Handbook on
Poultry Diseases

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ASA HANDBOOK ON

POULTRY DISEASES

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PREFACE

The second edition of the ASA Handbook on Poultry Diseases has been prepared for the American Soybean Association to assist veterinarians, students, and avian health professionals to diagnose, treat and prevent diseases in poultry flocks.

It is emphasized that in the context of Asia, some diseases such as avian influenza occur as epornitics. Most frequently, production is impacted by combinations of infections and parasites which are invariably complicated by intercurrent nutritional, environmental and managemental deficiencies. Careful evaluation of the history and application of modern techniques are necessary to diagnose and resolve complex infectious multi-factorial diseases.

The American Soybean Association encourages constructive comments on this 2nd edition of the Poultry Disease Handbook, including suggestions to be included in subsequent printings. Specialists and consultants affiliated to the American Soybean Association are willing to assist producers, cooperatives, poultry organizations, and universities with additional information on specific aspects of the control and prevention of poultry disease.

Simon M. Shane
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MANAGEMENT, NUTRITION AND BIOSECURITY

1.0 INTRODUCTION

The purpose of the ASA Handbook on Poultry Diseases is to acquaint veterinarians and poultry health professionals with current information on the diagnosis and prevention of poultry disease in commercial broiler and egg production flocks in emerging and established industries. Productivity and profitability are enhanced by application of sound principles of biosecurity, vaccination, and management. Improving efficiency increases the availability of eggs and poultry meat to supply the protein needs of populations in countries with expanding demand.

During the past two decades, primary breeders of broiler, egg and laying strains have eliminated vertically-transmitted diseases from their elite and great-grandparent generations. Unfortunately, infection of grandparent and parent flocks occurs in many developing countries resulting in dissemination of diseases including mycoplasmosis, salmonellosis and reoviral infection.

Improved biosecurity and an awareness of the need for appropriate vaccination programs, reduces the potential losses caused by both catastrophic and erosive infections on commercial-scale farms, village cooperatives and in integrated operations.

Angara disease, virulent infectious bursal disease, highly pathogenic influenza, reoviral stunting syndrome and swollen head syndrome are examples of emerging diseases affecting flocks in Asia, Africa, and Latin America. In addition, chronic, low-intensity infections such as coryza, pasteurellosis, and salmonellosis continue to erode profit margins.

Prevention of disease depends on a comprehensive program incorporating a sequence of planning, implementing and control in a repetitive cycle (Figure 1.1). Strategies to prevent infection are based on the purchase of breeding stock free of vertically-transmitted disease. Vaccination of parent flocks and progeny and appropriate levels of biosecurity represent the components of disease prevention subject to direct managemental control. The relative importance and contribution of these strategies can be calculated using simulation studies. These should incorporate projections of risk of infection and compare the production parameters and costs for diseased and healthy flocks.

The components of biosecurity comprise a hierarchy with each of 3 levels influencing the cost and effectiveness of the entire program (Figure 1.2):

- Conceptual Biosecurity: The primary level represents the basis of all programs to prevent disease. Conceptual biosecurity includes selecting the location of a complex or operation in a specific area to separate different types of poultry, reduce biodensity, and avoid contact with free-living birds. Siting of farms in relation to public roads and service facilities such as hatcheries, feed mills, and processing plants has a profound impact on the effectiveness of a program to maintain optimal standards of production. Decisions concerning conceptual biosecurity influence all subsequent activities relating to prevention and control of disease. Generally, defects in conceptual biosecurity cannot be changed in response to the emergence of new diseases which may result in severe losses or even failure of an enterprise.

- Structural Biosecurity: The second level of biosecurity includes considerations such as the layout of farms, erection of fences, construction of drainage, all-weather roads, equipment for decontamination, bulk feed installations, change rooms, exclusion of rodents and wild birds, and the interior finishes in houses. Structural biosecurity can be enhanced in the intermediate term with appropriate capital investment. Remedial action may often be too late to respond to the emergence of a new disease or an epornitic of a catastrophic infection such as highly pathogenic avian influenza.

- Operational Biosecurity: The third level comprises routine managemental procedures intended to prevent introduction and spread of infection within a complex or enterprise. These activities can be modified at short notice to respond to disease emergencies. Constant review of procedures, participation by all levels of management and labor and appropriate monitoring of the health status and immunity of flocks contributes to effective operational biosecurity.

FIGURE 1.1 CYCLE OF MANAGEMENT

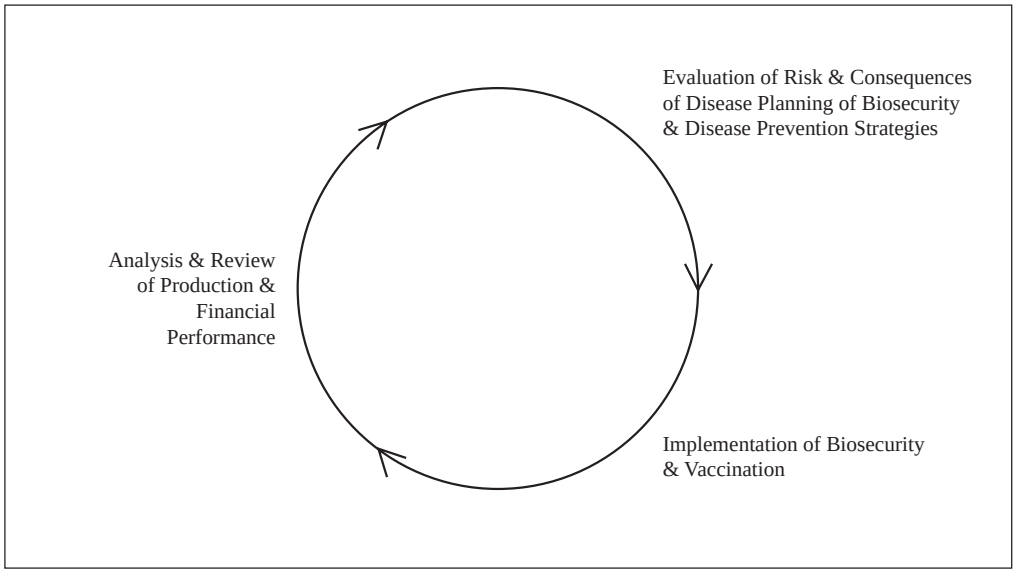
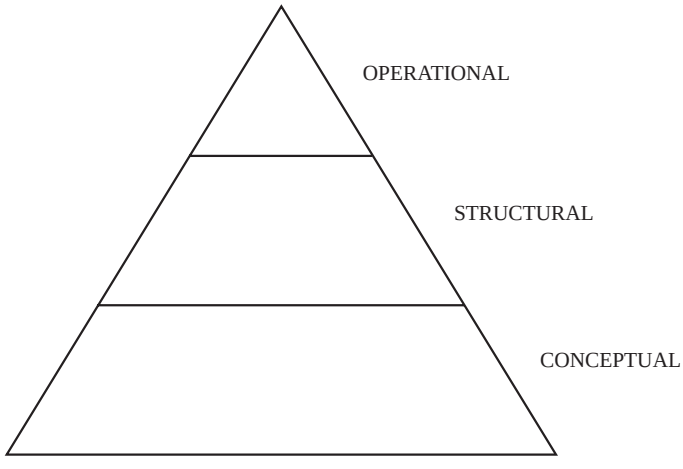


FIGURE 1.2 HIERARCHY OF BIOSECURITY



2.0 ECONOMIC CONSIDERATIONS IN THE PREVENTION AND CONTROL OF POULTRY DISEASES

2.1 General Principles

The primary purpose of any enterprise is to maximize return on investment over the long-term. It is therefore necessary to market poultry, meat products, and eggs at a price which allows farmers or integrators to maintain profitability in a competitive market. Cost-effective programs of biosecurity and vaccination are required to prevent or limit the impact of disease.

It is emphasized that the incremental return in the form of enhanced egg production, hatchability, liveability, growth rate, and feed conversion efficiency must exceed capital and operating expenditures on disease prevention. There is considerable difficulty in predicting the potential loss arising from a disease or projecting the probability of an outbreak. Risk of exposure and consequences of infection, are the two significant variables required to quantify the decline in production which may follow exposure to a disease. The benefit-to-cost ratio can be used to relate expenditure on resources and management efforts to the monetary value of improved performance. Programs of emergency treatment and long-term prevention are justified for severe diseases which have a profound impact on production. Aggressive counter measures are required under conditions which predispose to a high risk of infection, where the prevalence of endemic diseases severely affects production efficiency or where the value of eggs and meat is high in relation to expenditure on biosecurity and vaccination.

It is necessary to invest capital in adequate poultry housing and ancillary installations to attain a suitable level of biosecurity. Changing rooms, fences and equipment to decontaminate hatcheries and housing are examples of assets which reduce the probability of introducing disease. A decision to invest in improvements which promote biosecurity should be based on an anticipation of return within a defined, and preferably short to intermediate time period. The future cash flows, derived from improved performance attributed to the absence of disease, should be calculated for a period corresponding to the operating life of the investment. The net present value (NPV) of an investment in biosecurity can be calculated from the annual cash flows, discounted by an appropriate interest factor. If the NPV exceeds the cost of improvements, the investment can be considered justifiable. The NPV method can be used to select the most beneficial program to prevent disease from among

a range of alternatives. It is emphasized that the validity of any investment decision is dependent on selecting an appropriate value for the risk of infection and accurately projecting the consequences of disease, given prevailing production costs and revenue.

2.2 Fixed and Variable Costs in Poultry Production

Costs relating to live bird production can be classified into fixed and variable components. Fixed costs do not change as a result of an increase in the volume of production and include depreciation, interest on fixed capital, salaries, overhead, and lease payments.

Variable costs are proportional to the volume of production. Feed, labor, packaging material, fuel, vaccines and medication, purchase of day-old chicks and breeding stock, are examples of this category of production costs. The concept of apportioning expenditure is important in projecting the effects of disease on total production cost. A decrease in broiler weight delivered to a plant attributed to increased mortality or depressed growth rate will adversely affect production cost and efficiency. Processing plants, hatcheries, and feed mills operate at a break-even cost approximating 70% to 80% of design capacity due to their relatively high proportion of fixed costs.

Figure 2.1 shows the relationship between total cost, volume of production and profit. Fixed costs which are constant are illustrated by the line parallel to the horizontal (quantity) axis. Total costs are represented by the area which encompasses both fixed and variable costs. In this example, unit selling price is considered constant over volume of throughput and accordingly revenue is linear and proportional to the quantity produced. At the break-even point (quantity Qo) total revenue is numerically equal to total costs. At this level of production fixed costs represent approximately half of the total cost. At a higher throughput, variable cost assumes a greater proportion of total cost. Offsetting fixed costs by increasing production level is the basis of efficiency through economy of scale, which benefits progressive integrations and cooperatives in mature industries. In the context of individual farms, there are limits to increasing production volume. Altering stocking density from 20 to 25 birds/m^2 increases throughput by 25%. Delaying slaughter of a broiler flock to attain a higher live mass (1.75 to 1.95 kg) may increase biomass by 11%. Reducing intercrop interval from 10 to 5 days may result in an 8% increase in broiler live mass over a year. Implementing these management changes will increase the risk of disease and intensify the financial impact of infections. The severity of viral respiratory diseases such as bronchitis or

laryngotracheitis is influenced by environmental and clinical stress. The effect of intercurrent low-grade conditions such as pasteurellosis, mycoplasmosis or coccidiosis may be exacerbated by increased biodensity. Secondary infections such as E. coli septicemia will intensify losses in proportion to increased biomass. Ventilation, capacity, feeding space, drinking points and floor area represent the limiting health factors for flocks when output is increased.

2.3 Gross Marginal Analysis

This analytical technique can be applied to relate expenditure on disease prevention with output over a specific time period. Gross marginal analysis allows producers to project the possible outcome of a program with uncertain risks and consequences of infection. The technique evaluates alternative methods of preventing disease in the context of prevailing costs and revenue. The format table for gross marginal analysis is shown in Figure 2.2. The inputs required to determine the gross margin attributable to a specific program are listed for an ongoing poultry operation over a specific time period. A series of analyses can be performed reflecting alternative prevention strategies and probabilities of disease exposure. The values calculated from the gross marginal analysis are entered into a pay-off table which depicts the financial result of a selected option.

Figure 2.3 considers the effect of three alternative approaches to preventing a disease which has a 0.6 probability of occurrence. The options available to the producer include: no action ("base = 0"); biosecurity (#1) or vaccination (#2). It is determined that the respective gross margins derived from the flock under conditions of no action are $3,000 and $10,000 with and without exposure to disease. The corresponding gross margins generated when flocks are subjected to either biosecurity alone (strategy #1) or vaccination alone (strategy #2) can be calculated and entered into a pay-off table. The expected monetary value of each prevention strategy is calculated by multiplying the probability factor with outcome as shown. In the given example, vaccination costing $1,000 provides the highest of $8,400, compared to $6,460 for increased biosecurity, costing $2,000 and $5,800 for no action. Expected monetary values are influenced by changes in variable costs, unit revenue, and the probability of infection.

Variability in the impact of a disease occurs due to change in the pathogenicity of the causal organism, the presence of secondary agents, immuno-suppression or environmental stress. Changes in these factors influence the outcome of exposure of a flock to infection and requires relaxation or intensification of the preventive strategy depending on the

circumstances. Figure 2.4 depicting expenditure and return from control of disease shows the relationship between expenditure on prevention and control measures (horizontal axis) and the loss associated with introduction of disease (vertical axis). As expenditure on control of velogenic Newcastle disease (vvND) or highly pathogenic avian influenza (HPAI) by effective vaccination is increased, the loss in output is reduced. The low cost of ND and HPAI vaccination and the relative efficiency in improving liveability and enhancing the growth rate or egg production in infected survivors reduces losses associated with minimal expenditure as designated by the curve L_oL_1. Increased outlay on disease prevention and control, such as intensifying the vaccination program and implementing biosecurity will result in an incremental reduction of losses. Eventually the economic optimum is reached (point A) at which a monetary unit of expenditure on control generates only a single unit of return. Additional prevention and control activities will in fact reduce gross margin and generate a negative benefit:cost ratio.

Under certain conditions, such as the need to eradicate a vertically transmitted infection in breeding stock or to suppress a disease of zoonotic significance, control measures are extended beyond the economic optimum. Ultimately the technical optimum (B) is attained. At this point additional efforts to prevent disease will not achieve any measurable reduction in losses.

This sequence may be illustrated by the intensive programs to eradicate mycoplasmosis by the primary broiler breeders during the 1960's and 1970's. Control measures included pressure-differential treatment of eggs with antibiotics, and injection of embryos and chicks with mycoplasmacidal drugs. These measures together with pre-incubation heat-treatment of eggs to destroy *Mycoplasma* spp and enhanced biosecurity and monitoring of pure-line flocks maintained in strictly-isolated small groups eradicated the disease in elite lines.

FIGURE 2.1 CONCEPTUAL RELATIONSHIP BETWEEN COST AND REVENUE

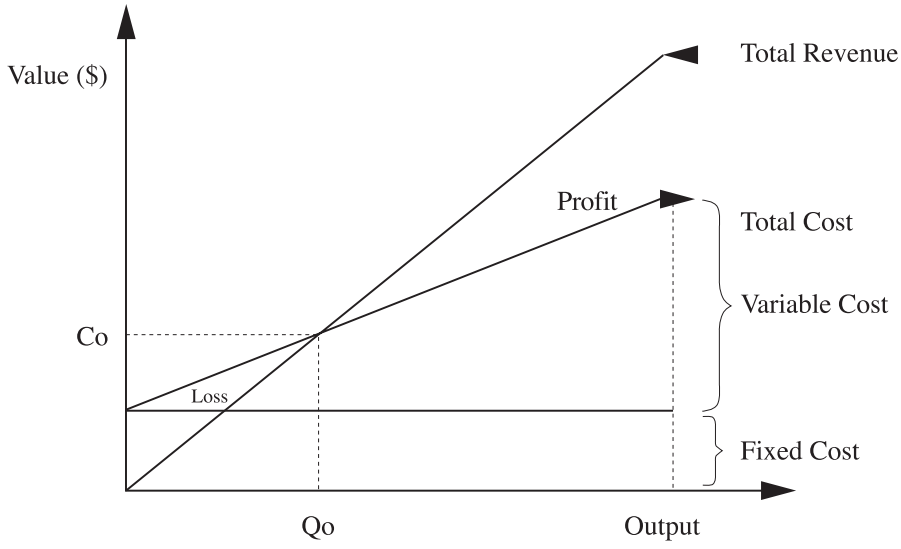


FIGURE 2.2 GENERAL FORMAT FOR GROSS MARGIN ANALYSIS

(1)	Value of inventory at the beginning of the period
(2)	Cost of chicks/flocks purchased
(3)	Variable costs (feed, management, health care)
(4)	Total value at the beginning of the period plus all costs [(1) + (2) + (3)]
(5)	Value of flock at the end of the period
(6)	Value of chickens and products sold
(7)	Revenue from by-products
(8)	Total value at the end of the period [(5) + (6) + (7)]
(9)	Gross margin [(8) - (4)]

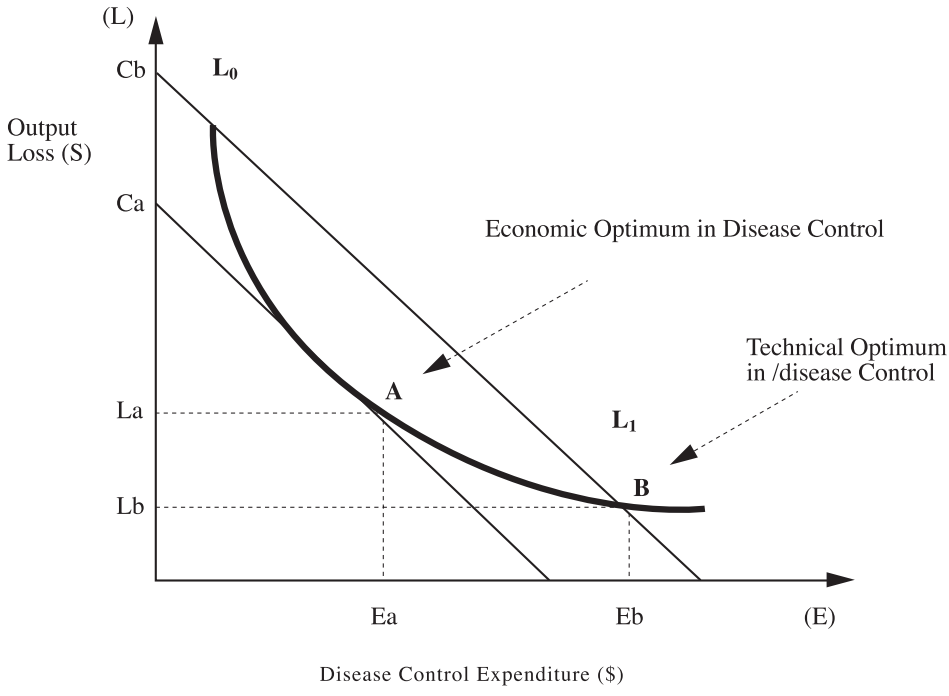
FIGURE 2.3 FORMAT OF PAY-OFF TABLE CONSIDERING ALTERNATIVE PREVENTIVE STRATEGIES

Possible outcomes	Probability of occurrence	Financial result of alternative strategies in designated monetary unit ($)		
		no action #0	biosecurity #1	vaccination #2
With disease	X = 0.6	a $3,000	b $6,000	c $8,000
Without disease	(1 -X) = (1 - 0.6) = 0.4	d $10,000	e $7,000	f $9,000

Expected monetary value associated with alternative strategies:

(Base #0)	=	(a x X)	+	[d x (1 - X)]	=	$5,800
		$1,800	+	$4,000		
(Strategy #l)	=	(b x X)	+	[e x (1 - X)]	=	$6,400
		$3,600	+	$2,800		
(Strategy #2)	=	(c x X)	+	[f x (1 - X)]	=	$8,400
		$4,800	+	$3,600		

FIGURE 2.4 RELATIONSHIP BETWEEN EXPENDITURE AND RETURN FROM DISEASE CONTROL



3.0 HEALTH AND PERFORMANCE OF POULTRY IN HOT CLIMATES

3.1 Physiological Effects of High Ambient Temperature

Exposure of poultry flocks to ambient temperature above the zone of minimum metabolism results in an increase in endogenous heat production. Convective transfer of heat is the major thermo-regulatory mechanism of chickens and depends on movement of air by natural or fan-powered ventilation. An increase in convective heat transfer as a result of air movement is proportional to air velocity of up to 100 m/minute, provided ambient air temperature is below body temperature.

Hyperpnea (panting) occurs in mature chickens exposed to temperatures exceeding 30ºC. Respiratory rate can increase from 22 breaths/minute (bpm) to 200 bpm when ambient temperature is increased from 27ºC to 45ºC within 20 minutes. Panting facilitates evaporative cooling, and above 38ºC, chickens are almost entirely dependent on latent heat loss for thermo-regulation. Prolonged hyperpnea results in excessive excretion of carbon dioxide resulting in respiratory alkalosis. Exposure to high ambient temperature has a profound economic impact on liveability, growth rate, egg production, egg shell quality, and feed conversion efficiency.

Exposure to high environmental temperature for extended periods will suppress the humoral immune response of chickens, reducing antibody titer. It is presumed that a reduction in circulating antibody is associated with a corticosteroid-induced change in serum ions. Cellular immunity is also suppressed by prolonged exposure to temperatures in excess of 36ºC. This effect is mediated through T-cell or regulatory amplifier cell response.

3.2 Design of Housing in Tropical Countries

Convection-ventilated housing is most frequently used in temperate and tropical areas where moderately high seasonal temperatures occur. Structures should be designed to permit passive airflow over the flock. Size and siting of houses in relation to local topography are critical to achieving satisfactory results. The significant design characteristics for convection-ventilated houses relate to internal dimensions, provision of adequate air inlets, and insulation. Convection houses should not exceed 10 m in width to facilitate cross flow of air at low velocity. Houses should be oriented in an east-west direction to limit solar heat load, and the interior height at the apex should not be less than 4 m to reduce air temperature at bird level. Roof overhang should extend at least 0.8 m to limit solar gain through the side walls. The lateral ventilation openings should comprise at least 60% of the side

wall area and should be fitted with impervious curtains. In modern units, the area of the side opening can be controlled automatically by a thermostatically activated motorized winch with an emergency high temperature release mechanism in the event of power failure. General recommendations for insulation in tropical countries include values of 2.5m^2 ºC/W (R = 14) and 1.2 m^2 ºC/W (R = 7) for roof and wall structures respectively. Fiberglass blanket insulation or polyurethane panels should be coated with a reflective radiant barrier of aluminum film on the exposed outer surface and should be provided with an impervious plastic protective lining for the inner surface.

Convention-ventilated houses are economically justified in many warm-climate areas with developing poultry industries. Although stocking density is generally low (eight to ten broilers or pullets or two to three mature breeders per square meter) compared with more advanced housing, the relatively low capital and operating costs optimize profitability. Simple mechanical and electrical installations and elementary technology for management and maintenance favor the basic convection-ventilated unit in tropical and subtropical areas.

To overcome high environmental temperatures, it is necessary to increase the rate of air movement in a house. When daily ambient temperatures exceed 30ºC with any frequency, mechanical ventilation is required. This can be achieved either by installing fans in closed housing or by selecting an appropriate configuration of air inlets in relation to the dimensions of convection-ventilated units.

Air movement facilitates convective heat loss by the bird. The efficiency of this process is proportional to the velocity of the air stream and the temperature differential which exists between the bird and its surroundings. Egg production, fertility, and feed conversion are improved in heat-stressed flocks provided with a direct stream of air.

Evaporative cooling is used to reduce the severity of heat prostration in areas where the maximum temperature exceeds 35ºC with seasonal regularity. All systems function on the principle of adiabatic cooling from a change of state of water from liquid to vapor. The physical relationship between dry bulb temperature, relative humidity, and heat content of air is depicted in psychometric charts. Generally, low humidity improves the efficiency of adiabatic cooling at high ambient temperature, but evaporative cooling can avert losses from heat prostration even in extremely hot and humid areas.

Air at 45ºC and 15% RH could theoretically be cooled to 25ºC assuming complete saturation. Due to restraints associated with the process of evaporation, commercial equipment functions with an efficiency ranging from 60 to 80%. Air at 45ºC and 15% RH could be cooled to a dry bulb temperature of 30ºC with an elevation in relative humidity to 60%.

The simplest evaporative cooling system comprises fogger nozzles which deliver up to 8 to 10 l/hr at a pressure of 5 to 8 bar. Nozzles are positioned in close proximity to turbulence fans to provide one discharge point for each 500 birds. This system is used in the U.S., where low cost is compatible with existing convection-ventilated houses. The low-pressure fogger nozzle produces a coarse spray. Although systems are capable of achieving a 5ºC reduction in temperature with ambient air of 37ºC and 30% RH, low-pressure fogger nozzles are inefficient with respect to the cooling effect relative to water consumed. Systems require frequent cleaning and descaling and litter becomes saturated in the vicinity of the nozzles. The system should only be operated when humidity is below 70% RH and with fans displacing 5 m^3/hr per broiler. Generally, the coarse-nozzle system is unsuitable in Middle Eastern countries due to inefficient utilization of scarce water and blockage of nozzles by mineral contaminants in artesian water.

Pad cooling systems are used extensively in Asia, the U.S.A. and Latin America, where seasonally high temperatures are encountered. The principal deficiency of the pad lies in the inherently lower efficiency of evaporation compared with the ultra-high pressure fogger. Modern cooling pads are composed of cellulose material in a honeycomb configuration to increase surface area. Although this enhances cooling, the system is susceptible to algae and mineral contamination in water. The efficiency of cooling may be enhanced by spraying pads with water from suitably placed nozzles.

3.3 Management of Flocks at High Temperature

The survival of birds at high temperature is strongly influenced by the volume of water consumed. Cold water functions as a heat sink in the intestinal tract and surface evaporation from the comb, wattles, and head exerts a cooling effect. It is essential to provide additional watering points to facilitate consumption in areas where ambient temperature exceeds 3ºʃC for more than 2 hours per day. Recommendations include 1 suspended drinker with a diameter of at least 40 cm, for 75 broilers or 50 breeders and 1 cup or nipple per cage of up to 5 commercial layers. Insulation of header tanks and supply piping is indicated if the temperature of water at the point of consumption exceeds 25ºC.

Research on integrating lighting programs and operation of feeders for broilers has been reported from Singapore. Performance was improved in convection-ventilated housing using nocturnal illumination and feeding. This reverse diurnal lighting program produced the highest live weight at 56 days, but feed conversion, mortality, and return were lower than with other combinations examined. Various lighting and feeding programs were investigated in Nigeria using medium-strain commercial layers. The use of night feeding with a reversed lighting program (18:00 to 6:00) supported a significantly higher level of egg production than conventional daytime feeding, which was accompanied by exposure to high diurnal temperature.

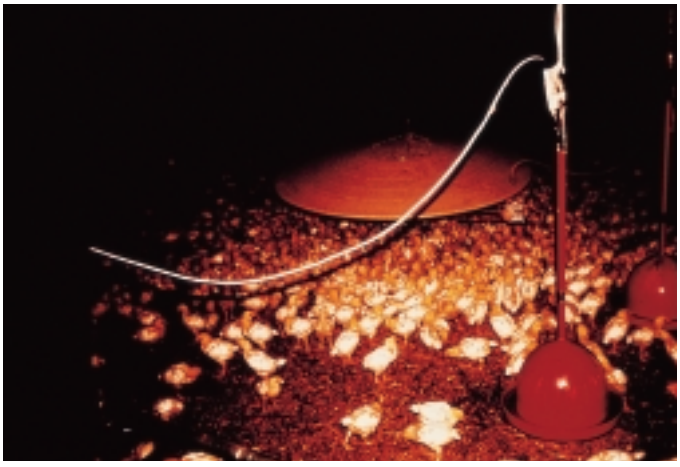
1. Exterior of breeder house showing relative size of sidewall ventilation openings, drainage, concrete apron, bulk-feed installation and grassed area surrounding the unit.

2. Convection ventilated broiler house on an island subject to hurricanes necessitates concrete construction. Note the relative size of ventilation

3. Interior of broiler house showing 2 rows of pan feeders and 4 rows of suspended plastic drinkers. Flock is uniform in size and shows feathering and pigmentation, consistent with health.

4. Equipment used to grow broiler chicks showing 20 year old overhead-filled pan feeding system and modern nipple-cup drinkers. Flock is uniform and well feathered for age.

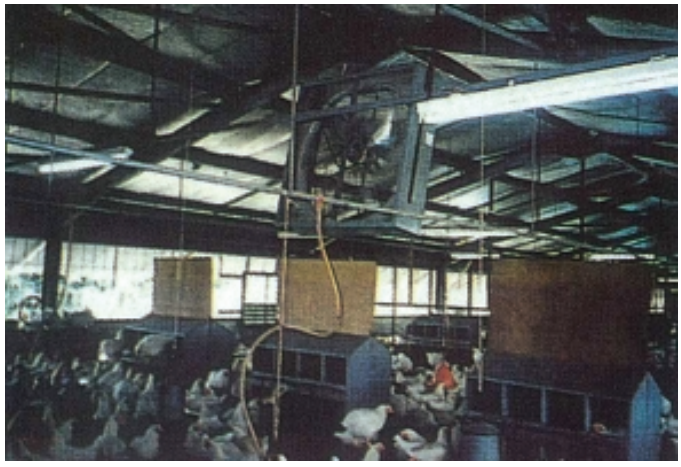
5. Broilers showing signs of heat stress.



6. Chicks beneath a gas-fueled pancake brooder show good distribution consistent with satisfactory growth and feed conversion efficiency.

7. Uniformity in flocks is achieved with adequate feeding space and a supply of clean water from a closed system through nipples or cups.

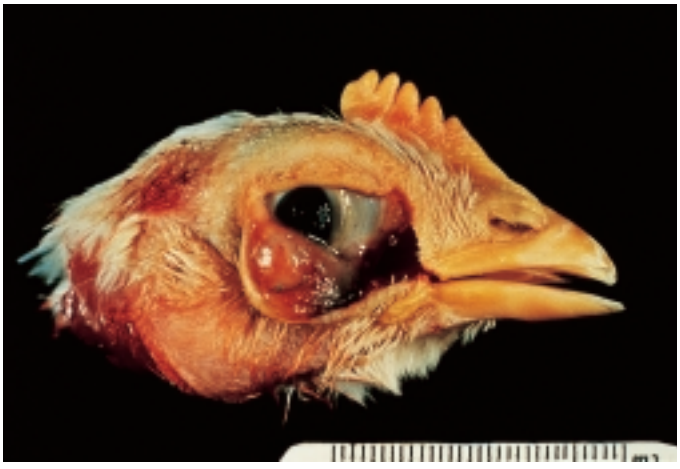
8. Modern high-density cage installation with mechanical feeding and egg collection and fan-powered ventilation controlled by electronic systems.



9. Breeder flock in convection-ventilated house showing ceiling insulation to reduce solar heat gain & belt-driven fan to create air movement over the flock. Fluorescent lamps require less electrical power than incandescent bulbs. Metal nest boxes with manual collection of eggs are easy to decontaminate. Vertical plywood boards prevent perching. Suspended manual feeders for cockerels. Hens obtain feed from troughs fitted with male exclusion grills.

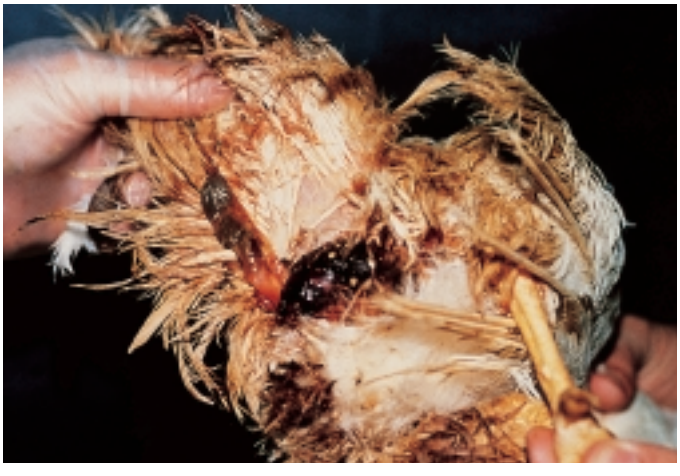
10. High level of dust due to inappropriate ventilation, results in respiratory stress and causes severe reactions to aerosol vaccination and viral respiratory infection.

11. Monitoring of atmospheric ammonia at litter level using a bellows pump and chemical indicator system which is sensitive to ammonia. High ammonia level results in respiratory stress and blindness.



12. Keratitis (inflammation of the cornea) and conjunctivitis following exposure to high levels of atmospheric ammonia.

13. Pododermatitis (Bumble foot) resulting from wet litter. Obese cockerels and hens are susceptible to this condition which reduces fertility.



14. Vent peck and disembowelment in cage-housed hens can be avoided by precision beak trimming at 7-10 days of age and adjusting light intensity to 20 lux.

4.0 PREVENTION OF DISEASE

Prevention of disease in commercial poultry operations requires the application of a coordinated program of biosecurity, vaccination and hygiene.

4.1 Mechanisms of Disease Transmission

In order to develop control procedures it is important to understand the mechanisms by which pathogens are introduced into commercial poultry farms and how disease agents are disseminated among units.

Biological transmission occurs when the pathogen multiplies in the infected host which then transmits the agent when placed in contact with susceptible flocks.

Mechanical transmission involves transfer of a pathogen from an infected source or reservoir host to a susceptible flock by contaminated personnel, equipment, insect vectors, rodents, wild birds, or dust carried by wind.

The following mechanisms of transmission are recognized.

4.1.1 Transovarial Route

Pathogens may be transmitted by the vertical route from hen to progeny via the egg. Mycoplasmosis, pullorum disease (*Salmonella pullorum*), reoviruses and adenoviruses are transmitted in this way. *Salmonella enteritidis (Se)* may also be transmitted vertically by incorporation of the bacterium into the albumen of the egg in the oviduct.

4.1.2 Transmission on the Egg Shell

Pathogens such as *E. coli* and paratyphoid *Salmonella* spp deposited from the cloaca or nest-box litter can penetrate the shell and infect the developing embryo. This form of vertical transmission results in contamination of the hatchery environment and direct and indirect infection of chicks. Omphalitis and salmonellosis may be introduced into brooding and rearing units by contaminated egg-shells.

