Poultry Health and Management

by

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Preface

The purpose of this *Poultry Health and Management book* is to provide an up-to-date accessible source of information about poultry diseases and other aspects of poultry health and management. The book is divided into subsections, each of which will deal specifically with single or related topics.

Introductory chapters provide a variety of information such as anatomy and physiology, clinical and postmortem examination, disease prevention and control measures, which include sanitation, disinfectants, vaccination programs, and other pertinent information useful for creating and maintaining sound poultry health programs.

Various chapters deal with diseases caused by a particular type of agent including bacteria, virus, fungi, protozoa, parasites, etc. Another section is devoted to "miscellaneous diseases" conditions in which the exact cause is not known or is not in a single entity group. The mention of any drug or vaccine in this handbook, whether by trade or by chemical name, is not to be construed as an endorsement of that product.

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1. Anatomy and physiology

Before you can treat a disease, one must make an accurate diagnosis. Diagnosis requires that you know the normal anatomy of the fowl (figure 1.0). This chapter will provide a general overview of avian anatomy and physiology.

![Figure 1.0. Normal anatomy of the domestic chicken](image)

The skeletal system

The bones of birds are rich in calcium salt and are dense. In many bones, the marrow is displaced by air spaces, which are connected with the respiratory tract. These hollow bones help lighten the skeleton to aid in flight (figure 1.1).

Skeleton of the wing

Scapula, coracoid, and clavicle compose the shoulder. The scapula, long, narrow and slightly curved, lies dorsal (back of) to the ribs. It articulates (connects) with the coracoid at an acute (severe) angle. The coracoid is the largest bone in this group and articulates by its proximal (close to its center of origin) end with a facet (a small smooth surface on the bone) on the sternum. This proximal end presents a foramen (aperature or perforation), which connects with the anterior (above) thoracic air sac. The distal (away from the center of the body) end of this bone has a hook-like process, which together with the proximal ends of the humerus and scapula, forms a ring of bone (Foramen triosseum) through which passes the tendon (connects muscle with bone) of the supracoracoid muscle. This is an elevator of the wing. Anterior to the coracoid is the clavicle, which by its proximal end articulates with the coracoid and humerus and by its distal end meets its fellow to form a forked bone (furcula, wish bone). A ligament (connects bone to bone) unites the furcula to the rostrum (beak-shape structure) of the sternum.
The humerus is present on the medial side of the proximal end, a Foramen pneumaticum, which connects with the anterior thoracic air sac. In the adult bird, a radial and an ulna compose the bones of the carpus. There are the second, third, and fourth incompletely developed metacarpal bones. The second metacarpal bone is very short and is attached to the fused proximal ends of the third and fourth. The latter two are also fused at their distal ends. Of the digits corresponding to the metacarpal bones, the second and third are each composed of two phalanges (long bones of the digits), and the fourth has one phalanx (singular of phalanges).

Figure 1.1. Skeleton of the domestic fowl (left) and psittacine bird (right).

Skeleton of the leg

Ilium, ischium, and pubis bones compose the pelvic girdle (belt). The ilium and ischium are fused with the regional vertebrae, and in the concavities (depression) of the ventral surfaces of these two bones the kidneys are lodged. A large sciatic foramen perforates the ischium posterior to the acetabulum. The pubis is a long, slender bone beginning antero-ventral to the acetabulum by a knob and extending posterior with its free end projecting beyond the ischium. Slightly postero-ventral to the acetabulum, it is separated by the obturator foramen from the ischium. There is attachment between pubis and ischium for a short distance posterior to the foramen, beyond which the shaft and extremity of the pubis are free.

The femur, patella, fibula, and tibia closely resemble those of mammals. Since the tarsal bones fuse early with the tibia and metatarsal bones, there are no separate tarsal bones. The tarsal joint is a ginglymus, or hinge joint, composed in the adult of the fused bones on the distal end of the
tibia and those on the proximal end of the metatarsus. The metatarsus is composed of a shortened first metatarsal bone connected by a ligament (connects bone to bone) to the medio-plantar border of the fused second, third, and fourth metatarsal bones. Very close and proximal to the first bone on the medial side of the shaft of the fused mass of the male is a hooked process. This is the base of the spur. The distal end of the metatarsus has three condyles (articular surface on the bone), which articulate with the first phalanges of the second, third, and fourth digits. In the chicken the first digit of the foot is directed posterior and carries two phalanges. The second, third, and fourth digits are directed interiorly and have, respectively, three, four, and five phalanges.

**Muscular system of fowls**

The pectoral muscles (figure 1.3) are highly developed in birds that can fly. There is a superficial one, which depresses the wing, and a deeper one, the supracoracoid muscle. This latter one has a tendon of insertion, which passes through the *Foramen triosseum* at the shoulder joint to be inserted on the humerus near its head. The supracoracoid muscle is an elevator of the wing. The abdominal muscles are very thin sheets of muscle. The thigh muscle (figure 1.2) is also well developed in avians.

![Figure 1.2. Thigh Muscle](image)

![Figure 1.3. Breast Muscle](image)
The digestive system

The beak

The beak, both upper and lower segments, is composed of a superficial epidermis (superficial portion of skin). This has the four typical strata (layers) ordinarily ascribed to it. *Derma* or *corium* intervenes between epidermis and periosteum. This layer contains blood vessels and nerves, and some touch corpuscles. The bone of the upper beak is the *Os incisivum* (*premaxillae*) and the lower is the *Os dentale* (*Corpus mandibulæ*). Since birds have no teeth, the beak serves to pick up food.

