K^+, ↑ Mg^{2+}, ischemia) • antiarrhythmics • pacemaker
5. ↓ contractility (↓ CO) • acute hypertension • ischemia, infarct • ventricular over distention • acute valvular dysfunction • myocardial depressants	5. systolic (inotrope) • eliminate cardiac depressants • relieve ischemia • inotropes • avoid ventricular distention diastolic (lusiotropic) • relieve ischemia • B1 agonist or phosphodiesterase inhibitor ↓ cardiac work • treat hypertension • IABP • LVAD

Premature Ventricular Complex (PVC)

This may reflect marginal coronary perfusion and ongoing myocardial ischemia. Check serum electrolytes, repeat 12 lead ECG and patient hemodynamic. Examine preoperative ECG and records as there may be pre-existing problem. Treat aggressively if multifocal and frequent.

- Check Swan-Ganz position (< 55 cm)
- Serum K^+ optimal (> 4.0 meq/L)

- Magnesium sulfate 2-4 g i.v., may repeat as it poorly correlates with serum level
- Lidocaine 100 mg i.v. bolus (may repeat 50 mg i.v. bolus), infusion 1-4 mg/min
- Amiodarone 150-300 mg i.v. slowly over 15-30 minutes, infusion 900-1200 mg/250 cc D5W over 24-48 h

Tamponade

The great masquerader of the postoperative cardiac surgery patient. Lethal but often difficult to diagnose so consider in the context of:

- sudden absence of bleeding from chest tubes
- rising filling pressures (CVP = PA_D > 20 mm Hg) with fall in BP
- fall in cardiac output (CO), poor peripheral perfusion
- falling urine output

An echo (2-D or TEE) may help distinguish right ventricular failure from tamponade or help determine if clot is compressing the atrium. The problem is more likely if the pericardium is closed, so even if negative 2-D echo the surgeon may choose to re-explore and release pericardium or evacuate clot.

GASTROINTESTINAL

Gastritis Prophylaxis

Patients who do not undergo mechanical ventilation for more than 48 h and who have no coagulopathy are at extremely low risk (0.1% vs. 6.0%) of clinically important bleeding and therefore do not require prophylaxis. Treat patients with active ulcer disease.

Both sucralfate and H_2 blockers are equally effective in decreasing the risk of bleeding by 50%. Sucralfate is cheaper and associated with 20% less risk of nosocomial pneumonia. Proton pump inhibitors are not effective due to variable oral absorption.

a) Sucralfate (Sulcrate) 1 gram po or NG qid is the first line of therapy

b) H_2 blocker can be used if patient unable to tolerate sucralfate (Table 32.4)

Table 32.4. H_2 blockers in gastritis prophylaxis

Generic	Trade	Oral prophylaxis	Parenteral
cimetidine	Tagamet	400 mg q h or 300 mg BID	300 mg i.v. q6 h
ranitidine	Zantac	150 mg q h	50 mg i.v. q8 h
famotidine	Pepcid	20 mg q h	20 mg i.v. q12 h

Nutrition

Start NG feeds early if prolonged recovery is anticipated. Regardless of bowel sounds, begin full strength formula at 10 cc/h and increase slowly. Calculate caloric requirement and monitor serum electrolytes to ensure optimal rate and amount of free water. Consider metoclopramide 10 mg i.v. q 8 h if delayed gastric emptying. Use regular NG or silastic feeding tube, but absolutely need CXR to confirm tube position prior to feeds commencing.

Total parenteral nutrition (TPN) is started only after enteral feeds have failed or if contraindicated.

Diarrhea

Most common causes in post cardiac surgery patients are enteral feeds, intestinal ischemia or pseudomembranous colitis.

Management includes a culture of stool for *Clostridium difficile* toxin, reassess drugs, and rule out hypoperfusion. Stop any gastric prokinetic agents and enteral feeds. Consider a rectal tube and Loperamide (Imodium) 4 mg PO initially then 2 mg PO after each bowel movement (max. 16 mg/d).

Liver Dysfunction

Unusual but may occur in patients with past history of liver disease (preexisting right heart failure), following an episode of hypoperfusion or use of high dose vasopressors. Watch for very high CO, low SVR, low blood sugar, persistent acidosis and elevations in liver enzymes. No specific treatment except supportive by minimizing hepatic congestion and avoiding hepato-toxins.

If preoperative ascites present, remember to fluid restrict and if possible avoid salt or saline.

RENAL

Low Urine Output

Urine output is an important reflection of overall patient condition. Management of low urine output includes:

- check volume status (filling pressures or give a volume bolus)
- check hemodynamics (BP and CO) may require alpha agonist to maintain adequate perfusion pressure and/or inotrope to optimize CO.
- ensure patency of urinary catheter (flush with saline if needed)
- stop all nephrotoxins (Indomethacin, aminoglycosides, ACE inhibitors)
- start Dopamine 1-3 ug/kg/h
- give Diuretics

⇒ furosemide 10-300 mg i.v. bolus
⇒ metolazone 5-10 mg po/NG prior to furosemide bolus
⇒ ethacrynic acid 50-100 mg i.v. bolus
⇒ furosemide drip 10-20 mg/h

High Urine Output

May occur due to mannitol in the CPB prime, furosemide given in the operating room, low dose dopamine infusion, or SIADH.

Try to keep patient volume replete, stop dopamine if possible and consider single dose desmopressin 4 μg i.v. for treatment of SIADH.

Dialysis

Required in postoperative patients who either have pre-existing renal failure or develop acute renal dysfunction unresponsive to diuretics.

Intermittent hemodialysis is used if the patient is hemodynamically stable. Continuous abdominal peritoneal dialysis (CAPD) can be continued if there is a catheter in situ. The peritoneal cavity may communicate with the mediastinum draining fluid from the chest tubes and not be effective.

Ultrafiltration or continuous hemodialysis may be started using continuous arterial-venous (CAVHD) or continuous venous-venous (CVVHD) approach. This may be better tolerated by hemodynamically unstable patients.

HEMATOLOGIC

Bleeding

See Appendix 8 on algorithm for management of microvascular bleeding.

Thrombocytopenia

Common occurrence postoperatively and usually self-resolving. Could be related to hemodilution, intra-aortic balloon pump (IABP), amrinone, heparin induced thrombocytopenia (HIT) or sepsis. Discontinue amrinone and heparin (test for HIT). Transfuse only if platelet count < 20,000 or if an invasive procedure planned. If the patient requires anticoagulation, use an alternative to heparin.

METABOLIC

Diabetes Mellitus

Patients are often seen by the endocrine service preoperatively and already have a sliding insulin scale written (Table 32.5).

Patients on multiple inotropes may need a more generous insulin dose.

Potassium (K^+)

Only 2% of total body K^+ is extracellular (70 meq of 3500 meq total lean body weight) and measured by serum levels (normal serum K^+ = 2 - 4 meq/L).

There is a nonlinear relationship between total body K^+ and serum K^+ (Table 32.6). Depletion causes less change in serum K^+ due to intracellular

Table 32.5. Insulin scale for perioperative hyperglycemia

blood sugar (mmoles/l)	blood sugar (mg/100ml)	infusion rate
> 20	400	2 units/h
15-20	300-400	1.5 units/h
10-15	200-300	1.0 units/h
<10	<100	none

Humulin R 25 units/250 D5W (1 unit = 10 cc)

translocation to replenish stores while excess results in a better correlation with serum K^+.

Estimate deficit as decrease 1 meq/L in serum K^+ causes a 10% decrease in total body K^+ (50 meq/L lean body weight).

Calcium (Ca^{2+})

Normal serum levels are indicated in Table 32.7.

Hypoalbuminemia decreases the protein bound fraction and results in a decrease in total serum calcium but no change in serum ionized Ca^{2+}. Correction factor is every decrease of 1 mg/dl albumin increases total serum Ca^{2+} by 0.8 mg/dl.

Ionized calcium be should measured quickly at body temperature with minimal heparin in sample as a measured decrease may be reported with alkaline pH, low serum sodium or heparin.

Magnesium (Mg^{2+})

Magnesium is the second most abundant intracellular cation (Table 32.8) but uneven distribution in the body: 1000 mmoles total in body, serum 2.6 mmoles (0.3%), bone 530 mmoles (53%). Serum levels are not checked routinely at TTH as there is a poor correlation with total body levels. Urinary Mg is likely a better indicator of total body stores.

Measured low serum levels are treated with magnesium sulfate 2.0-4.0 g i.v. This is also of therapeutic value in patients with atrial or ventricular arrhythmias and normal serum levels.

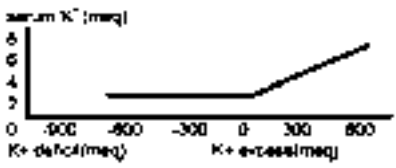
Metabolic Acidosis

Calculation of anion gap is based on serum levels of $Na^+ - (Cl^- + HCO_3^{2-}) = 10 - 12$ mmol/L = 140 - (105 + 25).

Most common cause in CVICU is hypoperfusion (increased lactate), renal failure or diabetes (increased ketones) (Table 32.9).

Physiological correction involves for every decrease in HCO_3 by 1 meq/L causes a decrease in pCO_2 by 1.1 mm Hg (though pCO_2 can not be < 10 mm Hg).

Table 32.6. Potassium homeostasis



K+ deficit		
serum meq/l	wt (lb)	meq
3.0	10	350
2.5	15	470
2.0	20	700

NB. 1 meq/L K+ = 1 mmol/L K+

Hypokalemia (< 3.5 meq/L)

Ddx	Transcellular shift ⇒ B agonists (epinephrine, dobutamine), alkalosis K^+ depletion ⇒ diuretics, NG, vomit, low CO_2, cirrhosis, steroids, diarrhea		
Sx	muscle weakness, CNS changes, AV block, ST segment depression "u" wave, T wave flat, potentiate digoxin-toxic arrhythmias		
Rx	postoperative protocol based on urine output and initial K^+ = 4.0-5.0meq/L		
IV	U/O (cc/h)	meq KCl	
	0-50	0-5	if $K^+ > 5.0$ KCl is held and recheck level
	50-100	5-10	if $K^+ < 4.0$ patients get 10 meq KCl/ h
	> 100	10-15	
	> 200	15-20	
PO	liquid: Kaochlor (10%)/Kciel: 15 cc = 20 meq tablets: Slow K 1 tab = 8 meq, KDur 1 tab = 10 meq		
	correct acidosis (increase PCO_2, decrease HCO_3) avoid > 40 meq KCL/L/ hour through central line give magnesium sulfate if refractory hypokalemia		

Hyperkalemia (>5.5 meq/L)

Ddx	Transcellular shift ⇒ myonecrosis, insulin lack, acidosis, drugs (β-blockers, succinylcholine, digoxin toxicity) ↓ Renal excretion ⇒ renal insufficiency, adrenal insufficiency, drugs (ACEI, diuretics, NSAIDS, heparin) Pseudohyperkalemia ⇒ hemolysis, increased platelets or WBC
Sx	skeletal muscle weakness, diarrhea peaked T wave, ↓ p wave, ↑ PR, ↑ QRS, asystole
Rx	should treat if > 6.0 meq/L or if any ECG changes consider a) $CaCl_2$ 1.0 gm i.v. b) $NaHCO_3$ 1 amp i.v. (only if able to eliminate additional CO_2 produced) c) Humulin R 10 units i.v. bolus with D50W if low glucose d) hyperventilate, salbutamol inhalation (renal failure) e) Furosemide 10-20 mg i.v. f) Kayexalate 20-50 g in 20% Sorbitol PO or PR q3 h g) dialysis (if renal failure)

Table 32.7 Calcium homeostasis

normal serum calcium levels	mg/dl	mmol/L
total serum calcium	8.5-10.2	2.1-2.5
ionized serum calcium	4.8-7.2	1.1-1.3

NB. Conversion factor to SI units is 0.25 x traditional unit

Ionized hypocalcemia

Ddx	sepsis, alkalosis, acute pancreatitis (↓ PTH) hypomagnesemia (↓ PTH) ⇒ Rx magnesium renal failure (↑ phosphorus) ⇒ Rx by lowering phosphate miscellaneous: blood transfusion, CPB, burns, drugs
Sx	neuromuscular excitability hypotension, left ventricular failure, increase QT interval
Rx	if heart failure or hypotension may promote vasoconstriction may decrease ventricular compliance and worsen diastolic dysfunction Calcium chloride ($CaCl_2$ 10%) ⇒ 272 mg (13.6 meq) Ca^{2+} Calcium gluconate (10%) ⇒ 90 mg (4.5 meq) Ca^{2+} initial dose 1 ampule $CaCl_2$ over 10 minutes

Hypercalcemia

Ddx	hyperparathyroidism, malignancy
Sx	altered mental status, coma, ileus, hypotension, renal failure
Rx	Rx > 13 mg/dl a) diuresis with saline and loop diuretics b) calcitonin 4 U/kg IM or SQ q12h x 2 doses (decrease bone resorption) c) mithramycin 25 μg/kg i.v. bolus q 2 days x 2 doses d) dialysis

Treatment depends on the underlying cause and hemodynamic profile of the patient.

- calculate total meq deficit of bicarbonate by using the measured base deficit on arterial blood gases and either of the following formulas.
  ⇒HCO_3 deficit x 40% TBW(where TBW = 60% total body weight)
  ⇒HCO_3 deficit x 0.3 x weight
- Remember that administered sodium bicarbonate ($NaHCO_3$) is converted to carbon dioxide increasing the patient's pCO_2. Give $NaHCO_3$ only if the patient can eliminate the additional CO_2 produced.
- Correct by administering 1 ampoule of $NaHCO_3$ (50 cc) which contains 44 meq HCO_3. Aim to correct at least one half the calculated deficit.

Table 32.8. Magnesium homeostasis

Normal serum levels = 0.8-1.2 mmol/L = 1.6 -2.4 meq/L
NB. Conversion factor to SI units is 0.50 x traditional unit

	Hypomagnesemia
Ddx	diuretics (loop, osmotic), alcohol, aminoglycosides, amphotericin, decrease intake (i.v. poor Mg), diarrhea
Sx	decreased serum potassium, phosphate, sodium, calcium arrhythmias in acute MI, intractable arrhythmias digitalis cardiotoxicity, ↑ QT on ECG muscle weakness, seizures tremors
Rx	normal 1 mmol = 2 meq deficit ⇒ 1-2 meq/Kg (70-140 meq) $MgSO_4$ 2 g (16 meq)/1-2 min $MgSO_4$ 5 g (40 meq)/6 h $MgSO_4$ 5 g (40 meq) q 12h reduce by 50% with renal insufficiency should normalize serum levels over 24 h
	Hypermagnesemia
Ddx	renal failure + Mg, diabetic ketoacidosis, pheochromocytoma
Sx	hypotension (3.0-5.0 meq/L) complete heart block (> 7.5 meq/L) respiratory depression, coma (> 10 meq/L)
Rx	i.v. calcium loop diuretics and volume

Table 32.9 Differential diagnosis of anion gap

normal anion gap (↑ chloride)	**↑ anion gap (normal chloride)**
HCO_3 loss	**H^+ gain**
gut ⇒ diarrhea, small intestine/ pancreas, ureterosigmoid, ileal loop renal ⇒ renal tubular acidosis, carbonic anhydrase inhibitor, HCl	ketoacidosis: starvation, alcohol, diabetes, lactate: a = hypoperfusion, b = no hypoperfusion intoxication: salicylate, methanol, ethylene glycol, paraldehyde, uremia

INFECTIOUS DISEASE

Fever

In the first 24 h postoperative it is reasonable to treat symptomatically. Continue preoperative antibiotics in patients with a pre-existing infection. Postoperative patients receive either:

- cefazolin 1 g i.v. q8 h (total 6 doses) if allergic, then vancomycin or clindamycin to be used:
  vancomycin 1 g i.v. q12 h (1 dose if CABG, 2 doses if valve procedure)
  clindamycin 600 mg i.v. q8 h 6 doses.

If the patient is febrile beyond 24 h, culture urine, blood, sputum and check white blood cell count. If the patient has prosthetic material consider early antibiotic treatment.

Invasive Line Changes

Central lines if not obviously infected prophylactic line changes should be every 7-10 days depending on patient condition and alternate available access.

Arterial lines are changed only if the site is inflamed, infected or in a position that interferes with mobilization.

Selected Readings

1. The ICU Book, Paul Marino, Lea and Febeiger, (1991).
2. Manual of Intensive Care Medicine, edited by Rippe and Csete, Little Brown Spiral, (1983).