4.1.3 Direct Transmission

Contact between susceptible flocks and clinically affected or asymptomatic reservoirs of disease will result in infection. This situation occurs in multi-age units and is a common method of transmitting salmonellosis, coryza, mycoplasmosis, laryngotracheitis and pasteurellosis.

4.1.4 Indirect Transmission

Introduction of contaminated transport coops, equipment or feed onto farms or movement of personnel between infected and susceptible flocks without appropriate biosecurity measures will effectively transmit disease. Imperfectly decontaminated buildings which have housed infected flocks often contain pathogens including infectious bursal disease virus (IBDV) and *Salmonella* spp which can infect successive placements especially when interflock intervals are less than 10 days in duration.

4.1.5 Dissemination by Wind

Infected flocks may excrete large numbers of viruses which can be entrained in dust and moved by wind for distances of up to 5km. Spread of vvND and ILT by wind has been documented in a number of outbreaks.

4.1.6 Biological Vectors

Wild birds are reservoirs of avian influenza and *Pasteurella* spp. Rodents carry a wide range of diseases including pasteurellosis and salmonellosis. Insects are responsible for transmission of various diseases. Pox, West Nile and Highland J arbovirus may be transmitted by mosquitoes and spirochetosis by *Argas* ticks. Litter beetles (*Alphitobius diaperinus*) are reservoirs of a wide range of infections including Marek's disease, IBD, salmonellosis, pasteurellosis and coccidiosis. House flies transmit campylobacteriosis. Argasid ticks (*Argas* spp) are vectors of spirochetosis.

4.1.7 Feed

Contamination of ingredients or manufactured feed with pathogens such as *Salmonella* spp, or IBD and paramyxovirus virus can result in infection of susceptible flocks.

4.1.8 Vaccines

Contaminated poultry vaccines prepared in eggs derived from non-specific pathogen free (SPF) flocks may contain pathogens including adenoviruses, reoviruses, or the agents responsible for chicken anemia and reticuloendotheliosis. Pathogens may also be transmitted among flocks as a result of contaminated vaccination equipment or personnel used to administer vaccines.

In the context of Asia, workers, supervisors, and dealers in live poultry are significantly involved in transmitting disease. Delivery of feed in bags requires manual handling. Sale of live poultry involves frequent visits to farms by dealers who ignore the most rudimentary biosecurity precautions.

4.2 Biosecurity

Evaluating the biosecurity of ongoing operations is important in developing effective programs to prevent the introduction of disease into a complex or to limit subsequent dissemination among farms.

A successful biosecurity program presumes an understanding of the principles of epidemiology and economics and requires teamwork to maximize benefits. Biosecurity programs require a structured approach involving the following sequence:

- Planning and evaluation of programs.
- Locating resources and training of personnel.
- Implementing including erection of facilities.
- Control involving review of results and analytical procedures.

The following items should be considered in evaluating a comprehensive biosecurity program for a breeder or growout complex:

4.2.1 Conceptual Biosecurity

- Location of the complex in relation to concentrations of poultry of the same or different species.
- Distance among breeder and growout farms and facilities such as hatcheries, feed mills, and processing plants or packing units.
- Location of major and minor roads and the movement of commercial and backyard poultry in relation to company facilities.
- Proximity to large lakes or waterways or migratory flyways.
- For commercial egg production consider the implications of multi-age on-line units or single-age, company-owned or contractor-operated facilities.

4.2.2 Structural Biosecurity

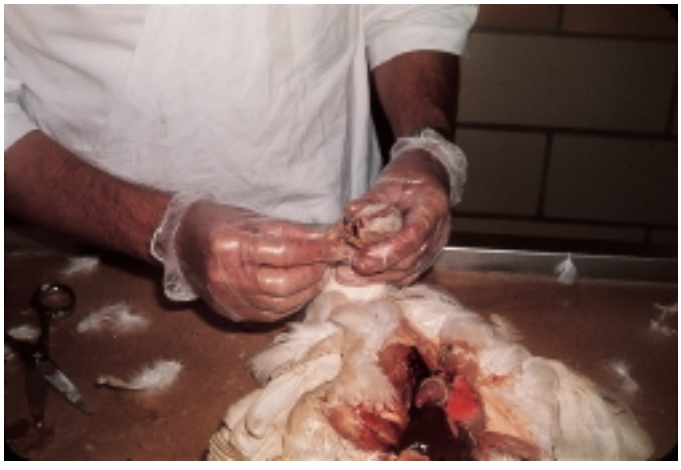
- Fenced farm area with notices to prevent trespass.
- Fencing of house area, with secured gates.
- Water supply free of pathogenic bacteria, and chlorinated to a level of 2 ppm.
- Farm service module comprising an office, storage, and change room-shower facilities.
- Concrete apron with a suitable water and power supply to permit decontamination of vehicles entering the farm.
- All-weather roads within secured perimeter to facilitate cleaning and to prevent dissemination of disease agents by vehicles and footwear.
- Appropriate location of bulk bins or secure, vermin-free storage areas for bagged feed.
- Installations for disposal of dead birds (incinerators, composters, pits).

- Secure housing with appropriate bird and rodent proofing.
- Concrete floors for breeding stock at the grandparent level. In many countries with endemic salmonellosis, concrete floors are required in both rearing and laying housing for breeders.
- Correct positioning of extractor fans to prevent airborne transmission of pathogens to flocks in adjacent houses.
- Impervious apron adjacent to the door of each house and installation of drains.
- Feed, unused litter and cleaned equipment should be stored in a module separated from the live-bird area of the house to prevent contamination of flocks by delivery and maintenance workers.

4.2.3 Operational Biosecurity

- Operational manuals should be developed for routine procedures carried out in feed mills, hatcheries, breeding and grow out facilities. Manuals should incorporate contingency plans in the event of a deviation from normal production parameters or outbreaks of disease on company farms or in units located in close proximity to the operation. Manuals should be developed for appropriate levels of management including company veterinarians and health maintenance professionals, service personnel, contractors, and employees.
- Standardized procedures should address specific aspects of operation including:
  - o Decontamination and disinfection of units following depletion of flocks.
  - o Storage, reconstitution and administration of vaccines according to recommended route.
  - o Specific procedures on entering and leaving farms should be designated for managers, supervisors, authorized visitors, work crews and permanent and part-time employees.
  - o Controls required to prevent contact with exotic avian species, and backyard poultry.

15. Clinical examination as part of a disease investigation requires evaluation of representative birds from a flock to determine the organ systems which are involved.



16. Structured post-mortem examination is necessary to determine the presence of lesions characteristic of a disease in the flock. Pathologists should take precautions to prevent personal infection and should exercise high standards of biosecurity to obviate transmission of pathogens.

4.3 Decontamination of Housing and Equipment

4.3.1 Definitions

Decontamination is the process of physically removing biological and inorganic material from the surfaces of a building or equipment.

Disinfection is the destruction of pathogenic organisms.

4.3.2 Decontamination

Thorough decontamination is necessary to achieve effective disinfection. Cleaning programs require planning followed by implementation and control to ensure satisfactory preparation of surfaces for subsequent application of disinfectants.

4.3.3 Disinfectants

A number of compounds are available commercially, each with characteristics for specific applications.

- Cresols, derived from petroleum distillation are cheap and effective biocides when applied to buildings and soil. These compounds should not be used in the presence of live poultry, eggs, or processed meat as tainting of products will occur.
- Organic phenols are suitable for use in hatcheries to decontaminate equipment.
- Quaternary ammonium compounds (QATs) are highly recommended to decontaminate housing, equipment, and in hatcheries provided that an anionic detergent precedes application of a QAT.
- Chlorine compounds are widely used in processing plants and to purify water on farms. Hypochlorite is only effective over a pH range of 6.5 to 7.5 in water free of organic matter and requires 10 - 20 minutes exposure to inactive bacteria
- Formalin is a corrosive and potentially carcinogenic compound suitable to fumigate eggs in purpose-designed cabinets. Use of formalin requires special precautions to avoid exposure and injury to applicators who must be provided with protective clothing, functional equipment and chemical monitors.

In selecting a disinfectant, it is necessary to take into account the chemical characteristics, toxicity, and the cost of application.

4.3.4 Public Health Considerations

In most countries the use of disinfectants and pesticides is controlled by legislation which restricts the use of products to specified and approved application in accordance with manufacturers' label directions.

Recommendations concerning disinfection and pest control should always conform to statutory regulations and should be designed to limit possible contamination of the environment, flocks, and products. In the absence of national or local rules, the US Department of Agriculture and the US Food and Drug Administration guidelines are recommended.

4.4 Disinfection of Poultry Houses

Complete depopulation of houses and decontamination of units and surroundings at the end of each broiler, rearing, breeder or layer cycle will contribute to enhanced liveability and performance in subsequent flocks.

The following procedures should be followed;

- The surface of the litter and the lower side walls should be sprayed with a 2% carbamate insecticide.
- Litter should be graded to the center of the house for removal either manually or with a front-end loader. Litter should be either bagged or alternatively transported in bulk from the house to a central site for composting or disposal.
- Equipment should be disassembled and removed from the house for cleaning and disinfection.
- Electrical units, motors, and switch gear should be cleaned using a high-pressure air spray and then sealed to protect installations from water damage.
- The floor of the house should be swept to remove residual litter.
- The house should be decontaminated by spraying a non-ionic detergent at a concentration recommended by the supplier. Detergent should be applied to the exterior in the sequence of roof, exterior walls, drains, and service areas. Cleaning the interior should follow the sequence of ceiling, internal walls, and then the floor.
- The interior structure and equipment should be rinsed with water and remaining detergent solution should be allowed to drain.
- The interior of the house should then be sprayed with a quaternary ammonium or phenolic disinfectant solution at a concentration recommended by the manufacturer. A cresolic disinfectant can be applied to earth floors.

- A 2% carbamate insecticide solution should be sprayed on the ceiling, walls, and floor to control litter beetles. (*Alphitobius spp*)
- Equipment should be reassembled and routine preventative maintenance completed. A clean, dry substrate (wood shavings, groundnut hulls, rice hulls, sawdust) should be spread to a depth of 3 - 10 cm, over the floor area.
- Breeder houses should be sealed and fumigated with formalin generated from heated paraformaldehyde or from a mixture of formaldehyde and potassium permanganate. A fog generator can also be used to distribute formalin in aerosol form through the house. It is emphasized that formalin is a toxic compound and is potentially carcinogenic. Appropriate protective clothing and respirators should be used and workers should be trained to use the compound in accordance with accepted procedures to protect health.
- Water lines and drinkers should be drained and cleaned. A quaternary ammonium compound (1 - 2,000 dilution) or chlorine solution (1 liter of 6% sodium hypochlorite per 8 liters of water as a stock solution, proportioned at 1%) should be used to flush water lines.
- Rodent control measures should be implemented including sealing of burrows and baiting. (See 4.5 below)

4.5 Control of Rodents

Rats and mice are significant pests in poultry facilities. They cause damage to building structures, including foundations, water lines, electrical cables, switch gear, and insulation.

Rodents are major vectors and reservoirs of poultry and zoonotic pathogens, including *Pasteurella multocida*, *Salmonella typhimurium* and *S. enteritidis*. Mice amplify environmental contamination and will infect poultry and products. Rodents serve as mechanical transmitters of infectious agents such as influenza and infectious bursal disease viruses and *Salmonella* and *Pasteurella* spp.

Rodents are nocturnal and are active after lights have been turned off. Rats and mice are seldom seen during the day unless infestation is very heavy. Colonization can be detected by the presence of active nesting sites in attics, in cracks in concrete slabs, under cages, in manure, in corners, or in burrows around the foundation walls. Fresh droppings may be observed around the inner perimeter of the poultry house. Outdoor burrows may be closed by filling with soil and observed for reopening of entrances. The frequency of catching rodents in traps may also be used to assess the level of infestation.

A continuous integrated program to control rodents includes rodent-proofing of buildings, elimination of nesting places, appropriate management and sanitation, and chemical and nonchemical elimination. Preventing access to feed, water, and shelter is an important part of a rodent-control program.

Chemical methods to control rodents include bait and tracking powder. All rodenticides are poisonous at various levels for poultry, livestock, and humans. Caution in the use of rodenticides is required, and manufacturer's label instructions should be strictly followed.

Rodenticides are available for single- or multiple-dose application. A single-dose rodenticide will kill rodents after one feeding if an adequate amount is consumed. Most single-dose compounds are toxic to nontarget animals and should be kept out of reach of children, pets, poultry, and livestock. Only extreme situations call for the use of a single-dose rodenticide with high toxicity.

Multiple-dose compounds have a cumulative effect and will kill rodents after several feedings. Bait has to be available continuously, and other feed sources must be removed. The rate of rodent kill depends on the type of rodenticide and the dose consumed. Some products kill within 1 hour, but most available anticoagulant rodenticides require 4 to 7 days after ingestion.

Baits are available in dry or wet form, in powder mixed with grain, in pellets, micro-encapsulated, in paste, in wax, or in water. For maximum effectiveness, bait should be available in both feed and water. Bait should be offered at stations located in the activity zone of rodents, in the routes between the nesting site and the common food source, and at the entrance to houses and near active burrows.

4.6 Control of Free-Living Birds

Free-living migratory and resident birds serve as reservoirs and disseminators of numerous infections of commercial poultry. These include Newcastle disease, avian influenza, duck viral enteritis, chlamydiosis, salmonellosis, and pasteurellosis. The following precautions can be applied to reduce the probability of infection:

- Water obtained from lakes or ponds on which waterfowl accumulate must be filtered and treated with chlorine to a level of 2 ppm.

- Buildings housing flocks and warehouses should be bird-proofed. This includes netting over air inlets, exhaust openings, and screen doors. A commercial product, Avipel® (9,10-anthraquinone) can be applied as a paint suspension to roof areas, gantries and structures where resident pigeons and sparrows congregate. Avipel® will repel birds by a process of aversion to the compound, which induces an irritation of the crop as a result of ingestion of minute quantities following preening. Since birds can differentiate between treated and non-treated surfaces by visualizing the UV spectrum of 9,10-anthraquinone, resident populations of potential reservoirs of infection are displaced from critical areas in feed mills, farms and processing plants.

4.7 Quality of Water

Water supply for farms and hatcheries should be obtained from a municipal source which is filtered and chlorinated or from a deep (+50m) cased well or from a filtered and treated source from a dam or river. Water containing mineral impurities can affect skeletal integrity, intestinal function and detract from optimal growth and feed conversion efficiency. Microbiological contamination including fecal coliforms and viable Newcastle disease and avian influenza viruses can result in infection of flocks. Standards for mineral and microbiological quality are shown in Table 4.1.

Chlorine can be added to drinking water at a level of 2 ppm using either sodium hypochloride or a gas chlorine installation. For effective treatment the pH of water should be adjusted within the range of 6.5 to 7.5. Water lines can be flushed and decontaminated with solutions as indicated in Table 4.2.

TABLE 4.1 STANDARDS OF WATER QUALITY FOR POULTRY

Component	Average Level	Maximum Acceptable Level
Bacteria		
Total bacteria	0 CFU/ml	100 CFU/ml
Coliform bacteria	0 CFU/ml	10 CFU/ml
Acidity and hardness		
pH	6.8 - 7.6	6.0 - 8.0
Total hardness	60 - 200 ppm	150 ppm
Naturally-occurring elements		
Calcium	60 mg/l	
Chloride	15 mg/l	250 mg/l
Copper	0.002 mg/l	0.6 mg/l
Iron	0.2 mg/l	0.3 mg/l
Lead	0	0.02 mg/l
Magnesium	15 mg/l	125 mg/l
Nitrate	10 mg/l	25 mg/l
Sulfate	125 mg/l	250 mg/l
Zinc	5 mg/l	1.5 mg/l
Sodium	30 mg/l	50 mg/l

TABLE 4.2 PREPARATION OF SANITIZER SOLUTIONS TO FLUSH WATER LINES SUPPLYING NIPPLE & BELL DRINKERS

Additive	Concentration of Stock Solution
35% hydrogen peroxide	50 ml/10 l
20% sodium hypochlorite (commercial)	500 ml/10 l
6% sodium hypochlorite (domestic)	1500 ml/10 l
18% iodine complex	150 ml/10 l

Stock solution to be metered at a dilution rate of 1% into water system using a proportioner.

17. Backyard poultry and gamefowl serve as reservoirs for a wide range of infections which can impact the health and profitability of commercial poultry.

18. Inadequate change room facilities may contribute to the introduction of infection to farms and hatcheries.

19. Neglecting maintenance will result in rodent infestation.

20. Accumulation of debris and discarded equipment encourages breeding of rodents.

21. Wet markets are a source of infection and special precautions should be taken to avoid introduction of disease onto farms by live bird traders.

22. Bulk delivery of grain reduces manual handling, is cost efficient and consistent with accepted standards of biosecurity.

23. Manual handling of feed bags by workers may result in introduction of infection onto farms.

5.0 VACCINATION AND MEDICATION

5.1 General Principles

Vaccination involves the administration of a specific antigen to stimulate the immune system to produce homologous antibodies against viral, bacterial, and protozoal diseases. Vaccination programs should be based on the following considerations:

- Diseases prevalent in the area of operation.
- Risk of exposure.
- Immune status of parent-level stock in relation to maternal antibody transfer.
- Cost of acquisition and administration of vaccines.
- Intensity and consequences of adverse vaccine reaction.
- Flock placement programs.
- Availability of specific vaccines.
- Cost to benefit ratio associated with vaccination taking into account the risk of infection and financial loses from disease.

5.2 Significance of Maternal Antibody in Relation to Flock Protection

Maternal antibody (parental immunity) is stimulated in breeding stock in response to exposure to pathogens (naturally acquired maternal antibody transfer), or by vaccination (artificially acquired immunity).

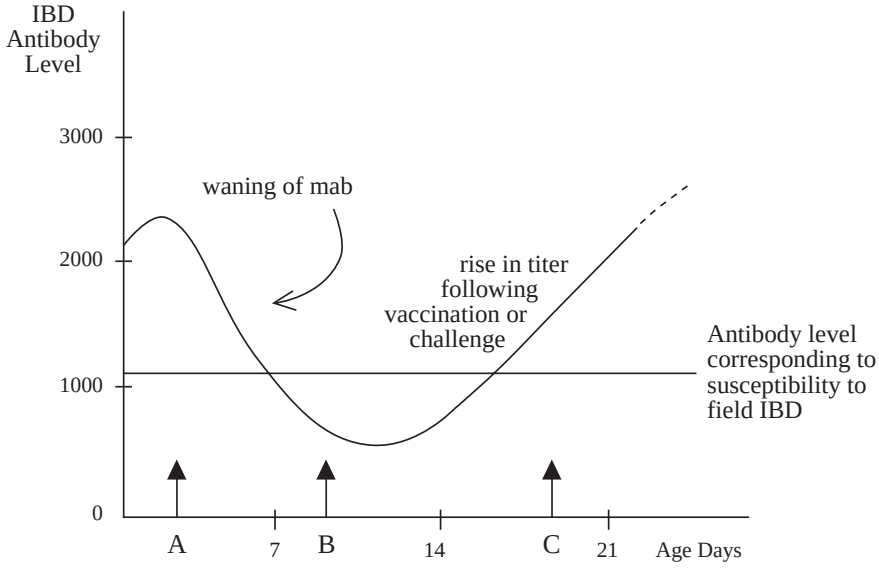
Maternal antibodies (MABs) are transferred from the serum of the hen via the yolk to the chick. Passively acquired maternal antibody may protect progeny against post-hatch exposure to certain pathogens for up to 2 weeks. Circulating antibodies derived from the hen increase from day 1 to day 3 as yolk is absorbed. A waning in titer occurs over the succeeding 1-3 weeks, according to a decay rate characteristic for the antibody.

High maternal antibody is reflected in uniform and proportionally elevated antibody levels (titers) in progeny. Low and variable immunity in parent flocks is associated with early susceptibility of chicks.

High levels of maternal immunity often inactivate mild attenuated vaccine virus administered to chicks. This interference phenomenon is important in timing the first “priming” dose of vaccine which stimulates immunity against IBD, IB and ND. The dilemma facing poultry health professionals in developing vaccination programs for chicks is to specify the age of administration relative to the level of maternal immunity. If the initial vaccine is administered too early in relation to the decline in maternal

antibody, the chick will not be protected. If the initial vaccination is delayed, field challenge of susceptible birds will occur. (Figure 5.1)

FIGURE 5.1 RELATIONSHIP OF MATERNAL ANTIBODY AND VACCINATION



A. Administration of mild IBD vaccine will be ineffective due to interference by high maternal antibody (mab) level. At this age exposure to vvIBD will result in infection due to the ability of virulent field virus to overcome maternal antibody protection.

B. Administration of a mild IBD vaccine will stimulate immunity due to waning of maternal antibody. Flock will be susceptible to field IBD challenge at this time.

C. Subsequent administration of intermediate IBD vaccine will reinforce immunity and raise circulating antibody level.

Other factors of importance in devising vaccination programs include the antigenicity ("strength") of the vaccine virus, the risk and consequences of field infection, and environmental and management factors which may induce adverse vaccine reaction.

Various strategies have been adopted in the US and Europe to immunize broilers. Flock application at day old with attenuated IB/ND is followed by one or more "boosters" during growout. For young breeder flocks, which are housed at high levels of biosecurity, the initial doses of vaccine may be delayed until 7-14 days of age to ensure active priming of the immune system. Administration of vaccines in drinking water or by spray are repeated successively during the growing period.

High uniform levels of maternal antibody are attained in breeders using attenuated live vaccines as "primers" followed by inactivated subcutaneous or intramuscular oil-emulsion "boosters" prior to onset of lay.

Intensive vaccination programs are required to protect broilers, breeders and pullets against exotic and catastrophic diseases including very virulent (vv) pathogenic IBD and vvND.

Although it is not possible to provide immunization protocols to suit specific circumstances, Tables 5.1 to 5.3 illustrate practical programs which integrate vaccination against the diseases that occur commonly in Asia. Avian health professionals are advised to consult with local specialists and suppliers of vaccines to develop appropriate programs.

Table 5.1 represents a comprehensive program to protect imported breeding stock from a wide range of diseases prevalent in an area of operation. This program should only be considered as a general guide to the types of available vaccine, sequence, routes, and ages of administration. The principle of using a mild attenuated vaccine to establish immunity is emphasized. The administration of oil emulsion vaccines to boost immunity is required to ensure satisfactory transfer of maternal IgM antibody to progeny.

Table 5.2 represents a comprehensive program to protect broilers in areas with both endemic vvND and vvIBD.

Table 5.3 represents a comprehensive program to protect commercial laying flocks against the usual range of diseases endemic in Asia.

5.3 Administration of Vaccines

Various methods of administering vaccines are used commercially, including;

- *In ovo* vaccination at 18 days of incubation using the patented Embrex InovoJect® system and alternatives.
- Post-hatch spray vaccination, in cabinets for mass-administration of aerosol vaccines to day old chicks.
- Subcutaneous injection, using a manual or automatic syringe, to administer either live or inactivated emulsion vaccines to chicks, rearing stock and breeders.
- Intramuscular injection, to administer inactivated aqueous and emulsion vaccines to replacement pullets or mature stock.
- Wing-web stab to administer live vaccines by the percutaneous route directly to each bird.
- Eye drop and intranasal routes, requiring handling of individual chicks, are applied in hatcheries and during brooding of chicks.
- Aerosol administration, using a knapsack or electric sprayer to deliver vaccines to flocks as a coarse spray.
- Drinking water administration can be implemented at low cost but is of limited effectiveness against some infections.

It is emphasized that appropriate control over the reconstitution of live vaccines is required to ensure potency. The actual administration of vaccines should be monitored by submission of serum specimens to a diagnostic laboratory for titer assay using ELISA or other acceptable technique.

5.4 Medication

Antibiotics and chemotherapeutic compounds are administered to flocks to treat outbreaks of disease.

The administration of drugs is generally a last resort to salvage the value of a flock and to reduce losses following infection. The availability of drugs varies according to regulations in a specific country. Over-reliance on medication is both expensive and has negative flock and public health implications. Medication should be used only after implementing accepted methods of prevention and control of disease.

Important considerations which contribute to effective medication include:

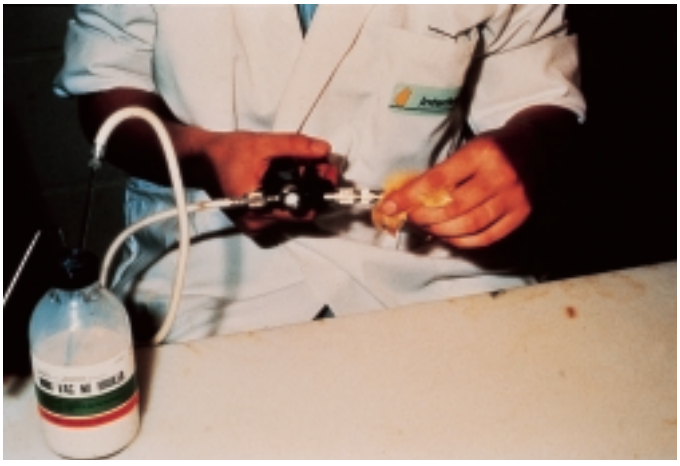
- The diagnosis should be established by isolation and identification of the pathogen by microbiological or other laboratory procedures.

- Pathogens should be shown to be susceptible to the selected drug using appropriate microbiological sensitivity assays.
- Medication should be administered in accordance with the manufacturer's dose recommendations and for the suggested duration of treatment.
- Response to medication should be monitored in relation to clinical improvement and, where possible, by assay of compounds in feed or water.
- Medication should only be initiated in anticipation of a positive benefit to cost ratio projection based on previous clinical response and a projection of losses. (Figure 5.2)
- Statutory withdrawal periods should be followed before the sale of live birds or processing to prevent drug residues in food products.

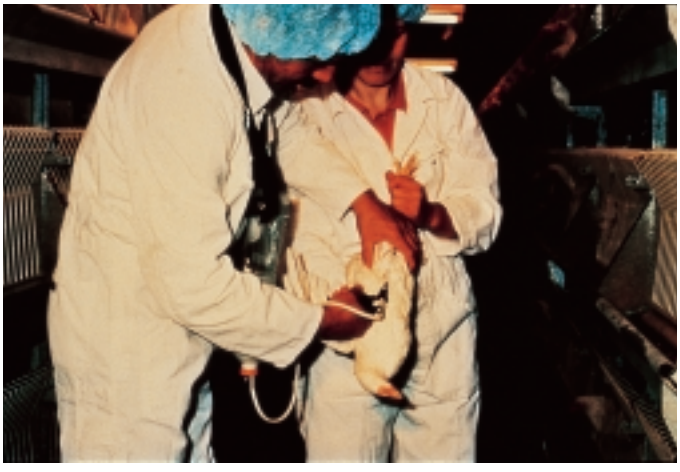
It is emphasized that if routine medication is required for successive flocks, deficiencies in management, biosecurity or vaccination exist. Alternatively, breeding stock may be infected with a vertically transmitted disease. Frequent or continuous administration of medication will result in emergence of drug resistant pathogens which will affect poultry, other livestock and consumers.

A schedule of therapeutic drugs and appropriate dose rates is depicted in Annex 41.2.

24. Hatchery vaccination using an automatic injector.



25. Administration of an oil emulsion vaccine using a manual syringe. (Courtesy Intervet)



26. Vaccination of immature breeding stock with an oil emulsion. (Courtesy Intervet)

27. Vaccination by the wing-web stab route using a rotating carousel to hold birds. (Courtesy Schering-Plough)

28. Vaccination by the intranasal route. (Courtesy Intervet)

29. Cleaning of drinkers prior to administration of vaccine by the oral route.

FIGURE 5.2 EVALUATION OF ANTIBIOTIC THERAPY

Consider antibiotic product with a concentration of 7% active ingredient @$100/liter

Suggested dose rate of 1 ml/liter of drinking water

Flock size, 10,000 birds with a daily consumption of 100 ml/bird

Total consumption of water over 5 days	= 5000 l
Antibiotic consumed	= 5
Cost of antibiotic therapy for flock	= $500
Value of bird	= $1
Number of birds to be “saved” as a result of therapy to break even	= 500
Reduction in mortality rate to break even	= 5%
Assume mortality reduced by	= 800 birds
Benefit : Cost ratio for therapy	= 1.6

TABLE 5.1 COMPREHENSIVE BREEDER VACCINATION PROGRAM

Age	Vaccine or Operation	Route
1 day	Marek's disease, Rispens strain	Subcutaneous, hatchery
	**Salmonella typhimurium (mutant)*	Aerosol spray
2 days	ND HB-1	Eye drop
	IBD(mild)	Eye drop
	ND oil emulsion	Subcutaneous
5 days	Coccidiosis oocysts	Drinking water
7 days	Beak trim	
7 days	Reovirus Live (mild)	Subcutaneous
10 days	IBD (Intermediate)	Eye drop or Drinking
14 days	ND HB-1	water
19 days	IB - H120	Eye drop
	**Salmonella typhimurium (mutant)*	Drinking water
21 days	ND HB-1	
24 days	IBD (intermediate)	Intranasal
5 wks	ND HB-1	Drinking water
5 wks	Reovirus Live	Aerosol spray
	ND oil emulsion	Wing web
6 wks	AE + Fowl Pox	Subcutaneous
	*Avian influenza emulsion	Wing web
8 wks	IB - H120	Intramuscular
10 wks	Pasteurella live	Drinking water
12 wks	ILT	Wing web
15 wks	EDS	Drinking water
	Pasteurella live	Subcutaneous
17 wks	Deworming	Wing web
18 wks	Multivalent ND, IB, IBD, Reo emulsion	Drinking water
	*Avian influenza emulsion	Subcutaneous
20 wks	Transfer	Intramuscular
21 wks	Deworming	
32 wks	NDHB-1 + IB - H120	Drinking water
42 wks	Multivalent ND, IB, IBD, Reo emulsion	Aerosol spray
(optional)		Subcutaneous
55 wks	NDHB-1 + IB H120	
65 wks	NDHB-1 + IB H120	Aerosol spray
(optional)		Aerosol spray

*where prevalent. No antibiotic should be administered within one week before or after live mutant *S. typhimurium* vaccine.

Specific modifications will be required to the program to protect against fowl typhoid, mycoplasmosis and coryza if prevalent in the area of operation.

Abbreviation

AE Avian encephalomyelitis (epidemic tremor)
EDS Egg drop syndrome
ND Newcastle disease
IBD Infectious bursal disease
ILT Infectious laryngotracheitis
ND Newcastle disease

TABLE 5.2 COMPREHENSIVE BROILER VACCINATION PROGRAM

Age (days)	Vaccine	Route
1	Marek's disease, HVT & SB 1 combination	Subcutaneous
	**Salmonella typhimurium (mutant)*	Aerosol spray
	ND oil emulsion	Subcutaneous
	ND HB-1	Aerosol spray
8	IBD (mild or intermediate)	Eye drop or Subcutaneous
10	IBD (intermediate)	Drinking water
12	ND HB-1	Drinking water or Aerosol spray
14	IB H120	Drinking water
16	*ILT	Drinking water
21	IBD (intermediate)	Drinking water
30	ND HB-1	Aerosol spray
(optional)	ND HB-1	Aerosol spray

* where prevalent

Abbreviation

IB	Infectious bronchitis
IBD	Infectious bursal disease
ILT	Infectious laryngotracheitis
ND	Newcastle disease

TABLE 5.3 COMPREHENSIVE VACCINATION PROGRAM FOR COMMERCIAL EGG PRODUCTION FLOCKS

Age	Vaccine or Operation	Route
1 day	Marek's disease	Subcutaneous, hatchery
	HVT & SB 1 combination	Drinking water or Eye drop
	**Salmonella typhimurium (mutant)*	Aerosol
	ND HB-1	
	*ND oil emulsion	Subcutaneous
5 days	*IBD oil emulsion	Subcutaneous
	*IBD mild strain	Eye drop
10 days	IBD intermediate strain	Drinking water
14 days	**Salmonella typhimurium (mutant)*	Drinking water
20 days	IB-HI20	Drinking water
24 days	ND	Drinking water or Aerosol spray
30 days	IBD intermediate strain	Drinking water
6 wks	*Avian influenza emulsion	Intramuscular
7 wks	Avian pox and AE	Wing web stab
10 wks	ILT	Drinking water
12 wks	ND - IB combination	Drinking water
16 wks	Transfer	
	Multivalent inactivated EDS, ND IB	Subcutaneous
	*Avian influenza emulsion	
30 wks	ND - IB combination	Drinking water or Aerosol spray
	(and at 12 wk intervals thereafter)	
	If required: Fowl typhoid at 7 wks	Intramuscular
	or: Coryza bacterin at 7 wks and 14 wks	Intramuscular

Small scale operations with available labor, and potentially exposed to a high risk of infection.

*if required in area of operation.

Abbreviation

AE	Avian encephalomyelitis (epidemic tremor)
IB	Infectious bronchitis
IBD	Infectious bursal disease
ILT	Infectious laryngotracheitis
ND	Newcastle disease

6.0 SPECIAL PROCEDURES RELATING TO CONTROL OF DISEASES IN POULTRY OPERATIONS

6.1 Control of Disease in Multiplier Breeder Farms

Suppliers of breeding stock in the USA and Europe maintain flocks free from salmonellosis, mycoplasmosis, and many other vertically transmitted diseases including specific retroviruses. Deficiencies in biosecurity, at the grandparent or parent level in the country of operation may lead to infection of breeding flocks, resulting in suboptimal production and transmission of disease to progeny.

Breeder farms should be operated on an all-in-all-out basis preferably with absolute separation of rearing and laying flocks.

6.1.1 Structural Biosecurity

Multiplier breeder farms should be at least 3 km from any commercial or backyard poultry. The facility must meet the following requirements:

- Perimeter of the site must be surrounded by a chain-link fence buried to a depth of 0.3 m to exclude burrowing wildlife and should be topped with barbed wire to prevent unauthorized entry.
- All openings in the perimeter fence must be secured.
- A 1 m wide strip on either side of the fence must be mowed so that any rodent or vermin activity can be detected.
- A 3 m wide area around building perimeters must be kept free of all vegetation other than mowed grass to inhibit rodent and wildlife activity.
- Entry of personnel into each single-age flock should be through a shower module and require a complete change of clothing. Rigid separation of the potentially "contaminated-outside" and the inner high security bird area should be maintained.
- No vehicles or equipment should be allowed within the farm area from the time of delivery of flock until depletion.
- Small tools and equipment for mowing, spraying, vaccination, and weighing must remain inside the fence during the life of the flock.
- All equipment, such as feed bins, gas tanks, electric and water meters and stand-by plants, requiring service by non-farm personnel must be located adjacent to the perimeter fence.
- Buildings housing flocks must be constructed to allow complete decontamination after depletion of flocks. Design features should include:

- Perimeter walls, 70 cm high, should be constructed of concrete blocks.
- Concrete floors should be smooth-troweled and sloped to lateral drains.
- The interior surfaces of the building, including ceilings and side walls should be cladded with an impervious material such as galvanized steel or aluminum that can be easily cleaned and disinfected.
- All openings into the building should be screened to prevent access of free-flying birds, rodents, and other wildlife. Metal personnel doors and installation of wire mesh screens over windows are recommended.