Cavity of the mouth

The lining membrane is similar to that of the mammals. Many posterior directed papillae (nipple-like projections) are present in the mucosa of the hard palate, which presents a median cleft. Lymphoid tissue is present in the *Lamina propria* of older birds. The food is swallowed immediately when taken into the mouth.

The tongue

The dorsum of the tongue has a mucous membrane, which although uneven, is devoid of papillae except posterior where there is a transverse row. The epithelium on the dorsum is highly keratinized. It continues over the tip to the ventral side where soon it becomes less cornified and smooth. Lymphoid tissue may be present in this part of the tongue of older birds. The muscles are poorly developed and are arranged about the arrow-shaped end of the *entoglossal* bone, which is the anterior end of the hyoid bone.

The salivary glands

The salivary glands all have a similar structure. The tubules of each lobule open into a central cavity, which is continuous with an excretory duct. Amylase is present in the saliva of the hen. Lymphoid tissue may be found both interlobular and intralobular in most of the salivary glands of adult birds.

The pharynx

The posterior-most transverse row of palatine papillae and the row on the base of the tongue may be taken as a division line between mouth and pharynx. The mucous membrane of the pharynx is similar in structure to that of the mouth. Dorsally on the median plane is a slit, which provides a common opening for the pharyngeal ends of the eustachian tubes. Continuity with the nasal cavity is through the cleft of the hard palate.

The esophagus

The structure of the esophageal wall is similar to that of mammals. The thick epithelial layer of the mucosa is highly keratinized (scleroprotein). Extending through this layer and into the
Lamina propria are large mucous glands. Some lymphoid tissue may be found in the Lamina propria. This organ serves as a tube for passage of food from the mouth to the crop (figure 1.4).

The crop

The esophagus of ducks and geese has, in its cervical portion, a long spindle-shaped dilatation. In pigeons and chickens a ventral diverticulum (a sac-like opening from a tubular structure) marks the junction of cervical and thoracic portions. In the chicken this crop, or ingluvies, has a structure similar to that of the esophagus with the exception that it is glandless in its greater part, and its greater curvature has a sparse blood supply. The crop serves as a food reservoir and moistens (softens) the food. The crop contains some mucous glands and produces some amylase. The crop of the pigeon has two lateral lobes.

The structure of the wall is similar to that of the esophagus. In the female pigeon, which has been setting eggs for 8 days, hypertrophy (increase in size) of the wall has already begun. This remains until several days after hatching. The most marked change is in the increased number of cells of the epithelial (avascular covering of surface) layer, which becomes markedly folded. The superficial cell-layers of the epithelial layer become laden with fat and are desquamated (scaled off) into the lumen to form pigeon "milk" for feeding the young.

The proventriculus

The proventriculus is the glandular stomach of the fowl. It differs from the similar mammalian structure, in that its lumen is scarcely larger than that of the esophagus. Its storage capacity is limited. There are the four typical layers in its wall. In the center of each lobule is a cavity, which receives the glandular secretion and is continuous with an excretory duct. Several excretory ducts empty into the lumen of the proventriculus through large papillae. The free ends of the cells of the tubular glands are directed toward the central cavity and do not touch adjacent cells, thus giving a separated appearance to the glandular epithelium. The glands of the proventriculus secrete gastric juice, which contains mucus, pepsin, certain salts, and hydrochloric acid for digestion of nutrients.

The gizzard

The gizzard (ventriculus) is a spheroidal organ, flattened in the lateral direction. Its two lateral sides are biconvex discs. There is a dorsal and a ventral muscular mass. These muscular masses are of a red color, but non-striated. The lumina (spaces in the interior of the structure) of the glands contain a material with cellular debris, which forms the so-called horny layer of the gizzard. It has wavy lines parallel to the surface and wider lines perpendicular to the surface. The gizzard has a mechanical function; it serves to crush grain.

The small intestine

The intestine of the fowl, although similar to that of mammals, differs markedly in some parts. It is about five to six times the length of the body. The duodenum, which does not have glands of
Brunner, presents a loop supporting the pancreas, and is generally considered to terminate at the entrance of the bile and pancreatic ducts. The *jejenum* and *ileum* are supported by a mesentery, and bounded by air sacs, which separate them from the abdominal wall. There is often a diverticulum (pouch) on this portion of the intestine, which is a remnant of the yolk stalk. The propria (sensory area) contains considerable lymphoid tissue and lymph nodules. *Crypts of Lieberkühn* open into the lumen of the gut between the bases of adjacent villi. The small intestine is the primary organ where digestion and absorption of nutrients takes place. Digestion takes about 10 hours to complete in the chicken.

**Figure 1.4. Digestive System**

The ceca and large intestine

Beyond the small intestine, the bowel presents two retrograde (going backward) portions (the ceca) and a continuing portion (the large intestine). The paired ceca extend interiorly for some nine inches parallel to the ileum. The ceca are attached by peritoneal folds. The villi are well developed near the mouths of the sacs, shorter and broader in the mid-portions, and in the fundus (lowest parts of the sac) the villi are low and blunt. The glands of *Lieberkühn* are poorly developed. In older birds, lymph and much lymphoid tissue are present in the *propria*. The large intestine has numerous villi for absorption of nutrients. The ceca absorb water from the stool.