Chest Reopening

Terrence M. Yau

INTRODUCTION

Chest reopening in the early postoperative period may be diagnostic, therapeutic, or both. Reopening may be indicated for hemorrhage, tamponade, shock or cardiac arrest. Postoperative sternal osteomyelitis or mediastinitis may prompt resternotomy and sternal dehiscence may require repeat sternal closure. Finally, early reoperation for graft thrombosis, failure of a valve repair or prosthetic valve dysfunction may occasionally be required.

INCIDENCE

In 1997 the overall incidence of chest reopening in 2460 patients was 5.4%. Incidence by procedure (Table 33.1) and indications (Table 33.2) are summarized for all patients.

INDICATIONS

Postoperative Hemorrhage

With the advent of intensive blood conservation efforts in the last decade, shed blood volume and the requirement for blood transfusion have dropped significantly.

Our tolerance for postoperative blood loss has decreased in parallel. Without a significant coagulopathy, or if adequate clot formation is noted in the chest drains, re-exploration is indicated for drainage of 200 cc of blood in 1 h, or sustained drainage of 100 cc per hour for 3-4 h.

Perioperative Care in Cardiac Anesthesia and Surgery, edited by Davy C.H. Cheng and Tirone E. David.

Table 33.1. Incidence of chest reopening by procedure at TGH

Procedure	# of patients	%
Isolated CABG	1613	4.3
Valve and combined CABG/Valve	467	7.1
Other procedures*	380	8.2

* includes cardiac transplantation, thoracic aortic surgery, congenital heart surgery, others

Table 33.2. Incidence of chest reopening by indication at TGH

Indications	Incidence
Postoperative hemorrhage	3.0%
Tamponade	0.6%
Shock or cardiac arrest	0.6%
Sternal osteomyelitis or mediastinitis	0.6%
Sternal dehiscence	0.04%
Redo surgery	0.2%
Other indications	0.3%

Clinical suspicion of a surgical site of bleeding or hemodynamic instability should prompt earlier reintervention.

The objectives of re-exploration are to establish hemostasis and to evacuate the hemopericardium and/or hemothorax, preventing further fibrinolysis and coagulopathy.

Tamponade

Cardiac tamponade remains a clinical diagnosis, although ancillary investigations including echocardiography (generally transesophageal) may at times be useful.

Immediate resternotomy in the CVICU or on the ward is indicated for critical hemodynamic instability or collapse with a clinical picture suggestive of tamponade. A more indolent course, frequently allowing confirmation by echocardiography, may allow transportation of the patient back to the operating room.

Constriction of the heart by pericardium that has been tightly closed may cause clinical signs identical to tamponade caused by hemopericardium.

Patients on warfarin may develop a large or symptomatic hemopericardium or pericardial effusion 4-7 days after surgery, well after removal of chest drains and after transfer of patients to the ward. Our practice has been to evacuate this collection in the operating room by reopening only the lower portion of the incision. This allows placement of a suction device into the pericardium through a subxiphoid approach and evacuation of the pericardial collection. A chest tube is left in the pericardium for 24-48 h.

Shock or Cardiac Arrest

Hemodynamic collapse may require emergent chest reopening for diagnosis and/or treatment. Inability to achieve adequate cardiac output by closed cardiac massage is an indication for chest reopening.

Resternotomy for shock or cardiac arrest may allow diagnosis of graft problems, including kinking, spasm or thrombosis, problems with a prosthetic valve, tamponade due to hemorrhage and/or pericardial constriction, or major hemorrhage.

Immediate treatment of a kinked or spastic graft, tamponade, or control of hemorrhage may then be affected.

Sternal Osteomyelitis or Mediastinitis

Diagnosis is clinical, with fever, sternal instability and leukocytosis with or without purulent discharge from the wound indicating the probability of deep sternal infection. CT scan may be useful to demonstrate bony destruction and to rule out mediastinal collections.

Early reoperation to debride the sternum with primary closure is preferable. In cases of extensive bone destruction, debridement with concomitant placement of pectoral flaps is performed. Patients with sternal osteomyelitis require 4-6 weeks of intravenous antibiotics.

Sternal Dehiscence

Dehiscence presents with copious serosanguineous drainage from the sternal incision coupled with varying degrees of sternal instability.

Treatment consists of early resternotomy, intraoperative cultures to rule out infection, sternal rewiring and routine wound closure.

Redo Surgery

Reoperation for early failure of a graft, valve repair, prosthetic valve, or other indication may in rare circumstances be required.

TECHNIQUE OF EMERGENCY CHEST REOPENING

Equipment

An emergency resternotomy tray should be kept on hand in the CVICU and on every ward. This tray should contain all the instruments necessary for rapid chest reopening, including scalpels, wire cutters and a sternal retractor.

In addition, a mobile cart with ancillary supplies such as skin preparation solutions, surgical drapes, sterile surgical gloves, gowns, masks, sutures and suction tubing should also be available.

Portable operating lights and cautery machines may be obtained from the operating suites.

Personnel

Besides the operating surgeon, a surgical assistant is helpful.

In addition, a nurse or respiratory therapist to ventilate the patient by bagging with 100% O_2 is required. If the patient had been extubated prior to the time of hemodynamic collapse, emergency reintubation by a physician or trained respiratory therapist is mandatory.

An anesthesiologist or intensivist, if available, can attend to anesthetic considerations and supervise pharmacological circulatory support.

Notification of the operating suites that an emergency reopening is underway will allow preparation of an operating room and a cardiopulmonary bypass circuit if one is required, as well as recall of operating room nurses, a perfusionist and an anesthesiologist.

Opening

Rapid skin preparation and draping is performed with the patient in the CVICU bed. A scalpel is used to cut all sutures down to the sternum. The sternal wires are cut and removed, and a sternal retractor placed.

Evaluation and Initial Management

Clinically significant tamponade, if present, can be immediately confirmed and treated by reopening the pericardium. The volume of blood and/or clot in the mediastinum and/or pleural spaces indicates whether significant hemorrhage has occurred.

The primary objective is to support and maintain adequate systemic perfusion, which may require reopening alone, internal cardiac massage, or further measures. Internal massage must be performed carefully to avoid injury to the right ventricle, any coronary artery bypass grafts, or the left ventricle in patients with a prosthetic mitral valve.

A careful evaluation of all surgical sites may allow identification and control of sources of ongoing hemorrhage if present.

Definitive Treatment

After chest reopening for hemorrhage or tamponade, the hemopericardium is completely evacuated and any surgical bleeding sites are controlled. The pericardial space is irrigated with warm saline and suctioned dry to ensure that hemostasis is adequate. When a coagulopathy is identified, administration of plasma, cryoprecipitate or platelets may be required. Fibrin glue or other topical hemostatic agents may be useful.

Resternotomy for hemodynamic collapse or cardiac arrest necessitates an exhaustive search for the precipitating cause including careful evaluation of coronary bypass grafts to rule out kinking, thrombosis or spasm, and/or evaluation of prosthetic or repaired valves, which requires echocardiography in addition to palpation. In a minority of cases, cardiopulmonary bypass may be required for definitive correction of the problem.

When chest reopening is performed for deep sternal infection or mediastinitis, all collections of blood, fluid or pus are drained and all necrotic bony and soft tissues are debrided back to healthy vascular tissue. Swabs from the mediastinum and sternum for microbiologic analysis will direct antibiotic therapy. In the majority of cases, one-stage closure with pectoralis major flaps can be accomplished, leaving drains in the mediastinum and under the flaps to prevent hematoma formation or reaccumulation of fluid collections.

Closure

Check temporary epicardial pacing wires, as these are frequently dislodged during re-exploration.

Flush chest drains to ensure patency, and replace if necessary.

Pericardial closure is at the discretion of the surgeon unless reopening was required for tamponade due to or exacerbated by pericardial constriction.

Sternal closure is performed in the routine manner, but a greater number of wires and/or heavy gauge wire is used in large patients or when the sternum has been unstable.

Skin closure is performed with either an absorbable subcuticular suture or a running vertical mattress closure with polypropylene suture.

OUTCOMES

Mortality

The need for chest reopening is a marker of unfavorable outcomes and was associated in our 1997 experience with a perioperative mortality rate of 18.5%. Mortality in patients undergoing resternotomy for hemorrhage was 12%, for tamponade or sternal osteomyelitis 7%, but patients undergoing reopening for shock or cardiac arrest had a mortality of 71%.

Deep Sternal Infection

Resternotomy was associated with a 3- to 4-fold increased rate of deep sternal infection. In multivariate logistic analyses, however, the requirement for chest reopening for reasons other than sternal infection was not an independent predictor of subsequent sternal osteomyelitis or mediastinitis.

Length of Stay

Patients requiring chest reopening stayed a mean of 3.5 days in the CVICU and 17.5 days in the hospital (vs. 1.3 and 8.3 days, respectively, for patients not requiring resternotomy).

SUMMARY

Chest reopening in the early postoperative period may be required for emergency diagnostic or therapeutic purposes. The CVICU and wards should have an emergency resternotomy tray and appropriate equipment on hand. Restoration of hemodynamic stability in unstable patients is the first priority, followed by careful evaluation of all surgical sites, grafts, and prosthetic or repaired valves to ascertain what further interventions are required. Reopening is associated with increased mortality, morbidity and length of stay. Meticulous attention to detail at the time of initial operation may minimize the need for resternotomy.

Sternal Debridement and Muscle Flap Coverage

Tracey A. Thompson, Bing Siang Gan

INTRODUCTION

Incidence of deep sternal infection ranges between 0.5% and 5.9%; most major centers report between 0.5% and 2.0%. At Toronto General Hospital (TTH) it is 0.8%. The risk factors for sternal wound infections or mediastinitis are listed in Table 34.1. It is associated with high mortality; it has decreased from as high as 50% to 5% to 10% with radical debridement, flap coverage and primary closure (preferred method in our hospital).

DIAGNOSIS OF MEDIAN STERNOTOMY INFECTION

Clinical Features

- redness and tenderness
- wound drainage/discharge (in 70-90% of cases)
- sternal instability ("click")
- frank dehiscence with exposed deep structures
- fever
- leukocytosis
- systemic sepsis

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Table 34.1. Risk factors for sternal wound infections/mediastinitis

ITA harvest (Bilateral > Unilateral)
Obesity
Diabetes mellitus
COPD/smoking history
Prolonged operative time
Early postoperative blood transfusions
Early chest re-exploration
Sterile dehiscence requiring sternal rewiring
Prolonged postoperative ventilation
Poor general health

Classic signs and symptoms of infection may be absent. A small percentage of patients only have fever and leukocytosis. These patients may still develop multisystem organ failure.

INVESTIGATIONS

- wound cultures and blood cultures if septic
- white blood cell (WBC) count
- rule out other septic foci (e.g., mesenteric infarction, empyema of gallbladder)
- CT/MRI to document substernal fluid/air collections, sternal integrity, associated pleural collections
- bone/gallium scan is of no value in the early postoperative period

SEVERITY OF INFECTION

- sterile dehiscence is wound breakdown with no clinical or microbiologic evidence of infection
- infected dehiscence is wound breakdown with clinical or microbiologic evidence of infected presternal tissue, sternal osteomyelitis, mediastinal sepsis, and/or unstable sternum

CLASSIFICATION

- superficial: skin & subcutaneous tissue
- deep: exposed bone with or without unstable sternum
- complicated: deep infected and/or necrotic, unstable sternum, or exposed heart
- systemic sepsis, hypotension, delirium

PRINCIPLES OF SURGICAL MANAGEMENT

Identify infecting organism(s) by wound and bone cultures

Reduce bacterial load by (a) copious irrigation, (b) removal of hardware, (c) debridement of all necrotic bone and costochondral cartilage

Eliminate dead space
Provide well vascularized tissue cover
Primary wound closure if patient stable
Delayed flap elevation and delayed primary closure is optional in unstable patients though a single stage procedure dramatically reduces length of hospital stay
Liberal use of suction drains

ANTIBIOTIC THERAPY

Perioperative antibiotic therapy guided by culture results

- Partial sternectomy: Postop i.v. antibiotics x 6 weeks
- Total sternectomy: Postop i.v. antibiotics x 2-3 weeks

SURGICAL INDICATIONS

Sterile Sternal Dehiscence

- May be adequately treated with simple sternal rewiring and reclosure
- Consider sternal debridement and muscle flap coverage if:
  a) Inadequate bone stock
  b) Obese patients or those with chronic obstructive pulmonary disease (COPD)
  c) Prior internal thoracic artery (ITA) harvest

Superficial Infections

- May be adequately treated with simple drainage, debridement, dressing changes and closure by secondary intention or delayed primary closure

Table 34.2. Sternal involvement

Upper 2/3	Pectoralis major Uni/bilateral depending on defect size Turnover, pedicled or combination
Lower 1/3	Rectus Abdominus Omentum Pectoralis major Turnover: split into 2 elements (smaller superior and larger L shaped inferior portions)
Total sternum	Combination of pectoralis major and rectus abdominus/omental flaps

Table 34.3. Flap options

	Advantage	Disadvantage	Contraindications
Pectoralis major:			
Pedicled flap:			
◊ Based on pectoral branch of thoraco-acromial artery ◊ Divide humeral and clavicular attachment to increase rotation	◊ Simple dissection in same operative field ◊ Large muscle ◊ Can be used when ITA used for previous procedure	◊ May not reach inferior sternum ◊ Contour deformity: breast & loss of anterior axillary fold	
Turnover flap:			
◊ Based on ITA perforators	◊ Preserves anterior axillary fold ◊ Preserves lateral 1/3 and neurovascular pedicle ◊ Segmental use: lower portion can cover the inferior sternum portion of muscle can be used for small defects		◊ side of ITA harvest
Rectus Abdominus			
Deep superior epigastric artery (abdominal extension of ITA)	◊ Simple dissection ◊ Reliable ◊ Covers inferior sternum	◊ Abdominal weakness/herniation ◊ Requires extension of incision	◊ Ipsilateral ITA harvest: may base flap on costomarginal supply
Omentum			
Right and/or left gastroepiploic arteries	◊ Fast flap harvest and inset reducing OR time ◊ Broad, well vascularized surface area ◊ Contours and fills deadspace much easier ◊ May be lengthened along vascular arcades ◊ Immunological contribution of B & T macrophages	◊ Requires laparotomy ◊ Hernia (20%) ◊ Hematoma (8%) ◊ Seroma (4%) ◊ Adhesions/Small bowel obstruction (rare) ◊ Intra-abdominal extension of infection (rare) ◊ Variable flap vascularity, size, and shape	◊ Previous abdominal operations ◊ Ascites secondary to liver failure

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Deep Infections

- Remove hardware
- Obtain bone biopsies
- Debride sternum to healthy solid bone with bleeding margins
- Provide well-vascularized tissue, eliminate dead space and close primarily

Table 34.4. Early complications of flaps

Hematoma	8-10%
Wound dehiscence	2-6%
Re-exploration/wound necrosis	6%
Flap loss	major = 3%; minor = 3-4%
Abdominal hernia	2-3%
Rectus abdominus	11% rate of true hernia formation 42% rate of fascial weakness/bulging
Omental flaps	11%
Recurrent wound infection	6.5%
Costochondritis	5-8%
Late osteomyelitis	1%
Flail chest segment	extremely rare

FLAP SELECTION

FACTORS

- Site of sternal involvement: upper 2/3, lower 1/3 or total sternum (Table 34.2)
- Previous ITA harvest
- Long term functional results

Flap options are summarized in Table 34.3.

COMPLICATIONS

Incidence of early complications are listed in Table 34.4

Hematoma has equivalent incidence following pectoralis major and rectus abdominous flaps

Risk factors for wound dehiscence include, COPD/smoking history, obese women with large pendulous breasts

Late patient complaints

- Sternal Instability 45%
- Chest discomfort/pain 40-50%
- Chest numbness/dysaesthesia 44%
- Arm weakness (operated side) 25-35% (pectoralis major flap patients)
- Chest deformity 56% (pectoralis major flap patients)
- General patient satisfaction 72% satisfied with chest appearance
75% rate scars good to excellent
83% satisfied with overall result
- Return to work 48% of eligible patients

OUTCOME

Mortality: 5.3-9.9% in the literature (TTH 0% for 1995-1997)

Morbidity: 20% early or flap closure complications (TTH 10%), 15% late complications

Length of hospital stay: still decreasing, 13 days (TTH 1993-1996) vs. 19 days (TTH 1975-1992)

SUMMARY

Debridement of infected sternal wounds with muscle or omental flap closure achieves permanent wound healing in more than 95% of patients. Indications for surgery depend on the depth and severity of infection as well as the degree of sternal involvement. Flap choice depends on the prior harvest of internal thoracic arteries and the level of sternum affected. Patients may be counseled on the risks of postoperative chest discomfort, sternal instability and diminished chance of return to work. An increased risk of abdominal wall complications can be expected after using rectus abdominous or omental flaps.