6.1.2 Operational Biosecurity

Disease-surveillance procedures must be followed for all consumables used or introduced onto the site. This requires examination of each load of floor and nest litter for insects or foreign material. Feed ingredients, and consignments of feed should be assayed monthly for bacterial and fungal pathogens.

Some integrators operate placement programs which house flocks from day old until the end of the egg-production cycle. This system has disadvantages relating to capital cost and management. Split-cycle rearing and laying is recommended. Moving flocks between farms can potentially expose birds to disease. All equipment, such as ramps, nets, coops and vehicles, must be thoroughly cleaned and disinfected after use. All equipment should be visually inspected, and bacteriological cultures should be performed to monitor the effectiveness of decontamination.

Movement of workers and crews among farms should be limited during each working day. Complete decontamination of equipment, showering of personnel, and provision of clean, site-clothing should be followed. Ideally, supervisors or managers should visit only one production facility, such as a farm or hatchery each day. If a production unit has birds of more than one age, movement must proceed from the youngest to the oldest flocks to limit cross-transmission of pathogens.

6.2 Control of Disease on Commercial Broiler Farms

6.2.1 Structural Biosecurity

The general considerations relating to construction of houses as outlined for breeding facilities should be followed.

6.2.2 Operational Biosecurity

It is impossible to maintain adequate biosecurity when live birds are marketed directly from growing farms. It is necessary to transport broilers intended for sale to a remote site for selection and purchase by dealers. This approach to live bird sales allows complete depopulation of farms with realistic interflock intervals of at least 10 days.

The installation of bins (silos) for bulk-delivery of feed is strongly recommended to reduce the risk of introduction of disease associated with manual handling of feed bags.

Appropriate programs of disease detection and vaccination should be implemented. These are adapted from the general biosecurity recommendations and the management guidelines issued by breeders. The intensity of disease prevention measures depends on the risks and consequences of infection.

30. Isolated breeder farm in a rural U.S. location remote from nearest public road. The entire farm is surrounded by secure fencing and a high level of biosecurity is imposed to prevent introduction of disease which could be spread vertically to progeny. (Courtesy of Ross Breeder Inc.)

31. Maintaining multi-age farms or establishing units in close proximity creates problems relating to control and eradication of chronic diseases such as mycoplasmosis and coryza.

32. Well-ventilated convection house using indigenous materials suitable for broiler growing. Materials used for construction are incompatible with disinfection.

33. Delivery vehicles should be disinfected before entry to farms to avoid introduction of pathogens.

34. Installation of mechanical washer to decontaminate coops before return to farms. Processing plant has a throughput of 100,000 birds/week.

35. Litter beetles (*Alphitobius diaperinus*) serve as reservoirs of Marek's disease virus, *Salmonella* spp., *Pasteurella* spp., and other infectious agents.

6.3 Control of Disease in Commercial Egg Production Units

6.3.1 Operational Biosecurity

- It is suggested that small-scale egg-production units in Asia should be operated on an all-in-all-out basis with separate rearing and laying units. If this is not possible, pullets should be obtained from a source known to be free of vertically transmitted infections or diseases characterized by a permanent carrier state such as coryza, salmonellosis or laryngotracheitis.
- Appropriate vaccination programs should be implemented to protect flocks against challenge. Exposure to disease will lower egg production and reduce quality following transfer to laying units. In view of the high investment in facilities and flocks, it is recommended that appropriate biosecurity procedures should be implemented. Movement of personnel should be controlled, and where possible, bulk-delivered feed is recommended to obviate manual handling and delivery in bags.
- Since rodents are reservoirs of salmonellosis, pasteurellosis and Marek's disease, effective programs to suppress vermin should be an integral component of a disease prevention program.
- Care should be exercised in using recycled egg packing material, especially when products are distributed through dealers. Plastic flats should be used, which can be decontaminated at the point of entry to the farm. Culled hens should be transferred from the production unit to a remote site for sale to live-bird dealers.
- Routine disease monitoring is necessary. Procedures include post-mortem examination of dead birds when mortality exceeds standard levels and periodic serum antibody assays to determine the immune status of flocks.
- Due to the low level of mechanization in laying units in Asia, relatively large numbers of laborers are employed to feed flocks and collect eggs. Change-room and shower facilities are required and protective clothing should be provided to prevent introduction of disease onto farms by workers. Laborers invariably have contact with backyard chickens which are reservoirs of disease.

36. Well developed comb of a healthy 25 week old Leghorn hybrid hen.

37. The undeveloped comb of a culled pullet.

38. Simple single-tier layer cage installed in open-sided house incorporating manual feeding and a trough drinker. These inexpensive systems are extensively used in Asia, but labor input is high and the system is associated with problems of manure disposal and houseflies.

6.4 Control of Disease in Hatcheries

6.4.1 Structural Biosecurity

- Access to the hatchery and any associated buildings should be restricted. The facility should have a secure fence, and all entrances to the building should be located inside the fenced area. Facilities should be provided to disinfect all entering vehicles.
- Hatcheries should be designed with a floor plan, ventilation system and interior finishes conforming to accepted standards of hygiene. Work flow should permit separation of potentially contaminated and "clean" areas. Hatchery design should allow for future expansion and incorporate provision for drainage, disposal of waste, washing of chick boxes and trays. The facility should be supplied with chlorinated water.
- Air intakes should never draw air from the vicinity of an exhaust duct. To prevent movement of air from "dirty" to "clean" areas, positive pressure should be maintained in egg setter bays, cold room for eggs, and chick dispatch area. Potentially contaminated areas are the chick takeoff, processing, and washing areas. Door seals should prevent cross-contamination by air movement.
- Adequate laundry, shower, and change room facilities for staff and visitors should be available and used routinely.
- Installations should be capable of adequately cleaning and disinfecting all setter and hatching trays, chick boxes, and egg flats used in the hatchery.

6.4.2 Operational Biosecurity

- A dedicated egg-collection vehicle should be used for fertile eggs, which are graded and decontaminated by spray or fumigation on the breeder site. Egg-delivery vehicles should be decontaminated and fumigated daily.
- Reusable plastic egg trays can be used to collect commercial broiler eggs but should be cleaned and disinfected after each use. To prevent mold growth, all egg flats, trays, and metal boxes should be thoroughly dried after disinfection. Since fiber trays and cardboard boxes cannot be cleaned and disinfected, these should not be reused. Where possible, plastic egg flats and packaging material should be color-coded to the farm of origin.
- All vehicles should be disinfected before entering a hatchery. A log book should be kept for entry of visitors or deliveries to the hatchery, recording date and time and the previous farm or site visited. One central point of entry adjacent to the change room should be designated.
- An appropriate cleaning and disinfection program should be followed, in accordance with the recommendations of suppliers of chemicals and equipment.

- Single-stage setters should be cleaned and disinfected after each transfer. Broken eggs should be removed from setters daily, with appropriate action to prevent cross-contamination. Setter rooms should be disinfected daily under supervision and inspected to ensure compliance with standard procedures. Setter racks should be cleaned and inspected before return to the breeding farm.
- Hatchers and hatching equipment should be cleaned and disinfected after each take-off.
- At the end of each hatching day, the chick-processing room should be cleaned and disinfected, including all work surfaces and installations. Vaccination equipment should receive special attention according to manufacturers' recommendations. Chick boxes should be cleaned on return to the hatchery.
- Chick-delivery vehicles should be decontaminated daily. Routine fogging or fumigation is necessary to prevent aspergillosis.
- The hatchery sanitation program should be incorporated into a hatchery manual. Procedures should specify disinfectants, concentration, and the method and frequency of application. Procedures should be reviewed and updated as necessary. Only properly trained personnel should clean and apply disinfectants.
- Routine monitoring of cleaning and disinfection should be carried out and appropriate remedial action should be taken if the prevalence of omphalitis or aspergillosis results in a 0.5% increase in first week mortality.
- Quality control procedures include examination of hatchery fluff, agar gel impression disks, exposure of media plates to air, centrifugal air sampling, and surface swabs. Routine tests should include incoming eggs, the egg room, setters, hatchers after disinfection, hatcher rooms, setters rooms, chick-processing rooms, vehicles, exhaust ducts, and the water supply. Correlations between the microbiological test results, hatchability, and chick livability should guide the choice of disinfectants and dilution rates.

39. Setter bay in small hatchery showing high standards of surface finishes consistent with acceptable hygiene and decontamination.

7.0 NUTRITION OF CHICKENS AND DIETARY DEFICIENCIES

7.1 Establishing Nutritional Specifications

Modern broiler, breeder and egg-production flocks require diets balanced in essential nutrients to achieve optimal reproductive efficiency, feed conversion, liveability, and immune response. Suppliers of stock provide printed management guides incorporating nutrient specifications appropriate to the various ages and types of poultry. Nutritionists satisfy dietary requirements by blending available ingredients into diets on a least-cost basis. Generally, linear programming is used to develop formulations containing the most critical nutrients. These include;

- Energy
- Crude protein
- Essential amino acids with specific reference to,
  - methionine
  - cystine
  - lysine
  - tryptophan
  - threonine
- Fats and essential fatty acids (linoleic acid)
- Macro Minerals
  - sodium
  - calcium
  - magnesium
  - potassium
  - chlorine as chloride
  - phosphorus as phosphate
  - sulphur as sulfate
- Micro Elements
  - copper
  - cobalt
  - manganese
  - zinc
  - selenium
  - iron
  - iodine as iodized salt
- Vitamins

7.2 Nutrient Deficiencies

7.2.1 Causes of Nutrient Deficiencies

- Diets may be erroneously formulated.
- Biological potency of specific vitamins or availability of minerals may

be sub-optimal.

- Deficiencies may occur due to deletion of specified ingredients or supplements from rations.
- Destruction of nutrients can occur in feed due to oxidation.
- Chemical antagonists in feed may increase the nutritional requirements of nutrients.
- The nutrient quality of ingredients may be depressed by excess moisture, mold contamination or inappropriate processing.

Under commercial conditions multiple deficiencies often occur and signs and lesions associated with suboptimal intake of a specific nutrient may not be clearly defined.

7.2.2 Low Energy Intake

Most poultry will compensate for low energy density by consuming a greater quantity of feed. Under conditions of feed restriction or extreme competition, mature birds will lose weight and hens will show a decline in both egg size and egg numbers. Male breeders will become infertile. Growth rate of immature stock will be depressed. The effect of restricting energy intake will be exacerbated by low environmental temperature or improper management of brooding and ventilation systems during the early growth phase. Flocks deprived of energy will show increased susceptibility to infection.

7.2.3 Deficiencies of Proteins or Amino Acids

Low protein intake will depress growth rate, feed conversion efficiency, immune response and reproductive efficiency. A deficiency in lysine may occur in wheat and maize-based diets and will result in depressed growth rate and feed conversion efficiency in broilers. Methionine deficiency in diets containing maize and soybean meal will result in a low growth rate. In the case of mature flocks, both egg size and egg numbers will be reduced.

It is emphasized that suboptimal levels of essential amino acids will not result in any specific clinical sign or lesion other than a failure to attain accepted production standards. Deficiencies in energy and essential amino acids will exacerbate the effects of viral malabsorption syndrome and intestinal damage caused by coccidiosis or endoparasites.

7.2.4 Fats

Suboptimal levels of essential fatty acids including linoleic and linolenic acid will depress egg size in high-producing hens. Under conditions of elevated temperature, essential fatty acid deficiency will result in

degeneration of the liver and possibly rupture of the capsule, with hemorrhage into the body cavity. (Fatty liver syndrome)

7.2.5 Oxidative Rancidity

Failure to stabilize ingredients such as fishmeal, carcass meal, rice bran, vegetable oils and animal tallow will allow oxidative rancidity to occur. Ingredients containing high levels of saturated fatty acids are susceptible to this process which yields toxic peroxide free radicals which damage cell membranes and overwhelm the inherent biological antioxidant systems at the cellular level. The initiation of oxidative rancidity characterized by free radical formation is stimulated by high ambient temperature, prolonged storage of diets, and the presence of metal catalysts in storage tanks. Autoxidation is prevented by supplements which chelate metallic ions and scavenge and inactivate free radicals. Most commercial antioxidant additives for feed use contain ethoxyquin, and/or butylated hydroxy toluene. These compounds are combined with a chelator such as citric acid, sodium bicarbonate as a buffer in a hydrated aluminum silicate carrier. Antioxidant products are required for ingredients containing in excess of 10% fat. Addition rates to diets at levels equivalent to 125 ppm ethoxyquin are suggested. Antioxidants can also be added to specific ingredients such as fishmeal or animal byproduct meal in liquid form during manufacture.

7.2.6 Vitamin Deficiencies

Deficiencies of vitamins may occur following inappropriate formulation, the use of impotent commercial preparations or destruction of nutrients in feed by oxidation. The significant deficiencies encountered in commercial poultry production include;

- **Avitaminosis A**

  Chicks will show poor growth and feathering and in advanced cases, ataxia (inability to stand), xerophthalmia ("dry eye") and chronic purulent conjunctivitis (accumulation of yellow caseous material beneath the eyelids).

  Laying hens subjected to avitaminosis A will show a deterioration in internal egg quality and a high prevalence of blood spots. Fertility and hatchability of breeders will be adversely affected. Since vitamin A is concerned with the integrity of respiratory and gastrointestinal mucosa, flocks subjected to avitaminosis A will show a high prevalence of *E. coli* and other bacterial infections and will be more severely affected by endoparasites and coccidiosis.

Diagnosis of avitaminosis A can be confirmed by microscopic examination of the trachea and oral mucosa. The characteristic change comprises squamous metaplasia, in which normal columnar epithelial cells regress to multiple layers of flattened cells.

Both immature and adult flocks will show kidney degeneration and the accumulation of urate in the ureters. In advanced cases urate deposit on the viscera (visceral gout) is observed at postmortem examination.

- **Vitamin D3 (Cholecalciferol) Deficiency**
  A deficiency in vitamin D3 will lead to rickets in immature flocks. Affected birds aged 4 to 7 weeks show a disinclination to walk. Swelling of the joints is noted together with depressed growth rate and poor feathering. On post-mortem examination decreased skeletal density is evident, costochondral (rib to spine) junctions are enlarged and the end plates of the long bones are irregular due to defective mineral deposition required for osteogenesis (bone formation). Vitamin D3 deficiency results in gross enlargement of the parathyroid glands.

  Rickets can be confirmed by histological examination of the proximal end plate of the tibia and parathyroid gland tissue.

  In mature laying and breeding stock, a deficiency of vitamin D3 results in osteomalacia characterized by decreased skeletal density. Affected flocks show a gradual decrease in egg production and a marked deterioration in shell quality. Ascending mortality is associated with paresis and paralysis in caged hens which are unable to stand to feed and drink.

- **Vitamin E Deficiency**
  Vitamin E is required in complex biochemical functions. Three specific conditions occur in young chickens.

  Encephalomalacia occurs in chicks fed diets in which vitamin E has been destroyed by oxidative rancidity. The presence of free radicals will result in destruction of vitamin E both in the feed and *in vivo*.

  Transudative diathesis occurs in chicks fed diets deficient in vitamin E or containing free radicals. Transudative diathesis results from degeneration of the endothelium (lining of the blood vessels) resulting in leakage of plasma into surrounding tissues. Transudative diathesis can be partly reversed by supplementation of diets with adequate quantities of selenium. (0.1 to 0.3 ppm), preferably in the organic

(selenomethionine) form which is more available than inorganic sodium selenite.

Muscular Dystrophy occurs in the skeletal muscles, the ventriculus (gizzard) and myocardium (heart muscle). The effect of avitaminosis E is exacerbated by concurrent deficiency of sulphur-containing amino acids and selenium.

Encephalomalacia is the most commonly encountered condition associated with avitaminosis E due to a deficiency or in vitro destruction in tropical countries. Onset is at approximately 10 - 20 days of age and may result in the death of up to 10% of the flock. Infected birds demonstrate ataxia, incoordination, and terminal recumbency with cycling motions of the legs. The characteristic lesion comprises punctate hemorrhages within the cerebellum and occasionally the cerebrum. Malacia (softening) of the brain is evident. The condition can be confirmed by histological examination of brain tissue from affected birds.

The differential diagnosis of ataxia in chicks includes avitaminosis A, avian encephalomyelitis (epidemic tremor), thiamine and pyridoxine deficiencies. Recently, arenavirus infection has emerged which leads to hypoglycemia which results in recumbency and tremors. Organophosphate toxicity which causes incoordination and death preceded by convulsions is usually peracute in onset and involves the entire flock.

Flocks showing nutritional encephalomalacia will respond to administration of water dispersable vitamin E and stabilization of diets with antioxidants and supplementary vitamin E. Affected birds do not recover.

- **Vitamin K Deficiency**
  This condition occurs in caged flocks fed rations deficient in vitamin K. Subcutaneous hemorrhages are noted on the head and beneath the wings of affected birds. On post-mortem examination sub-serosal hemorrhage is evident. The condition can be diagnosed by determining the prothrombin time which is delayed from a normal 20 - 30 seconds to values exceeding 5 minutes. Differential diagnoses for vitamin K deficiency include hemorrhagic syndrome, mycotoxicoses, and anticoagulant rodenticide toxicity.

- **Vitamin B1 (Thiamine) Deficiency**
  Avitaminosis occurs as a result of failure to add thiamine to vitamin premixes or occasionally as a result of excessive addition of the anticoccidial, amprolium, to diets. The principal sign of thiamine deficiency in 10 to 20 day old chicks comprises incoordination and an abnormal retraction of the head ("star gazing"). There are no macroscopic lesions associated with thiamine deficiency.

- **Vitamin B2 (Riboflavin) Deficiency**
  This condition is characterized by rotation of the legs in chicks aged 10 - 30 days, and is referred to as "club foot" or "curled toe paralysis". Affected flocks will demonstrate low growth rate and poor feathering. Breeding flocks fed diets deficient in riboflavin show low egg production and hatchability. Chicks from deficient flocks have abnormal development of down feathers. Histological examination of the major peripheral nerve tracts will show myelin degeneration.

- **Biotin Deficiency**
  Affected flocks demonstrate poor growth and feathering and elevated mortality. The principal sign comprises dermatitis of the feet and of the skin adjacent to the angle of the beak. These changes also occur with pantothenic acid deficiency and reoviral malabsorbtion syndrome. In breeding flocks, hatchability is lowered and embryonic malformations of the feet are noted.

- **Other Vitamin Deficiencies**
  Deficiencies of pantothenic acid, niacin, pyridoxine and folic acid can be reproduced under experimental conditions. These avitaminoses are seldom diagnosed as single entities in commercial poultry in tropical countries but contribute to a general pattern of poor growth and depressed reproductive efficiency in mature flocks.

- **Calcium and Phosphorus Deficiency**
  In immature flocks a deficiency of either calcium or available phosphorus or an imbalance in these nutrients will result in rickets. In laying hens and breeders, osteomalacia may occur. Calcium and phosphate deficiencies may be diagnosed by histological examination of bones and the parathyroid gland, bone ash determinations, and analyses of representative feed samples.

- **Manganese Deficiency**
  Manganese deficiency leads to chondrodystrophy which results in deformation of the distal tibiotarsus and proximal tarsometatarsus. In

extreme cases displacement of the gastrocnemius tendon occurs as perosis ("slipped tendon").

- **Sodium and or Chloride Deficiency**
  Failure to add supplementary salt to poultry diets composed of maize and soybean meal will result in depressed growth rate and decreased egg production. Young chicks will show tail picking and cannibalism.

- **Zinc Deficiency**
  Deletion of zinc from the mineral premix will result in decreased growth rate and chondrodystrophy.

7.3 Quality Control in Feed Manufacture

It is necessary to implement a comprehensive quality control program to monitor the nutritional value and composition of ingredients and to ensure thorough mixing and correct identification of diets. Standard operating procedures consistent with industry practice should be developed and a quality control program implemented based on laboratory analysis and a review of production records. A commitment to the principle of total quality management generates the following benefits:

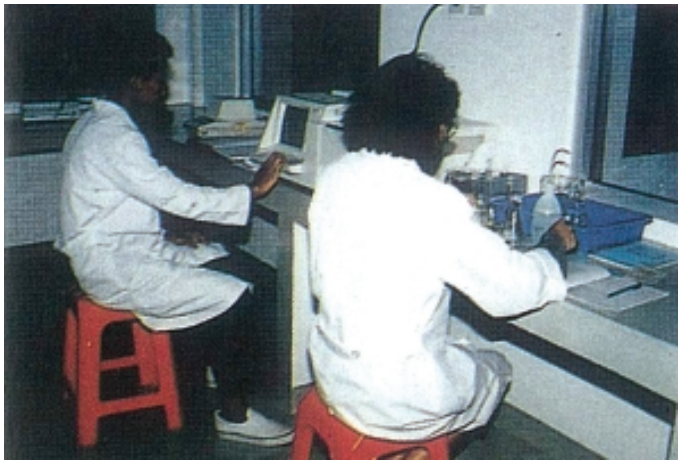
- enhanced performance of flocks by obviating toxicity and deficiency in diets.
- improving client and in-company acceptance of feeds.
- improved market share and profitability.

The direct and indirect benefits of comprehensive quality control invariably outweigh the capital and operating costs involved.

The quality of soybean meal incorporated into poultry diets is an important contributor to performance of broilers and mature stock. Routine assays should include:

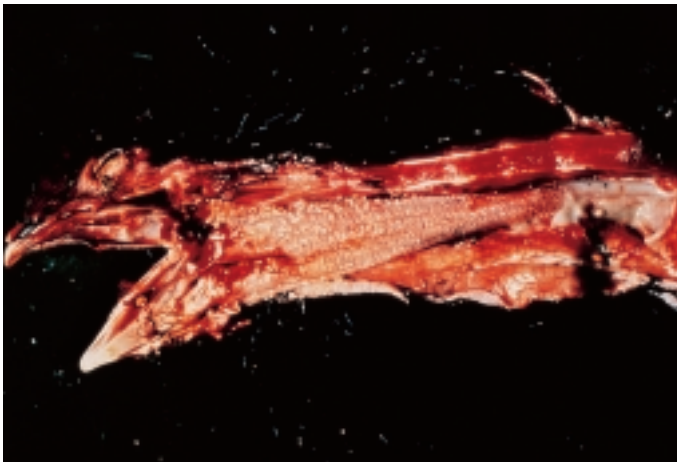
- moisture
- crude protein
- fat
- urease activity
- protein solubility in 0.02% potassium hydroxide

Information on analytical procedures, normal values, storage of soybean meal and formulation is available from an ASA International Office.



40. Quality control is an important component of feed production and requires trained technicians and suitable equipment.

41. Purulent conjunctivitis due to *Avitaminosis A.*



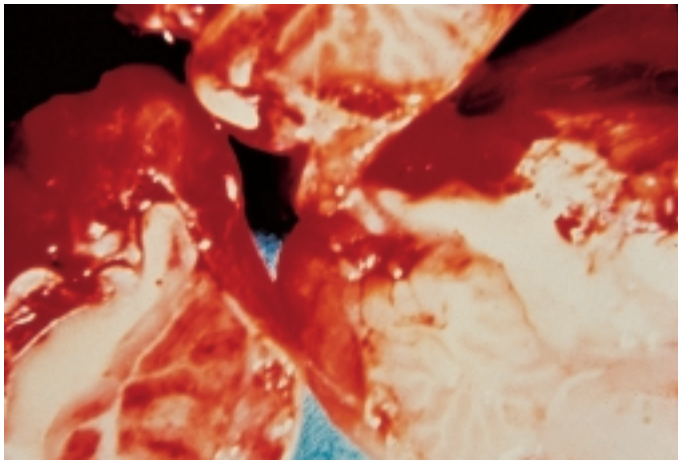
42. Avitaminosis A characterized by nodular lesions in the mucosa of the esophagus.

43. Chicks affected with rickets show lowered skeletal density. In this case, decreased mineralization of the mandible and maxilla allows extensive lateral movement of the beak.

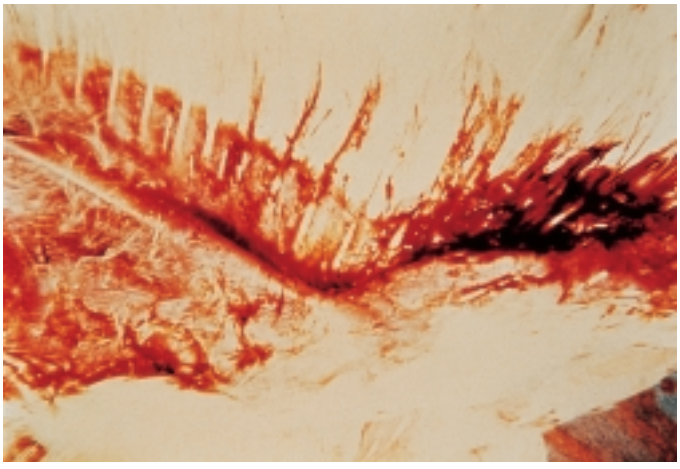
44. The long bones of chicks with rickets can be bent without fracture.

45. Installation of a large capacity black-painted metal storage tank for feed-grade blended oil in a tropical area will lead to rancidity with consequential destruction of fat-soluble vitamins. Horizontal orientation increases surface area and oxidation.

46. Broiler chicks aged approximately 14 days showing lateral recumbency associated with nutritional encephalomalacia.



47. Hemorrhage within the cerebellum characteristic of nutritional encephalomalacia due to a deficiency of Vitamin E or destruction of this nutrient by free radicals in oil undergoing rancidity.



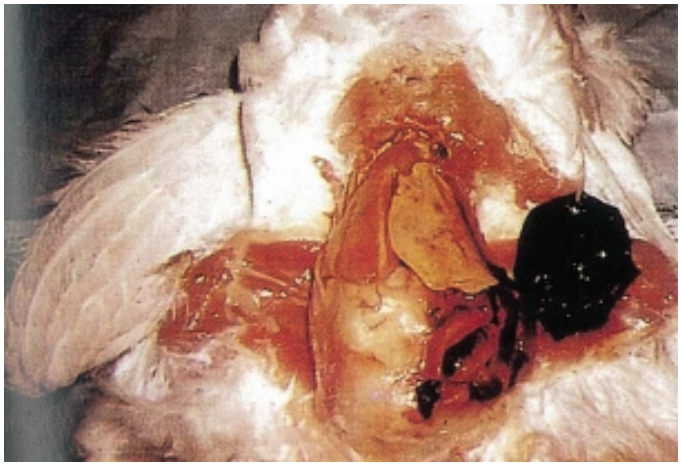
48. Hemorrhage and transudate beneath the wing associated with transudative diathesis due to destruction of Vitamin E following oxidative rancidity.

49. Chicks showing edema of the intermandibular region due to transudative diathesis.

50. Subcutaneous hemorrhage beneath the wing of a cage-housed pullet due to Vitamin K deficiency.

51. Hyperkeratosis (thickening of the skin) of the plantar surface of the feet associated with pantothenic acid or biotin deficiency.

52. Hen showing sternal recumbency due to osteomalacia.



53. Fatty liver syndrome in a mature hen results in rupture and hemorrhage into the body cavity. This condition can be prevented by lowering the energy value of the feed and ensuring that sulphur-containing amino acids and choline are at an appropriate dietary inclusion rate. Measures to reduce heat stress are required to minimize losses.

IMMUNOSUPPRESSIVE DISEASES

8.0 MAREK'S DISEASE

9.0 INFECTIOUS BURSAL DISEASE

10.0 INFECTIOUS ANEMIA

8.0 MAREK'S DISEASE

8.1 Etiology

An oncogenic (tumor-inducing) herpesvirus

8.2 Occurrence and Economic Significance

Marek's disease affects commercial chicken flocks from approximately 5 to 35 weeks of age in all areas of the world.

Highly pathogenic (vvMD) strains of the virus are responsible for acute outbreaks of mortality which may attain 50% in exposed, non-immunized flocks up to 60 weeks of age. Generally, erosive losses of up to 20% occur in non-protected or inadequately vaccinated flocks. Marek's disease virus is responsible for neural and visceral tumors. Marek's disease virus is immunosuppressive and infected broiler and pullet replacement flocks are susceptible to a wide range of viral and bacterial infections. Exposed broilers show increased mortality and condemnation rates at processing.

8.3 Transmission

Exposure to MDV occurs by horizontal infection. The virus is resistant to environmental exposure and can remain viable for long periods in houses especially if units are not decontaminated between cycles. Infected birds shed "dander" (feather dust) contaminated with virus which can be distributed by wind, equipment, and personnel.

8.4 Clinical Signs

Involvement of the peripheral nerves results in paresis (weakness) of the legs or wings which progresses to paralysis. Death occurs in both caged and floor-housed birds as a result of dehydration and persecution.

8.5 Pathology

Enlargement of the feather follicles is observed on the skin of de-feathered broilers and results in condemnation of carcasses in the USA, Canada, and Europe.

The characteristic MD lesion comprises enlargement of the peripheral nerves of the sciatic or brachial plexus. Occasionally visceral lesions are observed and the kidney, eye, proventriculus, ovary or other organs may be affected.

8.6 Diagnosis and Confirmation

The gross appearance of neural lesions is generally diagnostic. Histological examination of nerve and visceral lesions will show characteristic

lymphocytic proliferation. The condition should be differentiated from botulism and from "transient paralysis", an emerging condition of unknown etiology, but suspected to be an autoimmune response to vaccination in specific strains of commercial laying hens.

The causal virus may be isolated and identified by submitting tissues to a suitably equipped laboratory using specific tissue culture techniques.

8.7 Prevention

Vaccination of broiler embryos using *in ovo* administration on the 18th day of incubation or by subcutaneous administration of vaccine to broiler, breeder or replacement egg-strain chicks at day old.

Three types of vaccine are available:

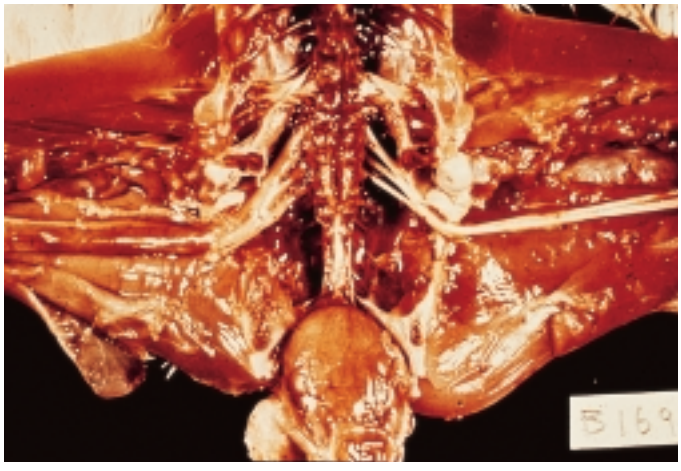
Type 1: attenuated chicken strain (e.g. Rispen's)
Type 2: apathogenic chicken strain (e.g. SB1)
Type 3: apathogenic turkey, strain (e.g. HVT)

Due to the interfering effect of maternal antibody on HVT it is advisable to alternate vaccine types in successive generations. In countries where highly pathogenic MDV occurs, parents should be vaccinated with Rispen's strain, allowing commercial progeny to be immunized using the less expensive HVT strain alone or in combination with the potentiating SB1 strain.

Cell-associated, frozen vaccines require special storage in a liquid nitrogen canister. Careful reconstitution using the diluents supplied by the vaccine manufacturer is necessary to maintain viability of the vaccine virus. Improper vaccination technique may lead to defective immunization with resulting "breaks".

It is essential to place day old chicks in houses which have been thoroughly decontaminated to allow vaccinated flocks to develop immunity. Rearing farms and broiler growing units should be operated as single-age units with all-in-all-out cycles.

54. Characteristic posture of an immature chicken in sternal recumbency with Marek's disease showing alternating extension and retraction of legs. (Courtesy AAAP)



55. Unilateral enlargement of nerves of the sciatic plexus is characteristic of Marek's disease. (Courtesy AAAP)

9.0 INFECTIOUS BURSAL DISEASE

synonyms IBD:Gumboro Disease

9.1 Etiology

Type 1 avibirnavirus strains

Type 1: Both classic and highly pathogenic (vvIBD) serotypes are recognized. Pathogenic Delaware variants A through E predominate in the USA and Central America. The vvIBD strains occur frequently in the Middle East, Asia and Africa.

Type 2: Turkey strains are apathogenic to chickens.

9.2 Occurrence and Economic Significance

The disease affects immature chickens world wide.

Acute infection with classic mild or variant strains results in up to 5% mortality. The variants are more immunosuppressive than the classic strains. Very virulent (vvIBD) virus may kill up to 50% of susceptible flocks.

Avibirnaviruses are immunosuppressive and predispose flocks to intercurrent viral pathogens and secondary bacterial infection.

Infectious Bursal Disease is a major restraint to productivity and profitability in the poultry industries of both industrialized and developing nations.

9.3 Transmission

Direct contact of young birds with infected flocks in multi-age units results in persistent “rolling” infection which is difficult to control. Indirect infection with IBDV occurs within days of placing chicks as the agent can survive in a contaminated environment for up to 3 months. Contaminated equipment, non-pelleted feed containing inadequately heat-treated poultry by-product meal, housing, and clothing of personnel are frequently sources of infection.

9.4 Clinical Signs

Flocks are affected acutely and show variable morbidity (5-50%) and rapidly ascending mortality (5-50%), depending upon the pathogenicity of the IBDV strain and the susceptibility of the flock. Affected birds are depressed and show recumbency, ruffled plumage and white diarrhea. There are no characteristic signs specific to IBD.

9.5 Pathology

Dehydration and muscular hemorrhages are evident in dead birds. In acute cases the characteristic lesion comprises enlargement of the bursa of Fabricius which is often surrounded by gelatinous exudate. Sectioning the organ may show hemorrhages. Recovered birds show bursal atrophy. Airsacculitis and *E. coli* septicemia are frequent complications following exposure to respiratory viruses especially with superimposed climatic or environmental stress.