The rectum

The rectum is that portion of the bowel between the cecal orifices (openings) and the beginning of the cloaca. The structure of the rectal wall resembles that of the small intestine. The glands of *Lieberkühn* are, however, much smaller and fewer. A slight constriction is usually present to mark the termination of the rectum.

The cloaca

Usually, definite circular folds delineate the three portions of the cloaca (the *coprodaeum*, the *urodaeum*, and the *proctodaeum*). The *coprodaeum* is the passageway between the rectum and *urodaeum*. The *urodaeum* continues the passageway between the *coprodaeum* and the *proctodaeum* and, in addition, receives the ureters and genital tubes. Between the *urodaeum* and
the proctodaeum, the limiting fold on the dorsal side guards the entrance to the bursa of Fabricius. The wall of the cloaca has a structure similar to that of the rectum and small intestine.

The bursa of Fabricius

The bursa (of Fabricius) cloacae in the chicken, extending dorsally from the roof of the proctodaeum, attains a maximum size in four to five weeks, and at about six months has disappeared. The mucosa is much folded, and these folds contain great numbers of lymph nodules. The nodules have a typical lymph-follicle structure (dense periphery with a lighter center). The bursa produces immature (B) cells, which later develop into plasma cells. Plasma cells secrete antibody into circulation. The bursa also produces B cells, which cede to other lymphoid organs such as the spleen, cecal tonsil, and gland of Harder (small gland located behind the eye). The bursa atrophies at sexual maturation. These other lymphoid organs take over the bursa’s function at this time.

The anus

The structure of the anus resembles that of mammals. A sheet of cross-striated muscle, making its appearance at the level of the bursa of Fabricius, extends in the fused propria and submucosa to the borders of the dorsal and ventral anal lips. The end product of protein digestion (uric acid) is voided together with other digested material. The urates are white in birds, the intestinal void is green, and the cecal excrement is light brown.

The liver

The liver (figure 1.5) is the largest gland of the body, and plays an important part in generation of energy. The liver is involved in the following: formation and secretion of bile; formation and storage of glycogen and regulation of glucose content in circulation; deamination of amino acids; desaturation of fatty acids; detoxification of poisonous substances brought to the liver by blood; and aiding the destruction of erythrocytes. The microscopic structure of the liver varies little from that of mammals. A cystic duct and a hepatic duct both empty into the intestine, near the terminations of the pancreatic ducts. The bile produced by the liver and stored in the gall bladder serves as a medium of excretion and as a digestive secretion.

Figure 1.5. Liver
The pancreas

The pancreas of the chicken is a compound tubulo-acinar gland with a structure similar to that found in mammals. There are islets of Langerhans, which secretes juices that can digest protein, carbohydrates and lipids. The chief function of the organ is the production of pancreatic lipases capable of hydrolyzing (saponifying) neutral fats.

The respiratory system

This avian respiratory system (figure 1.6) differs in many ways from mammals'.

The nostrils and nasal cavities

The anterior naris, elliptical in outline with the long axis antero-posterior, may be guarded by small feathers. The two nasal cavities are separated from each other by a septum (a thin wall which divides two cavities) composed of bone and cartilage. Three mucous-membrane-covered plates project from the walls; these correspond to mammalian turbinates or nasal conchae. On each lateral wall of the nasal cavities, ventral (undersurface) to the middle turbinate and at the level of the anterior end of the palate cleft, is the opening of the naso-lacrimal canal. Each posterior naris is connected with the cavity of the pharynx through the palatine cleft.

The larynx and trachea

The larynx is not guarded by an epiglottis. The larynx of the fowl, often called the cranial larynx, also is devoid of thyroid cartilages and vocal cords. The cricoid cartilage is segmented. The cartilaginous rings of the trachea are complete. Outside the tube, there are bundles of skeletal muscles running longitudinally. Two bronchi continue the air passageway into the lungs. At the point where these begin is the caudal larynx, sometimes called the bronchi-tracheal larynx, and syrinx. This is the organ of voice in the bird. It is a median crest at the bifurcation (forking) of the two bronchi, surmounted by an elastic lamina on each of its lateral sides. The syrinx, together with similar membranes laterally in each bronchial wall, serves as vocal cords.

The lungs

The lungs extend from the first ribs to the anterior poles of the kidneys. The medio-lateral dimension of the lungs is relatively short; consequently, they do not project far into the thoracic cavity. The ventro-medial surface is covered in part by the pulmonary diaphragm, while the dorso-lateral surface is deeply grooved by the ribs. The lungs are not divided into lobes, as in many mammals, but distinct lobules are formed about the terminal loops of the tertiary bronchi.

The bronchi are divided into primary, secondary, and tertiary segments. The primary and secondary bronchi each are connected with air sacs. The tertiary bronchi terminate by dividing into great numbers of interconnecting air tubes or capillaries. Avians do not have alveoli. The epithelial lining of these air capillaries may be considered as respiratory epithelium of the lung.
proper. Running between the epithelial layers of adjacent air tubes, are the capillaries of the pulmonary circulation.