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Difficult Weaning from Mechanical Ventilation

Richard M. Cooper

INTRODUCTION

Many patients presenting for cardiac surgery have co-existing pulmonary disease. Emergent surgery may not provide adequate time to optimize their respiratory status. Even with elective surgery, there may be little room for improvement.

Cardiopulmonary bypass can induce additional lung injury, due to

- microemboli
- complement, coagulation and fibrinolytic pathway activation
- leukoaggregation
- atelectasis

Early postoperative respiratory insults include:

- inhibition of cough by residual narcotics or pain from sternotomy
- impaired mucociliary clearance
- increased extravascular lung water

Most cardiac surgical patients are successfully weaned from mechanical ventilation soon after surgery under fast track cardiac anesthesia technique. The routine ventilation and extubation management has been discussed in chapter 30 and in appendix 6.

IDENTIFICATION OF THE SLOW-TO-WEAN PATIENT

Preoperative Predictors

Attempts at preoperative prediction of patients requiring prolonged mechanical ventilation have been inconsistent and largely unsuccessful. Several investigators have found that older patients, particularly those with impaired ventricular function, are slower to be extubated. The predictive findings in fast-track cardiac anesthesia is currently being evaluated (see chapter 2).

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Intraoperative Predictors

The following have not been prospectively evaluated with regard to their predictive value but might be expected to contribute to more complicated ventilatory course:

- prolonged cardiopulmonary bypass time
- adverse reaction to protamine, other drugs or blood components
- high oxygen requirements
- high ventilation pressures
- hemodynamic instability, particularly low cardiac output state

Postoperative Predictors

Hemodynamic instability, particularly low cardiac output state combined with anemia reduces availability of oxygen to respiratory muscles. As much as 25% of total body oxygen consumption may be involved in the performance of respiratory work.

Dysrhythmias and increased bleeding may delay extubation.

Preoperative nutritional depletion may be aggravated by delayed postoperative feeding.

MANAGEMENT OF THE SLOW-TO-WEAN PATIENT

Patients Requiring Suctioning or Unable to Protect Their Airways

Assess quantity and quality of pulmonary secretions. Is frequent suctioning of ETT required or can the patient clear his/her secretions? If pulmonary secretions are the only criterion preventing extubation, consider a mini-tracheostomy for pulmonary toilet.

Check for a leak around the ETT when the patient exhales with the cuff deflated and the tube occluded. The absence of a cuff leak reduces the likelihood of successful extubation. If intubation had been easy and the patient otherwise qualifies for extubation, it might be undertaken. Racemic epinephrine, helium/oxygen, head-up positioning and systemic steroids might be considered. If intubation had been difficult, consider extubation over tube exchanger or tracheostomy.

The gag reflex may be absent. If the patient is alert, this should not preclude extubation.

Discontinue enteral feeds for an appropriate period, prior to and following extubation.

Ventilatory Insufficiency

Patients may have inadequate alveolar ventilation and/or elevated $PaCO_2$ due to:

- increased dead space (alveolar overdistension, hypovolemia, low cardiac output, pulmonary embolism)
- excessive CO_2 production (fever, increased metabolic rate, excessive carbohydrate load)

- over-sedation, respiratory depression, splinting, or inadequate analgesia
- problem should be identified and corrected if possible

Work of breathing increases with inspiratory resistance, larger tidal volumes, or a reduction in respiratory compliance:

- increased inspiratory **resistance** may result from a small diameter, long or kinked endotracheal tube, respiratory secretions, airway edema or bronchoconstriction. These elements are dynamic and may vary with respiratory rate and degree of patient discomfort.
- **compliance** may be altered by intrinsic (atelectasis, infiltrates, pulmonary edema) or extrinsic factors (pleural effusions, muscle rigidity, gastric or abdominal distention, etc.). Increased compliance and/or reduced resistance can result in gas entering the distal airways more readily than it can exit, resulting in progressively increasing lung volumes and volo/barotrauma.

Intrinsic PEEP (auto-PEEP) may go unrecognized by the clinician and ventilator manometer but not by the patient. The distal airways are subjected to the sum of extrinsic PEEP (set on the ventilator) and intrinsic PEEP. Appropriate levels of PEEP will diminish venous admixture and reduce right and left ventricular afterload. Excessive PEEP may exacerbate pulmonary deadspace, further reduce venous return and augment ventricular afterload. In addition, overdistension of the lungs will increase the work of breathing and reduce diaphragmatic efficiency. The presence of intrinsic PEEP can be identified on a flow/time curve by incomplete exhalation at the onset of a respiratory effort and quantified by using a Braschi valve or expiratory pause.

Central causes of ventilatory insufficiency: narcotics, chronic hypercapnia ± metabolic alkalosis, hypothermia, hypothyroidism, sleep deprivation. Metabolic alkalosis and sleep deprivation are common problems in the CVICU patients due to diuretic administration, inappropriately timed or persistent daytime sedation and nocturnal stimulation resulting in disruption of diurnal patterns.

Muscular causes of ventilatory insufficiency: residual neuromuscular blockade, aminoglycosides, local anesthetics, antiarrhythmics, myasthenia gravis, nutritional depletion, hypophosphatemia, hypokalemia, hypomagnesemia, critical illness neuropathy, glucocorticoids. Hyperinflation may result in excessive diaphragmatic muscle length thereby diminishing strength. Inadequate oxygen transport or substrate depletion may lead to relative starvation of the respiratory muscles.

Effect of CPAP/PEEP on Hemodynamics

Provide some CPAP or PEEP to all postoperative cardiac patients as this will:

- help increase FRC and pulmonary compliance while reducing atelectasis
- translocate extravascular lung water
- diminish the work of breathing and maintain patency of conducting airways and alveoli

With severe ARDS, it has been demonstrated that the PEEP may only serve to over-distend the compliant alveoli, exacerbating acute lung injury.

Excessive PEEP may reduce venous return, increase alveolar deadspace, increase pulmonary vascular resistance and diminish cardiac compliance. These may be overlooked by the increase in PaO_2, but the product of cardiac output and arterial oxygen content better reflect the net effect of PEEP.

By reducing venous return and LV transmural pressure and therefore, LV afterload, PEEP may have a beneficial hemodynamic effect in the failing heart.

Modes of Weaning

Most common choices include a conventional "T-piece wean", synchronized intermittent mandatory ventilation (SIMV) and inspiratory pressure support ventilation (PSV). Selection is often empiric or based upon local preference. Transitions are most abrupt with T-piece and generally most comfortable and physiologically acceptable with PSV.

A trial of spontaneous breathing will assist in determining the probable success of the wean. This can be conducted with PSV, CPAP or T-piece. PSV can be at a low level to overcome the inspiratory resistance of the ventilatory circuit. Either PSV or machine-delivered CPAP provides continuous monitoring of respiratory rate, tidal volume and apnea alarms.

SIMV allows the clinician to determine the minimum minute ventilation (by presetting tidal volume and respiratory rate) while the patient breaths spontaneously between machine delivered breaths. Transitions are generally smooth, however the wean may be prolonged due to infrequent reductions in the respiratory rate. Patients weaned by SIMV may benefit by increased levels of ventilatory support at night to ensure sleep and respiratory muscle recovery.

PSV weaning allows variable inspiratory assistance, permitting the patient to establish their own rate and tidal volume. Tidal volumes may vary from breath to breath depending upon airway resistance and compliance. Respiratory rates greater than 30 probably reflect inadequate pressure support, while rates less than 20 may suggest that it is excessive. Increased nocturnal support may facilitate daytime weaning.

Cardiac Considerations

Increased extravascular lung water or large pleural effusions will put the patient at a mechanical disadvantage when attempting to assume spontaneous ventilation.

Review cumulative fluid balance. Compare this to the patient's preoperative weight. If aggressive diuresis is indicated, ensure that electrolyte abnormalities are corrected. Diuretic-induced metabolic alkalosis combined with chronic hypercapnia may interfere with central respiratory drive. Consider giving acetazolamide 250-1000 mg/d for a few days.

Patients with poor ventricular function, consider afterload reduction.

PEEP/CPAP may be reducing ventricular preload and afterload, benefits which may be lost after extubation.

Marked spontaneous inspiratory effort will increase venous return and may precipitate onset of volume overload. Manage the patient using accessory muscles

and acutely elevated RA or PCW pressures, with aggressive preload reduction (nitrates or diuretics).

Respiratory Considerations

Endotracheal tube patent, unkinked and of adequate diameter.

Review CXR for absence of parenchymal infiltrates, large pleural effusions, gastric or abdominal distention.

Wheezing should prompt consideration of bronchodilator therapy.

In weaning patients with chronic hypercapnia, base decisions on clinical assessment. Weaning can continue if not complicated by hemodynamic instability, cardiac dysrhythmias, significant tachy/bradycardia, myocardial ischemia, agitation, diaphoresis or arterial desaturation (< 90%).

If CPAP is used, select equipment which imposes a minimal resistive load.

Metabolic and Nutritional Considerations

Fever will increase oxygen consumption and carbon dioxide production.

Underfeeding will fail to provide metabolic substrate for weaning demands.

Overfeeding, particularly with an excessive carbohydrate load will increase CO_2 production.

O_2 consumption and CO_2 production measured with a metabolic cart may differ from the calculated value (Harris-Benedict equation). Ideally, do this during weaning to accurately assess metabolic rate. Occasionally patients with severe hypercapnia may need to receive higher proportion of calories from fat.

Correct levels of magnesium, potassium, calcium and phosphate if deranged.

Metabolic (e.g., hypothyroidism), neuropathic (e.g., Parkinson's disease, motor neuron disease, diaphragmatic paralysis, aminoglycosides, myasthenia gravis, critical illness polyneuropathy) or myopathic problems (e.g., steroids, atrophy) may play a role.

Correct significant anemia (to ≥ 100 g/L).

Psychological

Assure that patient has slept and is adequately rested prior to beginning to wean.

Patients will generally breath more efficiently when sitting upright. However, recognize that for a weakened patient, assuming the upright position may in itself be exhausting.

Set achievable goals and provide supportive atmosphere. Earn the patient's trust and confidence.

If the patient is clinically depressed, consider psychotherapy or antidepressant medication.

Mobilization with a walker, even if "bagging" is required, may improve motivation and emotional state.

Weaning should exercise, not exhaust the patient.

INDICATIONS FOR TRACHEOTOMY

Timing of tracheotomy is not absolute; there is no point at which this becomes mandatory. It is more important to make this determination on the basis of progress of wean, patient comfort, security of the airway, quantity of secretions.

Tracheotomy offers marginal reduction of dead space compared to endotracheal tube, however deadspace is significantly reduced compared with oral nasopharynx.

Tracheotomy may provide greater comfort, facilitate suctioning, and permit speech and eating. These are often very important and augment motivation.

Concern about sternal infection: some authors believe that percutaneous tracheotomies are less prone to peristomal and presumably sternal infection. Bedside tracheotomy is also more resource efficient.

Further discussion of this topic is presented in the following chapter.

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Tracheotomy Postcardiac Surgery

Jonathan C. Irish, Paul D. Warrick

DEFINITIONS

Tracheotomy is a surgical procedure performed to establish an airway conduit between the trachea and the external environment. The term *tracheotomy* refers to a procedure whereby an opening into the side-wall of the trachea is performed. It is considered temporary in that a cannula is required to maintain patency of the airway and removal of the cannula will result in closure of the tracheotomy. A *tracheostomy*, on the other hand, refers to a surgical procedure whereby the tracheal lumen is brought out as an end stoma to the skin surface. It is viewed as a permanent measure that does not require cannula placement to maintain patency.

Percutaneous dilational tracheotomy has become an alternative method to the traditional open tracheotomy technique with the apparent advantages of less cost, infection, bleeding, procedural time and resource consumption. Although some authors have found higher complication rates with the percutaneous technique, many studies confirm the safety of the procedure.

Tracheotomy may facilitate long-term intubation and enable ventilatory wean by improving tracheobronchial toilet and decreasing ventilatory dead space.

INDICATIONS

Current indications for tracheotomy include:

- To treat acute or chronic upper airway obstruction.
- To facilitate respiratory support when prolonged mechanical ventilation is necessary.
- To protect the airway from aspiration and provide access to improve tracheobronchial toilet.
- To eliminate ventilatory dead space.

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Table 36.1 summarizes some of the complications associated with prolonged endotracheal intubation. The risk of posterior glottic stenosis increases with duration of intubation: 5 days (0%), 5-10 days (4%), > 10 days (14%) Secondary chronic glottic incompetence leads to: aspiration, stridor, dysphagia, dysphonia.

The timing of tracheotomy in the context of median sternotomy is controversial. A suggested approach is shown in Figure 36.1. One must weigh the risks and benefits of prolonged intubation vs. premature placement of tracheotomy and consequent communication of tracheotomy and sternotomy wounds.

- when the procedures are performed simultaneously, the tracheotomy and sternotomy wounds can be seen to communicate, thus allowing a portal of entry for pathogens into the anterior mediastinum
- delay of tracheotomy until after 14 days of intubation may allow maturation of sternotomy wound, thereby reducing risk of wound infection
- use of cricothyroidotomy to further separate wounds, reduce risk of infection

There are no absolute contraindications for tracheotomy. Patients should be stable enough to tolerate a general anesthetic.

Table 36.1 Complications of prolonged endotracheal intubation

pressure necrosis of mucosa with ulcer formation
posterior glottic stenosis
subglottic stenosis
tracheal stenosis
cricoid abscess

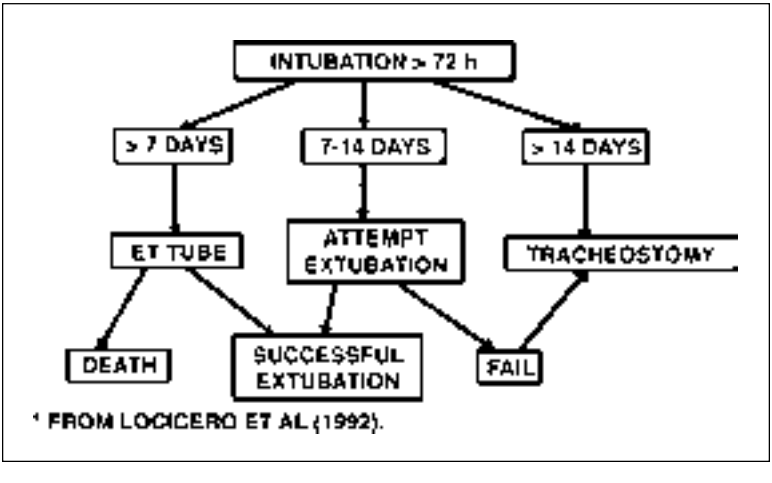

Fig. 36.1. Decision tree for tracheotomy following cardiac surgery.

COMPLICATIONS

Table 36.2 outlines some of the more common complications associated with tracheotomy.

Bleeding

< 72 h after procedure is from a previously undetected vessel that opens after straining, coughing or moving

> 72 h after the procedure

if < 10 mL/48 hr, suspect coagulopathy

if > 10 mL/48 hr, suspect innominate artery erosion with "sentinel" bleeding as greater than 50% of all cases are attributable to this cause

Tracheoinnominate Fistula (TIF)

Catastrophic with 75% mortality rate, usually 1-3 weeks after procedure (range 30 h-7 months)

Telltale sign is a pulsating tracheotomy tube. Immediately control bleeding by inserting a finger under the sternum and compressing the artery against the posterior sternum. Overinflating the tracheostomy cuff may be attempted, but is often unsuccessful. The patient must go to OR for emergency sternotomy and vessel ligation.

Prevent by making tracheotomy incision above third tracheal ring, use of adequate size trach tube, treatment of local infection, gentle suctioning and humidification of incoming air.

Table 36.2. Complications of tracheotomy

Intraoperative
- Hemorrhage (rare)
- Pneumothorax/Pneumomediastinum (0-4%)
- Tracheoesophageal fistula (TEF) (very rare)
- Recurrent laryngeal nerve injury (very rare)
- Cardiorespiratory arrest

Early Postoperative
- Hemorrhage (about 10% of all cases result in death)
- Tracheoinnominate fistula (TIF) (0.4-4.5%)
- Aspiration and dysphagia (up to 87%)
- Wound infection (rare)
- Tube obstruction (2.5%)
- Tube displacement (0-7%)

Late Postoperative
- Tracheal stenosis
- Tracheal malacia
- Tracheoesophageal fistula
- Tracheoinnominate fistula
- Tracheocutaneous fistula

Wound Infection

May be more common in the cardiac population, as approach in the presence of tracheostomy recommends either a limited median sternotomy technique or bilateral thoracotomy approach. A large series refutes the contention that an increased incidence of sternotomy dehiscence is observed among tracheostomy patients.