9.6 Diagnosis

Acute bursal changes are generally diagnostic. Histological examination of bursas from broilers or pullets at various stages of the disease will show edema progressing to atrophy. IBDV can be isolated in specific pathogen free embryonated eggs or on tissue culture. Serotyping carried out in reference laboratories using monoclonal antibodies, can identify specific IBDV isolates.

Antibody response in vaccinated flocks should be routinely monitored using ELISA serology.

9.7 Prevention

Parent flocks should be immunized with one or more attenuated live (mild or intermediate) vaccines followed by an oil-emulsion booster. This program will promote transfer of high and uniform levels of antibodies to progeny. Broiler and replacement layer chicks should be vaccinated with live-attenuated vaccine which primes the immune system. In North America a mild live multivalent (classic and variant) vaccine is administered by the *in ovo* route to provide initial stimulation of the immune system. This is required if chicks are exposed to IBDV at the time of placement. In areas of the world where relatively avirulent strains of IBD virus occur, mild strain vaccines may be administered from day-old to 14 days. Subsequent administration of intermediate strain vaccine may be necessary depending on factors including:

- Risk of infection.
- Strains of virus prevalent in the area of operation.
- Intercurrent exposure to respiratory viral infections.

A combination of live IBD virus with corresponding antibody (Bursaplex®) is available to be administered either *in ovo* or at day-old by the subcutaneous route. This vaccine is effective in the presence of high levels of maternal antibody.

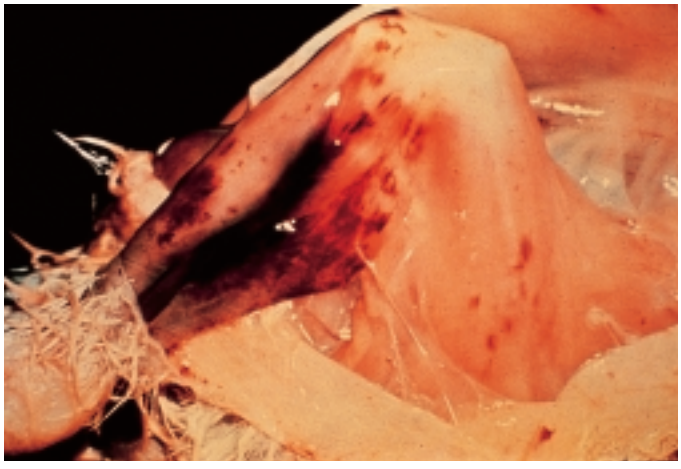
The age of administration of live attenuated vaccines depends on the level of maternal antibody and the risk of infection. In areas of the world where the very virulent (vvIBD) strain occurs, intermediate-plus ("hot") vaccines are administered in drinking water. Selection of the age of vaccination is determined by applying the following formula based on the results of ELISA serology at day old. This formula relates the initial antibody level to the rate of decline in maternal antibody to obtain the optimal age for vaccination.

$$\text{Age of vaccination: } \sqrt{\text{ELISA titre}} - 22.36 / 2.82 \ (+1) \text{ days}$$

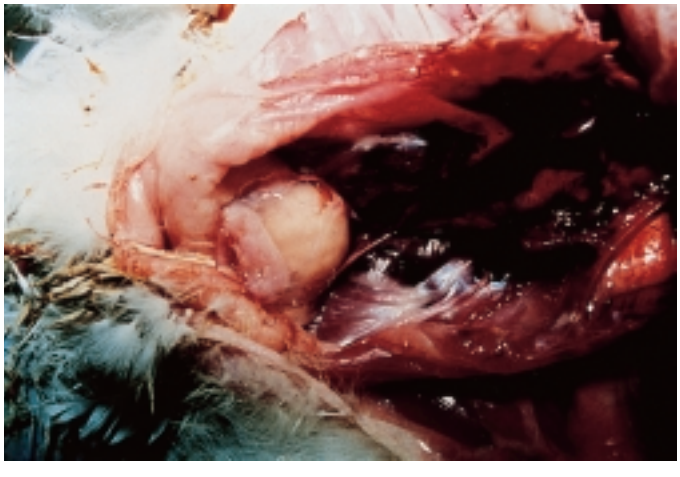
where 22.36 is the square root of 500 ELISA units, (the threshold of protection) and 2.82 is the mean half life (in days) of maternal antibody.

It is necessary to maintain strict levels of biosecurity and to operate flocks on an all-in-all-out basis in areas where severe infectious bursal disease is endemic.

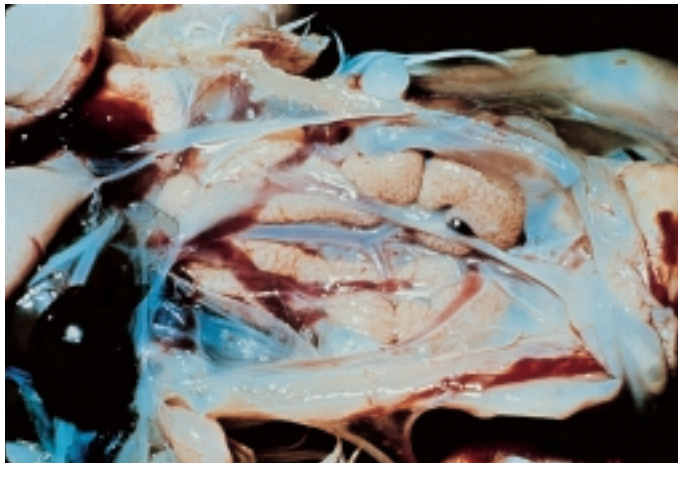
56. Acute infectious bursal disease showing chicken with copious white diarrhea. (Courtesy of Dr. P.D. Lukert, AAAP Slide Set)



57. Characteristic subcutaneous and intramuscular hemorrhages associated with infectious bursal disease. This condition should be differentiated from mycotoxicosis and anti-coagulant toxicity. (Courtesy of Dr. P.D. Lukert, AAAP Slide Set)



58. Enlarged bursa of Fabricius typical of acute infectious bursal disease infection. Note urate accumulation on vent plumage and enlarged kidneys.



59. Severe nephritis characterized by pallor and urate deposit may be associated with nephropathogenic strains of infectious bronchitis virus, infectious bursal disease or water deprivation.

10.0 CHICKEN ANEMIA

10.1 Etiology

A non-enveloped icosohedral DNA-virus, classified as a circovirus.

10.2 Occurrence and Economic Significance

The infection is widespread in broiler and replacement parent and laying-strain pullets.

Chicken infectious anemia virus (CAV) is immunosuppressive and is responsible for mortality of up to 10% in affected broiler flocks. The virus causes stunting and increased susceptibility to secondary viral and bacterial infections including dermatitis which results in downgrading. The pathogen is synergistic with IBDV, MDV and reticuloendotheliosis virus (REV) (a retrovirus).

10.3 Transmission

Both vertical and horizontal routes of infection occur under commercial conditions. Contaminated live vaccines prepared from infected embryos are thought to have been responsible for widespread dissemination of CA infection prior to recognition of the virus.

10.4 Clinical Signs

Morbidity is variable, with onset at 10 days. Primary CA mortality occurs during the age period 15-20 days. Affected chicks are pale and stunted. Gangrenous dermatitis of the extremities ("blue wing") is noted. Marked anemia may be observed with corresponding hematocrit values below 15%.

10.5 Pathology

Thymus and bone marrow atrophy and muscular hemorrhages are characteristic. Septicemia and gangrenous dermatitis occur in older birds, following secondary bacterial infection.

10.6 Diagnosis

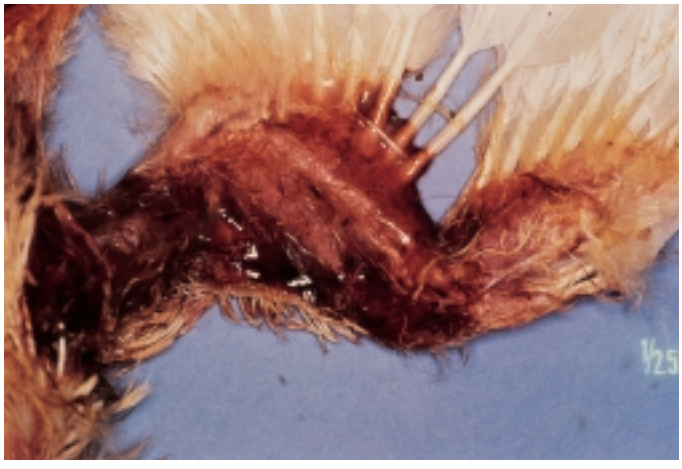
ELISA and VN serology can confirm infection of flocks. The PCR (polymerase chain reaction) assay can identify CAV T-cell and B-cell lymphoblastoid cell line tissue cultures can be used to isolate the agent in suitably equipped laboratories.

10.7 Prevention

Immunization of breeder flocks during the age period 12-15 weeks using an attenuated vaccine. Either vaccination or natural exposure will confer immunity to progeny through maternal antibody transfer.

Biosecurity procedures are required to prevent horizontal infection.

Appropriate management procedures can reduce the effects of primary CA infection. Control of respiratory infections and other immunosuppressive agents is essential to reduce the impact of CA.



60. Wing of 14-day old broiler showing edema, subcutaneous hemorrhage, and transudate due to chick anemia virus.

61. Pale bone marrow indicating immunosuppression associated with chick anemia virus or mycotoxicosis.

RESPIRATORY DISEASES

11.0 NEWCASTLE DISEASE

11.1 Etiology

Antigenically related strains of Avian paramyxovirus, type 1.

11.2 Occurrence and Economic Significance

Three categories of viral pathogenicity result in different clinical forms of the disease.

- Velogenic-viscerotropic virus (vvND) infection results in acute onset, highly lethal disease.
- Mesogenic virus causes acute, moderately lethal disease with nervous and respiratory signs.
- Lentogenic virus is responsible for mild respiratory infection.

Velogenic and mesogenic forms are exotic to the USA, Canada, the UK and other European countries but are widespread in Asia, Africa, and Latin America. The lentogenic form is encountered in most poultry-producing areas including the USA. Severe losses from mortality, depressed egg production and lowered feed conversion efficiency occur as a result of exposure to vvND.

The lentogenic form is responsible for erosive losses in broilers including lowered gain and feed conversion efficiency and elevated mortality and condemnation. The severity and financial impact depends on climatic and management stress and intercurrent exposure to pathogenic *E. coli* and other viral respiratory disease and immunosuppressive agents.

The cost and consequences (respiratory stress) of vaccination are significant, especially during winter and following immunosuppression. Disruption of trade and the cost of eradication of vvND in non-endemic countries imposes a significant burden on producers and the public sector after outbreaks.

11.3 Transmission

ND virus is highly contagious. Infection occurs either by the inhalation of virus in aerosol form or ingestion of contaminated feed or litter.

- Wind dispersal may occur over distances of 5 km.
- Direct and indirect contact with contaminated material (fomites) is associated with deficiencies in biosecurity.
- Companion birds, backyard flocks and gamefowl serve as reservoirs.

11.4 Clinical Signs

Velogenic Viscerotropic Newcastle Disease

This form is characterized by acute onset with up to 100% flock morbidity and rapidly ascending high mortality (20% in 2 days, 50% in 3 days, 80% in 5 days) accompanied by respiratory and nervous signs. In susceptible commercial egg production flocks and breeders, peracute cessation of production occurs with the presence of shell-less eggs due to premature oviposition. Exposure of immunized flocks results in variable decline in production.

Mesogenic

Variable to high morbidity is evident in an exposed flock which will show moderate mortality characterized by nervous and respiratory signs. An acute drop in egg production occurs in susceptible mature flocks with the presence of shell-less eggs.

Lentogenic

Acute onset with moderate to high morbidity. Mild to inapparent respiratory signs are noted but negligible mortality occurs in uncomplicated cases. Lentogenic ND may be responsible for asymptomatic drops in egg production in incompletely immunized commercial layer or breeder flocks.

11.5 Pathology

Velogenic

Prominent hemorrhages occur throughout the digestive tract especially in the mucosa of the proventriculus and gut-associated lymphoid tissue. Severe tracheitis and pulmonary congestion are evident in acute cases. These changes are not specific to vvND and may be observed with highly pathogenic strains of avian influenza and vvIBD.

Lentogenic

Mild conjunctivitis and tracheitis are observed. Recovered flocks show septicemia and airsacculitis due to secondary infection with *E. coli*.

11.6 Diagnosis

Isolation. The virus can be detected applying PCR technology to obtain a provisional diagnosis within a working day. Identification and characterization of the virus by a suitably equipped laboratory is the usual confirmatory procedure.

Retrospective serology (ELISA, hemagglutination inhibition and serum-virus neutralization) demonstrates the presence of antibodies which indicates exposure to ND virus and the titer (level) can differentiate between field infection and previous vaccination.

11.7 Prevention

Vaccination. Conventional programs:

- Lentogenic infection of broilers can be prevented by day old administration of aerosol or eye drop vaccine using Hitchner B1 with subsequent boosters in drinking water or by the aerosol route.
- Administration of a preparation comprising live virus with complementary antibody (Newplex®) by the *in ovo* route at 18-days of incubation is protective in countries where the vaccine is available.
- Recombinant pox and HVT-vector vaccines expressing the fusion (F) protein of NDV are available for either *in ovo* or subcutaneous vaccination.
- Lentogenic infection of breeders can be prevented by 10 day administration of Hitchner B1, by the aerosol or eye drop route.
- Subsequent vaccinations include 24 day, and 8 week Hitchner B1 or LaSota in non-chlorinated drinking water, followed by multivalent oil inactivated emulsion at 18-20 weeks. An optional 45 week multivalent oil inactivated emulsion may be administered to boost maternal antibody transfer, depending on antibody titer of the flock, risk of exposure, and other factors relating to the operation.
- In areas with a defective cold-chain the V-strain live thermostable mutant ND can be distributed to subsistence and backyard flocks.

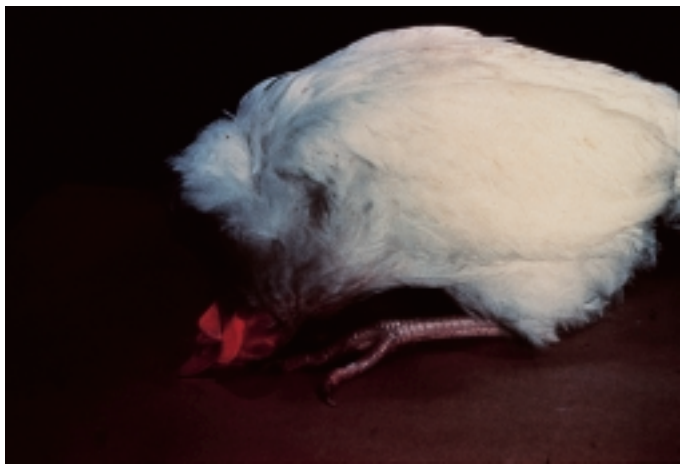
A variety of vaccination programs can be followed depending on the risk of infection, virulence of agent, management system, and economic factors.

In countries with endemic vvND, rigorous programs are implemented, incorporating day-old subcutaneous emulsion vaccine together with attenuated live vaccine by the eye-drop route. Hitchner or LaSota vaccine is administered to broilers by the aerosol route at 10 day intervals thereafter.

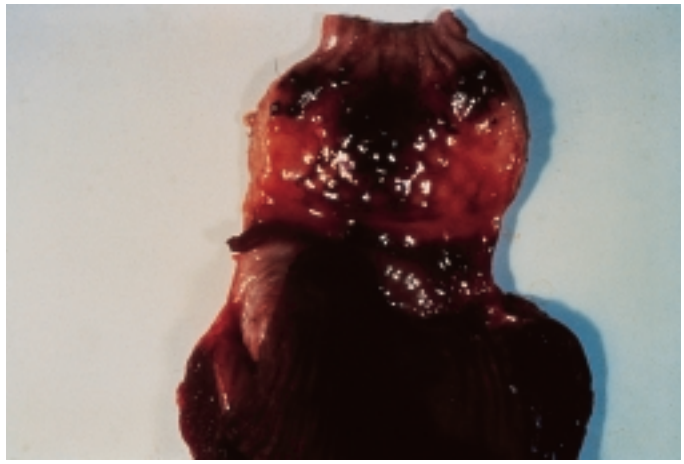
Breeders may be immunized with mesogenic-strain vaccines in some countries. This expedient is only justified if birds have previously received one or more live attenuated lentogenic vaccines.

62. The trachea yields virus for PCR analysis and for isolation and identification of viral and bacterial pathogens of the respiratory tract.

63. Broiler flock showing high morbidity and mortality due to velogenic Newcastle disease or HPAI. (Courtesy University of Pennsylvania)



64. Torticollis in an immature chicken infected with velogenic Newcastle disease.



65. Hemorrhage of the mucosa of the proventriculitis due to velogenic Newcastle disease. (Courtesy of the University of Georgia).

66. Acute onset of ocular discharge accompanied by high flock morbidity may denote infectious laryngotracheitis, infectious bronchitis, coryza, or mild avian influenza.

12.0 INFECTIOUS LARYNGOTRACHEITIS

Synonyms LT & ILT

12.1 Etiology

Gallid herpesvirus 1.

12.2 Occurrence and Economic Significance

Laryngotracheitis is distributed worldwide but is frequently regional in prevalence or seasonal in incidence.

Mild LT results in lowered growth rate and feed conversion efficiency and elevated mortality and condemnation in broilers. Decreased egg production occurs following exposure of mature susceptible flocks.

Moderate to severe strains of LT result in proportionately higher morbidity and mortality in both mature and rearing stock with losses approaching 50% with concurrent environmental stress and other infections.

12.3 Transmission

Direct contact with clinically affected chickens or recovered permanent carriers. Indirect contact through dust-laden vehicles, or contaminated personnel or equipment. Wind dispersal over 3 km has been documented.

12.4 Clinical Signs

The severity of LT is influenced by the strain of virus, immune status of the flock and environmental conditions. Mild to severe cases show acute onset with respiratory signs (snicking and gurgling), conjunctivitis and swollen heads. In severe cases, birds show expectoration of blood accompanied by cyanosis of the head due to dyspnoea.

12.5 Pathology

Hyperemia of the tracheal mucosa is present in most cases. Proportionally more severe lesions occur with pathogenic virus strains which produce severe hemorrhagic tracheitis with the presence of blood clots. Aggregations of desquamated epithelium and blood clots may obstruct the glottis resulting in asphyxiation.

12.6 Diagnosis

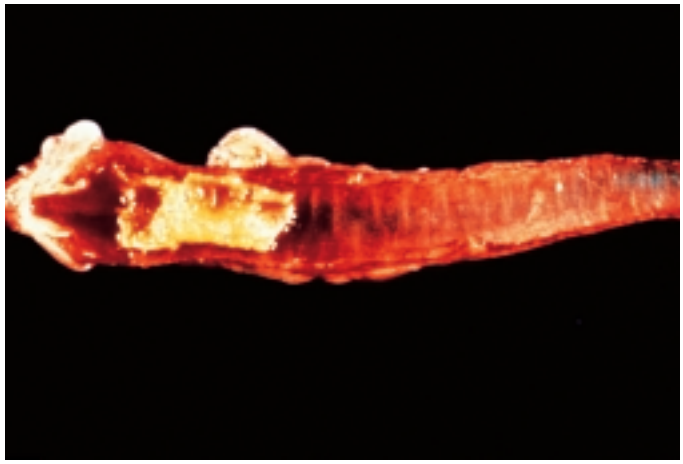
Histopathology usually reveals the presence of intranuclear inclusion bodies in the epithelium (cell lining) of the trachea. The fluorescent antibody technique may be used to demonstrate LT antigen in respiratory mucosa. The LT virus can be isolated using SPF embryos or tissue culture with identification applying immunofluorescence or serum-virus

neutralization. ELISA serology may be used to confirm infection by demonstrating a significant rise in antibody in paired serum samples obtained during the acute and recovery phases of infection.

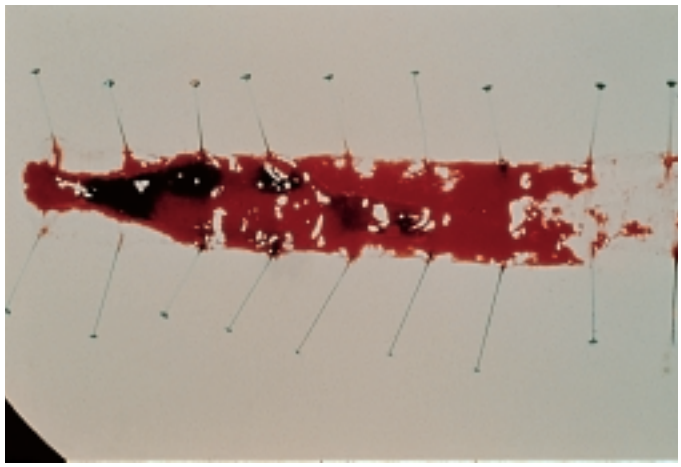
12.7 Prevention

Strict biosecurity measures are justified in endemic areas. Effective protection can be achieved using egg-embryo propagated vaccine administered in drinking water to broilers, immature breeders and commercial pullets at 14 - 20 days. The spray route is less effective especially with tissue-culture propagated vaccines. Commercial egg pullets and breeders are vaccinated at 6-10 weeks of age by administration of tissue-culture origin modified live virus which has a lower potential for reversion to virulence than chick-embryo origin vaccine virus. Since flocks immunized with chick embryo origin virus serve as permanent carriers of the vaccine virus, LT may be transmitted to susceptible flocks or to unaffected areas following movement of vaccinates. New vaccine candidates based on recombinant DNA technology should overcome the problem of reversion associated with live modified vaccines.

67. Broiler with swollen face and dyspnea due to laryngotracheitis. It is impossible to differentiate among the causes of respiratory diseases based on clinical observation only. Detailed laboratory evaluation with identification of pathogens is required to confirm a diagnosis. (Courtesy of the University of Georgia)



68. Caseous cast in the proximal trachea adjacent to the glottis is characteristic of laryngotracheitis but should be differentiated from diphtheritic pox infection and avian influenza. (Courtesy of Dr. D. N. Tripathy, AAAP slide set)



69. Severe hemorrhagic tracheitis characteristic of infectious laryngotracheitis.

70. Tracheitis which may be due to a range of viral agents including laryngotracheitis, infectious bronchitis, avian influenza Newcastle disease or adenovirus. Specific laboratory procedures are required to establish a diagnosis.

13.0 AVIAN INFLUENZA

13.1 Etiology

Diverse Type-A orthomyxoviruses characterized by hemagglutinating (HA) and neuraminidase (N) antigens occurring on the surface of the virus.

13.2 Occurrence and Economic Significance

Avian influenza is world-wide in distribution. Avian influenza viruses vary in their pathogenicity and their effects range from a mild respiratory disease (LPAI) to catastrophic losses associated with viscerotropic and pansystemic infection (HPAI or "fowl plague"). Sporadic outbreaks of HPAI result in severe losses in production, disruption in operations and high costs for control and prevention.

Avian influenza of low pathogenicity is an erosive disease reducing liveability and quality of either broilers or eggs and exacerbating secondary bacterial infection. Influenza adversely affects the financial return from flocks and a decline in quality of broiler carcasses or table-eggs following infection.

13.3 Transmission

Wild birds serve as reservoirs and transmit infection to subsistence flocks or commercial units which are operated with substandard biosecurity. Rapid multiplication of HPAI virus occurs in susceptible subsistence and commercial flocks, ultimately affecting all poultry operations in a region, unless appropriate controls are implemented. The virus is relatively resistant to environmental exposure and can infect birds placed in imperfectly decontaminated units housing a previously infected flock. Indirect infection can occur by moving flocks, equipment and personnel and by wind dispersal of virus-laden dust and feathers. Direct infection occurs following contact between infected carriers and susceptible flocks. This situation is common in countries with extensive distribution of live birds and where multi-age flocks are operated.

13.4 Clinical Signs

Highly pathogenic avian influenza (HPAI) results in an acute and precipitous decline in egg production with rapidly ascending mortality characterized by both respiratory and nervous signs.

Flock morbidity is apparent following exposure to AI virus but mortality is variable depending on the pathogenicity of the strain of AIV and intercurrent climatic and environmental conditions.

Mild strains (LPAI) result in low morbidity and mortality, and a decline in egg production. Elevated mortality follows secondary *E. coli* infection superimposed on flocks with a history of exposure to immunosuppressive infections (IBD, CA) and with concurrent respiratory pathogens (MG/MS, ND, ILT, IB).

13.5 Pathology

Highly pathogenic avian influenza is characterized by subcutaneous hemorrhages and edema of the head. Vesicles may be present on the comb and wattles. Hemorrhages are observed in the serosa of all viscera and in the mucosa and lymphoid structures of the intestinal and respiratory tracts.

Mild influenza results in tracheitis, pulmonary edema and if secondary bacterial infection occurs, airsacculitis is observed.

13.6 Diagnosis

Rapid presumptive diagnosis is based on solid-state antigen capture assay (Directigen®) and confirmed by RT-PCR assay. An alternative but more time-consuming approach involves isolation of a hemagglutinating virus from tissues using specific pathogen free embryos or avian-cell tissue culture systems. AI viruses are then identified and serotyped using serum-virus neutralization followed by more advanced sero-immunologic procedures conducted in suitably equipped laboratories.

The agar-gel immuno-diffusion test and the ELISA procedure are used to demonstrate group specific AI antibody in serum, denoting either exposure or vaccination.

13.7 Control in Areas Where Exotic HPAI is Diagnosed

Exotic outbreaks of HPAI are eradicated by implementing an intensive program comprising rapid diagnosis, slaughter and disposal of affected flocks, quarantine and concurrent surveillance with subsequent disposal of flocks demonstrating antibodies to AI. Restriction on movement of flocks and products from foci of infection should be imposed.

In areas where inadequate resources or extensive dissemination of infection precludes absolute eradication, flocks are immunized using autogenous inactivated vaccine or a recombinant vector product. Vaccination suppresses clinical occurrence of disease but the virus persists in the poultry population of the affected region, impeding exports.

Studies in industrialized nations have shown that strict biosecurity can limit dissemination of avian influenza virus among commercial farms

and within integrations. Preventing the spread of virus is extremely difficult in the context of industries in developing countries where feed is delivered in bags, and eggs, culled hens and live broilers produced by small-scale farmers are distributed through a network of dealers to regional markets.

Since 1997, the zootiotic potential of AI has been recognized. Human fatalities associated with the 2004 outbreak of the H5N1 strain of AI in Southeast Asia has created international pressure for extensive vaccination to suppress infection in regions and nations where HPAI has resisted traditional eradication programs and has become endemic.

13.8 Recent Outbreaks of H5N1 Avian Influenza in Asia

The 2003/2004 outbreak of H5N1 strain highly pathogenic avian influenza probably had its origins in China. Migratory waterfowl are considered to have introduced LPAI infection into free-living resident birds including sparrows and crows in the vicinity of feed mills and farms. These birds in turn transmitted virus to domestic subsistence chickens maintained under extensive management. Wild waterfowl may have also directly infected free-roaming domestic ducks and geese. Extensive movement of live domestic poultry to markets disseminated infection which eventually was introduced into large commercial operations. The H5N1 virus underwent mutation in large susceptible populations, resulting in a highly pathogenic agent.

Spread of infection from China to Vietnam, Cambodia, Laos and Thailand was associated with unrestricted cross-border movement of live poultry including fighting cocks. Failure to recognize the infection and to implement appropriate control measures including quarantines, depletion of affected and contact flocks and vaccination has resulted in the development of endemic HPAI status in most countries in Asia where the disease has emerged.

In Japan, Taiwan, Malaysia and Korea, limited outbreaks of H5N1 infection have been eradicated applying appropriate control measures for regions where the disease is exotic. China and Indonesia have adopted vaccination to suppress clinical outbreaks, recognizing this strategy to be the most cost-effective control measure.

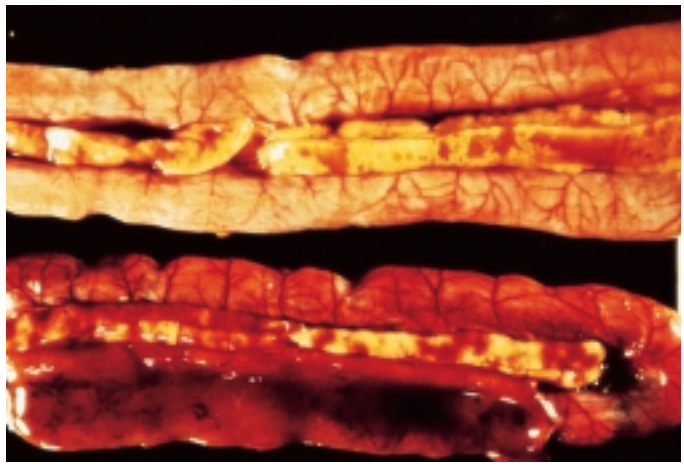
Asian outbreaks of H5N1 AI have been characterized by limited infection of humans following direct contact with diseased poultry. As of mid-2004 there have been no reports of direct human-to-human contact transmission. It is possible that a recombinant event between avian H5N1 and human influenza strains could occur in domestic swine or other animal species

yet to be identified in the chain of transmission. This could potentially result in a more pathogenic virus affecting humans, justifying vigorous suppression of infection in poultry populations and absolute separation of swine and poultry. Alternative strategies to control HPAI in countries with endemic infection should comprise detection and surveillance followed by depletion of infected flocks with fair compensation, quarantines and intensive vaccination appropriate to the circumstances.

In the event of extensive spread of HPAI in a region, administration of a HA-homologous inactivated vaccine will reduce the proportion of susceptible birds in a population. This will inhibit the multiplication and dissemination of HPAI infection. The extent and severity of H5N1 infection in Asia presumes persistence of virus in reservoir populations, requiring a commitment to long-term vaccination and intensified biosecurity for commercial farms.

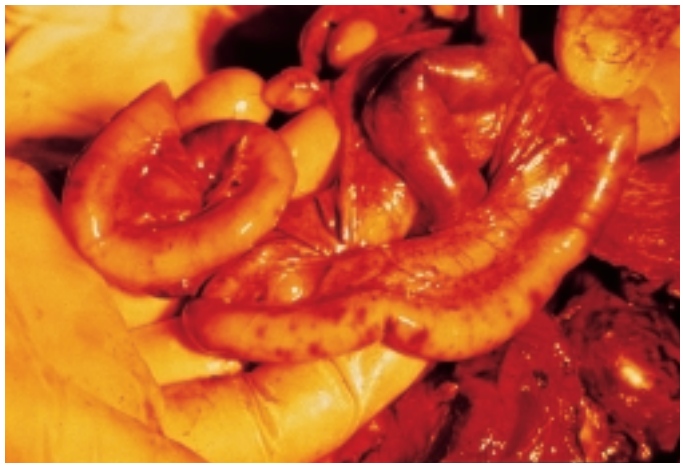
71. Subcutaneous hemorrhage characteristic of H5N2 strain of highly pathogenic avian influenza. (Courtesy of the University of Pennsylvania).

72. Cyanosis of the head observed in cases of H5N2 strain highly pathogenic avian influenza. (Courtesy of the University of Pennsylvania).



73. Severe hemorrhagic enteritis characteristic of highly pathogenic avian influenza or velogenic Newcastle disease. (Courtesy of the University of Pennsylvania).

74. Vesicle formation on the wattle of a bird infected with highly pathogenic influenza virus.



75. Visceral hemorrhages associated with velogenic viscerotropic Newcastle disease. Similar lesions are observed with highly pathogenic avian influenza. (Courtesy University of Pennsylvania)

14.0 INFECTIOUS BRONCHITIS

14.1 Etiology

Specific strains of an avian coronavirus.

14.2 Occurrence and Economic Significance

Infectious bronchitis (IB) occurs world wide and is responsible for depressed egg production and shell quality in susceptible commercial and breeder flocks. Infection of immature chickens causes a mild respiratory disease which may affect liveability and growth if exacerbated by adverse managemental, climatic stress or intercurrent mycoplasmosis.

14.3 Transmission

The virus can be transmitted from clinically affected birds to susceptible flocks either by direct contact or indirectly by fomites.

14.4 Clinical Signs

Moderate morbidity and low flock mortality associated with respiratory rales (gurgling and snicking) and ocular discharge. Mature flocks show reduced egg production with malformed shells.

14.5 Pathology

Hyperemia (red discoloration) of the trachea and accumulation of mucus in the nasal cavity. Chronic cases, complicated by secondary *E. coli* infection show airsacculitis.

14.6 Diagnosis

The diagnosis can be confirmed by immunofluorescence assay or isolation and identification of the causal virus using egg inoculation or tissue culture techniques. Where suitably equipped laboratory resources are available, RT-PCR is used to rapidly diagnose IB.

Retrospective diagnosis is possible by demonstrating a significant rise in circulating antibody in paired acute and recovery-phase sera applying ELISA or SN assay.

14.7 Prevention

Immature breeders and commercial layer flocks are routinely vaccinated with a mild attenuated product (H-120, Massachusetts, Connecticut strains or their combination) at 7 days in drinking water or by aerosol. The vaccination is repeated at 30-40 days. The initial live vaccine should always be administered to susceptible breeder and layer flocks before 12 weeks of age to avoid possible damage to the developing reproductive tract of

the pullet. Immunity in commercial layers can be boosted by administration of live attenuated vaccine either in drinking water or as a coarse spray during the production period. Potential breeder flocks receive inactivated IB vaccine as a booster, usually in the form of an injectable multivalent emulsion at the end of the rearing period and then at mid-cycle, as considered necessary, to maintain adequate maternal antibody transfer to progeny.

Broilers in endemic areas are vaccinated by aerosol at day-old or subsequently by coarse spray or in drinking water at a suitable time (10-20 days) depending on maternal antibody transfer or pattern of field challenge.

In many areas specific IB vaccines are required to prevent clinical problems attributed to variant strains.

15.0 MYCOPLASMOSIS

15. 1 Etiology

Mycoplasma gallisepticum and *M. synoviae* are the two significant species affecting commercial chickens.