The air sacs, which connect with the primary and secondary bronchi, have walls of a thin inner mucosa continuous with that of the bronchi, and an outer serous layer of either pleura (lining membrane) or peritoneum. The paired thoraco-cervical sacs are found at the cervico-thoracic junction. Prolongations extend interiorly through each *Canalis transversarius* as far as the third cervical vertebra, while those going posterior may reach the fourth thoracic vertebra. Many of the adjacent vertebrae are pneumatically connected with these two sacs, which also are connected with each other.

![Respiratory System](image)

**Figure 1.6. Respiratory System**

The anterior thoracic sac is in the anterior part of the thoracic cavity. It is related to the structures there and communicates with pneumatic cavities in the bones of the shoulder girdle, the humerus, the sternal ribs, and sternum. This sac has a right and left axillary diverticulum (sac), which projects into intermuscular spaces of the shoulder. The posterior thoracic air sacs are paired. These are bounded by the pulmonary and thoraco-abdominal diaphragms and do not have connections with air cavities of bones. The lesser abdominal air sacs are paired. These laterally compressed spheroids are located on the anterior part of the abdominal wall at about its middle third. The left is the larger of the paired greater abdominal air sacs. These sacs are co-extensive with the abdominal cavity on each side of the medially located viscera. The sacrum, hip bone, and femur have air connections with these sacs.

The active part of respiration in birds starts with automatic smooth muscle contraction. This causes the ribs to contract the body cavity resulting in negative pressure and the inspiration of (O₂) air through the nasal passages, trachea, bronchi, lungs, air sacs, and bones. When the muscles relax, the rib cage and body cavity are expanded and the air passes into the lungs where exchange occurs with air capillaries. Avians do not have a functional diaphragm or alveoli. The CO₂-containing air is then passed out through the bronchi, trachea, and nasal cavities. The avian respiration system acts like a bellows, expanding and contracting by the action of smooth muscles and the rib cage. The lungs are rather fixed and do not expand as with mammals.
The urinary organs

The kidneys usually have three lobes of unequal size. They are limited anteriorly by the lungs and extend posteriorly through the length of the pelvic cavity, filling in the fossae (trench) formed by the pelvic bones and the vertebrae. The ureters may easily be traced from lobe to lobe and to the terminations in the dorsal wall of the urodaeum of the cloaca. The finer structure of the kidney of the fowl closely resembles that of the same mammalian organ. The kidneys filter out poisonous substances and secrete uric acid, the byproduct of protein (nitrogen) metabolism, out the anus.

The reproductive organs

The male genitalia

The testes (figure 1.7) are intra-abdominal organs in fowls. Each testis is ellipsoidal and is suspended by the mesorchium ventral to the anterior lobe of the corresponding kidney. Growth of the testes correlates positively with age and sexual development and activity. The testes of most birds remain very small until sexual maturity, when they approach the size of those of the adult bird. The testes increase in size during breeding seasons. In chickens, testes begin to secrete semen at approximately 20 weeks of age. Pigmented areas are sometimes present in the testes. From a diminutive epididymis, each vas deferens pursues a flexible course posteriorly to enter the urodaeum through a papilla slightly lateral to the entrance of the ureter. In the chicken no penis is present. However, in the male goose, duck, and ostrich, a penis type of erectile organ may be found. The erectile tissue is composed of lymph spaces. The microscopic structure of the avian testis and vas deferens follows closely that of the same mammalian organs.

Figure 1.7. Testes
The female genitalia

Only the ovary (figure 1.7) and the oviduct on the left side persist in most birds. The ovarian mass of loosely connected yolk follicles lies immediately posterior to the left lung. It is supported dorsally by a fold of peritoneum. Medially it is bounded by the intestine and its mesentery, dorsally by the body wall, and laterally by the left abdominal air sacs. In some birds of prey the right ovary persists, but not the right oviduct. The ova from the right ovary have to enter the left oviduct. Changes in ovarian size and function occur in chickens at about 20 weeks of age. This time can be changed by modifying light and feeding programs. Increasing the light to about 14 to 16 hours per day, and increasing feed will generally bring chickens into production at 22 to 24 weeks of age, depending on breed and body weight. The day length must be kept at about 16 and 1/2 hours per day and constant amount of feed must be given to maintain optimal egg production through the laying cycle (usually about 60 to 65 weeks of age).

Figure 1.8. The reproductive tract of the female domestic fowl.

The newly laid egg is composed of various layers. These will be described going from the outside to the inside. The calciferous shell may be considered to have three strata (layers): an outer homogenous, a middle fibrous, and a deep particulate. An outer thicker part and an inner thinner portion, both composed of interlacing organic strands, compose the shell membrane. At the larger end of the egg, the two portions of the shell membrane are separated by an air space. The white of the egg, or albumen, which composes the greater part of the egg, may be seen to have an outer, more fluid albumen, an inner denser albumen forming the greater part, and the chalazae, which are coils of denser albumen extending from the yolk to the shell membranes at the poles of the egg. A structure-less vitelline membrane surrounds the yolk. In the yolk alternating narrow light and wider dark bands of white and yellow yolk may be seen, respectively. In one portion of the yolk, a mass of white yolk extends from near the vitelline membrane to the center of the yolk. Its central portion is expanded. This mass of white yolk is called the latebra. At the point of its contact with the vitelline membrane there is a pale disc of cells called the blastoderm. The cells of the blastoderm give rise to the embryo. The yolk is composed of microscopic granules or spheres with little interstitial fluid. The specific gravity of the parts of the yolk is such that shortly after turning, the blastodermal portion assumes a position on the upper side.