Tracheotomy undertaken after sternotomy, as opposed to sternotomy in the presence of a mature tracheostomy is unlikely to be associated with the same level of risk of dehiscence. However, very limited literature is available on this topic. As previously mentioned there is a high risk of infection associated with a median sternotomy approach:

- sternal wound infection (9-39x relative risk)
- mediastinitis (10-71% mortality)
- sternal osteomyelitis (exceedingly rare)

Prevent worsening infection through timely removal of flange sutures and avoidance of packing or dressings around trach stoma sites.

Tube Displacement

Most likely to present in first 72 h with signs of obstructed airway, sudden ability to speak, inability to pass the suction catheter. Prevent by suturing the tracheostomy tube in place for the first 72 h postoperative.

After first trach change (usually 5-7 days), keep trach in place by ties so that the trach tube flange is firmly applied against the neck skin.

OUTCOME

Highly variable among prospective analyses and depends on technique chosen.

Depends upon surgeon's experience; rates vary between 0-36% as to the risk of serious morbidity and mortality. Two important prospective studies of the incidence of complications of tracheotomy (Table 36.3) support the notion that the procedure is best performed by experienced surgeons to minimize the risks of surgery. Stauffer determined the risk of various complications of tracheotomy among a diverse group of surgeons while in the study by Stock, the surgeons were confined to otolaryngologists and experienced trauma surgeons

MANAGEMENT OF THE TRACHEOTOMY PATIENT

Accidental Decannulation

The critically ill patient may be best managed by emergency translaryngeal intubation followed by elective tube reinsertion using the following technique:

- have a Kelly's forcep in hand in case of loss of the airway in an immature tracheotomy

Table 36.3. Incidence of surgical complications of tracheotomy

Complication	Stauffer et al. (1981, N=51)	Stock et al. (1986, N=81)
displaced tubes	1*	0
moderate bleeding	36	2.4
obstruction	4	0
subcutaneous emphysema	9	0
pneumothorax	4	2.4
aspiration	8	0
perioperative morbidity	20	6

N.B. values reported in percent
* insertion into the pretracheal fascia resulted in cardiac arrest and death

- extend patient's neck
- cut all existing sutures or trach ties
- usually anterior tracheal opening can be visualized under optimal lighting conditions and retraction such that tracheotomy tube reinsertion can occur directly

Characteristics of Tracheostomy Tubes

Sizes vary from inner cannulas of 4, 6, 8, and 10 mm diameter. Choose a tube large enough:

- to prevent occlusion of the opening against the tracheal wall
- that the cuff does not need to be overinflated to occlude the airway, and thus increase the risk for tracheal stenosis

Cuffs are usually high volume, low pressure and air filled. There are special foam cuffs available. A non-cuffed tube can be substituted:

- whenever the risk of aspiration is deemed to be insignificant, usually 72 or more hours postoperatively, and much later in the critically ill patient
- when the patient no longer requires mechanical ventilation

Fenestrated tracheotomy tubes allow for speech and can be substituted:

- during spontaneous ventilation when the inner cannula is removed, the patient can place their finger (or the cork) over the stoma to produce speech
- double cannula tubes allow for mechanical ventilation with the inner cannula in place and spontaneous ventilation when the inner cannula is removed. Double cannulas typically include an inner cannula for easy removal and cleaning.
- note that fenestrated tubes have a greater curvature, which may not conform to the anatomy of some patients

Decannulation

Consideration of the timing for decannulation should begin immediately after discontinuation of mechanical ventilation and the criteria in Table 36.4 are met.

Table 36.4 Criteria for decannulation

- adequate ventilatory reserve
- adequate nutritional state
- patent upper airway
- minimal and well-tolerated aspiration
- absence of serious bronchopulmonary infection
- absence of impending need for mechanical ventilation
- cough adequate to clear secretions without suctioning

Adapted from Godwin and Heffner (1991).

Some patients tolerate decannulation 1-2 days after commencing fully spontaneous ventilation, but with underlying cardiorespiratory disease, a stepwise approach is more advisable.

Laryngoscopy may be necessary to confirm the clearance or resolution of an upper airway obstruction prior to decannulation.

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Postcardiac Surgery Pacing Therapy

Douglas D. Cameron

Temporary pacing has emerged as an important tool in the early management of arrhythmias following cardiac surgery. Applications vary from standard bradycardia indications, to hemodynamic support and tachyarrhythmia diagnosis and management. Consequently temporary ventricular epicardial pacing leads are routinely applied post cardiac surgery. Additional atrial leads are often placed in patients with excitable atria to allow optimal temporary pacing support. They also allow intracardiac atrial electrogram (IEGM) recordings to facilitate diagnosis of wide complex tachycardias as well as pace termination capabilities for various supraventricular tachyarrhythmias, chiefly atrial flutter. Knowledge of the indications, techniques, various applications and complications of temporary pacing is essential in the postoperative cardiac care setting.

TEMPORARY ELECTRODES

Pairs of teflon insulated stainless steel unipolar myocardial electrodes spaced 0.5-1.0 cm apart are used for temporary cardiac pacing. Needle conductors are attached to each end. The curved needle is used for intramyocardial placement of the bare wire, the needle is then removed. The straight needle allows passage externally typically to the epigastrium where the needles are blunted, capped with an insulating cover and bandaged for future use. By convention, atrial leads are exposed to the right of the sternum, ventricular leads to the left of the sternum.

All temporary pacing requires electrode pairs. Bipolar pacing uses paired myocardial electrodes; unipolar stimulation is achieved by one intramyocardial electrode (cathode) with a second myocardial electrode passed through the skin to function as the subcutaneous anode.

Electrode Care

- Daily dressing change with antiseptic cleaning of the electrode exit sites
- Insulating caps applied to electrode needle connectors when not in use
- Daily threshold/output and sensitivity checks while in use

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- Daily dependency checks
- Continuous arrhythmia monitoring while in use

Unused pacing electrodes are removed on day five by steady direct traction before achieving full systemic anticoagulation. Patient monitoring for cardiac tamponade and mechanically triggered arrhythmias is required during removal. The sinus site must be covered with occlusive gauze to avoid air tracking with potential pneumomediastinum and pneumothorax.

LEAD USES

Bradycardia Support

- Relative bradycardia: Immediate postoperative inappropriate sinus bradycardia (<80 bpm) associated with low output and/or atrial ventricular ectopy. Prefer atrial pacing or AV sequential pacing
- Traditional bradycardia: AV block, tachy/brady, sinus dysfunction, antiarrhythmic drug provoked bradycardia

Overdrive Pacing

- Hemodynamic: atrial or AV overdrive of accelerated junctional or idioventricular rhythm (restore AV synchrony), rate augmentation for relative sinus bradycardia
- Arrhythmia: Suppression atrial or ventricular ectopy, suppression of torsade des pointes VT
- Rate control: Acceleration atrial flutter to atrial fibrillation with improved rate control (concealed conduction), atrial rate increase to achieve 2:1 block response for rate slowing (inappropriate sinus/atrial tachycardia)

Arrhythmia Diagnosis

- Atrial IEGM: intra cardiac atrial electrogram recording to evaluate AV relationship during undefined tachycardias.

Prognostic Testing

- Bbradycardia: AV conduction and sinus node overdrive suppression
- Tachycardia: EP programmed ventricular stimulation study

PACER MANAGEMENT

Postoperative conduction abnormalities result either from pre-existing conduction disease, or acquired from direct surgical trauma, mechanical injury (annular calcification), ischemic injury, or are drug or electrolyte provoked.

Initiation of temporary pacing requires attachment of an electrode pair to the external pulse generator. Bipolar pacing uses paired myocardial electrodes. Where

only a single myocardial electrode is attached, unipolar stimulation is achieved by using one intramyocardial electrode (cathode) with a second myocardial electrode passed through the skin to function as the subcutaneous anode. Reversing polarity (anodal stimulation) is not recommended (higher risk ventricular arrhythmias), however may allow improved stimulation thresholds where high cathodal stimulation thresholds are encountered.

Temporary pacing generators use a 9 V battery source typically good for 14-16 days of pacing. Newer DDD temporary generators have a low battery indicator display that alerts at 1 day residual power. Gradually reducing the pacing rate to 30 bpm often allows emergence of an escape rhythm that will support the patient during the period of battery change. A new loaded pulse generator is switched rather than replacing the batteries in the active generator.

Pace termination is most frequently used for atrial flutter (Table 37.1).

EXTERNAL PULSE GENERATORS

Single Chamber: constant current outputs programmable up to 20 mA
variable rates 30-180 bpm.
variable sensitivity 0.5 mV—asynchronous.
High Output: variable output up to 50 mA. VOO mode.
VAT available using an external trigger from a second generator.
Dual Chamber: AV sequential: DVI mode
AV universal: DDD mode
High rate atrial burst stimulation up to 800 bpm available.

Typical temporary pacing codes are by three position code as summarized in Table 37.2.

Table 37.1. Atrial overdrive pacing

Arrhythmia	Method
Atrial flutter type I: Classical Rate: 230–340 bpm Morphology: inverted sawtooth inferior leads	Confirm atrial lead by IEGM recording: bipolar leads attached to R+L arm leads ECG lead I recording. Pace at maximum output low rates asynchronously to exclude cross chamber stimulation ventricles. Burst stimulate atrium starting 110% flutter rate. Increment rate until capture achieved. Pace minimum 30 seconds, with abrupt or gradual deceleration. Typical rates: 130%–140% flutter rate.
Atrial flutter type II: Atypical Rate: 340–450 bpm	Not amenable to pace termination. Pace acceleration to atrial fibrillation possible with improved rate control by increased concealed conduction

Table 37.2. Typical temporary pacing modes: 3 position code

	Chamber Paced	Chamber Sensed	Response Mode
AAI	**A**	**A**	**I**
AOO	**A**	**O**	**O**
VVI	**V**	**V**	**I**
DVI	**D**	**V**	**I**
DDD	**D**	**D**	**D**

Abbreviations: **A**: atrium, **V**: ventricle, **D**: dual, **I**: inhibited, **O**: off

AAI: Atrial bradycardia support, most physiologic pacing mode
Indications: Sinus dysfunction, excitable atrium, stable AV conduction.
Contraindications: atrial fib/flutter, frequent ventricular ectopy, AV block.

AOO: Asynchronous atrial overdrive pacing used to avoid oversensing
Contraindications: as per AAI mode

VVI: Ventricular bradycardia support, no AV synchrony
Indications: AV block, inexcitable atrium (atrial fibrillation) or lack of atrial pacing support.
Emergency and prophylactic (backup)

DVI: AV bradycardia support, no atrial sensing, loss AV synchrony during sinus acceleration with AV block
Indications: atrial bradycardia with unstable AV conduction.

DDD: AV bradycardia support with AV synchrony throughout programmed rate range. Limited atrial tachyarrhythmia algorithms, no mode switching available.
Indications: Variable sinus rates with unstable AV conduction.
Contraindications: Atrial fibrillation

The trouble shooting guide for temporary pacing is listed in Table 37.3.

PERMANENT CARDIAC PACING

Permanent cardiac pacing was required in 255 of 10,241 (2.4%) patients undergoing cardiac surgery at TTH between January 1, 1990 and December 1995. Indications by procedure are listed in Table 37.4. Risk factors predisposing to permanent pacemaker insertion are outlined in Table 37.5.

Permanent epicardial pacing electrodes should be prophylactically placed intraoperatively in patients with tricuspid valve surgery, vascular obstruction and intracardiac shunts.

Telectronic's accufix atrial leads should be routinely removed intraoperatively if present. These transvenous active fixation leads are under advisory for potential "J" wire fracture with risk of intrathoracic laceration. Use active fixation atrial leads.

Indications for a permanent pacemaker are as per ACC/AHA guidelines. The author's personal practice is suggested as follows:

- **Valve:** dense AV block > 3 days
  Persisting AV conduction impairment > 5 days

Table 37.3 Temporary pacing trouble shooting guide

Pattern	Checks	Etiology
I. Loss of Capture:		
Pacer spike absent:	**Visible LED output signal**	**Open circuit**
	check cable connectors	faulty contact
	trace/inspect leads	R/O fracture
	unipolarize	single lead fracture
	no LED output signal	**power failure**
	confirm power on	
	change battery	battery depletion
	change pulse generator	generator failure
Pacer spike present:	increase output	threshold rise
	check cable connections	poor/wrong contact
	reverse polarity	threshold rise
	unipolarize electrodes	exit block dislodged
	change battery	battery depletion
	high output unit	exit block
	correct	drug / metabolic
	correct	ischemia
II. Under sensing:	increase sensitivity	asynchronous
	increase sensitivity	rising threshold
	increase sensitivity/overdrive	VPB
	overdrive	loss sensing
	unipolarize	exit block/fracture
	appropriate adjust RP	refractory period
	reduce atrial output/change rate	vent blanking(DVI/DDD)
	secure connections	poor connections
III. Over sensing:	reduce sensitivity	farfield
	unipolarize	lead fracture
	Secure connectors	poor contact
	adjust permanent pacemaker	crosstalk
	Check grounding/patient isolation	EM interference

Table 37.4 Incidence of permanent pacemaker insertion by procedure

Type Cardiac Surgery	Frequency permanent pacing
Valve surgery	3 – 6 %
CABG	0.4 – 1.1%
Congenital surgery	1.0 - 2.2%
Cardiac transplant	4 – 10 %

Pacemaker dependent with developing exit block -inadequate safety margin
Sick sinus indications: day 5-7 depending on severity.
Tachy-brady indications: day 5-7 depending on severity.

- **CABG**: delay decision until day 5 if stable temporary pacing.
  If improving conduction consider VVI backup device.

Table 37.5 Risk factors for permanent pacemaker insertion

Valvular surgery (Tricuspid > Aortic > Mitral)
RVOT and ventricular septal surgery
Preoperative conduction disorders
Postop myocardial infarction
Redo surgery
Age > 75 years
Cold blood cardioplegia

- **Torsade VT:** Overdrive pacing if antiarrhythmic drugs required and clear metabolic trigger excluded.
- **Transplant:** Delay decision until day 7-10. Majority amenable to AAI, R pacing.
- **Congenital:** Mode switching DDD, R devices should be considered due to high incidence of atrial arrhythmias.

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Stroke

Charles M. Peniston

Apart from death, stroke is the most feared complication after cardiac surgery. Fortunately, the incidence of postoperative cerebral vascular accident (CVA) is low, approximately 1-2% overall. However, there are certain circumstances which considerably increase the likelihood of a postoperative CVA. All possible measures should be undertaken to prevent a perioperative stroke from occurring. This includes taking a careful history to determine if the patient has had previous strokes or transient ischemic attacks (TIAs), and if so, determining the possible source of any emboli (heart or great vessels). A stroke can greatly increase the length of recovery of the patient and can also place severe stress on the patient's family as well as the staff looking after the patient.

PERIOPERATIVE CONSIDERATIONS

If the risk of a perioperative stroke is high (Table 38.1) then it is helpful to discuss not only the risks of the surgery with the patient, but also to consider all possible outcomes after the surgery, especially the possibility of a severe stroke. It is particularly valuable to have established a good rapport with the patient and his family. Advanced directives and living wills can also be discussed and planned.

Strategies to avoid postoperative stroke are listed in Table 38.2.

DIAGNOSIS

Once a stroke has occurred, there is very little that can be done to reverse the process. It is usually obvious once a patient awakens that a stroke has occurred. Very rarely, a CVA may occur 2-5 days after the cardiac procedure.

Signs

- Slowness to awaken
- Unilateral or bilateral weakness or paralysis
- Persistent confusion

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Table 38.1 Risk factors for perioperative stroke

Previous stroke or TIA
Active TIAs
Diabetes mellitus
Age >75
Peripheral vascular disease
Left ventricular thrombus
Atheromatous disease of the ascending aorta and arch
Femoral artery cannulation

Table 38.2 Strategies to avoid postoperative CVA

Combined carotid endarterectomy and cardiac procedure
Alternate site of cannulation
Exploration of the left ventricle if thrombus present
"No touch technique" of cardiac surgery to avoid embolization
Replacement of the ascending aorta
Maintain adequate cerebral perfusion pressure

Physical Examination

- Orientation
- Cranial nerves
- Eye movements
- Motor power
- Sensory exam
- Deep tendon reflexes
- Babinski sign

A CT scan or MRI should be done within 1-2 days after the event to look for the following possibilities:

- Intracranial hemorrhage
- Size of the infarct
- Previous infarcts
- Single or multiple (shower) infarcts
- Assist in determining the prognosis
- Rule out subdural hematoma
- Help determine the cause of the stroke

MANAGEMENT

While there is little that can be done to treat a stroke once it has occurred, there are several important supportive measures that need to be considered while the patient is recovering.