15.2 Occurrence and Economic Significance

Chronic respiratory disease caused by *M. gallisepticum* and synovitis and airsacculitis due to *M. synoviae* infection, occur world-wide. These conditions are responsible for extensive losses in broiler operations especially where flocks are exposed to concurrent viral respiratory diseases and environmental stress. The economic impact of mycoplasmosis in broilers includes severely depressed growth rate and feed conversion efficiency, elevated mortality, and condemnation at processing. In commercial layers and breeders, liveability and egg production are depressed.

15.3 Transmission

Mycoplasmosis is transmitted by the vertical route from infected parent flocks to progeny.

Lateral transmission occurs by direct contact between clinically affected or recovered carriers and susceptible flocks.

Indirect infection occurs through contact with contaminated equipment, feed bags, and personnel.

Wild birds and rodents may transmit the disease to susceptible flocks.

Mycoplasmas do not survive outside the host for longer than 24 hours.

15.4 Clinical Signs

Mycoplasmosis is characterized by chronic respiratory signs including ocular discharge, tracheal rales (“gurgling and snicking”), markedly reduced growth rate and an increased susceptibility to intercurrent respiratory diseases. Chronic cases are emaciated and show purulent nasal discharge.

M. synoviae infection results in acute arthritis especially of the hock and stifle joints.

15.5 Pathology

Affected birds show congestion of the upper respiratory tract, mild tracheitis, and in chronic cases airsacculitis and colibacillosis. Acute cases of *M. synoviae* infection show serous arthritis. In advanced cases, sero-purulent exudate may be present in affected joints.

15.6 Diagnosis

Two to three weeks following infection, chickens demonstrate antibodies which can be detected using the serum plate agglutination test or the automated ELISA technique. These highly sensitive tests are used to screen flocks. The hemagglutination inhibition test is applied to confirm the provisional serologic diagnosis.

Mycoplasma spp can be isolated and identified by inoculating tracheal swabs or serous joint exudate from acute cases onto special selective media. This procedure may require up to 30 days and may be inconclusive due to technical problems including contamination. The polymerase chain reaction assay can be applied as a commercially available, sensitive and specific test procedure. Kits are available for *M. gallisepticum* and/or *M. synoviae* respectively.

15.7 Treatment

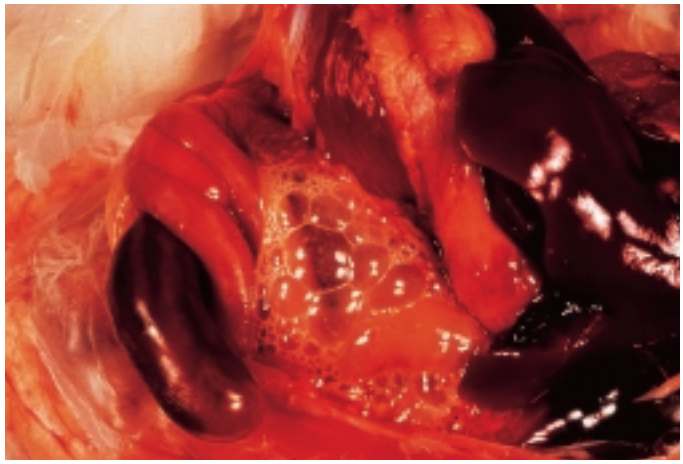
Clinical signs can be suppressed by administering tylosin or a fluoroquinolone compound in drinking water. Chicks derived from known infected parent flocks can be treated with a suitable antibiotic during the first 48 hours after placement and re-treated subsequently at 20 to 24 days of age for a 24 to 48 hour period. It is emphasized that treatment does not eliminate the carrier state in infected flocks but will suppress excretion of the organism in respiratory exudate and vertical transmission through eggs.

15.8 Prevention

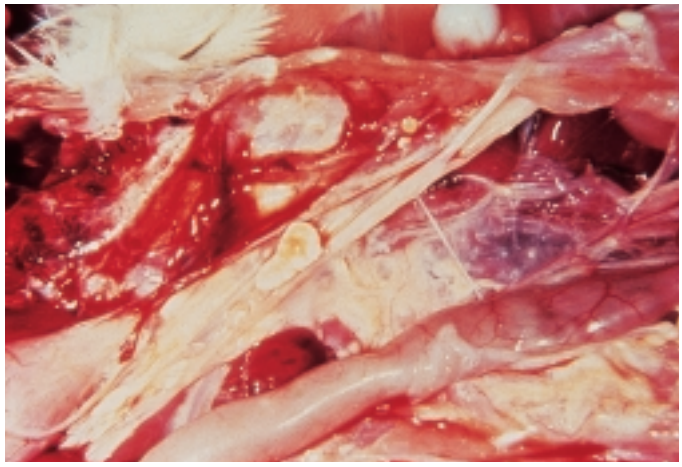
The world's primary breeders of broilers and commercial layers have eliminated mycoplasmosis. Infection of grandparent and parent level breeders occurs in developing industries due to deficiencies in biosecurity on farms operated by multipliers. It is essential to purchase parent and commercial stock from known mycoplasma-free breeder flocks.

Strict biosecurity will prevent lateral introduction of infection. Live *M. gallisepticum* vaccines are available for immature egg-production flocks which will be transferred to multi-age, infected layer farms. The live-attenuated F-strain vaccine administered in drinking water has been largely replaced in the USA by the milder TS-11 and 6/84 products. Inactivated vaccines administered as oil emulsions are available commercially but

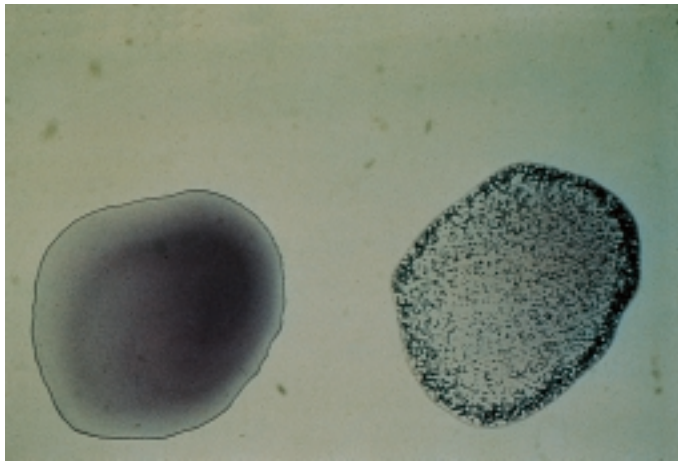
are of limited value. A pox-vectored recombinant Mg vaccine ("Vectormune") has recently been licenced for administration to immature egg-production pullets in the USA. It is noted that vaccination will suppress clinical signs of infection but will not eliminate the carrier state.



76. Acute foamy airsacculitis due to *Mycoplasma gallisepticum* infection.



77. Caseous airsacculitis associated with *Mycoplasma gallisepticum* infection.



78. Positive *Mycoplasma gallisepticum* Serum Plate Agglutination Test on right contrasted with negative test on left.

16.0 CORYZA

16.1 Etiology

Three *Haemophilus paragallinarum* serotypes designated A, B and C are recognized.

16.2 Occurrence

The disease is potentially encountered in any poultry-raising area but frequently occurs in specific regions or countries as a chronic or seasonal problem. Coryza results in decreased egg production in commercial multi-age laying and breeder operations.

16.3 Transmission

Infection follows direct contact with clinically affected or asymptomatic carriers or indirect contact with contaminated equipment or personnel. The pathogen does not remain viable outside the host for periods exceeding 24 hours.

16.4 Clinical Signs

Flock morbidity varies from 1 to 20%. Mortality is negligible in uncomplicated cases of coryza. Egg production in young commercial or breeder flocks is reduced following infection.

Clinically affected birds show unilateral or bilateral ocular discharge progressing to facial cellulitis and chronic sinusitis.

16.5 Pathology

Acute cases show severe conjunctivitis and inflammation of the periorbital fascia. Chronic cases show serous to caseous sinusitis.

16.6 Diagnosis

Haemophilus paragallinarum can be isolated from sinus swabs in acutely affected birds. Since the organism is susceptible to desiccation it is recommended that acutely infected live birds should be submitted to a diagnostic laboratory whenever possible. Alternatively, severed heads packed on ice can be forwarded to a laboratory. Isolation involves semi-aerobic culture on a blood agar medium streaked with *Staphylococcus* sp. incubated in a candle jar. The condition should be differentiated from pasteurellosis and viral infections including LPAI, and other respiratory agents.

16.7 Treatment

Immature birds can be treated with water-soluble sulfonamides. These

drugs should not be administered to mature flocks due to residues in eggs and the deleterious effect of sulfonamides on production and shell quality. Combinations of tetracyclines are frequently used to treat coryza by administration in water or injected directly by the intramuscular route. Compulsory or recommended withdrawal periods before marketing eggs should be followed after treatment of commercial flocks.

16.8 Prevention

Appropriate biosecurity measures will limit the possibility of introducing infection on to breeding and commercial egg production farms.

Immature flocks can be partly protected by administration of inactivated multivalent or homologous bacterins in aqueous suspension or oil emulsion. Two doses of inactivated vaccine should be administered by the subcutaneous or intramuscular route at four week intervals during the rearing period, as recommended by the manufacturer.

79. Ocular discharge and swelling of the nasal sinus associated with Coryza. Differential diagnosis includes *mycoplasmosis* or *laryngotracheitis*.

80. Accumulation of purulent material in the infraorbital sinus is characteristic of coryza.

17.0 ASPERGILLOSIS

synonym Mycotic Pneumonia

17.1 Etiology

Various fungi including *Aspergillus fumigatus*.

17.2 Occurrence and Economic Significance

The disease is world-wide in distribution but cases are more frequently diagnosed in tropical countries especially during warm and humid weather.

Severe outbreaks associated with hatchery contamination may result in up to 15% chick mortality during the first two weeks. Decreased growth rate and ascites complex are noted in affected survivors.

17.3 Transmission

Contamination of egg shells with *Aspergillus* spores results in colonization of the air cell. This is followed by subsequent infection of the respiratory tract of pipping embryos and hatching chicks. Horizontal transmission can occur in the hatchery or during handling and delivery. Chicks older than 48 hours are usually refractory to infection by inhalation of spores.

17.4 Clinical Signs

Morbidity may attain 10% of the flock with corresponding mortality during the first 3–12 days. Affected chicks are disinclined to move and show labored breathing with extension of the head, frequently accompanied by a whistling rale.

Mycotic encephalitis (infection of the brain) results in lateral recumbency, incoordination and coarse muscle tremors.

17.5 Pathology

Numerous 1mm diameter yellow to green nodules are observed in the lungs and air sacs and occasionally in other organs including the brain and eye.

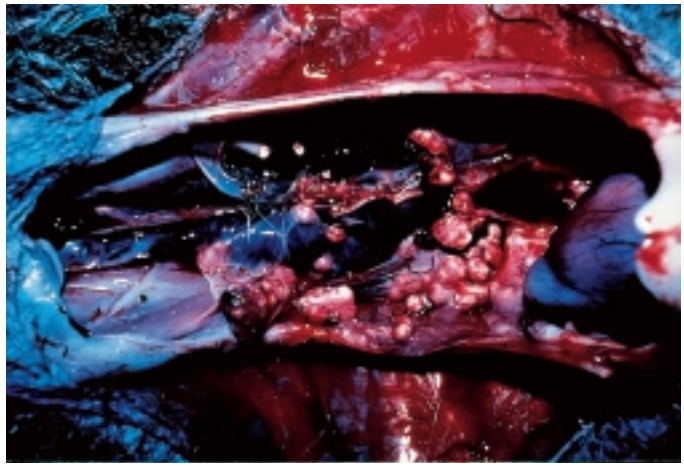
17.6 Diagnosis

Characteristic lesions are highly suggestive of aspergillosis. Confirmation of the diagnosis requires culture using an appropriate fungal medium (Sabouraud's dextrose agar). Histological examination of lungs will reveal characteristic hyphae.

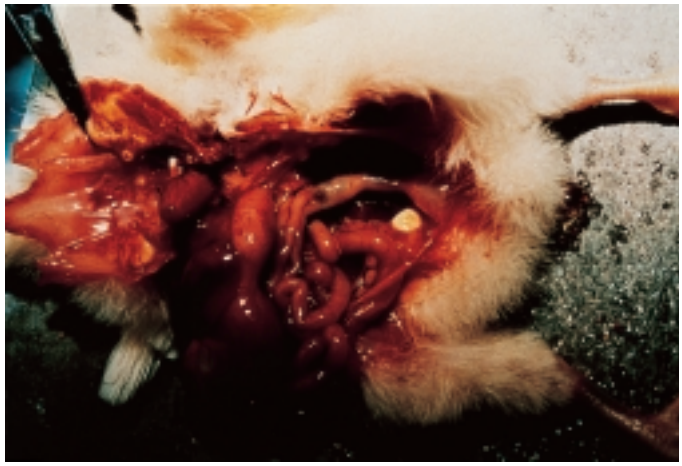
17.7 Prevention

Improving nest-box hygiene, increasing the frequency of collection of

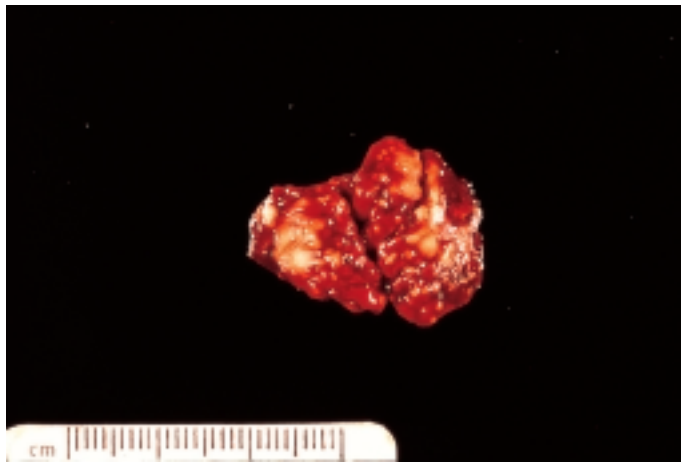
eggs to four times daily and where possible, substituting plastic nest pads for litter will reduce the prevalence of aspergillosis. Decontamination of eggs by formalin fumigation or application of a QAT or phenolic disinfectants is advised, but eggs should not be washed by immersion. Decontamination of setters, hatchers, and air ducts is recommended including the use of aerosol generators and medicated "candles." The efficacy of cleaning procedures can be monitored, using an appropriate microbiological detection procedure such as an air sampler or exposed petri-plates.



81. Multiple fungal granulomata typical of aspergillosis.



82. Diffuse airsacculitis of the thoracic air sacs and focal airsacculitis and peritonitis due to aspergillosis in a young chick.



83. Severe pulmonary aspergillosis showing numerous nodular foci and confluent lesions.

MULTIFACTORIAL CONDITIONS

18.0 SWOLLEN HEAD SYNDROME

19.0 SEPTICEMIA AND AIRSACCULITIS

18.0 SWOLLEN HEAD SYNDROME

18.1 Etiology

Swollen Head Syndrome (SAS) is a multifactorial condition involving the sequence of immune suppression (IBD, MD, CA) followed by exposure to a respiratory virus (IB, TRT, ND) and terminating in *E. coli* cellulitis of the subcutaneous tissues surrounding the eyes and of the head. Related metapneumoviruses including turkey rhinotracheitis virus (TRT) causing tracheitis and sinusitis in turkeys and a swollen head condition in broiler breeders are accepted as a precipitating agent of SHS in broilers. Outbreaks of facial cellulitis in chickens have been diagnosed in California without evidence of metapneumovirus infection. Environmental stress (low temperature and humidity, or high dust and ammonia levels) due to inadequate ventilation or climatic extremes exacerbate the prevalence and severity of SHS. Pathogenic *E. coli* strains, usually introduced through contaminated drinking water are responsible for the subcutaneous facial and occipital cellulitis which is characteristic of SHS.

18.2 Occurrence and Economic Significance

The condition occurs in southern Africa, the Middle East, Asia, and Latin America, especially in high-density broiler growing areas. Regionally, SHS often shows a seasonal (winter) occurrence. Losses due to primary mortality may attain 10% to 20% of the flock. Subsequent bacterial septicemia and airsacculitis which occur approximately 10 - 14 days after the onset of acute facial cellulitis may result in additional flock mortality of up to 20% of survivors of an acute outbreak.

18.3 Transmission

The immune suppressive and respiratory agents are transmitted by direct and indirect contact and usually are associated with defects in biosecurity, especially on multiple-age farms. Pathogenic *E. coli* or other opportunistic pathogens including *Ornithobacterium rhinotracheale* (ORT) can be introduced through contaminated water and litter.

18.4 Clinical Signs

Under commercial conditions SHS is characterized by acute onset of morbidity involving up to 10% of the flock in broilers aged 14 - 30 days.

Affected birds show ocular discharge and conjunctivitis progressing to periorbital swelling. Terminally, eyes are closed and enlargement of the head is a prominent sign in severely depressed or recumbent broilers.

18.5 Pathology

Subcutaneous accumulation of viscous sero-purulent exudate, which becomes caseous in chronic cases.

Acutely affected birds may show tracheal hyperaemia and pulmonary congestion. Chronic cases show caseous airsacculitis, perihepatitis and peritonitis. Bursal and thymic atrophy consistent with previous IBD or CAV infection, respectively, may be apparent.

18.6 Diagnosis

The obvious gross lesion comprising subcutaneous cellulitis of the head is highly suggestive of SHS. Attempts to isolate and identify primary viral pathogens and secondary bacterial pathogens should be carried out. Differential diagnoses include LPAI, ND, IB, coryza, pasteurellosis.

Serologic profiling of flocks is necessary to determine the pattern of maternal IBD antibody decay and the response to either vaccination or field challenge with a range of respiratory and immunosuppressive agents.

18.7 Treatment

Administration of water soluble antibiotics including fluoroquinolones will produce a transitory decline in flock mortality. Losses frequently resume after withdrawal of medication. Antibiotics should be used in accordance with the manufacturer's instructions and statutory restrictions relating to the withholding period before slaughter should be observed. Medication should be guided by anticipation of a positive benefit:cost ratio. Improper or prolonged use of antibiotics will result in emergence of drug-resistant *E. coli*. In small-scale operations acute cases can be salvaged by transfer to small pens where food and water are available and birds can be treated with parenteral antibiotic and protected from persecution by the remainder of the flock.

18.8 Prevention

Chlorination of drinking water to 2 ppm and installation of closed (nipple) drinking systems are recommended.

Alleviation of obvious managemental deficiencies and environmental stress factors will reduce the intensity of respiratory stress.

Appropriate vaccination programs are required to prevent immuno-suppressive and respiratory viral diseases.

Breeders can be vaccinated with commercial metapneumovirus vaccines. Attempts at immunization of broilers have not been successful. Control of IBD, ND, IB by vaccination and improved flock management and provision of chlorinated drinking water will reduce losses.

84. Swollen Head Syndrome in broiler showing cellulitis of the face and head with symblepharon (closed eyes). (Courtesy of Dr. S. Buys)

19.0 SEPTICEMIA AND AIRSACCULITIS

19.1 Etiology

Pathogenic strains of *E. coli*, superimposed on primary immunosuppressive and respiratory viral infections.

19.2 Occurrence and Economic Significance

Septicemia and airsacculitis resulting from *E. coli* infection are responsible for decreased growth rate and feed conversion efficiency, elevated flock mortality, downgrading and condemnation of carcasses in processing plants. Infection of commercial laying and breeding stock during the rearing period may adversely affect subsequent performance.

Flocks infected with vertically transmitted or acquired mycoplasmosis are extremely susceptible to *E. coli* airsacculitis.

Diets containing aflatoxins or free radicals evolved from peroxidation of lipids will lead to immunosuppression with increased susceptibility to *E. coli* infection.

19.3 Transmission

E. coli is introduced onto poultry farms through contaminated drinking water. High levels of infection occur following deficiencies in routine decontamination of housing, equipment and drinking systems. Immunosuppressive and respiratory viruses which precipitate infection are transmitted by direct and indirect contact especially on multi-age farms or where biosecurity is defective.

19.4 Clinical Signs

Flock morbidity of up to 10% occurs during the 10 – 40 day period accompanied by ascending mortality which may either plateau or decline but usually persists until depletion of the flock. Total losses may attain 50% in immunosuppressed broiler flocks subjected to environmental stress and previous exposure to viral respiratory pathogens and mycoplasmosis.

19.5 Pathology

Acute septicemia is characterized by pulmonary congestion, enlargement of the spleen and liver, and generalized venous congestion. Most birds which die of septicemia show perihepatitis, pericarditis and peritonitis. Bursal atrophy indicating previous exposure to IBDV is often observed. Birds surviving acute airsacculitis show stunted growth and develop a caseous exudate in the air sacs often accompanied by peritonitis, resulting in downgrading or condemnation at processing.

19.6 Diagnosis

Isolation, identification and serotyping of *E. coli* from heart blood, perivisceral exudate, and liver tissue. Evaluation of the epidemiology of immunosuppressive and respiratory infections by serology and isolation is recommended.

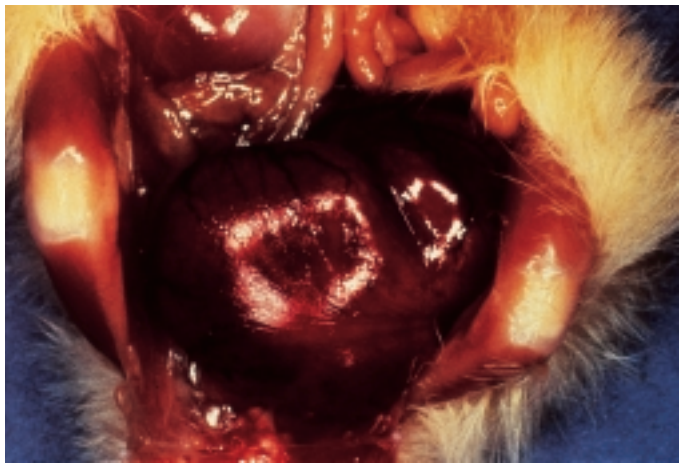
19.7 Treatment

Mortality can be suppressed by administration of water soluble furazolidone, sulfonamides, and fluoroquinolones where these drugs are permitted. It is necessary to perform antibiograms to ensure that selected drugs are effective. Medication should be administered in accordance with statutory restrictions concerning withdrawal and must comply with the manufacturer's recommendations.

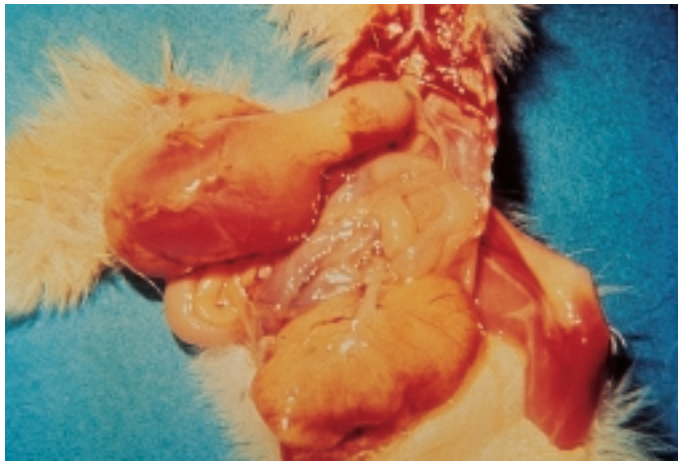
19.8 Prevention

Refer to 18.8, Swollen Head Syndrome.

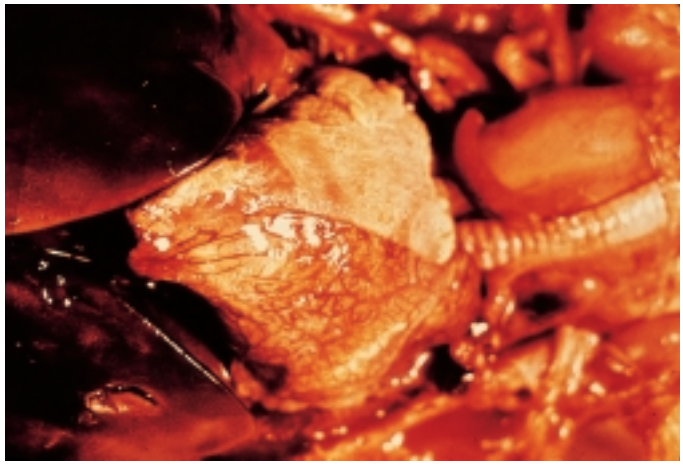
85. Hand cleaning of hatching eggs is an undesirable practice and leads to "exploders" in setters and hatchers and omphalitis in chicks due to bacterial contamination.



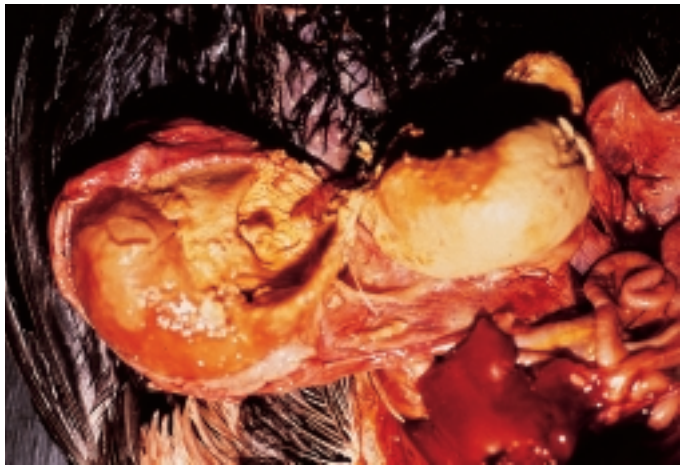
86. Grossly enlarged yolk-sac of a chick showing red-tinged contents associated with omphalitis.



87. Enlarged yellow yolk-sac, typical of omphalitis in a 3 day old chick.



88. Pericarditis which is usually associated with *E. coli* infection.



89. Chronic salpingitis occurs frequently in old breeder hens and is probably due to an ascending *E. coli* infection of the oviduct.

SYSTEMIC DISEASES

20.0 SALMONELLOSIS - PULLORUM DISEASE

21.0 SALMONELLOSIS - FOWL TYPHOID

22.0 SALMONELLOSIS - PARATYPHOID

23.0 PASTEURELLOSIS

24.0 SPIROCHETOSIS

25.0 AVIAN ENCEPHALOMYELITIS

26.0 ADENOVIRUS INFECTIONS
- RESPIRATORY INFECTION
- INCLUSION BODY HEPATITIS
- ANGARA DISEASE
- EGG DROP SYNDROME

27.0 RUNTING SYNDROME

20.0 SALMONELLOSIS-PULLORUM DISEASE

20.1 Etiology: *Salmonella pullorum*

20.2 Occurrence and Economic Significance

Pullorum disease (or "bacillary white diarrhea", BWD) is potentially world-wide in distribution but in practice is confined to non-commercial flocks in many countries. Infection results in high mortality in young chicks.

20.3 Transmission

Vertical transmission occurs by the transovarial route.

Horizontal transmission takes place by direct contact between clinically affected and recovered carriers and by indirect contact with contaminated equipment, housing, litter, and clothing of personnel. The pathogen can remain viable in soil for up to a year.

20.4 Clinical Appearance

Morbidity in affected batches of chicks often exceeds 40% with corresponding mortality commencing at hatch and extending through 21 days. Affected chicks are depressed and anorexic and tend to huddle under brooders. Birds may show copious white diarrhea and accumulation of fecal material adherent to the plumage surrounding the vent.

From 14 days of age onwards affected birds show stunting, poor feathering and frequently lameness due to arthritis.

20.5 Pathology

Chicks show enlargement of the spleen and liver. Omphalitis is often present.

Chronic cases show abscessation of the viscera (heart, internal serosa, lungs, liver) and chronic caseous typhlitis characterized by grey casts in the ceca.

20.6 Diagnosis

Isolation and identification of *S. pullorum* from liver, intestine or yolk sac using appropriate enrichment culture and standard microbiological techniques. Recovered carriers can be identified using the rapid whole blood plate agglutination test.

20.7 Treatment

None is recommended. Affected flocks should be depleted to eliminate chronic carriers.

20.8 Prevention

Breeding stock and chicks should be purchased from suppliers and hatcheries certified free of *S. pullorum* by a responsible government agency.

Breeder flocks can be monitored using the rapid whole-blood plate agglutination test.

Strict biosecurity should be enforced to prevent introduction of the pathogen from backyard flocks which serve as reservoirs. Rodent eradication is an important component of control.

21.0 SALMONELLOSIS-FOWL TYPHOID

21.1 Etiology: *Salmonella gallinarum*

21.2 Occurrence and Economic Significance

Potentially world-wide in distribution and frequently encountered in subsistence or semi-commercial flocks. The disease is responsible for serious economic losses in commercial units in organized poultry industries in endemic areas. Producers in Latin America and Asia experience mortality in both mature and immature flocks, loss of egg production and increased costs incurred by prevention and treatment.

21.3 Transmission

Vertical and lateral transmission occurs as for *S. pullorum*.

21.4 Clinical Signs

Acute onset of fowl typhoid occurs in susceptible flocks which are exposed to infection. Ascending morbidity and corresponding mortality may attain 5 - 10% within a week. No characteristic prodromal signs are noted. Diarrhea, depression and a decline in egg production are observed in mature flocks but these signs are not diagnostic.

21.5 Pathology

Gross enlargement of the spleen and liver are observed in affected cases.

Oophoritis (inflammation of the ovary) followed by ovarian regression is noted in mature stock. Peritonitis may be present in chronic cases.

21.6 Diagnosis

Isolation and identification of *S. gallinarium* is required to confirm the diagnosis. The rapid whole blood plate agglutination test will demonstrate antibodies approximately 2 weeks after infection and can be used to screen flocks for reactors.

21.7 Treatment

Treatment is inappropriate for breeding flocks, which should be depleted.

Commercial laying flocks may be salvaged under specific conditions by administering furazolidone or tetracycline in feed at 400 g/ton, for two weeks, where permitted. Eggs should not be marketed during medication or the subsequent withdrawal period.

21.8 Prevention

Appropriate biosecurity measures should be implemented as for *S. pullorum* infection, to prevent introduction of infection.

Administration of live 9R strain *S. gallinarum* vaccine during the rearing period will eliminate outbreaks of clinical disease. Bacterins are generally ineffective in preventing fowl typhoid.

22.0 SALMONELLOSIS-PARATYPHOID

22.1 Etiology

Salmonella spp other than *S. pullorum* and *S. gallinarum.*

22.2 Occurrence

A world-wide problem in integrated commercial-egg and broiler operations and also on small-scale farms.

22.3 Economic Significance

Some *Salmonella* spp including *S. enteritidis*, phage type 4, may result in high chick mortality consistent with S. Pullorum infection. Generally paratyphoid Salmonella spp will result in up to 3% losses during the first 14 days. Paratyphoid *Salmonella* spp are responsible for food-borne infection in consumers of eggs (S. Enteritidis) and poultry meat (*S. typhimurium, S. agona, S. heidelberg* and possibly up to 50 other frequently encountered serotypes).

22.4 Transmission

S. enteritidis is transmitted vertically by the trans-ovarial and trans-oviductal routes. Other *Salmonella* spp may be transmitted mechanically by fecal contamination of egg shells, or in cases of immunosuppressed flocks, infrequently by the transovarial route.

Feed containing contaminated ingredients of animal-origin is often responsible for introduction of paratyphoid salmonellosis into integrations or entire countries. The condition can be perpetuated by recycling contaminated by-product meal from infected broilers and commercial laying flocks.

Rodents and litter beetles serve as reservoirs of infection. Paratyphoid salmonellosis can be introduced by contaminated equipment, personnel and wild birds.

22.5 Clinical Signs

Elevated chick mortality and unevenness in brooding flocks are observed. No specific signs are associated with paratyphoid infection in mature flocks. Vertically transmitted *S. enteritidis* pt 4 infection resembles pullorum disease in chicks.

22.6 Pathology

Acute cases show enlargement of the spleen and liver and occasionally enteritis and peritonitis. Chicks may show omphalitis.

22.7 Diagnosis

Confirmation is based on isolation and identification of *Salmonella* spp from liver, spleen, intestine, or heart blood. Routine microbiological screening of liver/spleen/intestinal pools from post mortem submissions to laboratories is strongly recommended. Specific ELISA-based test kits are available for assaying for *S. enteritidis* antibody.

22.8 Treatment

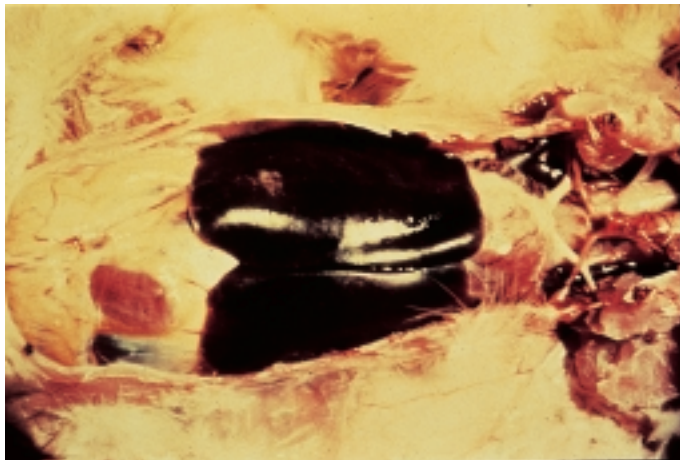
Furazolidone if allowed will suppress mortality but will not eliminate infection.

22.9 Prevention

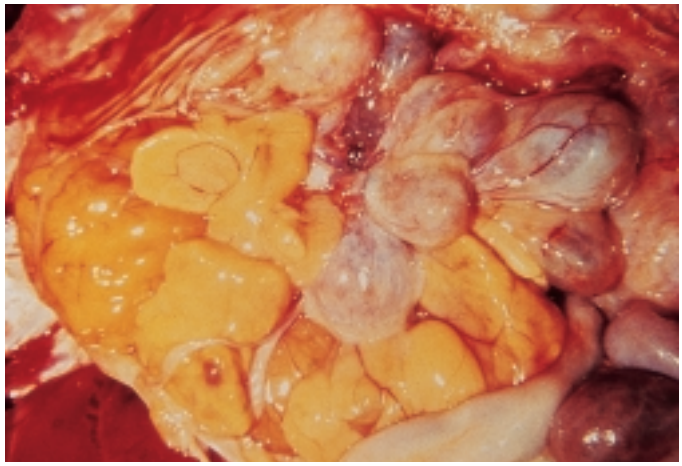
Reduction in prevalence in breeding flocks is possible (absolute elimination of S. Enteritidis and progressively *S. typhimurium* carriers) by implementing intensive programs of microbiological screening together with appropriate biosecurity procedures.