The oviduct of the fowl is a twisting tube that extends on the left side of the abdominal cavity from the ovary to the urodaeum, into the cavity of which it empties. It is suspended by a dorsal
peritoneal ligament, which is attached to the abdominal roof, while the ventral border of the ventral ligament is free. The oviduct in the laying hen may be as much as 30 inches in length. The clutch cycle of a hen is the number of eggs in a breeding cycle. For wild birds the cycle is normally 1 to 6 eggs, whereas for high producing commercial leghorns it may be as many as 250 eggs. Heavy meat-type birds range from 130 to 180 eggs per clutch. The clutch cycle is generally extended for 40 weeks in commercial poultry by manipulation of feed and light.

The infundibulum, which is the first part of the oviduct, is characterized by a very thin wall and many folds in the mucosa. It receives the ovum. The second portion, called the pars albuminifera, or albumen-secreting region, is characterized by the density of its glandular region in the propria. The third portion, the isthmus, is similar in structure to the preceding region. Most of the albumen is laid down in the second and third segments and the shell membrane in the third portion. In the uterus (the fourth segment), which also, has a glandular propria, the shell is formed. The final or fifth portion is termed the vagina. The mucosa of the vaginal portion presents many longitudinal folds, and the propria (receives sensation) soon becomes free of glands. This portion of the oviduct apparently contributes little, if any, to the structure of the egg, serving simply as a passageway. At the distal end of the oviduct are sperm storage glands. Sperm can be viable as long as 10 days. Sperm swim up the oviduct, and fertilization may take place at the anterior portion of the oviduct.

Molting in laying hens is a system whereby the oviduct atrophies and ceases to produce functional ova. This time (about six weeks in length) is a resting period for commercial poultry. Also during this time the feathers are shed and replaced in a symmetrical pattern. Commercial hens may be force molted by withholding feed and water and/or reducing the photoperiod to about eight hours per day. Commercial meat-type breeders are usually held off feed until they lose 25% of their body weight. This corresponds to about 10 to 14 days off feed depending on the time of year. Water is normally withheld for no more than four days depending on environmental temperatures, or considerable mortality may occur. After feathers have been shed, hens are stimulated to re-enter production by gradually increasing light and feed. Commercial egg layers may be molted as often as two times, whereas broiler breeder hens are only molted once, or not at all, depending on the need for hatching eggs. There is continual pressure from various “animal welfare” groups to discontinue the practice of feed or water withdrawal to induce molting. Other more “humane” practices are currently under investigation.

The organs of the blood and lymph vascular system

The Heart

The heart (figure 1.8) of birds is similar to that of mammals. It may equal as much as 25 percent of the body weight in small rapid-flying birds, while in the chicken it equals 4 to 8 per cent, as compared to 1.5 to 1.7 per cent for man and large domestic animals.

The origin of the arteries carrying blood anteriorly is from the aorta, which first gives off a left brachio-cephalic and then a right brachio-cephalic artery. These give rise to their respective common carotids and subclavians. The latter give off large pectoral arteries and are continued by the brachial arteries to the wings. The common carotids in the neck run close together, ventral to the longus colli muscle for most of the distance. The thoracic portion of the aorta beyond the
origin of the right brachio-cephalic artery and the abdominal aorta gives off the ordinary segmental paired and the unpaired arteries. The external iliac arteries (*S. femoral*) are small. The ischiadic arteries are large arteries, each passing through the sciatic foramen paralleling the sciatic nerve to supply pelvic and posterior-limb muscles. The internal iliac arteries (*S. hypogastric*) are small and supply the pelvic wall. The medial sacral artery is the direct continuation of the aorta.

The larger veins differ somewhat from those of mammals. The jugular veins are not satellites of the common carotids, which are more medially located. These veins parallel the trachea, which are one on each lateral side. They always anastomose (open one structure into another) at the base of the cranium. The right jugular may be of a larger caliber than the left. The jugulars unite with veins, which are satellites of the branches of the brachycephalic arteries, to form two similar trunks, called *anterior venae cavae*. These two, with the posterior vena cava, return the blood of the systemic circulation to the right atrium. The posterior vena cava receives hepatic veins from the liver and two common iliac veins.

Coming from the tail, the *coccygeal* vein sends an anastomosing branch to both internal *iliac* veins, and through the *coccygeo-mesenteric* vein joins the posterior mesenteric vein. The posterior mesenteric, the anterior mesenteric, and the gastroduodenal veins form the portal vein, which is continuous through the hepatic vessels with the hepatic veins. Each internal iliac vein drains the pelvic wall and, after receiving the aforementioned anastomosing (connecting by channels) branch from the coccygeal vein, passes anteriorly through the substance of the kidney, receiving the femoral vein at the junction of the anterior and middle lobes, and later the renal vein, to form a common iliac vein. The two common iliac veins unite to form the short posterior vena cava just anterior to the kidneys. The pulmonary veins, one from each lung, enter the left atrium through a common opening.

![Figure 1.8. Heart](image)

*The lymphatic system*

Ducks and geese have cervico-thoracic and lumbar lymph nodes in which lymphoblastic follicles and germinal centers appear in the first month after hatching. Lymph nodules are present throughout the entire intestine, often replacing parts of the mucous membrane. Birds have two
thoracic ducts, a right and a left, which are divided into thoracic and lumbar portions. Each of these ducts empties into the vena cava cranialis corresponding to its side. The larger lymph vessels of the hen drain either into the thoracic ducts or directly into the venous system, as lymph nodes are not present in the chicken. The spleen is small, brownish-red, and roundish, oval or somewhat disc-shaped. It lies to the right of the glandular stomach. It filters antigens from the blood and produces plasma cells, which later produce antibody.