Ventilation

If the patient has an inadequate ventilatory drive, then he will require prolonged ventilation until his respiratory drive center recovers. This will also apply if the patient is unable to protect his airway and clear secretions. If after 2 weeks of intubation and mechanical ventilation, the patient has still not progressed to weaning, then a tracheostomy should be performed in order to avoid the long term complications of intubation. These patients seem to be more prone to developing bouts of sepsis from infected lines, pneumonia or urinary tract infections.

Nutrition

Ideally, this should be orally if possible, or by a NG tube. If long term, then should use a soft silastic tube to avoid irritation of the nose. If there is inadequate absorption of feeds, or contraindication to enteral feedings, then TPN will be required. Consult a dietitian for nutritional requirements. Stop enteral feeds when planning to extubate.

Deep Vein Thrombosis (DVT) Prophylaxis

Heparin 5000 units s.c. q12h

Physiotherapy

Assistance is very important to help with clearing of secretions and to maintain range of motion of limbs and ambulation where possible.

Support

The patient and his family will require support as they deal with this potentially terrible complication. It is particularly in this situation that nursing staff and other allied health personnel (physiotherapists, occupational therapists and social workers) are very helpful.

Rehabilitation

Once the patient has been weaned from the ventilator and shows signs of recovery, then planning can begin for transfer to the ward and then to a stroke rehabilitation unit. Early consultation with the rehabilitation staff can be very helpful in facilitating recovery and timely transfer.

POSTOPERATIVE COGNITIVE DEFECTS

If specifically looked for, they are found in up to 50% of patients undergoing cardiac surgery. Usually the findings are subtle and transient, disappearing in 3-6 months. Delirium may appear in 7-50% of patients. It usually lasts about 1 week and resolves spontaneously. Usually only supportive measures are necessary.

Clinical Findings:

- Delayed awakening
- Confusion
- Disorientation
- Anxiety
- Hallucination
- Personality and behavioral changes
- Delusions
- Depression

Risk Factors

- Age > 60
- Alcohol or narcotic addiction
- Previous cerebral damage or disease
- Visual or auditory impairment
- Preoperative depression

Exacerbating Factors

- Communication deficit
- Sensory deprivation or overload
- Sleep deprivation
- Anxiety
- Pain

Possible Precipitating Factors Causing Delirium

- Drugs-virtually any class
- Drug withdrawal
- Metabolic disturbances: fluid, electrolytes, glucose, pH, renal, liver, endocrine, hypoxia, sepsis
- Acute cerebral disorders: CVA, edema, epilepsy
- Nutritional or vitamin deficiency

Lab Investigations

- CBC
- BUN, Cr, electrolytes, Ca^{2+}, phosphate, glucose
- AST, ALT, ALP
- Urinalysis
- Folate, vitamin B_{12}
- Thyroxine, cortisol, ammonia, protein
- Serum osmolality
- Blood cultures
- CT scan
- EEG

Management

- Rule out reversible causes
- Hepatic cause: consider short acting benzodiazepines
- Alcohol withdrawal consider benzodiazepines. Give thiamine 100 mg i.v. or i.m. daily for three days
- Haloperidol 2-15 mg po BID. If urgent, may be given as 1-5 mg i.v. q1hr until desired affects are achieved. Beware of extrapyramidal side effects which may also require treatment with benztropine.
- Avoid excessive analgesia or sedation
- Close observation

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Routine Surgical Ward Care

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Tirone E. David, Lynda L. Mickleborough, Susan Kerwin-Lenkei

GENERAL CARE

Patients who are hemodynamically stable and do not need assisted ventilation are transferred out of the CVICU to surgical ward (median 18 h). CVICU discharge orders (Appendix 3) and clinical form (Appendix 5) are reviewed. They are connected to ECG telemetry during the first two or three postoperative days (POD). Vital signs are recorded every 4 h, and body weight every morning. Ambulating with assistance is commenced on the 1st POD.

CHEST DRAINS

Chest drains are usually removed on the 1st POD. A chest x-ray is obtained after removal of the drains and checked for pneumothorax, atelectasis and other abnormalities. Mediastinal drainage in excess of 100 ml/8 h is abnormal and requires further observation and/or intervention. Air leak through mediastinal drains is abnormal and if it persists beyond 2 h, it is preferable to insert a pleural drain to avoid potential contamination of the sternotomy.

FOLEY CATHETER

Foley catheter is usually removed on the 1st POD unless urinary output is inadequate or the patient is on renal dose of dopamine to increase urinary output.

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EXTERNAL PACER WIRES

They are usually removed on the 4th POD unless there is cardiac dysrhythmia that may necessitate pacing. Pacer wires should be removed completely and should not be cut flush with the skin and left in because it can cause late infection and other complications.

WOUND CARE

Surgical wounds should be kept covered with sterile dressings during the first two days and then open to air and inspected daily thereafter. Patients with sternal click on palpation have a higher risk of dehiscence and/or infection. Wound infection usually occurs from the 5th to the 10th POD, and unfortunately most patients are already home when this happens. Patients should not be discharged if the sternum is unstable, have serous drainage, or if deep sternal infection is suspected. In our unit, deep sternal infection is uncommon (< 1%).

Leg wound problems such as numbness, fluid collection, edema and infection are common. Numbness is due to damage to the femoral nerve if the groin vessels are inappropriately dissected or the saphenous nerve when this vein is harvested. It is generally self-limited and resolves slowly over 6-12 months. Collection of fluid or a large hematoma should be drained. Edema is managed with leg elevation and diuretics.

Leg wound infection is treated with drainage, local antiseptic, and systemic antibiotics if the risk of sepsis and/or endocarditis is an issue. Large necrotic areas in the distal leg may be treated with debridement and skin graft after local sepsis is controlled.

NAUSEA AND VOMITING

Nausea and vomiting are common after open-heart surgery and is treated with anti-emetic drugs. If persistent, the cause must be searched and treated accordingly.

CONSTIPATION

Constipation is common particularly in elderly patients. Patients should be given a laxative daily and enemas if needed.

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PAIN CONTROL

We use morphine sulphate 1-4 mg IV q 1-2 h on the 1st and 2nd POD. We then change to Tylenol #3 1-2 tablets q 3-6 h prn or Percocet 1-2 tablets q 3-6 h prn if codeine is not tolerated or causes gastrointestinal problem.

Indomethacin suppositories 50-100 mg BID prn are effective for severe incisional chest pain. NSAIDs should not be used in patients with elevated creatinine, diabetics or peptic ulcer disease. It can also be given by mouth along with Cytotec or Zantac.

CHEST PHYSIOTHERAPY

Atelectasis is very common after open-heart procedures. Patients must be instructed preoperatively about respiratory exercises and reminded daily to cough and to take deep breaths. Inspection, palpation and auscultation of the lungs must be done daily during rounds and twice daily in those with COPD or other pulmonary illness.

ANTICOAGULATION

Patients who undergo surgery using cardiopulmonary bypass are fully anticoagulated with heparin and thrombosis of calf veins during the operation is uncommon. However, these patients develop a hypercoagulable state postoperatively and should be treated with mini-dose of heparin (5,000 units BID sc). This regimen can be discontinued on the 3rd POD if the patient is ambulating.

Patients in atrial fibrillation or those who had heart valve surgery may need intravenous heparin or oral anticoagulation with warfarin sodium. We start intravenous heparin on the 1st POD after removal of the chest drains. Anticoagulation with heparin requires monitoring of partial thromboplastin time (PPT). It should be maintained at around 65-95 seconds (with control of 30 seconds). Platelet count should also be monitored in patients on heparin because of the risk of heparin induced thrombocytopenia (HIT). If platelet count is below 100,000, heparin should be withheld (see chapter 13).

Anticoagulation with warfarin sodium requires monitoring of PT (prothrombin time) or INR (international normalized ratio). We give warfarin sodium 5-10 mg on the 1st POD. The INR should be maintained at 2-3 when anticoagulation is given because of atrial fibrillation, in patients with biological mitral valves or mechanical aortic valve. It should be increased to 3.0-3.5 in patients with mechanical mitral valves. It takes approximately 4-5 days to raise the INR to therapeutic level. Drugs that interact with warfarin sodium should be prescribed with care. If the INR rises to rapidly, liver function tests should be obtained (see appendix 9).

Table 39.1 Criteria for hospital discharge

Afebrile for 24 h
Weight below preoperative level
No drainage from wounds
Absence or control of heart failure
Optimal blood pressure and rhythm control
Therapeutic anticoagulation if indicated
Lab values in normal range, 2D echo done for valve patients
Oxygen saturation satisfactory on room air
Patient ambulating independently

LABORATORY TESTS

Our standard postoperative orders include daily CBC, electrolytes, INR (if anticoagulated) during the first four postoperative days. ECGs are done daily during the first 3 days and one on the day before discharge. Chest x-rays are done after removal of the chest tubes and on the 3rd POD. Any other test is ordered as needed.

DISCHARGE PLANNING

Discharge planning should start preoperatively. Most patients can safely go home on the 5th or 6th POD but daily monitoring by telephone conversation with a nurse clinician or personal visit is needed for early diagnosis of postoperative complications.

The patients and their families should be instructed about diet, physical activities, medications and the recovery. The "dos" and "don'ts" should also be given in writing (see Appendix 13).

Patients who need long-term anticoagulation with warfarin sodium must be carefully instructed on how this medication works, its side effects, the interactions with other drugs and the importance of close monitoring of INR.

A schedule of appointments with the family physician, cardiologist and surgeon should be given at the time of discharge.

Common Ward Complications

Tirone E. David, Lynda L. Mickleborough and Susan Kerwin-Lenkei

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POSTOPERATIVE DYSRHYTHMIAS

Cardiac dysrhythmias are the most common complications after open-heart surgery. Premature atrial and ventricular beats, tachyarrhythmias and conduction disturbances occur in 30-50% of patients after coronary artery bypass and in higher proportion of patients after valve surgery and combined procedures.

Sinus Tachycardia

Sinus tachycardia reflects increased adrenergic tone. It may adversely effect ventricular function. Treatment should be directed at the cause which can be pain, anxiety, hypovolemia, adrenergic rebound (withdrawal of beta-blocker), administration of chronotropic drugs, sepsis and anemia.

Premature Atrial Contraction

Premature atrial contractions (PAC) are benign but may herald the onset of atrial flutter or fibrillation. Therapy consists in optimizing the patient's biochemical milieu (K^+, Mg^{++}, pH, temperature). Atrial pacing, beta-blockers, or calcium antagonists may also be effective.

Atrial Flutter and Fibrillation

Atrial fibrillation or flutter affects approximately 30% of patients following isolated coronary revascularisation and 50% of patients after valve surgery. The peak occurrence is 24-72 h postoperative. Table 40.1 summarizes some clinical predictive factors for atrial fibrillation.

Atrial flutter (atrial rate < 380 beats per minute) or fibrillation (atrial rate > 380 bpm) is the most common arrhythmia following coronary artery bypass surgery. It seldom causes hemodynamic problem in patients with normal ventricular function, but it can be dangerous for those with impaired left ventricular

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Table 40.1. Clinical predictors of atrial fibrillation

Clinical Parameter	Odds Ratio/Relative Risk
Preop	
Age	1.24 (5 yr intervals)
Male	1.41
Hx AF	2.28
Hx CCF	1.31
Pre CPB HR > 100	1.59
Withdrawal of β blockers	2-5
Interatrial conduction delay	
Inducible AF	
Dispersion of refractoriness of atrial tissue	
Intraop	
Cross clamp time	1.06 (per 15 min)
Increased number of grafts	
CABG + CEA	
CABG + Valve replacement	
Pulmonary venous venting	1.44
Bicaval cannulation	1.40
Postop	
Atrial pacing	1.27
Failure to reinstate β blockers	
Not Predictive	
Hypertension, LV function, postop atrial pressures	

function. Treatment is targeted at reducing ventricular rate and restoring sinus rhythm.

Hemodynamically stable patients are treated with digoxin. The loading dose of digoxin is dependent upon body mass and age, but independent of renal function. A total loading dose of 1.0-1.5 mg is given over a 12-18 h period. It is started with 0.5 mg i.v. or PO and followed with 0.25 mg q 4 h to the total loading dose. Maintenance dose is of 0.25 mg daily or reduced if the patient has renal function impairment or is over 80 years old. All these patients should be placed on telemeter if they are not already monitored.

In the absence of severe COPD or poor left ventricular function, a β blocker such as sotolol should be added. It can be started at 40 mg BID and increased to 80 mg BID if needed.

Patients with a ventricular rate >150 bpm should receive digoxin as above and intravenous esmolol. The patient should also be started on oral sotolol 40-80 mg BID.

If ventricular function is compromised, intravenous amiodarone should be given. Electrical cardioversion should be attempted in patients who are hemodynamically unstable.

Current atrial fibrillation management at Toronto General Hospital (TGH) is according to protocol as outlined in Appendix 10.

Paroxysmal Supraventricular Tachycardia

Paroxysmal supraventricular tachycardia is infrequent after open-heart operations. It can be paroxysmal atrial tachycardia or atrioventricular nodal reentrant tachycardia at rates of 150-250 bpm. A simple vagal maneuver or adenosine 6-12 mg i.v. bolus can interrupt it. Rapid atrial pacing as well as cardioversion are also effective.

Premature Ventricular Complexes (PVC)

Unifocal premature ventricular contractions (PVCs) are usually benign and need no treatment unless their frequency is more than 6 per minute. The patient's biochemical milieu (K^+, Mg^{++}, pH, temperature) should be corrected. Frequent, multi-focal PVCs or triplets seldom progress to ventricular tachycardia but the cause must be determined. In addition to the above causes, perioperative infarction should be contemplated. If ventricular function is abnormal, lidocaine, procainamide or amiodarone should be given. Widespread use of these drugs is not advisable because they can precipitate complex ventricular arrhythmias, particularly tordsade des pointes in a small subset of patients.

It is important to check the serum electrolytes, specifically the serum potassium (K^+) and magnesium (Mg^{2+}).

- If K^+ 3.2-3.5, give 20 meq KCl
- If K^+ 3.6-3.8, give 15 meq KCl
- If K^+ 3.9-4.1, give 10 meq KCl.

Give potassium supplementation as intravenous additive (maximum KCl 10 meq in 500 ml of solution), or orally as K^+ tablets or KCl elixir.

Serum magnesium level may not accurately reflect intracardiac magnesium. Despite serum levels treat most patients for one or two days with Magnesium Rougier 15 cc PO TID.

Ventricular Tachycardia/Ventricular Fibrillation/ Pulseless Electrical Activity

Ventricular tachycardia (VT) and ventricular fibrillation (VF) are uncommon after open-heart surgery and usually indicate myocardial ischemia due to thrombosis of a coronary artery graft or perioperative myocardial infarction.

Treatment consists of cardiopulmonary resuscitation including establishing proper oxygenation, external massage, cardioversion, and appropriate drug therapy. (Appendix 11D) If the patient does not respond, the chest should be opened and direct cardiac massage instituted. If this fails, consideration to place the patient on cardiopulmonary bypass should be given if a correctable problem is identified.

Bradyarrhythmias

Patients may be asymptomatic with stable hemodynamics or symptomatic (presyncope, syncope) with hemodynamic compromise. Atrial pacing is the most effective treatment. Isoproterenol is a powerful chronotropic agent.

If the patient is hemodynamically unstable with a bradycardia, try pacing or administer atropine 0.5 mg intravenously up to a total of 2 mg. An absolute con-

traindication to atropine is glaucoma. Assess the patient for consideration of a permanent pacemaker.

CONGESTIVE HEART FAILURE

Common postoperative problem and usually due to fluid overload. Check daily weight; it should be lower than preoperative by the 4th POD.

Signs and symptoms: weight gain, paroxysmal dyspnea or orthopnea, pulmonary rales, distention of neck veins, S^3 gallop, pulsatile liver, acute pulmonary edema, and a positive hepatojugular reflex.

Echocardiography to rule tamponade or other mechanically correctable cause of heart failure.

Management include:

- Fluid restriction to 1000-1200 ml/day
- Sodium restriction of 1 gm/day
- Diuresis. Furosemide 40-80 mg i.v. or po in am. Consider adding Metolazone 5-10 mg po, 40 minutes before giving Furosemide to enhance the diuretic action. Use i.v. furosemide for patients more than 2 kg above their dry preoperative weight, as the absorption of po furosemide with this amount of fluid overload is minimal.
- Check potassium daily. Give KCl as needed.
- Left ventricular dysfunction: ACE inhibitors after diuresis is completed. Start the patient on captopril 6.25 mg po as a test dose. Check the patient's blood pressure before and q 15 minutes for one hour after the first dose. If the patient tolerates 6.25 mg of captopril for one dose, increase to 12.5 mg TID. Do not increase dosage sooner than 48 h. If tolerated, change to one of the once daily ACE inhibitors, for dosing convenience.