Salmonella enteritidis is controlled in commercial level stock using live, modified *S. typhimurium* vaccines alone or in combination with inactivated *S. enteritidis* emulsion boosters at point of lay.

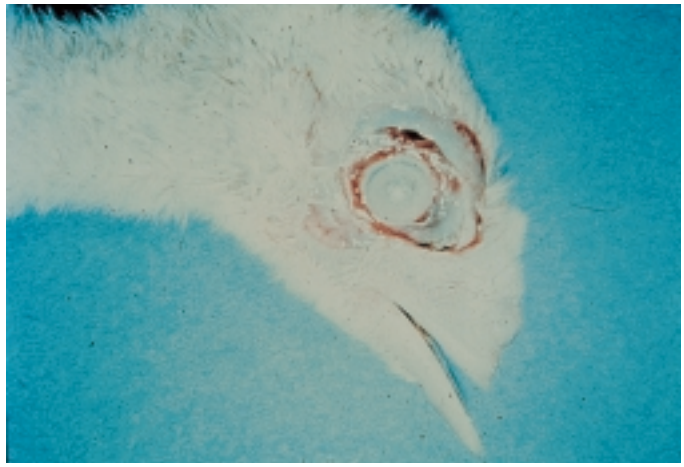
90. Accumulation of urate and excreta in the vicinity of the vent in chick with *Salmonella pullorum* infection, hence the designation “Bacillary White Diarrhea” (BWD) in the British commonwealth.



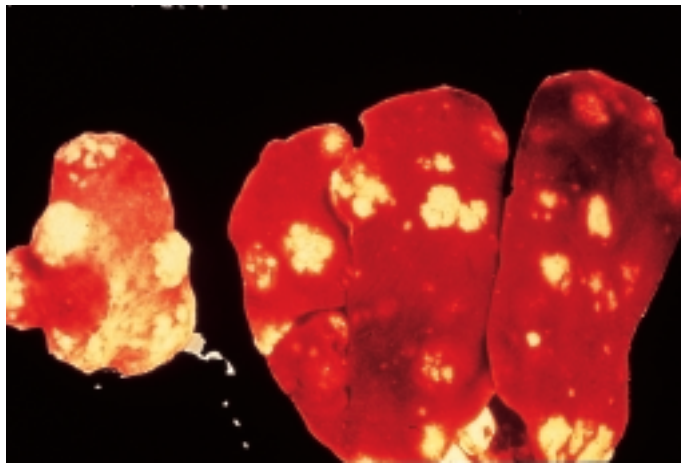
91. Enlargement of the liver with *Salmonella gallinarum* (Fowl Typhoid). This change is not characteristic of salmonellosis but may be observed with a number of bacterial infections including *E. coli* and *Pasteurella* spp. (Courtesy of the University of Georgia)



92. Regression of the ovary in a mature hen due to *Salmonella pullorum* infection.



93. Panophthalmitis in a turkey poult is characteristic of *Salmonella arizona* infection, but may occur with *S. pullorum* in chicks.



94. Multiple focal abcessation of the liver and heart following *Salmonella gallinarum* infection. These changes may also be seen with *S. pullorum.*

23.0 PASTEURELLOSIS

synonym Fowl Cholera

23.1 Etiology

Pasteurella multocida serotypes (including 1, 3, & 4) which vary in pathogenicity.

23.2 Occurrence and Economic Significance

World-wide in distribution, pasteurellosis is encountered as an endemic infection in many intensive poultry producing areas and frequently persists as an infection in specific integrations or farms. Mortality occurs in floor-housed replacement commercial laying and breeding stock and extends into mature flocks. Acute outbreaks associated with environmental or managemental stress, may result in depression in egg production. In breeders, reduced mating activity lowers fertility and depresses productivity of flocks as measured by the number of chicks produced by each hen placed.

23.3 Transmission

Infection occurs following direct contact between susceptible birds and clinically affected or recovered carriers. Environmental contamination, rodents, and wild birds are sources of indirect infection. Contaminated feed bags, equipment, and the clothing of personnel may introduce infection onto farms or into integrations. Intraflock transmission is enhanced by handling birds for vaccination and weighing and by open watering systems such as troughs and bell drinkers.

23.4 Clinical Signs

Morbidity and mortality rates depend on the pathogenicity of the strain and the susceptibility of the flock. Newly introduced infections may result in up to 10% mortality. Prodromal signs are not observed in peracute cases. Chronic infection may be recognized by enlargement of the wattles, lameness caused by arthritis and torticollis (twisted necks) due to otitis interna (infection of the inner ear).

23.5 Pathology

Acute cases show enlargement of the spleen and liver with punctate hemorrhages of the viscera including the heart. Subacute cases may show gray granulomatous foci in the liver. Caseous cellulitis of the wattles and seropurulent arthritis may be present in chronic cases.

23.6 Diagnosis

Laboratory examination is required to isolate and identify *P. multocida* from specimens of heart blood, liver, and spleen. In acute cases, characteristic bipolar organisms may be observed in Giemsa-stained smears of heart blood.

23.7 Treatment

Tetracycline incorporated into feed at a level of 200 - 400 g/ton or in water at 250 - 500 mg/l will suppress clinical signs and reduce mortality.

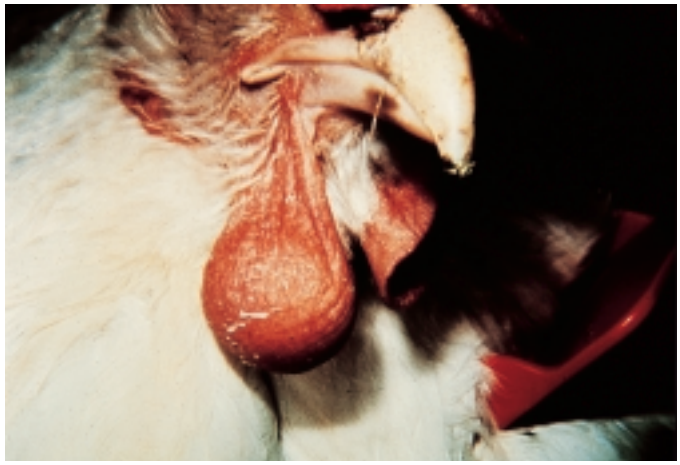
23.8 Prevention

Stringent biosecurity procedures are necessary to prevent introduction of infection. Eradication of rodents is critical to reducing the exposure of flocks to *P. multocida*.

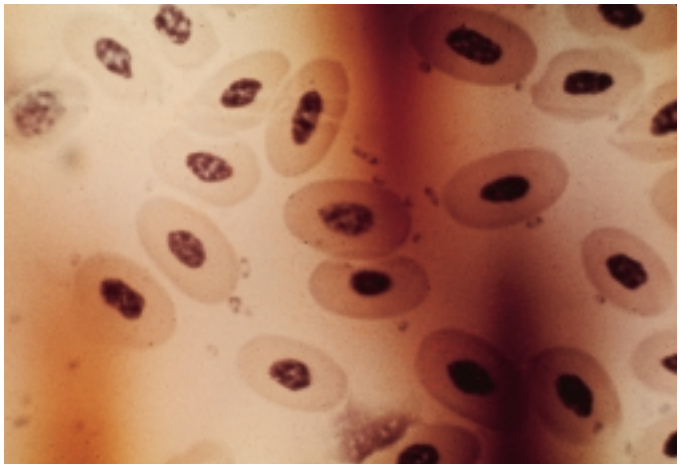
Immunization of flocks in endemic areas is recommended. Routine vaccination is essential on farms where previous cases have occurred.

Live-attenuated *P. multocida* vaccines (CU; PM-1; PM-9 strains) are administered by wing-web stab twice during the rearing period, at approximately 10 and 14 weeks of age. Breeder males or flocks subjected to environmental stress may be vaccinated with the relatively milder PM-1 and PM-9 strains in place of the CU strain to avoid adverse vaccine reaction. Antibiotics should not be administered one week before and one week after administration of a live attenuated vaccine.

Inactivated vaccines can be used to protect flocks if an undesirable reaction to a live vaccine occurs. It is emphasized that for effective control of pasteurellosis inactivated bacterins must be homologous with the endemic strains of *P. multocida*. In some areas or integrations, autogenous, inactivated vaccines are required.



95. Enlargement of the wattle due to *Pasteurella multocida* infection.



96. Characteristic bipolar staining *Pasteurella multocida* organisms in a Giemsa-stained blood smear.

97. Torticollis in a hen which might be due to infection of the inner ear by *E. coli* or *Pasteurella multocida*. This change is also observed in flocks exposed to velogenic Newcastle disease.

24.0 SPIROCHETOSIS

24.1 Etiology

A spirochete, *Borrelia anserina*.

24.2 Occurrence and Economic Significance

Spirochetosis is widespread in tropical countries due to the prevalence of the soft-shell tick vector *Argas* spp. The condition is responsible for sporadic losses in subsistence flocks and small scale commercial units.

24.3 Transmission

Ticks of the genus *Argus* are most frequently implicated in transmission of spirochetosis. Studies have confirmed that mites including *Dermanyssus* spp and *Culex* spp mosquitoes may also be involved in transmission.

24.4 Clinical Signs

Young birds are apparently more susceptible than older stock. Acutely affected birds show depression with cyanosis (blue discoloration) of the head. Mortality may attain 30% of the flock. In sub-acute and chronic cases, birds show paresis (weakness) terminating in paralysis and death.

24.5 Pathology

A grossly enlarged spleen with mottling due to subcapsular hemorrhage is the predominant lesion. Focal necrotic hepatitis may also be present.

24.6 Diagnosis

Demonstration of the organisms in Giemsa-stained blood smears. The pathogen can be propagated from a spleen homogenate injected into the yolk sac of embryonated eggs at the 6th day of incubation.

24.7 Treatment

Oxytetracycline by injection (1-2 mg/kg body weight) or chlortetracycline in drinking water are effective.

24.8 Prevention

Eradication of vectors and dusting birds at frequent intervals with 5% carbamate (Sevin®) powder. In some countries, locally prepared vaccines are available but vary in efficacy.

25.0 AVIAN ENCEPHALOMYELITIS

synonyms Epidemic tremor, AE

25.1 Etiology: A picornavirus

25.2 Occurrence and Economic Significance

Avian encephalomyelitis (AE) occurs world-wide, resulting in an asymptomatic egg production decline in commercial layers and breeders and elevated mortality in vertically infected batches of chicks.

25.3 Transmission

Vertical transmission occurs by transviral passage of virus from viremic hens to their progeny. Lateral spread from birds which shed AE virus in feces results in direct and indirect infection of susceptible flocks.

25.4 Clinical Signs

Mortality occurs in chicks aged 3 - 20 days. Morbidity varies according to transmission rate but seldom exceeds 5% under commercial conditions, especially in areas where AE vaccination is an accepted practice. Affected chicks show depression progressing to prostration in lateral recumbency. Terminally, chicks demonstrate fine muscular tremors of the head, neck, and feet. Recovered birds may show lenticular opacity (cataracts).

Infection of susceptible breeder or commercial egg flocks results in an asymptomatic decline in egg production.

25.5 Pathology

No gross lesions are observed. This is important in differentiating AE from encephalomalacia.

25.6 Diagnosis

Histological examination of brain and spinal cord tissue reveals perivascular cuffing and degeneration of neurons. Lymphoid aggregations are observed in the proventriculus, pancreas and other organs.

Epidemic tremor virus can be isolated from brain tissue by inoculation of embryonated SPF eggs. Commercial ELISA test kits are available to monitor the antibody titer of flocks to determine susceptibility or following vaccination or challenge.

25.7 Prevention

A live-attenuated vaccine can be administered to replacement commercial laying and breeding stock during the 10 - 14 week period.

Vaccines are available for administration in drinking water or as a combination product with avian pox using the intradermal, wing-web stab route.

Care should be taken to avoid introducing AE vaccine onto multi-age breeder farms. Lateral spread of vaccine virus will result in a drop in egg production in susceptible hens and mild outbreaks of epidemic tremor in progeny. For the same reason, pullets should not be vaccinated after 12 weeks of age since intestinal shedding of vaccine virus can occur for at least 4 weeks following vaccination.

98. Incoordination and lateral recumbency in a chick which may be due to avian encephalomyelitis, avitaminosis A, nutritional encephalomalacia or arenavirus infection (Spiking Mortality Syndrome). Detailed laboratory examination including histopathology is required to obtain an accurate diagnosis. (Courtesy AAAP)

26.0 ADENOVIRAL INFECTIONS

26.1 Etiology

Type 1 adenovirus:	Different strains produce specific conditions including mild respiratory infections, inclusion body hepatitis and hydropericardium-hepatitis syndrome (HHS) in chickens.
Type 2 adenovirus:	Hemorrhagic enteritis of turkeys.
Type 3 adenovirus:	Egg-drop syndrome in chickens.

26.2 Occurrence and Economic Significance

Egg drop syndrome in mature flocks occurs in Europe, Asia, Africa, and Latin America. This infection has not been diagnosed in the USA and Canada.

Inclusion body hepatitis and mild adenoviral respiratory infection may occur in all areas where commercial chickens are reared.

Hydropericardium-Hepatitis syndrome is responsible for severe losses in India (Lychee disease), Pakistan (Angara disease), and Latin America and is emerging as a significant restraint to production in areas where intercurrent problems of immunosuppression due to vvIBD and vvMD occur.

26.3 Transmission

All adenoviruses are potentially transmitted by the vertical route. Under commercial conditions, direct transmission occurs from fecal shedders to susceptible flocks. Indirect infection is possible on contaminated personnel, equipment and housing.

Vaccines produced using infected, non-SPF embryos have been implicated in outbreaks of EDS and HHS in Asia.

26.4 Adenoviral Respiratory Infection

26.4.1 Clinical Signs

Mild respiratory signs (moist rales) occur in a few birds in a flock following infection. The condition is characterized by slow onset and spread within and among flocks.

26.4.2 Pathology

Mild inflammation of the tracheal mucosa is observed following primary infection.

26.4.3 Diagnosis

Isolation and identification of the causal agent using SPF eggs.

26.4.4 Prevention

No specific vaccine is available.

26.5 Inclusion Body Hepatitis

26.5.1 Clinical Signs

Moderate (5 - 20%) morbidity and slightly elevated mortality occur in broilers and replacement rearing and breeding flocks aged 2 - 6 weeks. In the presence of intercurrent immunosuppressive viruses, morbidity and mortality may exceed 10%. No specific clinical signs are demonstrated. Affected birds are depressed, with ruffled plumage and are disinclined to move.

26.5.2 Lesions

Enlargement of the liver, with mottling due to petechial hemorrhages under the capsule interspersed with areas of necrosis. Nephrosis, characterized by enlargement of the kidneys and urate retention may be observed in chronic cases. The more severe HHS infection is characterized by hydropericardium and focal hepatic necrosis.

26.5.3 Diagnosis

Histological examination of affected livers often shows intranuclear inclusions. Adenovirus can be isolated from the respiratory and digestive tracts by inoculating embryonated SPF eggs.

26.5.4 Prevention

All-in-all-out placement programs and appropriate biosecurity procedures are recommended. No specific vaccine is available. Effective control of Marek's disease and IBD together with early exposure of breeders to mild prevalent adenoviral strains have contributed to a decline in the severity of adenoviral inclusion body hepatitis in broiler flocks in the USA. Specific inactivated oil-emulsion HHS vaccines are available for administration in endemic areas.

26.6 Egg Drop Syndrome

26.6.1 Clinical Signs

Other than acute drop in egg production in mature flocks, no specific clinical abnormalities can be detected following direct or indirect exposure to EDS virus. Eggs produced by brown and tinted-shelled strains show

lack of pigment and shells have a “chalky” appearance. Failure to attain peak production may be associated with activation of latent infection or lateral introduction of infection at onset of sexual maturity.

26.6.2 Lesions

Examination of sacrificed, clinically unaffected birds will show regression of the ovary. Histological changes in the oviduct occur following infection.

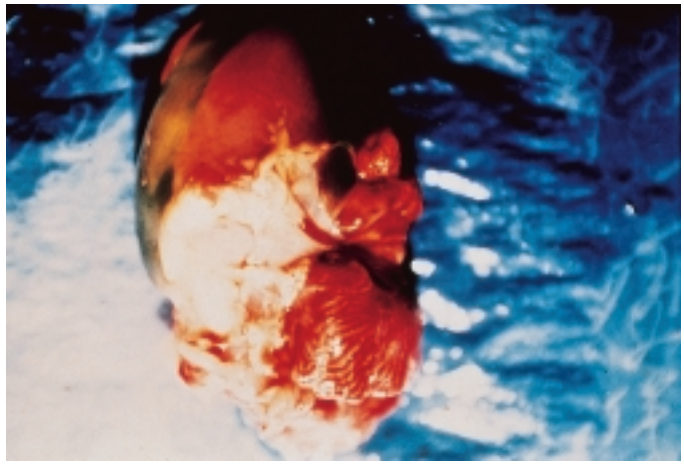
26.6.3 Diagnosis

Isolation of the causal virus in duck eggs or on liver cell tissue culture.

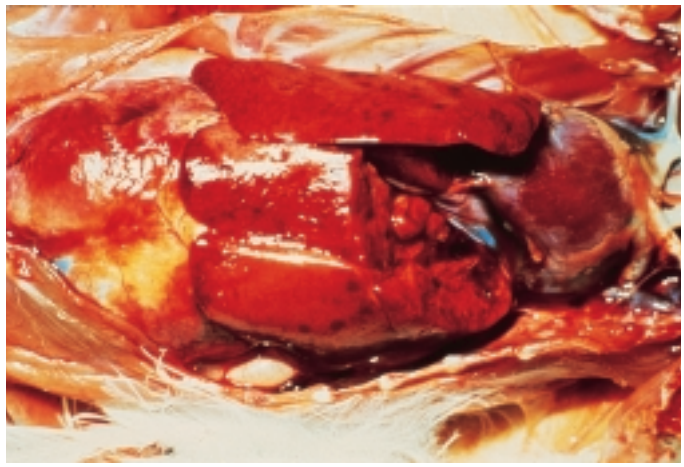
Confirmation of a diagnosis is based on demonstrating a rise in antibody titer in paired sera applying VN or ELISA procedures.

26.6.4 Prevention

Vaccination of immature breeding and laying flocks with an inactivated oil-emulsion vaccine is recommended before onset of production.



99. Characteristic hydropericardium associated with Angara disease.



100. Characteristic mottled appearance of liver (subcapsular hemorrhage) associated with Adenoviral hepatitis infections including HHS.

27.0 RUNTING SYNDROME

synonyms Malabsorption; Infectious Stunting Syndrome

27.1 Etiology:

The specific causal agents responsible for Stunting Syndrome have not been identified, although most poultry health professionals accept that specific reovirus strains (including 1733) are responsible for the condition, possibly in association with as yet unidentified viruses or anaerobic bacteria in the intestinal tract. It is noted that the condition can be reproduced by infecting specific-pathogen free chicks with intestinal homogenates from affected birds. It is not possible to reproduce the typical stunting syndrome by administering reovirus isolates from field cases, suggesting a multi-factorial etiology.

27.2 Occurrence and Economic Significance

The condition has been diagnosed in most broiler-raising areas and is responsible for decreased growth rate and elevated mortality in broiler chicks usually derived from young parent flocks. The severity and prevalence of the condition generally abates within 1 to 2 years after initial appearance in an area.

27.3 Transmission

The reovirus presumed to be responsible for the condition is transmitted by the vertical route from infected hens to progeny in addition to lateral infection among broiler chicks. It is known that reovirus infection can remain latent in replacement pullets during the rearing stage with viremia appearing at the onset of production and persisting for approximately 4 to 6 weeks thereafter.

27.4 Clinical Signs

Affected chicks show decreased growth rate which is evident by the 5th to 7th day of brooding. Feather abnormalities are obvious in affected chicks and include breakage of the shafts of the primary feathers of the wings and persistence of yellow down on the head, through 30 days of age. Abnormal wing feathering gives rise to the term “helicopter disease” since the abnormal feathers resemble rotor blades. By 4 weeks of age, affected chicks which may comprise up to 25% of the flock may weigh only 250 g and are less than half the size of normal pen mates. Examination of the orange-colored, loose droppings from infected birds shows the presence of undigested grain particles. A high proportion of affected birds show a disinclination to walk due to a rickets-like syndrome characterized by osteopenia. In extreme cases fracture of the proximal epiphysis of the

femur occurs. Affected birds show decreased pigmentation of the skin which is evident on the shanks and beak.

27.5 Pathology

Stunting syndrome attributed to malabsorption is characterized by a wide range abnormalities including:

- A rickets-like syndrome involving decreased skeletal density, beading of the ribs, osteopenia resulting in fracture of the proximal epiphysis of the femur (incorrectly termed “femoral head necrosis”).

- Pale colored skin and feather abnormalities. These changes which are observed clinically are confirmed on post-mortem examination. Despite obvious malabsorption, enteritis is not a primary lesion although affected birds may be concurrently infected with coccidiosis or may undergo secondary bacterial infection. Enlargement of the pancreas may be noted.

There are no characteristic gross or histological lesions associated with the stunting syndrome.

27.6 Diagnosis

A diagnosis of stunting syndrome is based on the history of young parent flocks producing affected chicks and the appearance of up to 20% stunting in a flock with typical clinical presentation in affected birds ranging in age from 7 to 35 days. A wide range of pathogens can be isolated from affected flocks. Since the range of etiologic agents have not been identified, there is no definitive laboratory diagnostic procedure.

27.7 Treatment

There is no specific treatment for stunting syndrome. Affected chicks can be gathered from the flocks at approximately 10 days of age and placed in a common pen where they can be provided with feed and water and protected from competition from normal pen-mates. Stunted chicks will grow slowly, and can be salvaged for live-bird sale or processing as low-weight birds. Isolation of affected chicks may reduce the probability of lateral transmission of virus.

Evaluation of dietary formulations is advised to ensure nutritional adequacy. The following components should be considered:

- levels of methionine and lysine should attain or exceed breed specifications

- selenium level should range from 0.1 to 0.3 ppm, with at least half of this dietary contribution in the form of selenomethionine.
- vitamin E supplementation should conform to NRC levels for stressed flocks (20 IU/kg)

All supplementary fats and animal byproducts should be stabilized with 300-600 ppm ethoxyquin or an equivalent compound.

27.8 Prevention

It is recommended that parent-level pullets receive an attenuated reoviral arthritis vaccine at 7 days followed by a multivalent live vaccine during the 14 to 30 day period consistent with the immunization program for the area. Inactivated reoviral vaccines administered during the late rearing period should contain antigenic components which are known to be protective against the reovirus strains (1733) considered responsible for stunting-malabsorption syndrome.

ENTERIC DISEASES

28.0 COCCIDIOSIS

28.1 Etiology

Various *Eimeria* spp which parasitize specific portions of the intestinal tract of chickens.

28.2 Occurrence and Economic Significance

Coccidiosis occurs world-wide and is a major cause of mortality and suboptimal growth and feed conversion efficiency in immature flocks unless appropriate preventive measures are implemented. The cost of anticoccidial feed additives and treatment is estimated to exceed $400 million annually in all poultry producing areas of the world.

28.3 Transmission

The sporulated oocyst is the infective stage of the life-cycle. Infected, recovered chickens shed oocysts representing a problem in multi-age operations. Oocysts can be transmitted mechanically on the clothing and footwear of personnel, contaminated equipment, or in some cases, by wind spreading poultry-house dust and litter over short distances.

Factors contributing to outbreaks of clinical coccidiosis include:-

- litter moisture content exceeding 30% due to ingress of rain or leaking waterers.
- immunosuppression (Marek's disease, IBD, mycotoxins)
- suboptimal inclusion of anticoccidials or incomplete distribution (poor mixing) in feed.
- environmental and managemental stress such as overstocking, inoperative feeding systems, inadequate ventilation.

28.4 Clinical Signs

Coccidiosis is generally acute in onset and is characterized by depression, ruffled plumage, and diarrhea. Birds infected with E. tenella show pallor of the comb and wattles and blood-stained cecal droppings.

28.5 Lesions

E. acervulina and *E. mivati:* 1-2mm areas of hemorrhage interspersed with white foci visible through the serosa of the distal duodenum and proximal jejunum.

E. necatrix: severe distention of the mid-jejunum with hemorrhages in the mucosa and red-stained fluid in the lumen.

E. maxima: distention of the mid-jejunum with hemorrhages in the mucosa.

E. tenella: hemorrhagic typhlitis (inflammation of the cecum).

E. brunetti: hemorrhages of the mucosa of the distal jejunum and colon. Fibrinonecrotic enteritis may occur in chronic cases.

28.6 Diagnosis

Gross lesions of *E. tenella*, *E. necatrix* and *E. brunetti* are diagnostic.

Microscopic examination of intestinal and cecal scrapings reveals oocysts.

To confirm a diagnosis in a commercial operation the following specimens should be submitted to a laboratory:

- Intestine from a sacrificed, affected bird preserved in 5% potassium dichromate for culture and identification of Eimeria sp.
- Intestine showing gross lesions in 10% formalin for histological examination.
- Representative feed samples for anticoccidial assay.
- Litter samples for oocyst counts.

28.7 Treatment

Administration of amprolium solution, 0.024% of the active ingredient in drinking water for 3 - 5 days. Sulfonamides (sulfamethazines , 0.1% for 2 days, 0.05% for 4 days or commercial combinations of sulfa drugs) in drinking water.

Administration of water dispersable vitamin A and K supplements may enhance recovery.

28.8 Prevention

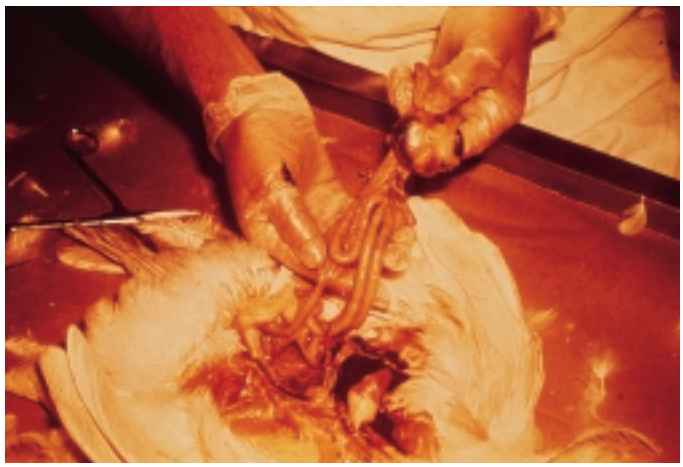
Management procedures which limit saturation of litter include:

- Appropriate installation and management of watering systems. Nipple drinkers reduce spillage of water onto litter compared to bell and trough drinkers.
- Acceptable ventilation rate.
- Maintaining recommended stocking density.
- Providing adequate feeding space.
- Inclusion of anticoccidials in diets at recommended levels will prevent clinical infection.
- Chemical and ionophoric anticoccidials for broilers in shuttle programs.
- Synthetic coccidiostats for breeders and floor-reared commercial egg-production flocks which allow the development of premunity.

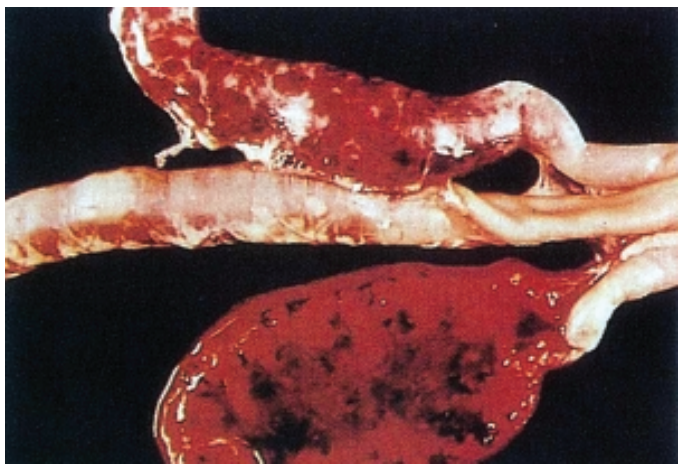
Anticoccidial vaccines are appropriate for replacement breeding stock and roasters. This approach is cost-effective but requires experienced and

diligent management and monitoring especially if the vaccine is applied over feed. Intraocular administration by spray or the insertion of a gelatine cylinder impregnated with oocysts in the chick delivery box contributes to an even distribution of vaccine through the flock.

Future control measures will include *in ovo* vaccination of broiler embryos with a highly purified oocyst suspension (Inovocox®). Administration of a vaccine (Coxabic®) derived from gametocytes to replacement pullets has been shown to confer immunity to progeny, obviating the need for anticoccidials.

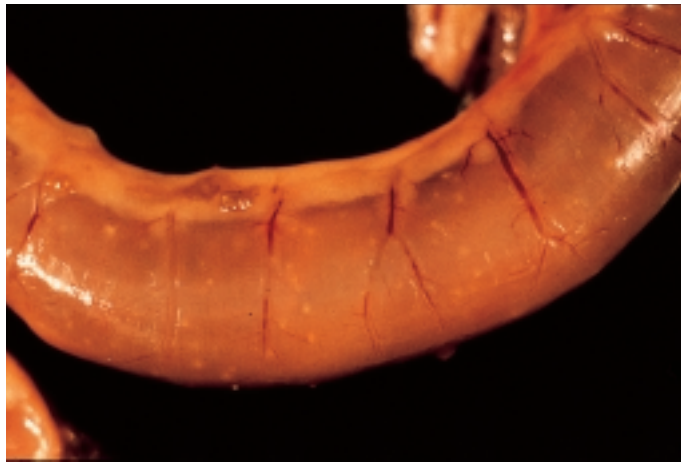


101. Careful examination of intestinal tracts from at least 5 clinically normal sacrificed birds per flock is necessary to monitor for coccidiosis.

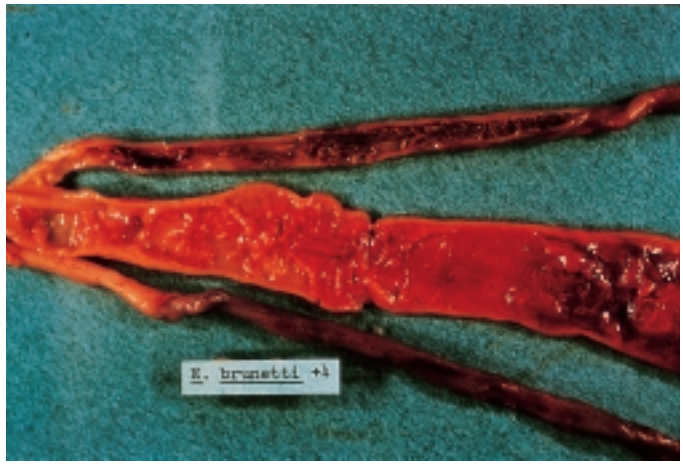


102. Severe *Eimeria tenella* infection showing hemorrhagic ceca. (Courtesy AAAP)

103. Obvious distention and hemorrhage of the ileum (middle of the intestinal tract) due to infection with *Eimeria necatrix*. These lesions should be distinguished from the changes caused by *Eimeria maxima*. This condition is associated with concurrent *Clostridium perfringens enterotoxemia* leading to necrotic enteritis.



104. Lesions of either *Eimeria acervulina* or *E. mivati* infection showing white foci visible through the serosa of the duodenum.



105. Severe Eimeria brunetti lesion in the intestine, proximal and distal to the cecal bifurcation. (Courtesy of Dr. W. Malcolm Reid, AAAP slide set).

29.0 CLOSTRIDIAL ENTEROTOXEMIA

29.1 Etiology

Clostridium perfringens is the principal pathogen responsible for necrotic enteritis (NE) although the condition is multi-factorial in origin and is usually preceded by mild intestinal coccidiosis. Necrotic enteritis is often initiated by an alteration in the feeding program (commencing skip-a-day feeding of replacement breeder pullets or accidental starvation) environmental stress, overstocking, withdrawing anti-coccidial growth-stimulating feed additives, vaccination, movement or weighing of flocks, or saturation of litter. *Clostridium botulinum* is responsible for botulism, an enterotoxemia resulting in progressive paralysis.

29.2 Occurrence and Economic Significance

Both NE and botulism can occur world-wide, in areas where chickens are reared on litter. Successive flocks in some regions show frequent or persistent outbreaks possibly due to high levels of *Clostridium* spp in soil or the presence of drug-resistant strains. The economic significance of clostridial enterotoxemia varies, but erosive losses of up to 4% can occur in broiler and immature breeder flocks due to direct mortality or concurrent infection with systemic bacteria.

29.3 Transmission

Clostridium perfringens and *C. botulinum* are ubiquitous soil contaminants. Pathogenic strains of *C. perfringens* may be introduced onto farms by deficiencies in hygiene and biosecurity. Ingestion of the vegetative form of the organisms invariably result in colonization of the intestinal tract.

29.4 Clinical Signs

Mortality is acute with no specific prodromal or clinical signs. Chickens affected with NE develop rigor ("stiffness") within 1 hour of death. Chickens with botulism show ascending paresis and then paralysis extending craniad from the legs, impairing locomotion. Terminally affected birds are in sternal recumbency with flaccid necks, ruffled plumage and extension of the nictitating membrane over the cornea.

29.5 Pathology

The mucosa of the intestine shows changes ranging from focal hyperemia (redness) to mucosal ulceration. In extreme cases NE is characterized by extensive pseudomembranous enteritis which resembles a coarse yellow coating. Focal hepatic necrosis may be observed. There are no characteristic lesions associated with botulism.

29.6 Diagnosis

Histological examination of affected mucosa will demonstrate the presence of characteristic clostridial organisms applying Gram stain. *Cl. perfringens* can be isolated and identified by a suitably equipped laboratory using anaerobic culture.

Botulinum toxin can be identified in the blood of severely affected broilers by injecting 0.5 ml of serum into susceptible mice using the intraperitoneal route. Toxin results in paralysis and death within 24 hours.