The nervous system

Meninges

As in mammals, birds have three meninges (membranes covering CNS), Pia mater, arachnoid, and dura mater. The subarachnoid space contains cerebrospinal fluid.

Brain

The brain (figure 1.8) of birds is lissencephalic (smooth) with virtually no convolutions (twists). The lateral ventricles are displaced dorsally by the large corpus striatum. The olfactory bulbs are pointed at the rostral extent of the brain, and the olfactory center of the brain is rather underdeveloped. The cerebral cortex is also underdeveloped, when compared to mammals. In contrast, the corpus striatum is well developed, and is the center for association in birds. Birds do not have a corpus callosum or septum pellucidum.

The diencephalon is the location of the pineal body, which lies dorsally and medially between the caudal (tail end) aspects of the cerebral hemispheres. It is composed of secretory cells, which resemble rudimentary photo-receptors. The nonmyelinated axons are responsive to light. The pineal body is involved in reproduction, migration and circadian rhythms. Its secretions exert their effect on the hypothalamus. The hypothalamus is located caudally (or ventrical) to the optic chiasm.

Midbrain

The midbrain is the location of the optic tectum. The avian optic lobes are massive and responsible for the well developed, monocular vision. The optic tectum is equivalent to the rostral colliculus of mammals, and birds have no caudal colliculus.

Cerebellum

The cerebellum is the center for coordination of movements and is large. It has a single median lobe with transverse sulci (groves), and is divided into three main lobes (not four as in mammals) by the fissura prima and fissura secunda. Each lobule is believed to be responsible for coordination of a specific part of the body.
Pons

The pons is poorly developed and present only as a broad band of fibers at the rostral (beak-shaped) portion of the medulla oblongata.

Cranial Nerves

The cranial nerves of birds correspond to those found in mammals. The olfactory nerve (CNI) is sensory; the optic nerve (CNII) is sensory and large; the oculomotor nerve (CNIII) is motor to the dorsal, central and medial rectus, and supplies the muscles to the eyelids. The trochlear nerve (CNIV) is motor to the dorsal oblique muscle of the eye. The trigeminal nerve (CNV) contains both sensory and motor fibers. The abducent nerve (CNVI) is motor to the lateral rectus muscles and muscles of the third eyelid. The facial nerve (CNVII) is motor with a sensory component and parasympathetic fibers. The vestibulocochlear nerve (CNVIII) has separate ganglia for the vestibular and the cochlear nerves. The glossopharyngeal nerve (CNIX) is sensory and motor. Cranial nerves IX, X, and XI arise from a row of small roots. Cranial nerves X and XI combine in a common ganglion (group of nerve cell bodies). The lingual branch of the glossopharyngeal nerve receives sensory input from taste fibers.

The vagus nerve (CNX, CNXI) and jugular vein course down the neck in a common sheath to the level of the nodose ganglion. The spinal accessory nerve (CNXI) is enclosed with the vagus and supplies the superficial muscles of the neck. The hypoglossal nerve (CNXII) and (CNIX) innervate the tracheal muscles. The former has a lingual branch that innervates the tongue muscles, and a syringeal branch, which connects to the syrinx and tracheal muscles.

Figure 1.9. Brain

Spinal Nerves

The lumbar spinal cord contains a dorsal gelatinous structure (glycogen body), which is nestled in a deep cleft between the dorsal columns. The brachial plexus (interjoining nerves) is formed by ventral branches of three to five spinal nerves. There are three nerve plexuses in the lumbosacral region (lumbar, ischiatic, and pudendal). These nerve roots lie embedded in the pelvis surrounded by the kidneys. All three plexus are connected to the sympathetic chain. The
The lumbar plexus is composed of three to four nerve roots (the last two lumbar and first sacral roots). It innervates the cranial thigh muscles and the muscles of the body wall. The obturator nerve, femoral nerve, cranial gluteal nerve and saphenous nerve arise from this plexus.

The ischiatic plexus, or sacral plexus, is usually made up of six sacral nerve roots. The first root arises with the last root of the lumbar plexus. The roots combine to form the ischiatic nerve, which is the largest nerve in the body. This nerve should be faintly striated, and a loss of striations is suggestive of an inflammatory process. The caudal gluteal nerve also arises from the sacral plexus.

**Organs of special sense**

The eye

The lower eyelid is somewhat larger than the upper. Eyelashes are replaced by fine feathers. A very extensive nictating membrane is present. A gland commonly called the gland of Harder is located behind the eye. It produces lymphoid tissue and plasma cells, which develop into antibody producing cells.

The structure of the wall of the eyeball differs somewhat from mammals. The sclera has an inner layer of hyaline cartilage, and a ring of body plates is found at the corneo-scleral junction. Projecting into the vitreous, from an otherwise avascular retina, is the pecten. It is a vascular and pigmented folded structure with its base on the optic disc.

**The ear**

The auricula (external part of the ear) is absent. The external acoustic meatus is guarded by some small feathers in most birds. One ossicle (bone), termed the columella, spans the space between the tympanic membrane and the fenestra ovalis. The middle ear extends into air spaces in the basi-occipital and basi-sphenoid bones. A slightly bent tube forms the cochlea.