POSTOPERATIVE CHEST PAIN

Obtain a detailed history of the pain. The most important aspect is to differentiate incisional pain from other types of pain such as myocardial ischemia, pericarditis, acute aortic dissection, etc. Most patients who had angina preoperatively can differentiate ischemic from other types of pain.

Obtain a 12 lead ECG and compare with previous ones.

Include in the differential diagnosis: incisional pain, pericarditis, pleuritis, pneumothorax, pulmonary embolism, aortic dissection, esophagitis, and peptic ulcer.

If ischemic pain is suspected, give sublingual nitroglycerin 0.3-0.6 mg and/or morphine 1-5 mg IV. Consider transfer to ICU, consult cardiology for further investigation.

PLEURAL SPACE COMPLICATIONS

Almost all patients will have two or three chest tubes in the mediastinum connected to suction postoperatively. These tubes are generally removed on the first postoperative day in the CVICU, but some patients are transferred to the ward with chest tubes in situ. Remove the chest tubes when the drainage is < 50 cc/4 h and no air leak is present. High clinical suspicion, especially at time of chest tube removal, will minimize morbidity and maximize therapeutic benefit.

Pneumothorax

This complication is most common following the removal of chest tubes, but may also be found while chest tubes are still in situ. See Figure 40.1 for an algorithm for management of this complication.

Signs and symptoms include tachypnea, dyspnea, resonance to percussion and decreased air entry on affected side, subcutaneous emphysema, and a mediastinal shift. Confirm diagnosis with stat CXR; if pneumothorax is greater than 20%, a chest tube should be inserted; if smaller than 20% and patient is comfortable, observation is adequate; if patient is symptomatic, a drain should be inserted.

Tension pneumothorax can cause hypotension, tachycardia, high jugular veins similar to the findings of cardiac tamponade, and possible agitation and/or lethargy. There is no time for CXR. A diagnostic needle tap should be done; if positive, a chest tube should be inserted immediately.

Pleural Effusion

The most common cause of pleural effusion in the postoperative patient is retained blood or serousanguineous fluid particularly when the pleural cavities have been entered. Heart failure is also a common cause of effusion. Chylothorax is rare.

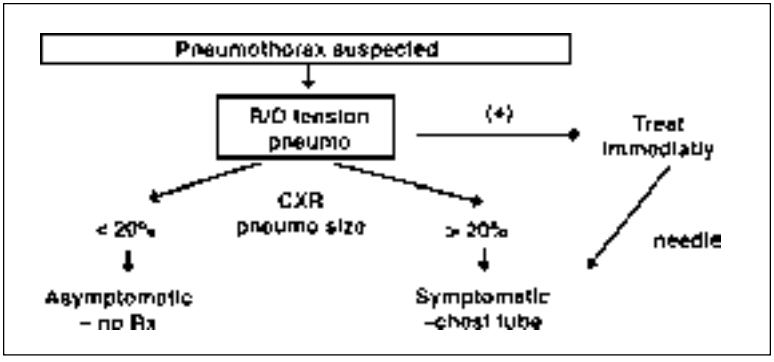


Fig. 40.1. Pneumothorax algorithm.

Of special interest, due to the rapidity of discharge of most patients are the accumulations of serosanguinous fluid secondary to postcardiac injury syndrome. This usually occurs within 7-14 days postoperatively, and chylothorax due to thoracic duct injury, most often with aortic surgery presenting 7-14 days following resumption of normal diet.

Signs and symptoms of fluid in the pleural space include pleuritic chest pain, dyspnea, tachypnea, tachycardia, falling hemoglobin, low grade fever, leukocytosis, absence of breath sound on affected side.

A chest x-ray (PA and lateral) should be obtained. A diagnostic pleural tap with drainage of as much fluid as possible should be done. Figure 40.2 outlines the management options for pleural effusion. Fluid should be sent for culture. If pus is obtained, it should be treated as empyema (chest tube + antibiotics).

Pericarditis

Nonsuppurative pericarditis is common after open-heart procedures. It can be diagnosed by a loud friction rub, low grade fever, pericardial effusion, and increased erythrocyte sedimentation rate. It is usually a self-limited problem but sometimes can be chronic and lead to constriction. Treatment is with anti-inflammatory agents such as a high dose of aspirin (2 or 3 gm/day). Corticosteroids are seldom necessary.

Delayed Pericardial Tamponade

Delayed pericardial tamponade is uncommon after open-heart surgery but it can be fatal if not recognized and treated appropriately. It is more common in patients on warfarin sodium and usually occurs one to two weeks after surgery. Since most valve patients are discharged on the 5th to 7th POD, it is important to obtain a transthoracic echocardiogram to rule out effusion before discharge. If effusion is detected, the patient should be followed closely.

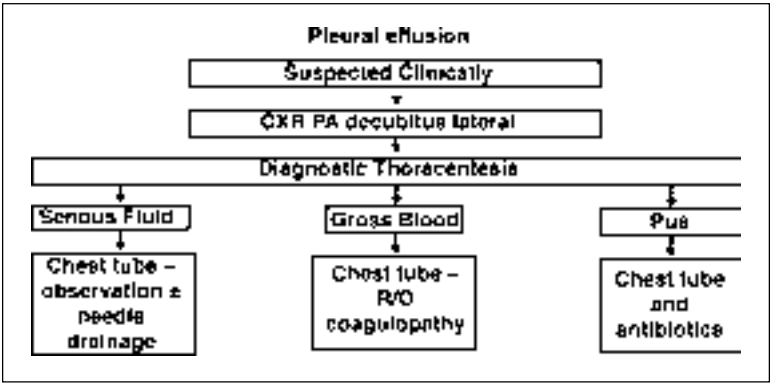


Fig. 40.2. Pleural effusion.

Most cases of delayed tamponade are due to accumulation of serous sanguineous fluid secondary to pericarditis. In this case, simple insertion of a large drain in the subxiphoid area under local or general anesthesia is all that is required. The drain should be left in for 24 to 48 h.

Delayed tamponade secondary to hemorrhage should be treated with reopening of chest, evacuation of blood clots, lavage of the pericardial cavity and hemostasis.

POSTOPERATIVE HYPERTENSION

Consider the following four factors in the management of hypertension: etiology, LV function, renal function, age. In most cases, restart the preoperative antihypertensive medications unless there have been postoperative changes in LV and/or renal function.

Table 40.2 summarizes current treatment of patients with postoperative hypertension at TGH.

All postoperative patients with a Type A or B dissection, take beta-blockers, regardless of age, unless otherwise contraindicated.

Carefully evaluate the use of ACE inhibitor, if BUN and creatinine are not within normal limits.

Table 40.2. Management of postoperative hypertension

For patients < 65 years of age: Left Ventricular function	Renal Function	
	good	**poor**
good	β-blockers Calcium channel blockers ACE inhibitors	β-blockers Calcium channel blockers Nitrates and hydralazine
poor	ACE inhibitors Amlodipine Hydralazine and nitrates	Hydralazine Nitropaste
For patients > 65 years of age: Left Ventricular function	Renal Function	
	good	**poor**
good	Calcium channel blockers ACE inhibitors	Calcium channel blockers hydralazine Nitrates
poor	ACE inhibitors Amlodipine	Hydralazine Nitropaste

OTHER POSTOPERATIVE COMPLICATIONS

Fever of undetermined origin after open-heart procedures is extremely worrisome particularly in patients who had prosthetic devices implanted. These patients should not be discharged from hospital before exhaustive work-up.

Deep venous thrombosis and pulmonary embolism are uncommon after cardiac operations but do occur from time to time. This complication must be included in the differential diagnosis of hypoxia, chest pain, leg edema and fever.

Confusion and disorientation are now uncommon with early extubation and resumption of physical activities while in hospital but still a problem from time to time. One should check the electrolytes, glucose, renal function, and oxygen saturation. All medications should also be reviewed. Stroke should be ruled out.

APPENDIX 1
CARDIAC SURGICAL ADMISSION–HISTORY AND PHYSICAL

Main Complaint:_____

History of Present Illness:_____

_____

_____

Past History:	**(yes)**	**(no)**	**Comments:**
Previous operations	( )	( )	_____
Allergies	( )	( )	_____
Central Nervous System	( )	( )	_____
Respiratory System	( )	( )	_____
Endocrine System	( )	( )	_____
Hematologic System	( )	( )	_____
Renal System	( )	( )	_____
Gastrointestinal System	( )	( )	_____
Peripheral Vascular System	( )	( )	_____
Genitourinary System	( )	( )	_____
Musculo-skeletal System	( )	( )	_____

Coronary Artery Risk Factors:

Smoking	( )yes	( )no	( )quit
Hypertension	( )yes	( )no	( )don't know
Diabetes mellitus	( )yes	( )no	( )don't know
Hyperlipidemia	( )yes	( )no	( )don't know
Family history	( )yes	( )no	( )don't know

Family History:_____

_____

Social History: ( ) Single ( ) Working Profession:_____
( ) Married ( ) Retired Living conditions:_____
( ) Divorced ( ) Unemployed_____

Current Medications:_____

_____

Physical Examination

General appearance: _____

Weight: _____ *Kg* _____ *Lb*
Height: _____ *cm* _____ *ft* ____ *in.*
Heart Rate: _____ *bpm*
Blood pressure: ___ /___ *mmHg*
Temperature: _____ *Celsius*

APPENDIX 1–Cardiac Surgical Admission–History and Physical *(continued)*

HEENT: _____

NECK: _____

CHEST: _____

Lungs: _____

Heart: _____

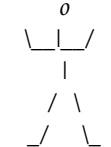
ABDOMEN: _____

Liver: _____

Spleen: _____

EXTREMETIES:

Pulses: (0 - 4)



Arterial Bruits: _____

Saphenous veins: _____

Allen's Test: Right _____

Left _____

Clinical Impression: _____

_____

_____

APPENDIX 2
ROUTINE POSTOPERATIVE CVICU ADMISSION ORDERS

TORONTO GENERAL HOSPITAL

A University of Toronto Teaching Hospital

Doctor's Order Sheet

POST-OPERATIVE STANDING ORDERS FOR CARDIAC SURGERY

ALLERGIES
NO KNOWN ALLERGIES

Surgery _____
LV Grade _____

DATE & TIME ORDERED	PHYSICIAN'S ORDERS	ACTION TAKEN	SIGNATURE AND POSITION
	Post-op order for CVICU I and II entered on computers		
	1. Vital signs q 15 min x 1 h, then q 1 h when stable. Postextubation: vital signs q 2h when hemodynamically stable.		
	2. Urine output q 1 h Post extubation: urine output q 2 h when hemodynamically stable.		
	3. Chest tubes @ -20 cm H20 suction. Record chest loss q 15 min x 1 h then q 1 h if patient hemodyna-mically stable.		
	4. Auto-transfuse chest drainage as per CVICU policy No Yes		
	5. Nasograstric tube to gravity drainage (if present)		
	6. Cardiac outputs and hemodynamic calculations now and q 6 h & prn (if PA catheter present)		
	7. IABP frequency _____		
	8. Check peripheral pulses q 1 h x 4 then q 4 h		
	9. Central line IV D5W @ KVO		
	10. K^+ replacement as per guidelines. First hour post-op give KCl __ mmol/L		
	11. Peripheral IV N/S 0.9% NaCl @ KVO		
	12. 12 lead ECG now then daily x 3 dats then reassess		
	13. Ventilation VT ____ Rate _____ min FiO_2 ___ PeeP _____ cm/H_2O		
	14. Titrate FiO_2 to Keep $PO_2 \geq$ _____ OR O_2 Saturation ≥ ___		
	15. Suction ETT prn and chest care as per assessment		
	16. Physiotherapy: assessment & treatment		
	17. Pacemaker connected and checked by M.D.		

APPENDIX 2
ROUTINE POSTOPERATIVE CVICU ADMISSION ORDERS *(continued)*

ALLERGIES Surgery _____
NO KNOWN ALLERGIES LV Grade _____

DATE & TIME ORDERED	PHYSICIAN'S ORDERS	ACTION TAKEN	SIGNATURE AND POSiTION
	Medications		
	a) Morphine sulphate _____ mg IV q 1 h prn No Yes		
	b) Indomethacin supp ___ mg pr q _ h x __ doses		
	c) Acetaminophen supp __ mg pr q 4 h prn if T > 38.5°C		
	d) Gravol ____ mg IV/IM q 4h PRN x 48 hrs		
	e) Antibiotics: Cefazolin 1 g IV q 8 h in 50 cc D%W x 6 doses No Yes OR Vancomycin __ mg IV q 12 h in __ 50 cc D5W over 12 hrs x _ doses No Yes Last dose antibiotic in OR at __ hr		
	f) Propofol _ mg IV q 12 h in _ cc D5W at _ cc/h No Yes D/C at __ h		
	g) Sodium Nitroprusside _ mg IV in _ 250 cc D5W to keep SBP < __ mmHg No Yes		
	h) Nitroglycerin __ mg IV in _ cc D5W at 5-50 cc/h No Yes		
	i) Dopamine _ mg IV in _ cc D5W at __ mcg/kg/h if necessary to keep SBP > __ mmHg		
	j) Nifedipine __ mg intranasally / po q 4 h prn for SBP > 130 mmHg		
	k) Protamine _____ mg IV No Yes		
	l) Albumin 5% ___ cc IV now No Yes OR Pentaspan ___ cc IV now No Yes Reversed in O.R. No Yes		
	1. Extubation		
	2. Extubation No Yes as per CVICU policy		
	3. Respirations q 15 min x 1 hr then q 1 h when stable		
	4. FiO2 _____ via facemask. Nasal prongs when eating		
	5. Oxygen saturation monitor until AM		
	6. Incentive spirometry q 1 h		

APPENDIX 2
ROUTINE POSTOPERATIVE CVICU ADMISSION ORDERS *(continued)*

ALLERGIES Surgery _____
NO KNOWN ALLERGIES LV Grade _____

DATE & TIME ORDERED	PHYSICIAN'S ORDERS	ACTION TAKEN	SIGNATURE AND POSITION
	7. D/C N/G tube No Yes D/C chest tubes No Yes (to decide in AM rounds) (if yes, CXR) 8. post removal 9. D/C Pulmonary artery catheter No Yes 10. Total IV / po _ ml/24 hr Clear fluids No Yes Advance to LAF/NAS if tolerated No Yes		
	Medications a) PCA treatment as per protocol No Yes OR Morphine sulphate _____ mg IV q 1 h prn No Yes b) Tylenol #3 __ tabs PO q 3hrs PRN No Yes c) ECASA __ mg PO OD No Yes OR ASA supp ____ mg P/R OD No Yes (start day after surgery for post-op ACB) d) HS sedation ____ mg PO qhs PRN e) Heparin ____ u S/C q 12hrs when chest tubes D/C No Yes		
	Physician's Signature Date		

APPENDIX 3
ROUTINE CVICU DISCHARGE ORDERS

TORONTO GENERAL HOSPITAL

A University of Toronto Teaching Hospital

Doctor's Order Sheet

POST-OPERATIVE STANDING ORDERS FOR CARDIAC SURGERY

ALLERGIES Surgery _____
NO KNOWN ALLERGIES LV Grade _____

Date & Time Ordered	PHYSICIAN'S ORDERS	Action Taken	Signature and Position
	1. Cancel all previous orders		
	2. Transfer orders (24 h or 48 h post-op) entered on computers.		
	3. Daily PT, PTT entered on computer No Yes		
	4. Daily Glucose entered on computer No Yes		
	5. Clear fluids, LAF, NAS DAT or Diabetic diet __ kJ		
	6. Vital Signs q 4 hrs, then qid PRN.		
	7. Intake and output as per unit guidelines		
	8. Peripheral IV 2/3-1/3 or D5W plus __ mmol/L KCL at _ ml/hr.		
	9. D/C central line No Yes (if no, D5W __ ml/h)		
	10. Total daily fluid allowance (IV & PO) __ ml/24 h.		
	11. Weight as per unit guidelines.		
	12. AAT No Yes Other _____		
	13. Wound care as per unit guidelines		
	14. ECG tomorrow No Yes and on 4th & 6th postop days		
	15. D/C pacemaker No Yes (if yes, cover pacer wires)		
	16. Telemetry No Yes		
	17. Remove pacer wires prior to discharge.		
	18. Inspirometer q 1 -2 hrs		
	19. Physiotherapy assessment and treatment.		
	20. FiO_2 _____ via face mask / nasal prongs Tandem flow No Yes		
	21. D/C arterial line.		
	22. D/C Foley catheter No Yes (Leave in situ if 24 hrs transfer)		
	23. D/C Chest tubes No Yes (if yes, CXR post-removal)		