29.7 Treatment

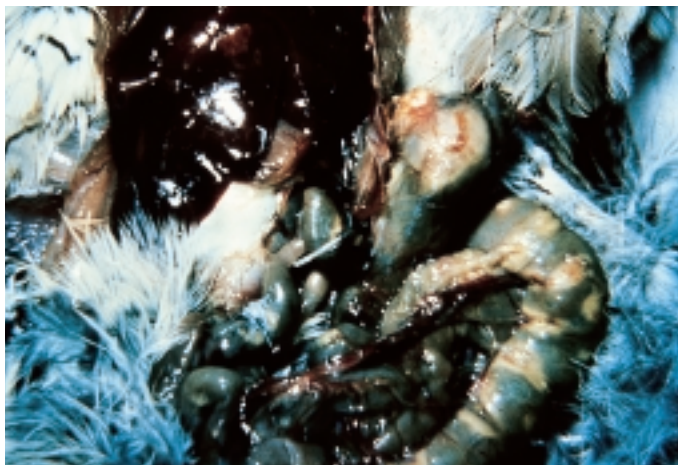
Administration of flocks with water soluble zinc bacitracin, lincomycin , virginamycin or penicillin for 72 hours reduces morbidity and mortality. Losses may reoccur following withdrawal of treatment.

29.8 Prevention

Addition of zinc bacitracin, lincomycin, or virginamycin to feed at levels approved by local regulatory authorities will suppress the clinical occurrence of clostridial infections.

Botulism and NE can be prevented by the management procedures recommended to prevent coccidiosis.

106. Mucosa of the intestine showing lesions characteristic of necrotic enteritis.



107. Focal ulceration characteristic of clostridial necrotic enteritis.

30.0 ENDOPARASITES

30.1 Capillariasis

Infection of the crop (*Capillaria contorta*) and the intestine (*Capillaria obsignata*) will result in severe emaciation and mortality in both immature and producing flocks. In floor-housed breeders and commercial layers reduction in egg production occurs. Ingluvitis (inflammation of the crop) is associated with *C. contorta*. Mucosal thickening and focal enteritis occurs with *C. obsignata*. Parasitism can be diagnosed by examination of mucosal scrapings and fecal flotation, which reveal characteristic bi-operculated ova.

Treatment

Fenbendazole in feed or levamisole or ivermectin (where permitted) in drinking water.

30.2 Ascaridiasis

Ascaridia galli occurs in the jejunum and *Heterakis gallinarum* in the cecum. Extensive *A. galli* infection may reduce egg production in floor-housed breeders and commercial layers. Death may occur due to intestinal obstruction in birds which are immunosuppressed or are affected by an intercurrent debilitating condition.

Other nematodes which may be encountered in subsistence or small-scale flocks include:

Oxyspirum mansoni – a 1.5 cm ( ) nematode beneath the nictitating membrane of the eye.

Syngamus trachea – a 2 cm ( ) nematode in the trachea.

Tetrameres americana – a 3 mm ( ) spherical nematode beneath the mucosa of the proventriculus.

Cheilospirura hamulosa – a 2.5 cm nematode beneath the mucosa (koilin layer) of the ventriculus.

Treatment

Piperazine, levamisole, or ivermectin (where permitted) in drinking water.

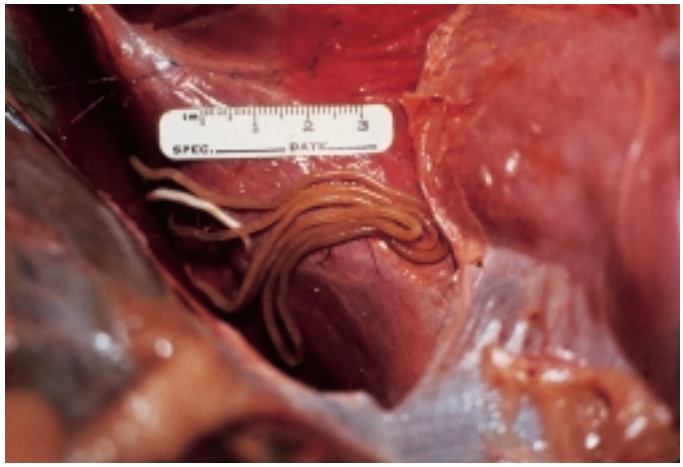
30.3 Cestodiasis

Numerous cestode species may occur in the intestinal tract and can be diagnosed at postmortem or by examination of feces. Cestodiasis results in emaciation in mature flocks, especially if severe infestation is exacerbated by malnutrition or immunosuppression.

The most commonly diagnosed cestodes include:
Davainea proglottina - a 4 mm cestode located in the duodenum.
Choanotaenia infundibulum - a 25 cm cestode located in the distal duodenum and jejunum.
Raillietina tetragona - a 25 cm cestode located in the distal jejunum.
Raillietina echinobothridia - a 30 cm cestode of the jejunum resulting in nodular granulomas and catarrhal enteritis.

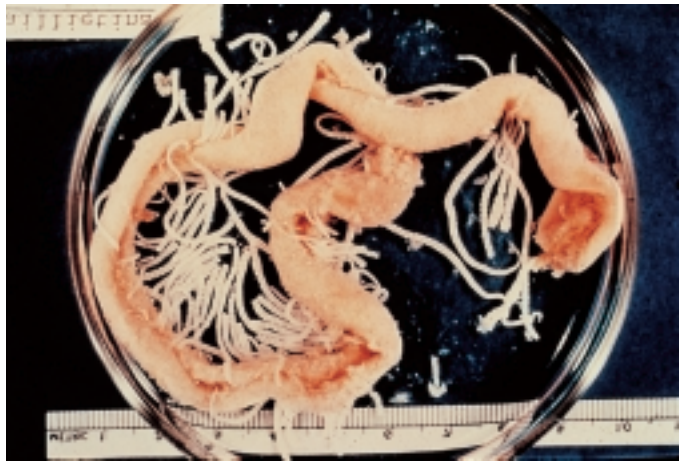
Treatment
Niclosamide in feed.

108. Microscopic examination of intestinal scrapings is necessary to determine the presence of endoparasites.

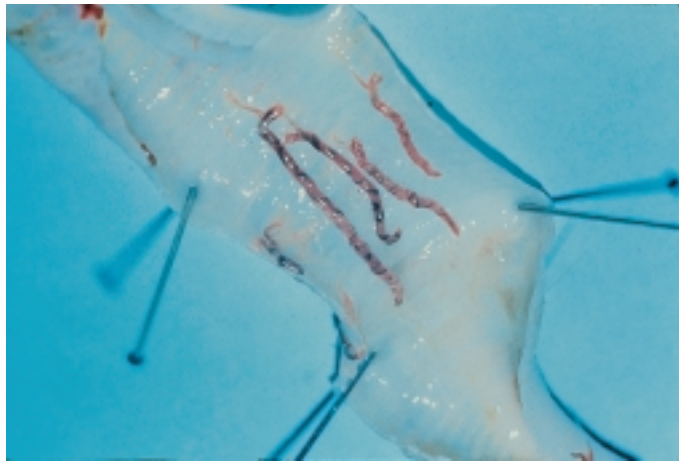


109. Filarid parasites are infrequently observed in the body cavity of backyard fowl and exotic birds at post mortem examination.

110. Severe ascaridiasis may cause obstruction of the intestinal tract.



111. Severe cestode infestation (*Raillietina* sp).



112. Presence of Syngamus trachea parasites in the trachea of an infected backyard fowl.

LOCOMOTORY ABNORMALITIES

31.0 SKELETAL DEFORMITIES AND ARTHRITIS

31.1 NUTRITIONAL ETIOLOGY

31.2 INFECTIOUS ETIOLOGY
- MYCOPLASMOSIS
- REOVIRAL ARTHRITIS
- STAPHYLOCOCCAL ARTHRITIS

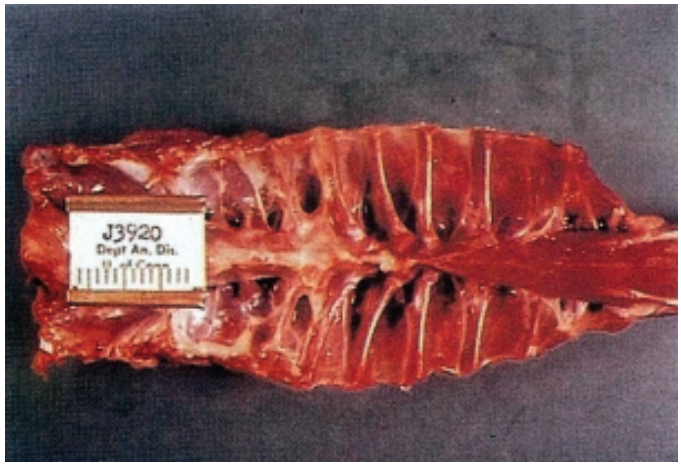
31.3 PODODERMATITIS

31.4 DEVELOPMENTAL ETIOLOGY
- TWISTED LEGS
- ROTATED TIBIA

31.0 SKELETAL DEFORMITIES AND ARTHRITIS

31.1 Nutritional Etiology

- Calcium or phosphorus deficiency or an imbalance in these nutrients will result in rickets in immature birds or osteomalacia in mature breeders and commercial egg-production flocks.
- Vitamin D3 (cholecalciferol) deficiency results in rickets in immature flocks housed in controlled environment units.
- Thiamine (vitamin B1) deficiency results in an abnormal gait progressing to recumbency and paralysis with hyperextension of the neck.
- Riboflavin (vitamin B2) deficiency results in a deformity of the feet termed “curled toe paralysis”.
- Pyridoxine (vitamin B6) deficiency results in abnormal gait and convulsions. This condition can also occur following administration of toxic levels of nitrofurans to immature flocks.
- Manganese deficiency results in chondrodystrophy. This occurs in growing chicks due to decreased formation of bone below the growth plates of the tibiotarsus and tarsometatarsus. Mildly affected chickens show stunting and enlargement of the hock joint with reduction in the length of the leg bones. The lesion progresses to severe deformation of the hock joint culminating in displacement of the gastrocnemius (Achilles) tendon (perosis). Chondrodystrophy is characterized by a high prevalence in the flock, bilateral involvement of the hock joints and reduction in length of the long bones. Confirmation of the diagnosis requires analysis of feed to determine manganese content. Dietary level should range from 80 to 120 ppm for optimal growth.
- Choline and pyridoxine deficiency may result in bilateral enlargement of hock joints, sometimes with displacement of the gastrocnemius tendon.
- Chondrodystrophy should be differentiated from valgus-varus abnormality of genetic origin.



113. Lesions of rickets showing, bending of the tibiotarsus, distortion of the ribs and enlargement of the costo-condral junctions. (Courtesy of Dr. C. Riddell, AAAP slide set)

31.2 Infectious Etiology

31.2.1 Mycoplasmosis

Mycoplasma synoviae results in serous arthritis. The condition can be diagnosed by serology (ELISA or plate agglutination test for flock screening, and hemagglutination inhibition for confirmation) or identifying the organism in synovial fluid by culture or by applying PCR technology.

For information on diagnosis, treatment and control of *M. synoviae*, refer to the mycoplasmosis section under respiratory diseases.

31.2.2 Reoviral Arthritis

Specific serotypes (S1133 and WVU 2937) are responsible for arthritis and tenosynovitis.

Occurrence and Economic Significance

Viral arthritis occurs world-wide and is responsible for losses in both commercial broilers and replacement breeding stock.

Transmission

Vertical transmission is the principal route of infection. Lateral spread from infected carrier chick occurs, especially during the first 48 hours after hatch. Under commercial conditions, indirect transmission is possible through contaminated equipment and from improperly cleaned housing.

Clinical Signs

Affected birds aged approximately 30 days onwards show an increasing prevalence of lameness characterized by unilateral or bilateral arthritis of

the hock and stifle joints. Up to 10% of the flock may be affected and lame birds generally die from dehydration or persecution.

Pathology

Both serous arthritis and teno-synovitis are observed, especially involving the hock and gastrocnemius tendon. The extent of the lesion progresses from acute inflammation to chronic fibrosis. Rupture of the tendon occurs in severe cases, and may be responsible for losses in hens at onset of sexual maturity.

Diagnosis

The causal organism can be isolated from synovial (joint and tendon) fluid.

Histopathology of affected tissues shows lymphocytic infiltration and reticular cell proliferation. Chronic cases show fibrosis of the tendon sheaths which can be palpated in birds which have recovered from the infection. Retrospective serological diagnosis is based on ELISA assay of serum from acute phase and recovered flocks.

Prevention

Breeding stock and broilers should be obtained from parent flocks immunized against reoviral arthritis.

High levels of biosecurity including operation of all-in all-out placement programs will prevent lateral transmission.

Breeding flocks should be immunized at approximately 4-5 days of age with a mild attenuated reoviral arthritis vaccine administered by the parental route. This should be followed by a second dose of less-attenuated vaccine at approximately 30 to 40 days of age. High levels of parental immunity for breeders is stimulated by administration of an inactivated emulsion vaccine prior to point of lay, and if required, at mid-cycle.

31.2.3 Staphylococcal Arthritis

Etiology

Staphylococcus aureus is a primary pathogen but often occurs as an opportunist, following reoviral arthritis or mycoplasmosis.

Occurrence and Economic Significance

The condition occurs world-wide but is a problem in specific broiler breeder flocks subject to immune suppression or previous exposure to reoviral arthritis.

Transmission
Introduction of *S. aureus* occurs through skin abrasions and lacerations and is often a consequence of parenteral vaccination with contaminated needles or contact with improperly cleaned equipment used for weighing.

Clinical Appearance
Increasing incidence of lameness occurs from 8 to 16 weeks and losses may attain 20% of the flock. Affected birds are characterized by unilateral or bilateral hock arthritis and occasionally pododermatitis. Affected birds invariably die of dehydration and persecution.

Pathology
Affected joints yield purulent, viscous yellow or green exudate.

Diagnosis
Diagnosis is based on isolation and identification of *S. aureus*. Concurrent diagnostic procedure should include serology and culture to determine the possibility of previous exposure to reovirus or *M. synoviae*.

Treatment
Parental administration of antibiotics to infected birds is only palliative, and is not cost effective. Culling of crippled birds is recommended.

Prevention
Purchase of Ms-free stock and effective vaccination against reoviral arthritis will reduce the occurrence *S. aureus* as a secondary infection. Immunosuppressive disease should be controlled by appropriate biosecurity including isolation of flocks and vaccination of breeders.

Acceptable biosecurity procedures including decontamination of houses and equipment are recommended. Needles used to vaccinate flocks should be sterilized before use and after administering vaccine to 50 consecutive birds, to reduce the probability of direct inoculation with *S. aureus*.

31.3 Pododermatitis

Etiology
This condition which occurs in mature broiler breeders, is multi-factorial in etiology. Predisposing factors include wet litter and obesity.

Occurrence and Economic Significance
Pododermatitis is responsible for lameness resulting in death and lowered fertility in broiler breeder flocks, from 40 weeks of age onwards.

Clinical Appearance

Males are more frequently affected than females, presumably due to their relatively higher weight. Pododermatitis is characterized by gross enlargement of the foot pad. The initial lesion is a superficial erosion which progresses to ulceration of the plantar skin with abscessation and chronic fibrosis of underlying synovial structures.

Pathology

The lesion is characterized histologically by fibrosis.

Prevention

Dry litter, free of ammonia reduces damage to foot pads. Implementing post-peak feed restriction is necessary to restrict the weight of both males and females within limits recommended by the supplier of breeding stock. The incidence of pododermatitis has decreased in the USA since the introduction of separate male and female feeding systems for broiler breeders.

31.3.1 Alleviation of Locomotory Problems Through Nutrition

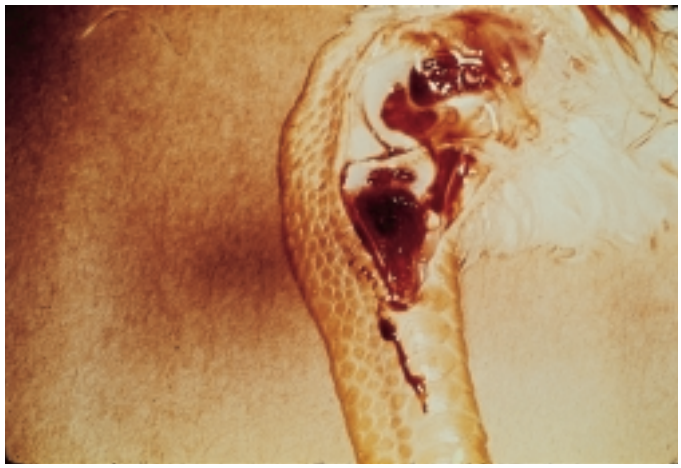
Changes in dietary formulation will alleviate specific nutritional deficiencies but will have no effect on genetic, environmental or infectious causes of locomotory abnormalities. It is advisable to review formulations, quality control of ingredients and feed in the event of acute episodes of locomotory dysfunction involving a high proportion of a flock.

Areas requiring specific attention include:-

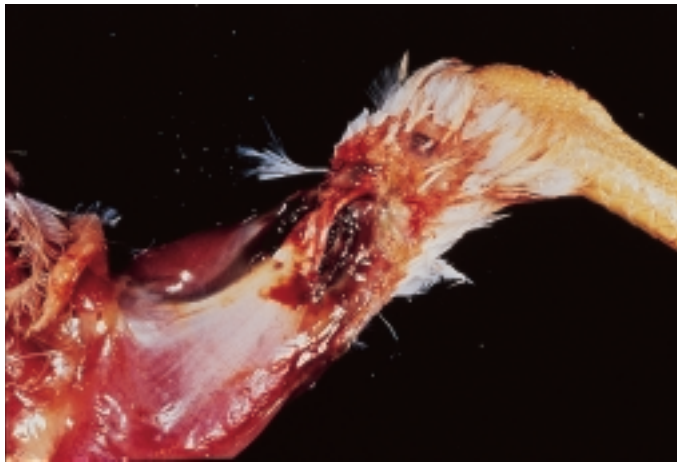
- Calcium and phosphorus levels in the diet should be in accordance with breed specifications. (Usually 1.0% calcium and 0.4% to 0.5% available phosphorus for immature flocks.)
- Limestone should have less than 3% magnesium content.
- Vitamin premixes should contain acceptable levels of potent D3; biotin; and riboflavin.
- Trace mineral premixes should contain adequate levels of zinc, manganese, iron.
- Essential amino acid content must conform to breed specifications.
- Ingredients should not contain mycotoxins at toxic levels.
- Water should conform to acceptable standards of purity and mineral content.
- Anticoccidial levels must conform to accepted inclusion rates. Ionophore toxicity results in paresis and paralysis in chicks.

114. Characteristic “hock-sitting” posture of broiler with *Mycoplasma synoviae* arthritis. (Courtesy AAAP)

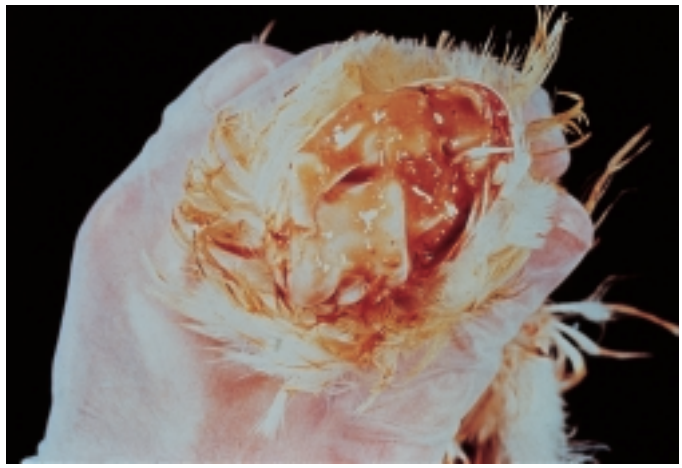
115. Enlargement of the hock joint characteristic of the arthritis caused by reoviral infection or *Mycoplasma synoviae*. (Courtesy AAAP)



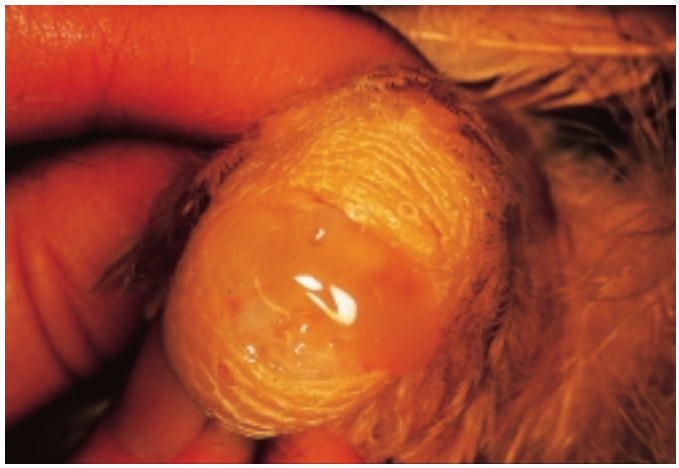
116. Serous arthritis associated with reoviral arthritis or *Mycoplasma synoviae* infection. (Courtesy AAAP)



117. Viral arthritis leads to inflammation of the tendon sheath characterized by the presence of serous exudate and hemorrhage.



118. Purulent arthritis attributed to *Staphylococcus aureus* infection.



119. Serous arthritis associated with reoviral arthritis or *Mycoplasma synoviae* infection.

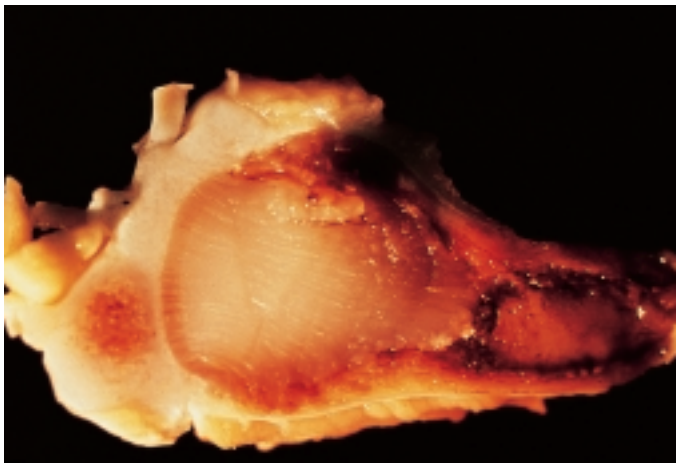
31.4 Developmental Etiology

31.4.1 Twisted Legs

Valgus (x-legged) and Varus (bow-legged) deformities occur in rapidly growing broilers. The long bones (tibiotarsus and tarsometatarsus) show obvious defects in 0.5% to 2% of broilers in otherwise normal flocks. The condition can be detected at approximately 3 weeks of age and progresses from angular deformation to displacement of the gastrocnemius (Achilles) tendon. Valgus and varus deformities are genetic in origin but severity may be influenced by intercurrent nutritional and managemental factors.

31.4.2 Rotated Tibia and Crooked Toes

These changes are observed in turkeys and heavy broilers and are probably genetic in origin. Generally they are not regarded as a significant cause of losses.



120. Tibial dyschondroplasia lesion in the proximal end plate of the tibiotarsus. (Courtesy of Dr. C. Riddell, AAAP slide set)

INTEGUMENTARY CONDITIONS

32.0 AVIAN POX

33.0 ECTOPARASITES
- LICE
- MITES
- ARGASID TICKS
- SCALY LEG MITES

34.0 DERMATOMYCOSIS

35.0 LEUCOCYTOZOONOSIS

32.0 AVIAN POX

32.1 Etiology: An avipoxvirus.

32.2 Occurrence and Economic Significance

The disease occurs in most countries with warm and humid climates. Broilers are frequently affected by the diphtheritic form of the infection. Losses are associated with a depression in growth rate and downgrading due to dermatitis although avian pox does not result in primary mortality. Infection of susceptible mature commercial-egg and breeder flocks results in a decline in production.

32.3 Transmission

The virus is mosquito-borne. Direct intraflock transmission by contact between infected and susceptible birds may occur.

32.4 Clinical Signs

Pink focal lesions occur on the comb and wattles and non-feathered portions of the body. These foci enlarge to become 0.5 to 1.0 mm diameter, black scab-like lesions, which persist for up to two weeks followed by desquamation and healing. Broilers may show confluent and extensive lesions of the back especially in the slow-feathering males of the auto-sexing strains. Mild respiratory rales (sounds) may occur in broiler flocks especially with suboptimal ventilation due to tracheitis.

32.5 Pathology

Histological examination shows characteristic intracytoplasmic inclusion bodies in infected skin and tracheal mucosa.

The diphtheritic form is recognized by the presence of nodular hyperplasia of the mucosa of the pharynx and trachea. Chickens which die of diphtheritic pox may show a plug of desquamated epithelium which lodge in the glottis resulting in asphyxiation.

32.6 Diagnosis

Cutaneous lesions are characteristic. Histological examination of affected tissue will confirm the presence of intracytoplasmic inclusions (Bollinger bodies) in the respiratory mucosa and skin.

32.7 Prevention

Immunization is recommended in endemic areas using a mild-attenuated avipox, chicken-strain virus vaccine administered at approximately 8 weeks of age. In areas where early exposure occurs, the age of vaccination

can be advanced. In some areas, broilers are routinely vaccinated against avian pox by subcutaneous injection at day-old. The efficacy of this procedure is questionable based on demonstrated maternal antibody interference.

In areas where flocks are affected with vertically transmitted mycoplasmosis, adverse vaccine reaction from avian pox vaccine can be prevented by administration of a pigeon-pox vaccine.

121. Focal lesion of avian pox on the comb of a hen.

33.0 ECTOPARASITES

33.1 Mites

Ornithonyssus spp remain on chickens permanently. *Dermanyssus* mites parasitize chickens nocturnally. Heavy mite infestation is characterized by anemia and the appearance of black mite exoskeleton casts and excreta and dermatitis in the vicinity of the vent.

33.2 Argasid Ticks

Soft-shelled ticks (*Argas* spp) occur in tropical areas and may affect cage-housed laying flocks or birds maintained on litter. Argasid ticks are nocturnal feeders and favor the soft unfeathered skin beneath the wings. Parasitized birds show multiple hematomas associated with feeding sites. Ticks transmit spirochetosis.

33.43 Scaly Leg Mites

Chronic infestation of the legs of free-roaming chickens with *Knemidocoptes mutans* results in proliferation of scales overlying the shanks and feet. The gross appearance of the lesion is pathognomonic. The diagnosis may be confirmed by microscopic examination of detritus from scales

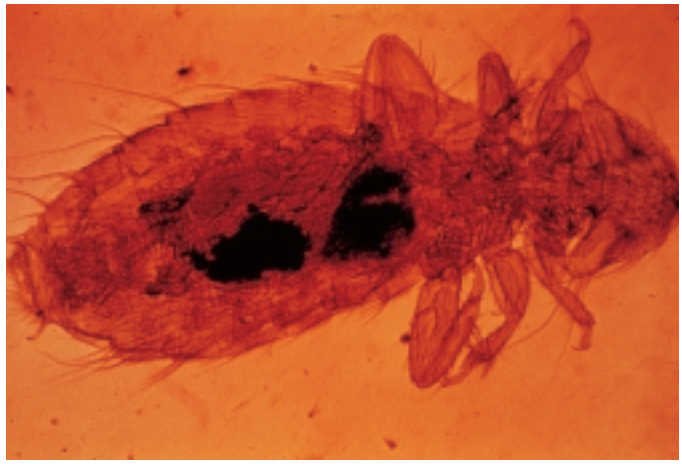
33.4 Lice

Lice are frequently encountered in subsistence flocks. Lice are responsible for irritation and damage to feathers. Mature adults are evident on examination. Egg clusters (“nits”) are observed as spherical white structures adherent to the shafts of feathers.

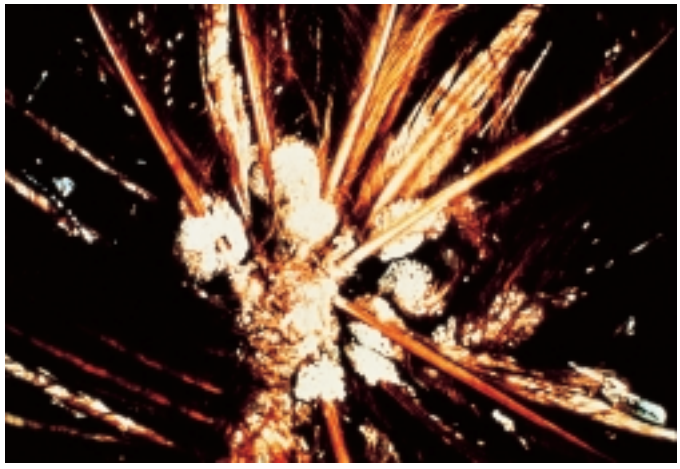
33.5 Treatment

Ectoparasites may be treated with a carbamate insecticide such as Sevin®, applied as a 5% powder to birds at two week intervals. Cages and housing can be treated with 2 - 7% carbamate suspension administered by spray.

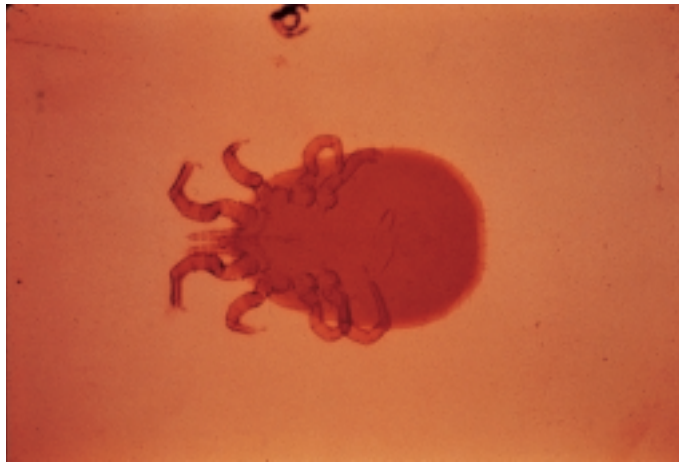
Only approved insecticides should be applied to poultry or used in the vicinity of housing to avoid contamination of the food chain. Insecticides should be used in accordance with manufacturers’ label instructions.



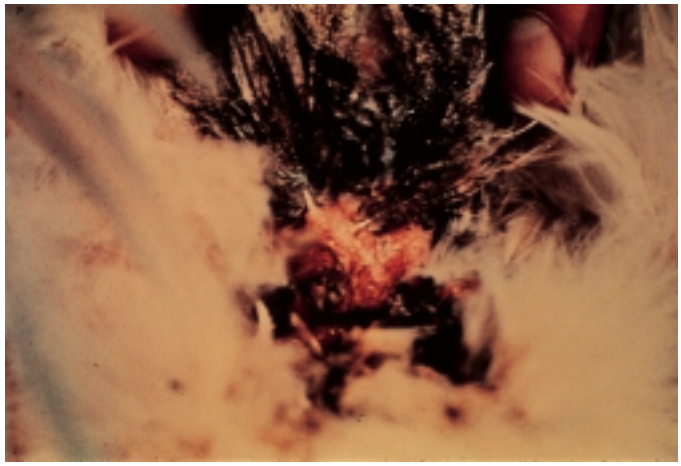
122. Lice are frequently present in backyard flocks and may depress production if infestation is severe.



123. Deposit of louse eggs ("nits") on the shafts of feathers.



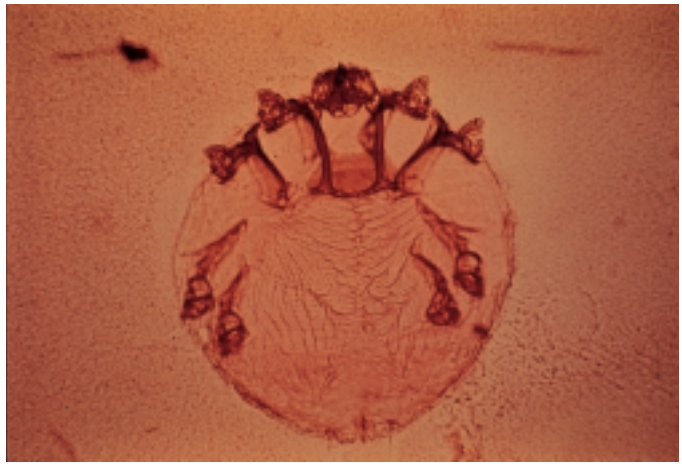
124. Blood-sucking *Ornithonyssus* spp mite.



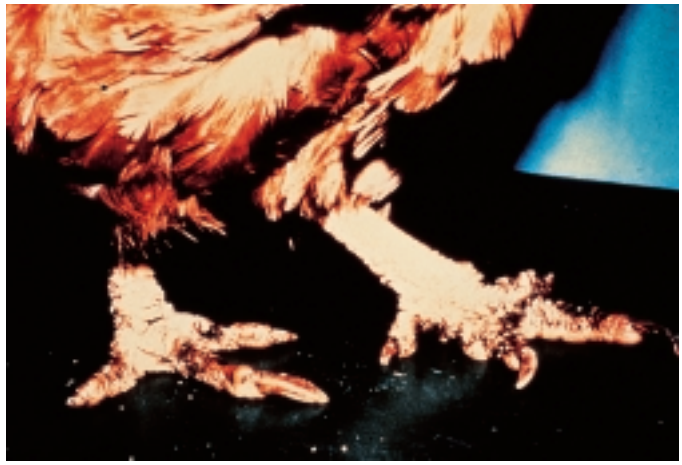
125. Severe infestation with *Ornithonyssus* spp mites in the region of the vent showing eggs, feather debris, and excreta.

126. Ticks of the genus *Argus* are vectors of spirochetosis.

127. Subcutaneous hemorrhage on de-feathered carcass due to nocturnal feeding of *Argas* spp ticks.



128. Scaly-leg mite. *(Knemidocoptes mutans)*



129. Advanced dermatitis of the feet (“scaly leg”) due to *Knemidocoptes mutans* infestation.

34.0 DERMATOMYCOSIS

synonyms Favus or "Ringworm"

34.1 Etiology: Fungi including *Trichophyton* spp.

34.2 Occurrence and Economic Significance

The condition occurs infrequently in commercial flocks but occurs mainly in subsistence chickens.

34.3 Transmission

The fungus is spread by direct contact or by contaminated cages or transport coops.

34.4 Clinical Signs

Grey to white scaly lesions appear on the comb and wattles, spreading progressively, especially in young birds. Feather loss may occur if lesions extend to the neck and body of the bird.

34.5 Diagnosis

The causal fungus can be cultured from lesions using Sabourauds dextrose agar. Histological examination of Gridley-stained tissue will show characteristic hyphae.

34.6 Treatment

Application of a 2% quaternary ammonium disinfectant, 1% tincture of iodine, or 5% formalin will eliminate the infection.

34.7 Prevention

Biosecurity precautions should be implemented to avoid introducing infected birds to the flock. Transport crates and other equipment should be thoroughly decontaminated and disinfected to prevent lateral transmission of the agent.