**The olfactory organ**

The distribution of the olfactory nerves is similar to that for mammals.

**The organ of touch and structure of the skin and its appendages**

Touch corpuscles are found in large numbers in various places in the skin. They are especially plentiful in the beak, in head and neck appendages, and in the wall of the cloaca. The structure of the skin is similar to that of mammals. However, sudoriferous (sweat) glands and Sebaceous (secrets fatty substances) glands are not evident. There is, however, a large uropygial gland (oil gland) located dorsally to the last sacral vertebra. This is a bilobed gland of the sebaceous type. From a sinus in the center of each lobe, the secretion passes by way of a tube to the outside through a single papilla common to the two lobes. It is most highly developed in waterfowl. Oil from this gland aids in body flotation and thermoregulation.
The skin of the metatarsal and of the digital regions of the foot presents horny scales. These scales, especially on the dorsal surfaces of the appendages, become harder and coarser with age. Many fowl have a spur on the medial side of the metatarsal region. It is a bony process on the metatarsal bone. Its horny covering appears to grow from base to apex (tip). The size of the claws varies from rudimentary in waterfowl to well-developed structures in birds of prey. Pads form cushions on the plantar (sole) sides of the digits for the absorption of shock. Webs between the digits vary in extent in different waterfowl. In the deeper layers of the skin, there is considerable muscle, which is attached to the follicles of the feathers.

The comb of fowls has a small core of adipose (fat) tissue, which is narrowed posteriorly. Thin, dense connective tissue laminae separate this core from a layer of muco-elastic tissue. This latter layer is composed of large anastomosing cells with a fine reticulum in their cytoplasm. In the meshes there is clear mucus that is slightly stainable by ordinary methods, but specifically stained by mucicarmine and mucihematin. There are many elastic fibers, from which collagenous fibers, extend from the laminae to the subepithelial zone of connective tissue. Large blood vessels of the core are connected by capillaries with a great number of sinusoidal vessels in the subepidermal connective tissue. There is a well-developed nerve supply with many pacinian corpuscles.

The cone-shaped comb of the turkey, called the snood, has large fasciculi (sheet) of smooth muscle around a dense connective tissue core. This core contains erectile tissue. Its arteries are medium-sized with heavy muscular walls. The subepithelial sinusoids (thin-walled vessel) are sparse while the subepithelial arteries are more numerous. The muco-elastic layer may be absent.

Wattles have a structure somewhat similar to that of the comb. The fatty core may be reduced or interrupted, and the muco-elastic layer may be more hyaline in character. Combs, snoods, and wattles serve in the thermo-regulation and are accessory sexual attraction organs in the male. During courtship the snood of the male may become dark red and erect to attract the female. This is accomplished by increasing circulation of blood to the organ.

Feathers (figure 1.10) are epidermal structures similar in that respect to hair, and also similar in that the proximal end is in a follicle. A typical feather has a tubular shaft or axis. The proximal portion of the shaft is called the quill or calamus. It is a cornified, somewhat translucent tubule, the lumen of which contains cellular debris and air spaces. The follicular end presents an opening, the proximal umbilicus, into which a dermal papilla projects. At the distal part of the quill, there is a foramen called the distal umbilicus. Closely associated with the distal umbilicus and projecting from the shaft is a small feather termed the hyporachis.

Distal (toward the periphery) to the quill or calamus is the part of the shaft called the rachis. It is grooved on the side facing the body. This portion, with its two rows of barbs, forms the vane or vexillum. The barbs are lamellae, which arise from the rachis and are directed obliquely distally. In many feathers each barb has a distal row of barbules, which have several hooklets near their terminal ends. The distal row of barbules overlaps a row of proximal barbules located on the succeeding barb. The barbules of this proximal row are curved. The concavity of the curve lies immediately deeply to the ends of the distal row of barbules. This interlocking device makes of the vane an elastic membrane providing resistance to air during flying. In downy types of feathers, barbules may be partly or wholly absent. The shaft, too, may be considerably reduced.
Molting (shedding of feathers) is accompanied by the formation of a new feather, which arises in the follicle already present and pushes out the old feather. As is the case of the distribution of hair in mammals, feathers are arranged in areas and lines, which are termed *pteryleae*. "Apteria" are the intervening bare areas.

![Image of feathers of the domestic fowl.](image1)

**Figure 1.10. Feathers of the domestic fowl.**

![Diagram of a Male and Female Chickens.](image2)

**Figure 1.11. Diagram of a Male and Female Chickens**
The endocrine organs

The endocrine system consists of the hypothalamic-hypophyseal complex, the gonads, pancreatic islet cells, adrenal glands, thyroid glands, parathyroid glands, ultimobranchial glands, and the endocrine cells of the gut. These organs release hormones into the bloodstream, which act on target tissues by interacting with receptors on the surface of the cell (peptide hormones), or within the cytoplasm or nucleus of the cell (steroid hormones).

The hypothalamus is a small structure that occupies about 3% of the total brain volume and forms a large portion of the ventral diencephalon. Various neural cell clusters can be recognized in the hypothalamus. Anteriorly, the most prominent are the preoptic nucleus, the infundibular nucleus, and paraventricular nucleus. The infundibular nucleus and the medial posterior hypothalamic nucleus are found in the posterior, or tuberal hypothalamus.