APPENDIX 3
ROUTINE CVICU DISCHARGE ORDERS *(continued)*

ALLERGIES Surgery _____
NO KNOWN ALLERGIES LV Grade _____

Date & Time Ordered	PHYSICIAN'S ORDERS	Action Taken	Signature and Position
	Medications a) PCA Treatment as per protocol No Yes OR Morphine sulphate _ mg IV/IM q _ hrs x 24 h No Yes OR Demerol ___ mg IV/IM q __ x 24 h No Yes b) Tylenol #3 _ tabs po q 3 hrs prn x 14 days OR Percocet ___ Tab po q 4 hrs prn x 14 days c) Gravol __ mg IV/IM q ____ h PRN days No Yes d) HS sedation __ mg po q hs prn x 14 days e) Colace __ mg po bid f) MOM ___ ml po qhs PRN g) Lasix ____ mg po daily x ___ days start on h) Slow K 1-2 ___ tabs po q __ hrs x days start on _____ i) Antibiotics _____ j) Anticoagulation 1. Heparin __ u s/c q 12 h. Re-assess if platelets < 100,000 D/C Heparin when fully ambulatory 2. ECASA ___ mg po daily No Yes 3. Coumadin ____ mg po daily order (Target INR _____) No Yes 4. Heparin __ u IV in ____ cc D5W at u/h No Yes		

Physician's Signature Date

APPENDIX 4
POSTOPERATIVE CVICU ADMISSION FORM

CVICU ADMISSION
Age: _____ Sex: ____ M _ F __
Dx: _____
Surgery: _____

LV grade: I ( ) II ( ) III ( ) IV ( )

ADDRESSOGRAPH

Concomitant Diseases:

IDDM Yes ( ) No ( ) NIDDM Yes ( ) No ( )

Smoker Yes ( ) No ( ) Allergies Yes ( ) No ( )

Hypertension Yes ( ) No ( ) PVD Yes ( ) No ( ) TIA/CVA Yes ( ) No ( )

Renal dysfunction Yes ( ) No ( ) Peptic Ulcer Yes ( ) No ( ) Thyroid Yes ( ) No ( )

Other: _____

INTRA-OP COURSE: _____

POST CPB: Paced: A ( ), V ( ), A-V ( ) PaO_2 _____

Hct: _____ K^+: _____

CVICU: Intubated ( )

HR _____ S-G position < 50 cm ( ) IABP position ( )

BP _____ Chest: A/E Bilateral ( ) (Check on chest x-ray)

T° _____ Hemostasis _____

CVP _____ Intraop U/O _____ Cross-clamp time _____

PAP _____ Fluid Balance _____ CPB time _____

CO/CI _____ Inotropes: _____

SVR _____ _____

EKG _____ _____

PLAN: _____
_____
_____
_____
_____
_____
_____
_____

APPENDIX 5
CVICU DISCHARGE FORM

CVICU DISCHARGE

Age: _____ Sex: M( ) F( )

Dx: _____

Surgery: _____

LV grade: I( ) II( ) III( ) IV( )

ADDRESSOGRAPH

CVICU COURSE

Extubated: Day of Surgery ( ) Postop Day # _____

Uncomplicated ( )

Complicated ( )

Comments: _____

_____

Fluid balance/ weight (at transfer): _____

_____

Allergies: No ( ) Yes ( ) _____

_____

Discharge medications: _____

_____

Anticoagulation: _____

Target INR: _____

Pacemaker: _____

_____

Telemetry: No ( ) Yes ( ) _____

_____

Complications: _____

_____

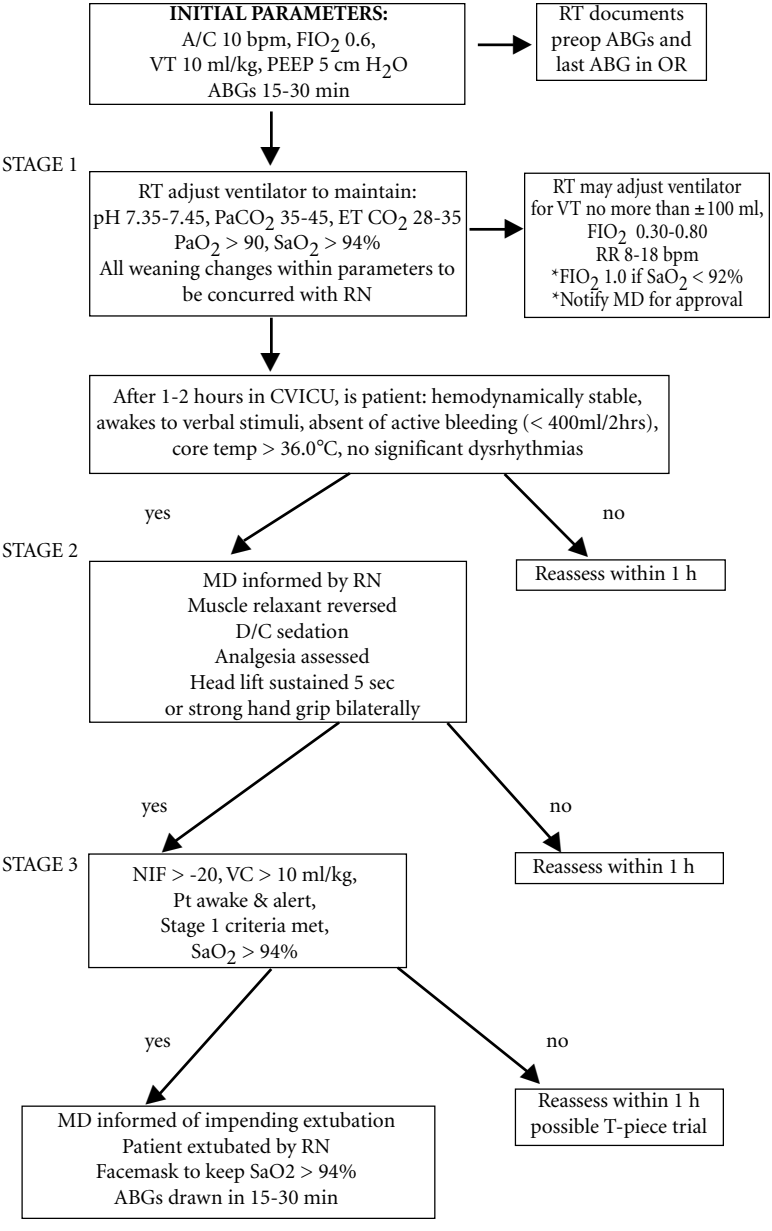
Consulting services: _____

_____

Plan: _____

_____

APPENDIX 6
VENTILATION WEANING AND EXTUBATION PROTOCOL



APPENDIX 7
CVICU ADMISSION CHEST X-RAY PROTOCOL

Admission chest x-ray will be ordered only if one of the following is NOT present, following physical assessment of the patient by the attending physician:

Respiratory system:

1. Endotracheal tube position - maximum 25 cm at the teeth for men and 24 cm for women
2. Symmetrical air entry by auscultation into both lungs
3. Post CPB operating room ABGs done on 100% $FiO_2 \Rightarrow PaO_2 > 200$ mmHg
4. Hemoglobin oxygen saturation (FiO_2 0.6) above 95%
5. On $FiO_2 < 0.6$, $PaO_2 \geq 90$ mmHg
6. Airway pressure during controlled ventilation < 30 cm H_2O

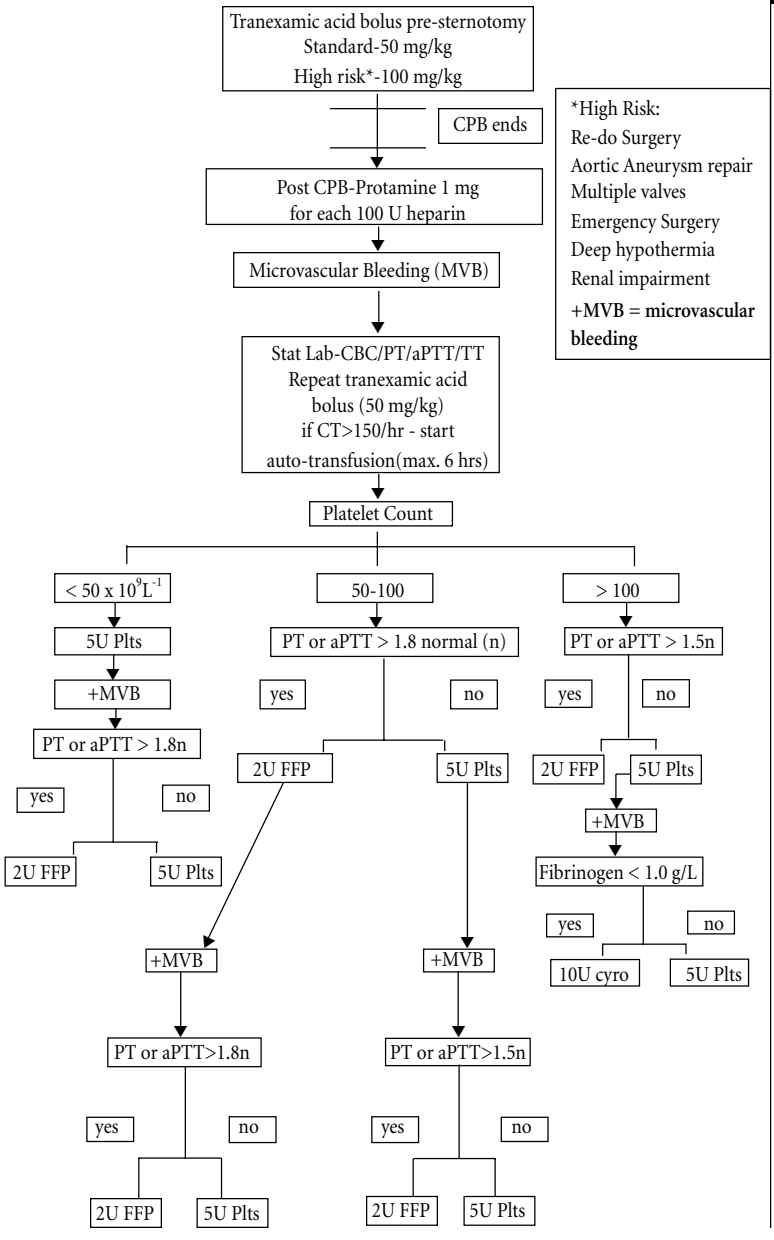
Circulatory system:

1. Patient hemodynamically stable (Syst BP > 90 mm Hg, CI > 2.0 L/m^2)
2. No excessive bleeding—over 200 ml first hour
3. PA catheter at maximum 50 cm from cordis
4. Proper waveform of PA catheter
5. No problems floating PA catheter in the OR

Admission chest x-ray should be ordered for these patients:

1. After complex congenital procedures and repeated complex valvular/ aortic surgery
2. To assess the position of IABP
3. In all patients after removal of the chest tubes to exclude post removal pneumothorax
4. When it is clinically indicated (as per attending physician assessment) e.g., before starting nasogastric feeding

APPENDIX 8
ALGORITHM FOR MANAGEMENT OF MICROVASCULAR BLEEDING



APPENDIX 9
ANTICOAGULATION GUIDELINE POST-CARDIAC SURGERY

1. VALVES

VALVE TYPES	
MECHANICAL	BIOPROSTHETIC
St. Jude Carbomedics Sorin Medtronic Hall Bjork-Shiley	Hancock Porcine Carpentier Edwards Pericardial Medtronic Mosaic Porcine Toronto Stentless Porcine Valve (SPV) Autograft
All Positions: Warfarin permanently INR 2.5-3.5 (Closer to 3.5 for mitral and tricuspid) (Closer to 2.5 for aortic)	Aortic position: ASA 325 mg od permanently Mitral (A. fib): Warfarin permanently INR 2.0-3.0 Mitral (sinus): Warfarin for 3 months INR 2.0-3.0; then ASA 325 mg od permanently
Heparin until INR therapeutic: Mitral position 8,000 units SQ tid Aortic position 5,000 units SQ tid	Heparin 5,000 units SQ bid until fully ambulatory

2. ANNULOPLASTY RINGS

Types: Duran, Carpentier Edwards
All positions: Warfarin x 3 months target INR 2-3. Continue warfarin only if indicated for other reasons.

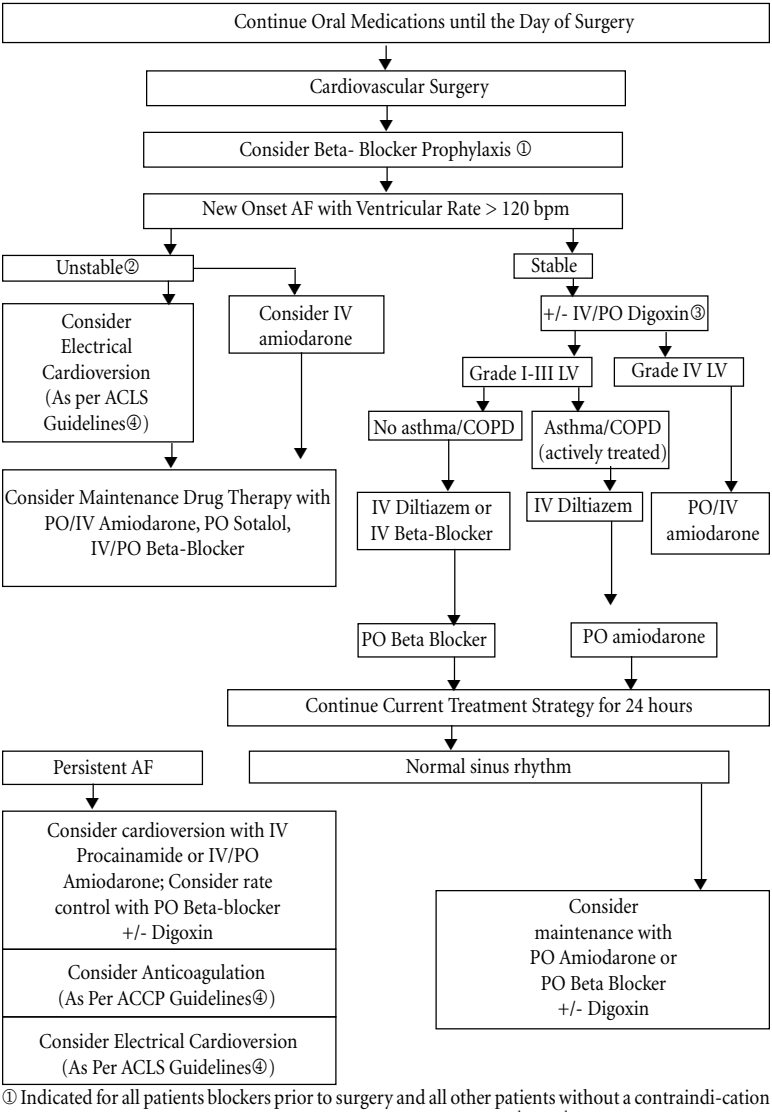
3. AORTOCORONARY BYPASS

Heparin 5,000 units SQ bid until ambulatory
ASA 325 mg po od
Dipyridamole 75 mg po tid (if endarterectomy in the OR only)

Additional Comments:

1. Coumadin should be started when the patient can swallow. If the patient cannot swallow by 24 hours after surgery then heparinization is recommended, but the therapeutic level and duration of therapy should be determined by the specific circumstances.
2. Patients on Heparin and Coumadin should not receive ASA.
3. Starting dose for Coumadin should be 5.0 mg or 7.5 mg if any of the following variables exist:
   - high LFTs of known liver disease or congestion
   - patients over 70 years of age
   - general condition of patient is frail, undernourished and underweight
   - patients of Chinese descent where dietary habits include mushrooms
   - preop or postop Amiodarone or medications affecting coagulation
   - fluid retention status is still significantly over pre-op weight, or other signs of heart failure
   - documented coagulopathy of known or unknown origin
4. On discharge letter to family doctor write valve type and anticoagulation required. e.g., : Aortic valve replacement with St. Jude mechanical valve permanent coumadin INR to be 2.5-3.5.