130. Dermatomycosis (favus or ring worm) characterized by gray scaly appearance of the comb, wattles, and non-feathered areas of the head. (Courtesy of the University of Georgia)

MISCELLANEOUS CONDITIONS

35.0 MYCOTOXICOSES

36.0 LEUCOCYTOZOONOSIS

35.0 MYCOTOXICOSES

Mycotoxicosis, a widespread problem in the poultry industry is caused by ingestion of toxins produced by molds which contaminate cereals and some oilseeds before and subsequent to harvest.

Mycotoxins are a diverse group of chemical compounds which adversely affect liveability, growth rate, feed conversion, immune response, egg production, and carcass quality. The acute and chronic effects of mycotoxins depend on the type of compounds present, level of contamination, and duration of ingestion.

Maize, wheat, rice, and peanut meal are most frequently implicated in cases of mycotoxicosis.

Immature chickens and ducklings are most susceptible to mycotoxins, but age, intercurrent health and environmental stress also influence the response to various toxins in feed.

Aflatoxins, ochratoxins, trichothecenes, and rubratoxins may result in high mortality if lethal levels of these compounds are present in feed. Low levels produce economically significant reduction in growth rate and feed conversion in broilers, and low egg production in breeders and commercial egg flocks.

Specific mycotoxins may product characteristic lesions in affected flocks:

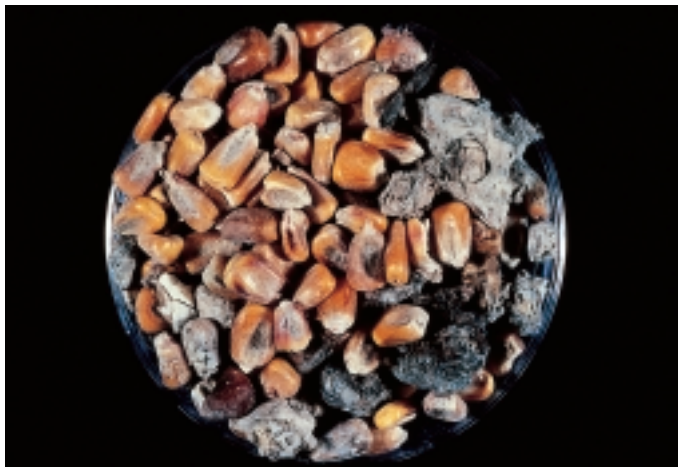
- Fusarium T-2 toxin is associated with stomatitis (ulceration of the lining of the oral mucosa).
- Ochratoxin results in kidney degeneration.
- Chronic aflatoxicosis is responsible for cirrhosis of the liver and ascites.

Generally, low-level mycotoxicoses are difficult to diagnose but should always be considered in cases of a chronic decline in growth rate, immunosuppression, suboptimal feed conversion, egg production or hatchability.

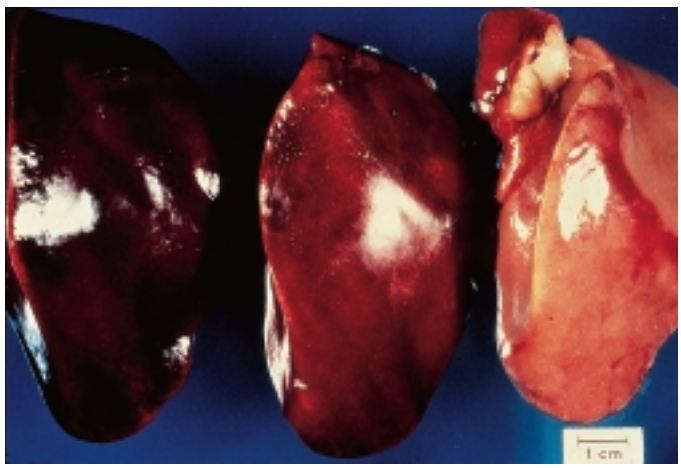
Nutrient content of grain is degraded when mold growth occurs on ingredients even in the absence of mycotoxins. Maize with moisture levels over 13% may be contaminated with mycotoxins including aflatoxin.

Prevention is based on detection of contaminated ingredients and exclusion from diets if this is practical or financially justified.

Correct storage of ingredients prevents post-harvest proliferation of molds. Feed additive inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Salvage of feed contaminated with aflatoxin is possible using high temperature ammoniation or adding commercial aluminosilicates to diets. Zeolite compounds and extracts from the cell wall of Saccharomyces cerevisiae (Mycosorb®) specifically bind aflatoxin in the intestine, inhibiting absorbtion. Zeolites have limited ability to inactivate other mycotoxins including the fusariotoxins. Charcoal and clay additives are generally ineffective as mycotoxins binders. Clay compounds may be contaminated with dioxins.

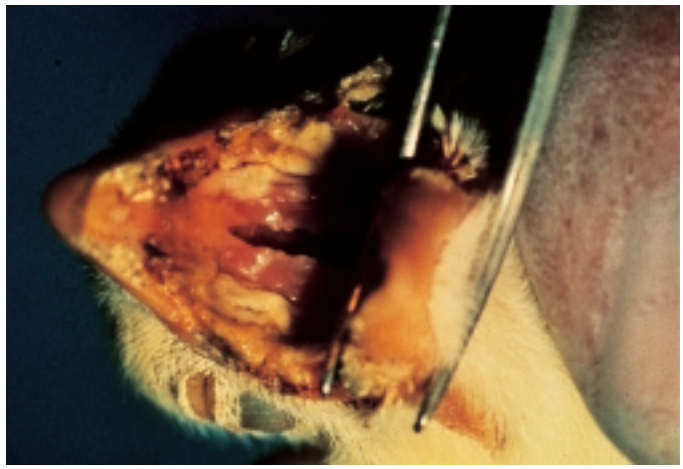


131. Contaminated corn showing severe fungal infection.



132. Pale liver of bird receiving 200 ppb aflatoxin in feed (right) compared to liver of bird receiving low level

133. Detailed examination of the oropharynx as necessary to exclude conditions such as T2 fusariotoxicosis, mycosis or avitaminosis A.



134. Stomatitis following consumption of T2 fusariotoxin (courtesy of Dr. Fred Hoerr)

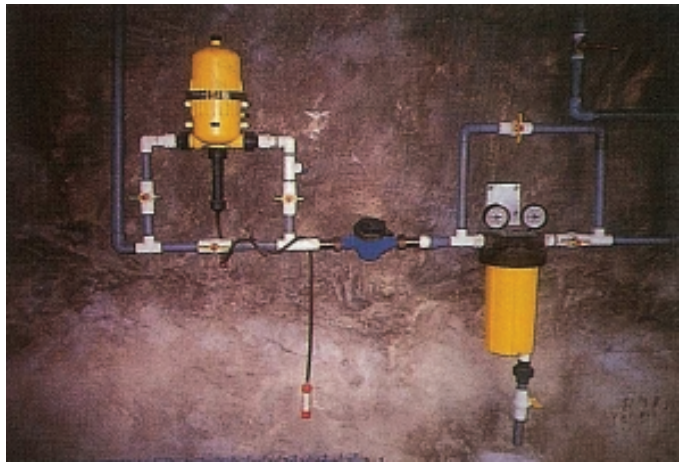
135. Chick showing stomatitis attributed to T2 fusariotoxicosis. (Courtesy of Dr. Fred Hoerr)

136. Abnormal feather formation (left) due to fusariotoxicosis. Compare with normal plumage on right (Courtesy of Dr. Fred Hoerr)

137. Diarrhea in chick fed fusariotoxin (Courtesy of Dr. Fred Hoerr)

138. Commercial test kits are available to monitor for the presence of mycotoxins.

139. Monitoring ingredients in feed for the presence of mycotoxins is a necessary quality control procedure.



140. Water proportioner can be used to distribute antibiotics and other medication through the drinking system. Water proportioners should be calibrated and their operation should be monitored.

36.0 LEUCOCYTOZOONOSIS

36.1 Etiology

Protozoa of the genus *Leucocytozoon*. *L. caulleryi* occurs in chickens and *L. simondi* in waterfowl in Asia.

36.2 Occurrence and Economic Significance

Leucocytozoonosis is frequently encountered in tropical countries especially where farms are located near lakes or ponds. Affected flocks show depressed growth rate and elevated mortality which may be influenced by immuno-suppression and intercurrent primary viral and secondary bacterial infection.

36.3 Transmission

Leucocytozoon is transmitted by dipterids of the genera *Simulium* and Culicoides.

36.4 Clinical Signs

Flock morbidity may exceed 25% in broilers and replacement egg production flocks. Batches of ducklings and goslings may show up to 35% mortality. Clinical signs include depression and decreased feed intake. Muscular incoordination occurs in extremis. Introduction of infection into a susceptible breeders or commercial egg-flocks may result in variable reduction in egg production.

36.5 Pathology

Affected birds show anemia, splenomegaly and hepatomegaly.

36.6 Diagnosis

Identification of the gametocytes which occur as extra-erythrocytic parasites in stained bloodsmear preparations. Gametocytes are elongated structures with prominent nuclei. Schizonts may be observed in preparations of kidney and liver tissue processed with Romanowski stain.

36.7 Treatment

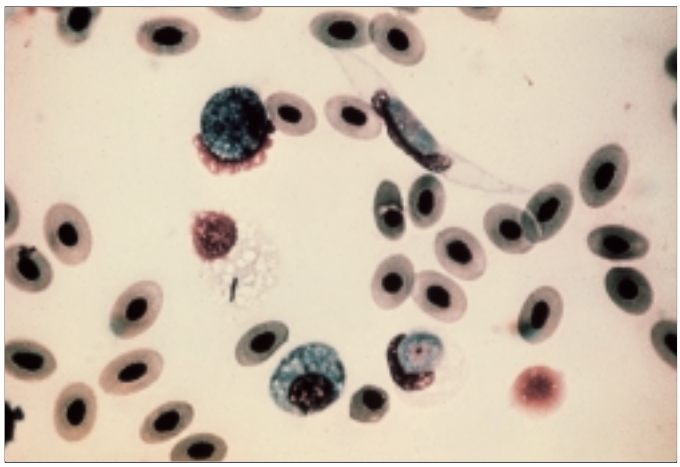
Pyrimethamine in combination with sulfadimethoxine.

36.8 Prevention

Control of insect vectors should be attempted using approved insecticides and draining of standing water. Exclusion of insect vectors in open-sided houses is impossible.

Clopidol anticoccidial incorporated in feed at levels ranging from 125 to 250 ppm has been used in the USA to prevent leucocytozoonosis in turkeys.

NOTE: In many countries, leucocytozoonosis is referred to incorrectly as “malaria.” Malaria caused by *Hemaproteus* spp are not associated with clinical infections in commercial chickens.



141. Leucocytozoon parasite visible as extra-erythrocytic structure in Giemsa-stained blood smear.

DISEASES OF WATERFOWL

37.0 DUCK VIRAL ENTERITIS

38.0 DUCK VIRUS HEPATITIS

39.0 DUCKLING SEPTICEMIA

40.0 CHLYMADIOSIS

37.0 DUCK VIRAL ENTERITIS

synonym “duck plague” DVE

37.1 Etiology

A herpes virus.

37.2 Occurrence and Economic Significance

Duck viral entertitis occurs in all areas where ducks and geese are raised. The infection is responsible for severe losses in susceptible flocks.

37.3 Transmission

Duck viral enteritis is transmitted directly by contact of susceptible birds with infected viremic ducks or recovered carriers. Concentration of ducks in intensive production areas and common use of ponds by commercial flocks and migratory and free-living resident waterfowl predisposes to infection.

37.4 Clinical Signs

Morbidity varies according to the strain of virus and the susceptibility of the flock and may range from 10% to 100% with a corresponding mortality rate. Ducks are usually infected from three weeks onwards with an incubation period of 5 to 7 days.

Mature breeding flocks show a precipitous decline in egg production immediately preceding a significant rise in mortality.

Affected ducks demonstrate extreme depression, ruffled plumage, diarrhea and photophobia.

Clinical signs in mature ducks composed paresis, flaccidity of the neck and terminally, tremors of the head and limbs. These signs should be differentiated from botulism. Highly pathogenic strains of the virus may cause hemorrhages from the nares and cloaca.

37.5 Pathology

Characteristic lesions of duck viral enteritis include:

- Free blood in the body cavity.
- Hemorrhages of the ovary in mature ducks.
- Free blood in the lumen of the intestine.
- Hemorrhages of the serosa of the intestine, pericardium and capsule of the liver which are evident as petechiae and ecchymoses.

Hemorrhagic changes are observed in all lymphoid tissues including the thymus, bursa of Fabricius in ducklings and the annular lymphoid bands (gut associated lymphoid tissue) of the intestine. Degenerative changes in the intestinal tract extending from the esophagus to the cecum include hemorrhagic areas in the early stages of the disease, progressing to confluent maculae resembling a diphtheritic pseudomembrane reminiscent of clostridial enteritis in chickens.

37.6 Diagnosis

Characteristic lesions are highly suggestive of the diagnosis. Histological examination discloses the presence of intranuclear inclusion bodies in hepatocytes.

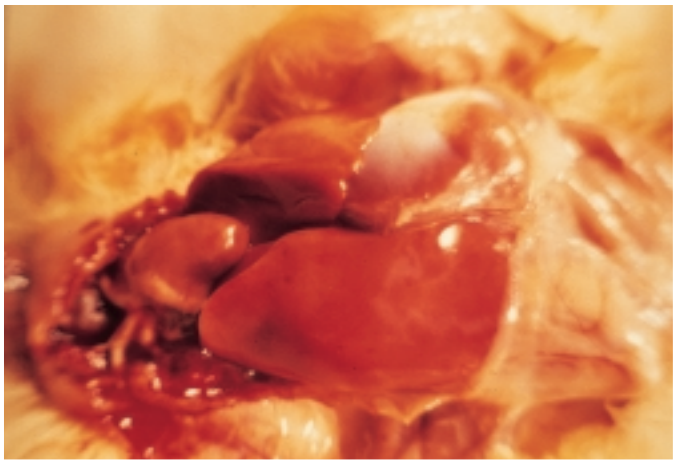
The viral agent can be isolated in 10-day old embryonated duck eggs inoculated by the chorioallantoic route. Retrospective diagnosis is based on demonstrating a marked increase in neutralization titers comparing sera from acute, newly infected and recovered ducks. It is important to differentiate duck viral enteritis from highly pathogenic avian influenza, botulism, pasteurellosis and duck viral hepatitis.

37.7 Treatment

None.

37.8 Prevention

A live attenuated chicken-embryo derived vaccine has been used in Europe to prevent outbreaks. Where possible, commercial ducks should be isolated from free-living waterfowl which are reservoirs of infection.



142. Hemorrhage from nares and also from cloaca is characteristic of DVE.

38.0 DUCK VIRUS HEPATITIS

38.1 Etiology

A picornavirus is responsible for duck virus hepatitis Types 1 and 3. This agent is distinct from hepadnavirus (duck hepatitis B). An astrovirus-like agent has been implicated in outbreaks of duck virus hepatitis Type 2 in the UK.

38.2 Occurrence and Economic Significance

Duck virus hepatitis Types 1 and 3 occur in ducklings under 4 weeks of age in all intensive duck-rearing areas of the world. Mortality results in extensive losses especially with intercurrent bacterial infections including chlamydiosis, *Riemerella* and *E. coli* infection, mycotoxicosis and environmental stress.

38.3 Transmission

Free-living waterfowl introduce the virus. Rodents serve as reservoir hosts on affected farms. Direct contact between infected and susceptible flocks especially in multiage operations predisposes to disease.

38.4 Clinical Signs

Morbidity in susceptible flocks may range from 50% to 100%. Mortality is dependent on the age of the flock, with losses of up to 90% in batches under one week of age, declining in severity to under 10% at 4 weeks of age. Mortality is exacerbated by intercurrent bacterial infections.

Ducklings demonstrate peracute mortality preceded by lateral recumbency and occasionally, opisthotonos (hyperextension of the neck, “stargazing”).

38.5 Pathology

The significant lesion comprises enlargement of the liver with punctate or ecchymotic hemorrhages. In the presence of chlamydiosis or *Riemerella* spp infection, airsacculitis and peritonitis may be observed.

38.6 Diagnosis

The picoronaviruses responsible for duck virus hepatitis can be isolated from livers using 9-day old embryonated SPF chickens inoculated by the allantoic sac route. Serologic procedures include agar gel diffusion precipitin test, virus neutralization in duck embryos and a plaque reduction test to quantify neutralizing antibody.

38.7 Treatment

No specific treatment is available. Supportive therapy is recommended.

38.8 Prevention

Hyperimmune serum from flocks surviving duck viral hepatitis can be administered to ducklings. An injection of 0.5 ml filtered serum is recommended using the intramuscular route. Breeders can be immunized with a live attenuated chicken-embryo origin vaccine. Some vaccines have shown reversion to virulence when applied to large flocks. Where possible, single-age isolated placement programs should be followed. Rodents should be eradicated.

143. High mortality in newly placed ducklings has a wide differential diagnosis including *DVH.*, *salmonellosis*, *E. coli*, and *Riemerella infection*, *aspergillosis* and *mycotoxicosis*.

39.0 DUCKLING SEPTICEMIA

39.1 Etiology

Riemerella anatipestifer is the principal pathogen responsible for duckling septicemia. Concurrent infections include *E. coli*, septicemia, salmonellosis and duck virus hepatitis.

39.2 Occurrence and Economic Significance

Duck septicemia occurs in all areas where ducklings are reared commercially, resulting in variable morbidity and mortality during the first two weeks of the brooding period.

39.3 Transmission

Direct contact of susceptible ducklings with a contaminated environment. Footpad lesions from defective wire floors predispose to percutaneous infection.

39.4 Clinical Signs

Affected ducklings show depression, ataxia, ocular and nasal discharge and respiratory rales.

39.5 Pathology

Septicemic changes characterized by perihepatitis, pericarditis and fibrinous airsacculitis, hepatomegaly and splenomegaly are observed. In some cases, fibrinous meningitis occur, especially in ducklings which display nervous signs.

39.6 Diagnosis

Diagnosis is based on isolation and identification of Rimerella anatipestifer from heart blood, liver or brain tissue on either blood agar or trypticase soy agar.

39.7 Treatment

Supportive therapy and administration of water soluble tetracycline may be attempted. Enrofloxacin can be administered in drinking water at a level of 50 ppm for the first 2 days followed by 25 ppm for 4 subsequent days.

39.8 Prevention

Managemental interventions including effective sanitation between placements, avoiding overcrowding and chilling should be implemented. Multivalent or homologous bacterins have been prepared for administration to ducklings at 1 to 2 weeks of age. A live attenuated vaccine against serotypes 1, 2 and 5 has been developed, which is administered to ducklings by the aerosol route or in drinking water at day-old.

40.0 CHLAMYDIOSIS

40.1 Etiology

Chlamydia psittaci, an obligatory intracellular organism.

40.2 Occurrence and Economic Significance

Outbreaks of chlamydiosis occur in poultry, including waterfowl, throughout the world. This infection is transmitted to humans and is regarded as an important zoonotic infection. Commercial transport and handling of waterfowl previously in contact with ornamental and companion species may result in outbreaks in distribution centers associated with trade in live birds.

40.3 Transmission

The disease is transmitted by both acutely infected birds and latent carriers. Infection requires inhalation of viable elementary bodies. Periodic epornitics have been described in commercial operations, attributed to transmission of virulent strains from indigenous or migratory bird species by direct and indirect contact and presumably by insect vectors.

40.4 Clinical Signs

Acute infection in young ducks is characterized by ataxia, diarrhea, purulent ocular and nasal discharge. Chronic cases show emaciation and chronic respiratory involvement.

40.5 Pathology

Chlamydiosis should be suspected if the following lesions are observed in waterfowl:

- Fibrinous pericarditis and perihepatitis
- Hepatomegaly and splenomegaly
- Airsacculitis

40.6 Diagnosis

Diagnosis is confirmed by isolation of the organism from homogenates of organs obtained using sterile necropsy technique. Laboratory host systems include embryonated eggs inoculated by the yolksac route and mice inoculated by the intraperitoneal route. Laboratory techniques include fluorescent antibody detection and retrospective diagnosis using the complement fixation procedure.

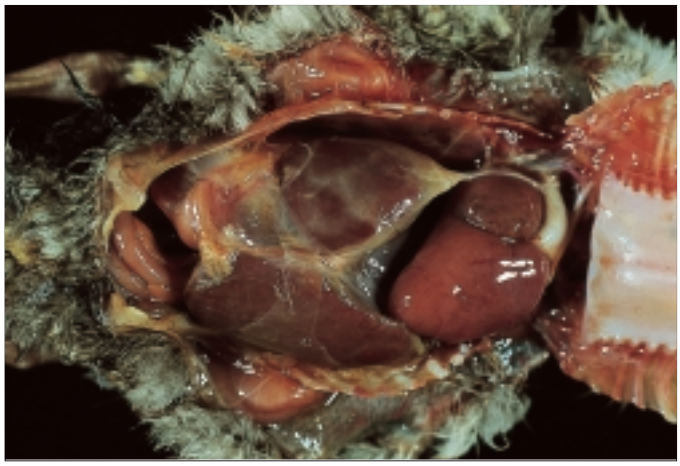
40.7 Treatment

Commercial duck flocks can be treated by administration of

chlortetracycline in feed at a level of 400 grams per ton for a period of up to two weeks. It is necessary to follow statutory requirements regarding withdrawal periods following administration of antibiotic before sale or slaughter of flocks, and obligatory reporting of outbreaks.

40.8 Prevention

Appropriate biosecurity procedures should be imposed to prevent transmission from infected reservoirs including companion bird species, free-living Psittacines and Colombibormes. It is virtually impossible to control chlamydiosis in multiage flocks with the opportunity for direct and indirect contact.



144. Chlamydiosis should be considered in cases of fibrinous peritonitis and airsacculitis.

ANNEXURES

41.1 CONVERSION OF U.S. METRIC WEIGHTS AND MEASURES

41.2 SCHEDULE OF THERAPEUTIC DRUGS

41.3 DIFFERENTIAL DIAGNOSES OF AVIAN DISEASES

41.4 ABBREVIATIONS

41.1 CONVERSION OF U.S. TO METRIC WEIGHTS AND MEASURES

Linear Measure

1 inch	= 25.4 mm
1 yard	= 0.914 m
1 mile	= 1.61 km

Square Measure

1 sq. inch	= 6.45 cm^2
1 sq. foot	= 929 cm^2
1 sq. yard	= 0.836 m^2
1 acre	= 0.41 Ha

Cubic Measure

1 cu. inch	= 16.4 cm^3
1 cu. foot	= 0.028 cm^3
1 cu. yard	= 0.765 m^3

Liquid Measure

1 pint	= 0.57 litres
1 quart	= 1.14 litres
1 US gallon	= 3.8 litres

Weight

1 ounce	= 28.4 g
1 lb.	= 0.453 kg
1 long ton	= 2,205 lb
	= 1000 kg
1 short ton	= 2000 lb
	= 907 kg

Heat Units

1 B.t.u	= 0.252 calorie
1 calorie	= 3.968 B.t.u.
	= 4.184 joule

Power

1 h.p.	= 33,000 ft. lb.min.
	= 746 watts
	= 1.01 metric h.p.
1 kilowatt	= 1000 watts
	= 1.34 h.p. watts

Temperature

Centigrade = 5/9 x (Fahrenheit - 32)

EXAMPLE:

C^o = 5/9 x (86ºF - 32)

C^o = 5/9 x (54)

C^o = 30º

Fahrenheit = (9/5 x Centigrade) + 32

EXAMPLE:

F^o = (9/5 x 30ºC) + 32

F^o = 54 + 32

F^o = 86º

Bushel Weight

Shelled maize	56 lb. = 25.4 kg
Cracked maize	50 lb. = 22.7 kg
Cottonseed meal	48 lb. = 21.8 kg
Soybeans	60 lb. = 27.2 kg

Conversion Factors

To convert	Into	Multiply by	Reciprocal multiply by
B.t.u.	Calories (kg)	0.250	3.968
B.t.u. per sq. ft.	Calories (kg) per m^2	2.712	0.369
Cu. ft. per minute	m^3 per second	2118	0.0004719
Cu. ft. per minute	m^3 per minute	0.02832	35.31
Cu. ft. per minute	m^3 per hour	1.699	0.5886
Ft. per minute	Metres per minute	0.3048	3.281
Ft. per second	Centimetres per second	30.48	0.03281
Horsepower	Force de cheval	1.0139	0.9863
Inches water column	Millimetres water column	25.40	0.03937
Pounds per sq. inch	kg per cm^2	0.07031	14.22
Pounds per sq. ft.	kg per m^2	4.883	0.2048
Pounds per cu. ft.	kg per cm^3	16.02	0.06243

41.2 SCHEDULE OF THERAPEUTIC DRUGS

Drug	Dose	Administration	Duration (days)	Suggested Withdrawal Period (days)
Feed				
Bacitracin methylene disalicylate	20-50 g/ton	feed	continuous	none
Chlortetracycline	100-500 g/ton	feed	10-14	5
Erythromycin	100-400 g/ton	feed	5-10	2
Furazolidone (where permitted)	150-250 g/ton	feed	7 max.	10 not used in EU, or USA
Gentian violet	8-12 g/ton	feed	10-14	none
Hygromycin B	2 g/ton	feed	continuous	none
Lincomycin	200 g/ton	feed	10-14	5
Niclosamide	200-400 g/ton	feed	1 dose	5
Novobiocin	50 g/ton	feed	1-10	none
Nystatin	100-200 g/ton	feed	10-14	3
Oxytetracycline	125 g/ton	feed	10-14	5
Sulfadimethoxine* with ormetroprim	800-1000 g/ton	feed	1-5	5
Tylosin	200 g/ton	feed	2-7	20
Virginiamycin	20 g/ton	feed	continuous	none
* Not for egg-producing or breeder flocks.				
Injectable (for therapy of individual high-value birds only. Not for flocks)				
Ampicillin	10 mg/kg	i.muscular	daily	5
Erythromycin	100 mg/kg	i.muscular	daily	5
Gentamycin	0.2-0.5 mg	s.cutaneous	day old	none
Levamisole	30 mg/kg	s.cutaneous	dose	none
Spectinomycin	2-5 mg/chick	s.cutaneous	day old	none
Streptomycin	100 mg/kg	i.muscular	daily	5
Sulfadiazine*	30 mg/kg	i.muscular	daily	5
Tylosin	10-40 mg/kg	s.cutaneous	daily	none
* Not for egg-producing or breeder flocks.				

Drug	Dose	Administration	Duration (days)	Suggested Withdrawal Period (days)
Drinking Water Administration				
Ampicillin	1.5 g/l	drinking water	2-5	5
Amprolium 17%	1 g/l	drinking water	3-5	5
Chlortetracycline	250-500 mg/l	drinking water	3	3
Dimetridazole* soluble	0.2 g/l or 200 mg/kg body wt.	drinking water pill individual bird	3-5 1 dose	not used in US 5
Enrofloxacin 10%*	0.5 - 1 ml/l	drinking water	3-5	5
Flumequin 12%*	0.5 - 1 ml/l	drinking water	3-5	5
Furaltadone 12%*	1 - 2 ml/l	drinking water	2-5	not used in US
Levamisole 10%	1 g/l	drinking water	1 dose	5
Nitrofurazone sol* 9%	10 g/l	drinking water	2-5	5
Oxytetracycline	50 mg/l	drinking water	5	5
Piperazine 17%	1 g/l	drinking water	3 hours	5
Spectinomycin 25%	1 g/l	drinking water	3	5
Sulfamethazine 12%	5 - 10 ml/l	drinking water	5-7	5
Tylosin 50%	1 g/l	drinking water	3	5

Notes

- * Drug to be administered only where permitted by law or the regulations of an importing country. Ascertain the legal or statutory restraints concerning drug in the area concerned especially for nitrofurans and fluroquinolones.
- The first value in the dose range usually represents the preventive level, the second figure is the appropriate therapeutic dose.
- The availability of commercial forms of these compounds should be determined for the area of operation.
- Specific rates of addition to feed and water may vary according to the concentration of the active compound in the commercial presentation. PRINTED LABEL DIRECTIONS SHOULD BE FOLLOWED.
- Comply with species and age restrictions.
- Observe withdrawal periods for egg-producing and poultry-meat flocks.
- Select drugs on a cost-effective basis.
- Monitor clinical response. Where practical compare with untreated, exposed controls.

41.3 DIFFERENTIAL DIAGNOSES OF AVIAN DISEASES & CONDITIONS ASSOCIATED WITH SPECIFIC AGE GROUPS & TYPES OF COMMERCIAL POULTRY

Diseases & Conditions characterized by:-

1. Mortality in chicks day old - 10 days

- Arenavirus infection
- Aspergillosis
- Avian encephalomyelitis
- Mismanagement: (temperature, water, feed)
- Omphalitis
- Paratyphoid including *Salmonella enteritidis*
- Pullorum disease
- Smothering
- Toxicity

2. Mortality & morbidity in chicks 10 - 30 days with nervous signs

- *Argas persicus* infestation
- Avian encephalomyelitis
- Encephalomalacia
- Infectious bursal disease
- Mycotoxicosis
- Newcastle disease
- Pyridoxine deficiency
- Thiamine deficiency
- Toxicity

3. Mortality & morbidity in chicks 20-50 days with locomotory abnormalities

- Arthritis - *Staphylococcus* sp.
  - Mycoplasma synovia
  - Reovirus
- Botulism
- Calcium deficiency
- Cholecalciferol (D3) deficiency
- Congenital deformities
- Manganese deficiency
- Marek's disease
- Mechanical injury
- Niacin & choline deficiency
- Phosphorus deficiency
- Riboflavin deficiency
- Spondylolisthesis
- Tibial dyschondroplasia

4. Mortality & morbidity in chickens with respiratory signs

- Adenovirus Type-1
- Avian infectious bronchitis
- Avian influenza
- Avian pox
- Coli-septicemia
- Infectious coryza
- Infectious laryngotracheitis
- Mycoplasmosis
- Newcastle disease
- Pneumoviral,\ Swollen Head Syndrome
- Vaccine reaction

5. Mortality & morbidity in broilers & pullets
(other than with locomotory, respiratory & nervous signs)

- Adenoviral hydropericardium - Hepatitis syndrome
- Ascites complex
- Coccidiosis
- Coli-septicemia
- Conjunctivitis
- Histomoniasis
- Infectious bursal disease
- Liver & kidney syndrome
- Pasteurellosis
- Salmonellosis
- Spirochetosis
- Sudden death syndrome (Right heart failure)
- Toxicity
- Ulcerative & necrotic enteritis

6. Decline in egg production
Asymptomatic or with mild signs & low or inapparent mortality

- Adenovirus Type-1
- Avian encephalomyelitis
- Avian influenza
- Infectious bronchitis
- Newcastle disease
- Nutritional (Ca; P; Na; Cl deficiency)
- Water & feed deprivation

7. Decline in egg production with mortality & morbidity

- Avian influenza
- Avian pox
- Coryza
- Infectious bronchitis
- Infectious laryngotracheitis
- Newcastle disease
- Pasteurellosis
- Salmonellosis

8. Conditions resulting in mortality & morbidity encountered predominantly in caged layers

- Infectious conditions as above:-
  (ILT, ND, IB, Salmonellosis, Coryza, Pasteurellosis, Adenovirus, Avian encephalomyelitis)
- Ectoparasitism
- Endoparasitism
- Fatty liver syndrome
- Leucosis complex
- Mycotoxicosis
- Nutritional deficiencies (Ca; P)
- Vent peck
- Water deprivation

9. Conditions affecting the integument

Avian pox
Avitaminosis A; Biotin; E (Transudative diathesis); Pantothenic acid deficiency
Cannibalism/trauma
Dermatomycosis
Ectoparasites
Gangrenous dermatitis/*Staph.* complex
Managemental factors
Marek's disease
Pasteurellosis - wattle lesion

10. Conditions associated with subsistence & free-ranging flocks

Arthritis
Avian pox
Candidiasis
Cannibalism
Coccidiosis
Coli-septicemia
Ectoparasites
Emphysema (trauma)
Endoparasite
Histomoniasis
Lymphoid leucosis
Marek's disease
Mycoplasmosis
Mycotoxicosis
Pasteurellosis
Pendulous crop
Pododermatitis
Predator loss
Salmonellosis
Spirochetosis
Thermal injury
Toxicity
Traumatic ventriculitis
Tuberculosis
Visceral gout

11. Conditions associated with waterfowl

Aspergillosis
Avian influenza
Botulism
Chlamydiosis
Colibacillosis
Duck viral enteritis
Duck virus hepatitis & food-borne infections
Erysipelas
Coccidiosis
Ectoparasites
Endoparasite
Lead poisoning
Mytotoxicosis
Nutritional deficiencies
Pasteurellosis
Salmonellosis

12. Zoonoses & food-borne infections associated with avian species

DISEASE	TYPE OR SPECIES INVOLVED
* Avian influenza	p
* Chlamydiosis	c, p
Tuberculosis	c, p
* Salmonellosis (food-borne)	c, p
* Campylobacteriosis (food-borne)	
* Arboviral encephalitides (including West Nile disease)	p
Listeriosis (food-borne)	p
* Erysipelothrix infection	p
Aspergillosis	p
Histoplasmosis	z, w
Newcastle disease	c, p
Sarcosporidiosis	w
Toxoplasmosis	c
Tularemia	z, w

* Significant disease

c – Companion species
p – Commercial poultry
w – Wild and free-ranging species

41.4 ABBREVIATIONS

AE	Avian encephalomyelitis (epidemic tremor)
AI	Avian influenza
bpm	breaths per minute
DVE	Duck viral enteritis
EDS	Egg drop syndrome (Type 3 adenovirus infection)
ELISA	Enzyme Linked Immunosorbent Assay
HHS	Hydrophericardium-hepatitis syndrome (Type 1 adenovirus infection)
HPAI	highly pathogenic avian influenza
IBD	Infectious bursal disease
ILT	infectious laryngotracheitis
MD	Marek's disease
Mg	Mycoplasma gallisepticum
Ms	Mycoplasma synoviae
ND	Newcastle disease
PCR	Polymerase Chain Reaction
QAT	Quaternary ammonium disinfectant
RT-PCR	Reverse Transcriptase- Polymerase Chain Reaction
SPF	specific pathogen free
VN	Virus neutralization
vvIBD	very virulent Infectious bursal disease
vvND	velogenic viscerotropic Newcastle disease

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