APPENDIX 10A
ALGORITHM FOR MANAGEMENT OF ATRIAL FIBRILLATION (AF) POST-CARDIAC SURGERY



① Indicated for all patients blockers prior to surgery and all other patients without a contraindi-cation to the use of a beta blocker (including bradycardia, hypotension, 2nd or 3rd degree A-V block asthma/ COPD, brittle diabetes, grade III to IV LV or any condition which, in the professional opinion of the treating physician, placed the patient at risk if beta-blockers are used) ②Includes patients who have hypotension (systolic blood pressure < 80 mm Hg), ischemia, low output or pulmonary congestion ③ Digoxin has a long onset of action and it effective for rate control or conversion in the presence of high sympathetic tone (as occurs after surgery) ④See attached ACLS and ACCP Guidelines.

APPENDIX 10B
FOR MANAGEMENT OF ATRIAL FIBRILLATION (AF)

Suggested Prophylactic Regimens

PO Beta Blocker

⇒ Give first dose 4 to 6 hours after surgery (via NG tube) or when patient is able to take oral medications (initiate by the morning after surgery)

- give metoprolol 25 mg bid

OR

- give sotalol 40 mg bid

Suggested Treatment Regimens

IV Amiodarone

- start with 150 mg bolus
- give additional 150 mg bolus
- consider giving 900 mg infusion over 24 h
- consider oral therapy (see below)

PO Amiodarone

- start with 400 mg TID for 3 to 5 days
- decrease to 200 mg od after loading period

IV Beta Blocker

- start with metoprolol 5 mg IV every 15 minutes for 3 doses
- consider oral therapy (see below)

PO Beta Blocker

- start with metoprolol 25 mg bid for 24 h
- titrate dose to achieve heart rate control without hypotension

IV Digoxin

- give 0.5 mg once then 0.25 mg q6h for 2 doses
- consider oral therapy (see below)

PO Digoxin

- dose based on body weight and renal function
- 0.125 to 0.375 mg daily

IV Diltiazem

- start with 0.25 mg/kg bolus
- give additional 0.35 mg/kg bolus if inadequate response
- consider 10 mg/hr continuous infusion for up to 24 h

IV Procainamide

- give 100 mg q5min until conversion or maximum 1 g dose given
- consider maintenance with 20-80 ug/kg/min infusion

PO Sotalol

- start with 40 mg BID
- increase to 80 mg BID on day 2

Two Common and significant Amiodarone drug interactions in cardiac patients:

1. Coumadin: reduce warfarin dose by 50% and monitor INR closely
2. Digoxin: reduce digoxin dose by 50% and monitor for signs of toxicity

** Other drug interactions must be considered with any drug therapy, on an individual patient basis

APPENDIX 11A-11D
MODIFIED ADVANCED CARDIAC LIFE SUPPORT
APPENDIX 11A: BRADYCARDIA ALGORITHM

- Assess ABCs
- Secure airway
- Administer oxygen
- Start IV
- Attach monitor, pulse oximeter, and automatic blood pressure
- Assess vital signs
- Review history
- Perform physical examination
- Order 12 - lead ECG
- Order portable CXR

(< 60 beats/min) ⇓

bradycardia, either absolute (< 60 beats /min) or relative

⇓

serious signs or symptoms ? a,b

⇓ no

type II 2° AV heart block? or 3rd° AV heart block? [c]

⇓ no

observe

⇓ yes

Intervention sequence:
Atropine 0.5-1.0 mg[c,d] (I& IIa)
TCP, if available (I)
Dopamine 5-20ug/kg/min (IIb)
Epinephrine 2-10ug/min (IIb)
Isoproterenol[f]

⇓ yes

- prepare for transvenous pacer
- use TCP as a bridge device [g]

a) serious signs or symptoms must be related to the slow rate. Clinical manifestations include:
- symptoms (chest pain, shortness of breath, decreased LOC)
- signs (low BP, shock, pulmonary congestion, CHF, acute MI)

b) Do not delay TCP while awaiting IV access or for atropine to take effect if patient is symptomatic

c) Denervated transplant hearts will not respond to atropine
Go at once to pacing, catecholamine infusion, or both

d) Atropine should be given in repeat doses in 3-5 min up to a total of 0.04 mg/kg.
Consider shorter dosing intervals in severe clinical conditions. It has been suggested that atropine should be used with caution in atrioventricular block at the His Purkinje level (type II AV block and new third degree block with wide QRS complexes). (Class IIb)

e) Never treat 3rd-degree heart block plus ventricular escape beats with lidocaine

f) Isoproterenol should be used if at all, with extreme caution. At low doses it is class IIb (possibly helpful); at higher doses it is class III (harmful)

g) Verify patient tolerance and mechanical capture. Use analgesia and sedation as needed

Some modifications are necessary for post cardiac surgery patients specifically:

1. **Bradycardia/ Asystole:** epicardial pacer wires are in place and should be the first line of treatment, followed by pharmacological options.
2. **Ventricular tachycardia/fibrillation:** defibrillate/cardiovert ASAP, start closed massage and pharmacotherapy⇒ lidocaine, amiodarone, epinephrine, magnesium sulfate, bretylium if refractory
3. **PEA:** volume, pace, epinephrine

REMEMBER: if the patient is not responding to initial resuscitative efforts then have a surgeon (staff, fellow) open the chest ASAP. This may help diagnosis and facilitate resuscitation.

APPENDIX 11B: ASYSTOLE TREATMENT ALGORITHM

- Continue CPR
- Intubate at once
- Obtain IV access
- Confirm asystole in more than one lead

⇓

Consider possible causes
- hypoxia
- hyperkalemia
- hypokalemia
- preexisting acidosis
- drug overdose
- hypothermia

⇓

Consider immediate transcutaneous pacing (TCP)[a]

⇓

- Epinephrine 1mg IV push[b,c] repeat every 3-5 min

⇓

- Atropine 1 mg IV repeat every 3-5 min up to a total of 0.04 mg/kg[d,e]

⇓

consider termination of efforts[f]

Class I: definitely helpful
Class IIa: acceptable, probably helpful
Class IIb: acceptable, possibly helpful
Class III: not indicated, may be harmful

a) TCP is a Class IIb intervention. Lack of success may be due to delay in pacing. To be effective TCP must be performed early, simultaneously with drugs. Evidence does not support routine use of TCP for asystole.
b) The recommended dose of epinephrine is 1 mg IV push every 3-5 min. If approach fails, several Class IIb dosing regimens can be considered:
   - Intermediate: epinephrine 2-5 mg IV push, every 3-5 min
   - Escalating: epinephrine 1mg-3mg-5 mg IV push, 3 min apart
   - High: epinephrine 0.1 mg/kg IV push, every 3-5 min
c) Sodium Bicarbonate (1Eq/kg) is Class 1 if patient has known pre-existing hyperkalemia
d) Shorter atropine dosing intervals are possibly helpful in cardiac arrest (class IIb)
e) Sodium bicarbonate (1 meq/kg)

Class IIa
- if known preexisting bicarbonate responsive acidosis
- if overdose with tricyclic antidepressants
- to alkalinize urine in drug overdoses

Class IIb:
- if intubated and continued long arrest interval
- upon return of spontaneous circulation after long arrest interval

Class III
- hypoxic lactic acidosis

f) If the patient remains in asystole or other agonal rhythms after successful intubation and initial medications and no reversible causes are identified, consider termination of resuscitative efforts by a physician. Consider interval since arrest.

Post Arrest:

1. **Failed resuscitation:** Inform staff surgeon promptly and family ASAP. Request autopsy from family. Call coroner's office for all deaths.
2. **Successful resuscitation:** Establish etiology if possible (ie. tamponade, ischemia, electrolyte, respiratory)
   - ventilate if needed to maintain adequate PO_2, minimize ↑ PCO_2 (cerebral protection)
   - CVS: optimize hemodynamics (antiarrythmics, inotropes)
   - CNS: do not sedate unless patient is responding or seizing. It is important to have an early assessment of neurological status
   - Renal: ensure adequate urine output, if not then volume ± diuretics
   - complete investigations when stable including: CBC, PT/PTT, lytes, Cr, glucose, Ca, Mg, Liver Function tests, ECG, CXR

APPENDIX 11C: PULSELESS ELECTRICAL ACTIVITY (PEA)

Includes:

- Electromechanical dissociation (EMD)
- Pseudo EMD
- Idioventricular rhythms
- Ventricular Escape rhythms
- Bradyasystolic rhythms
- Post-defibrillation idioventricular rhythms

- continue CPR
- intubate at once
- obtain IV access
- assess blood flow using Doppler Ultrasound

⇓

Consider possible causes
(parentheses = possible therapies and treatments)

- Hypovolemia (volume infusion)
- Hypoxia (ventilation)
- Cardiac tamponade (pericardiocentesis)
- Tension pneumothorax (needle decompression)
- Hypothermia (see hypothermia algorithm)
- Massive pulmonary embolism (surgery, thrombolytics)
- Drug overdoses (TCA, digitalis, β-blockers, calcium channel blockers)
- Hyperkalemia[a]
- Acidosis[b]
- Massive acute myocardial infarction

⇓

- Epinephrine 1 mg IV push[a,c] repeat every 3-5 minutes

⇓

- if absolute bradycardia (< 60 beats/min) or relative bradycardia, give atropine 1 mg IV
- repeat every 3-5 min to a total of 0.04 mg/kg [d]

Class I: definitely helpful
Class IIa: acceptable, probably helpful
Class IIb: acceptable, possibly helpful
Class III: not indicated, may be harmful

a) Sodium Bicarbonate (1 meq/kg) is Class 1 if patient has known pre-existing hyperkalemia

b) Sodium bicarbonate (1 meq/kg):

Class IIa
- if known preexisting bicarbonate responsive acidosis
- if overdose with tricyclic antidepressants
- to alkalinize urine in drug overdoses

Class IIb:
- if intubated and continued long arrest interval
- upon return of spontaneous circulation after long arrest interval

Class III
- hypoxic lactic acidosis

c) The recommended dose of epinephrine is 1 mg IV push every 3-5 min. If this approach fails, several Class IIb dosing regimens can be considered:
  - Intermediate: epinephrine 2-5 mg IV push, every 3-5 min
  - Escalating: epinephrine 1mg-3mg-5 mg IV push, 3 min apart
  - High epinephrine: 0.1 mg/kg IV push, every 3-5 min

d) Shorter atropine dosing intervals are possibly helpful in cardiac arrest (Class IIb)

APPENDIX 11D: VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA ALGORITHM (VF/VT)

- ABCs
- Perform CPR until defibrillator attached[a]
- VF/VT present on defibrillator

⇓

Defibrillate up to 3 times if needed for persistent VF/VT (200J, 200 -300J, 300J)

⇓

Rhythm after the first 3 shocks ?[b]

⇓

Persistent or recurrent VF/VT

- continue CPR
- intubate at once
- obtain IV access

⇓

- Epinephrine 1 mg IV push[c,d] repeat every 3-5 min

⇓

Defibrillate 360 J within 30 - 60 sec[e]

⇓

Administer meds of probable benefit (class IIa) in persistent or recurrent VF/VT[f,g]

⇓

Defibrillate 360 J, 30-60 sec after each dose of medication,[e] Pattern should be drug-shock, drug-shock

return of spontaneous circulation

- assess vital signs
- support airway
- support breathing
- provide medications appropriate for BP, HR and rhythm

PEA

Asystole

Class I: definitely helpful
Class IIa: acceptable, probably helpful
Class IIb: acceptable, possibly helpful
Class III: not indicated, may be harmful

a) Precordial thump is a Class IIb action in witnessed arrest, no pulse, and no defibrillator available

b) Hypothermic cardiac arrest is treated differently

c) The recommended dose of epinephrine is 1 mg IV push every 3-5 min.

d) Sodium Bicarbonate (1meq/kg) is Class 1 if patient has known pre-existing hyperkalemia

e) Multiple sequenced shocks (200, 200-300 J, 360 J)

f) Medications:
- Lidocaine 1.5 mg/kg IV push. Repeat in 3-5 min to total loading dose of 3mg/kg: then use
- Bretylium 5 mg/kg IV push. Repeat in 5 min at 10mg/kg
- Magnesium Sulfate 1-2 gms IV in torsades de pointes or suspected hypomagnesemic or severe refractory VF (maximum total 17 mg/kg)

g) Sodium bicarbonate (1 meq/kg IV):

Class IIa
- if known pre-existing bicarbonate responsive acidosis
- if overdose with tricyclic antidepressants
- to alkalinize urine in drug overdoses

Class IIb:
- if intubated and continued long arrest interval
- upon return of spontaneous circulation after long arrest interval

Class III
- hypoxic lactic acidosis

APPENDIX 12
PATIENT CONTROL ANALGESIA ORDERS

TORONTO GENERAL HOSPITAL
A University of Toronto Teaching Hospital

Doctor's Order Sheet

PATIENT CONTROLLED ANALGESIA STANDING ORDERS

ALLERGIES
NO KNOWN ALLERGIES

Surgery _____
LV Grade _____

Date & Time Ordered	PHYSICIAN'S ORDERS	Action Taken	Signature and Position
	1. While on PCA device, patient is to receive No further supplemental Narcotics unless approved 2. PCA DRUG: Morphine _____ mg/mL Meperidine _____ mg/mL Other _____ mg/mL 3. PUMP SETTINGS: Dose ____ mg to ____ mg Initial Lockout Interval ___ min Continuous dose _____ mg/h Start continuous dose when patient returns to floor postop Four hour limit _____ mg 4. MONITORING: Respiratory Rate, Sedation Score q 2 h x 24 h, then q 4 h. Record on PCA flow sheet 5. TREATMENT OF SIDE EFFECTS: Have Narcan (Naloxone) 0.4 g/mL vial readily available at Nursing station NAUSEA/VOMITING: Gravol ____ mg IV/IM q 3-4 h prn. IV dose to be infused in 15-30 min. ITCHINESS: Benadryl _____ mg IV/IM q 3-4 h prn. IV dose to be infused in 15-30 min. Discontinued Gravol and Benadryl when PCA stopped 6. If confused–HOLD PCA and treat with __ mg IV/SC q _ h prn. 7. Call Acute Pain Service (APS) Physician Beeper # for: a) Respiratory Rate less than 10/min b) Blood Pressure less than 90 systolic c) Pulse less than 50/min d) Sedation Score of 3 (somnolent, difficult to rouse) e) Unsatisfactory analgesia f) If 4 h limit of drug is reached before 4 h elapse 8. In an emergency, if No response after calling APS, call Anesthesia resident through locating 9. If side effects of slow respiratory rate, hypotension or somno-lence occur, STOP PCA pump immediately and inform attending service as well as Acute Pain Service 10. RN will check and verify PCA setting once per shift 11. Acute Pain Service Beeper Numbers: xxx-xxxx xxx-xxxx 12. Office and answering machine: xxx-xxxx 13. When tolerating fluids well, D/C PCA then start __ I-II q 3 h prn.		

Physician's Signature Date

APPENDIX 13
HOSPITAL DISCHARGE INSTRUCTONS POST-CARDIAC SURGERY

Cardiovascular Surgery Services—Toronto General Hospital, Toronto General Division

Discharge Instructions	**Patient: XXX, XXXX**
Date of Birth: XX/XX/XX	**TTH No. 000000**
Admissions Date: XX/XX/XX	**Date of Procedure: XX/XX/XX**
Discharge Date: XX/XX/XX	**Left Ventricular Function:**

Reason for Hospitalization (Admission Diagnosis):

Procedure(s) Performed:

Postoperative Course:

Activity:
SLOWLY increase your daily activities to return to normal within 2-3 months. Try to walk daily. Rest when you need to. Do not lift objects weighing greater than 5 kg (10 lbs). Do not work with your arms above shoulder height. Return to work 2-3 months after you have been seen by your Cardiologist.

Diet:
Healthy Diet (low fat, no added salt)

Anticoagulation:
COUMADIN daily order if indicated
Dosage adjusted to maintain Target INR:
Length of Therapy:

Leg Staple Removal:
Removal to be performed by family physician. Recommended Removal Date: XX/XX/XX

Medications Upon Discharge:

Additional Instructions:
Patient will be followed by home care nursing.

Please Call your Family Doctor if any of the following should occur:
- you develop a FEVER
- you experience RAPID HEART RATE
- you experience WEIGHT GAIN OF GREATER THAN TWO POUNDS IN 24 HOURS
- you experience SHORTNESS OF BREATH
- there is ABNORMAL DRAINAGE from you incision(s)

BE SURE TO ARRANGE FOLLOW UP APPOINTMENT WITH:
- FAMILY DOCTOR: Within 7 days of discharge.
- CARDIOLOGIST: Within 4 weeks of discharge.
- SURGEON: Approximately 8 weeks after discharge.

YOUR CARDIOVASCULAR SURGEON IS: Dr.
Toronto General Hospital, Division of Cardiovascular Surgery
200 Elizabeth Street
Toronto, ON M5G 2C4 Tel: _____

Completed by: _____ (signature) Date completed: XX/XX/XX

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