The hypophysis, or pituitary gland, is connected to the hypothalamus. The pituitary gland consists of an adenohypophysis and a neurohypophysis (pars nervosa, neural lobe). Avian neurohypophyseal hormones, arginine vasotocin (ART) and mesotocin (MT), reduce urine production. Adenohypophyseal hormones are either glycoproteins or polypeptides. The glycoprotein hormones are luteinizing hormone (LH), follicle stimulating hormone (FSH) and thyroid stimulating hormone (TSH) and thyrotropin. The Adenohypophyseal polypeptide hormones are growth hormone (GH, somatotropin), prolactin- and proopiomelanocortin-(PANC)-derived hormones such as adrenocorticotropic hormone (ACTH) and B-melanocyte stimulating hormone.

Both LH and FSH stimulate avian steroid synthesis. Prolactin affects reproduction and osmoregulation in birds. ACTH stimulates corticosterone and aldosterone production by the adrenal cortical cells. The parathyroid hormone (PTH), calcitonin (CT) and 1,2,5 dihydrocholecalciferol (the active metabolite of vitamin D₃) effect calcium metabolism. In the chicken, the left parathyroid gland (figure 1.13) is in contact with the thyroid. During egg laying, PTH functions in the resorption of medullary bone. CT protects the skeleton from excessive calcium resorption from the bone.

The thyroids are paired organs that lie on each side of the trachea in the thoracic inlet. The thyroid produces T₃ and T₄, which are important growth hormones. The right and left adrenals are yellow organs located cranial to the kidneys. The glands receive blood from branches of the renal artery. The secretion of corticosterone is regulated by ACTH. Corticosterone is essential for survival in times of stress, and regulates intermediary metabolism and hemodynamic functions. It has mineralocorticoid activity. Corticosterone balances the production and action of biologically active substances produced during stress (i.e., catecholamine, prostaglandin).

Molting is the result of complex hormonal influences. Molting occurs during a period of depressed sexual activity. It can be suppressed by sex hormones or induced by administration of progesterone. When the duration of light is decreased naturally or artificially and/or the amount of feed and water are decreased, sexual activity declines or ceases and molting begins. An increase in thyroid activity is observed during this process and is connected with the development of new feathers to replace feathers lost during the early stages of molting. Males
can also be forced into a molt resulting in a reduction of their sexual activity and a replacement of body feathers. This process, from the onset to the end of molt and regeneration of feather and sexual reproduction, takes about six weeks. Birds are restimulated to begin reproduction and replace their feathers with increase in photoperiod and/or feed.

The thymus (figure 1.12) is composed of lobules, which may extend throughout the cervical region. Its structure and involution resemble that of mammals. The thymus contains both T and B lymphoid cells involved in the immune response. T cells are involved in processing antigens, stimulation or suppression of B cells to produce antibody, activation of macrophages, and killing of infectious organisms and tumor cells. The thymus involutes at the onset of sexual reproduction. The thymus can regenerate in adults during molting, corresponding to the involution and then regeneration of the reproductive tract.

![Immune system of the chicken](image1)

**Figure 1.12. Immune system of the chicken**

![Parathyroid gland and trachea](image2)

**Figure 1.13. Parathyroid gland (pt) and trachea (t)**
2. Clinical examination

Before examining birds it is important to obtain from the case submitter as much information as possible (figure 2.1). Particular attention should be paid to the following questions:

• What have farm workers or service persons noticed wrong with the birds?

• How many of the flock are ill or have died?

• Has the flock been ill before and has it had any other treatments?

• Have there been any changes in the environment, which may have put it under stress?

• Has the farmer recently received a new feed supply?

• Has there been any recent change in eating habits? Have the droppings changed in character (size, color, consistency).

Other questions will occur to the experienced person, and the answers should be sought from the farmer. However, individuals vary greatly in their powers of observation and the diagnostician may find it rewarding to bring back additional ill and healthy birds to the lab so that a more accurate observation can be carried out.

Examination of the birds and cage

Figure 2.0. Soiled vent area (diarrhea)                  Figure 2.1. Normal laying hen
The character of the droppings

Always try to examine fresh droppings. The cloacal excreta (figure 2.0) usually consist of a dark-colored central part (from the rectum) and an off-white-colored surrounding portion consisting mainly of urate crystals from the kidneys. The consistency, and to some extent the color of the droppings, vary with the species and the diet of the bird.

As is to be expected in birds with enteritis, the dark, central part of the droppings becomes more fluid; the reverse is true in constipation. However, in birds that are not feeding or that are feeding inadequately, the central part of the droppings tends to be of a watery, greenish nature. Birds with pancreatic disease show excessive droppings that are grey in color and waxy in texture. Test these for starch with Lugol's iodine. Excessive or decreased urate crystals indicate a renal problem. Undigested corn in the droppings is always abnormal and may indicate a malfunction of the gizzard.

Blood in the droppings may come from the intestines, the rectum, the cloaca, or the oviduct. This may indicate ulceration, possibly involving neoplasm. Sharp foreign bodies, such as pieces of metal, can be ingested and can reach the rectum in some birds. Blood may also indicate bacterial, viral, or protozoal enteritis. Yellow colored droppings are associated with blackhead, Psittacosis, cholera, or typhoid.

Blood in the coops or carry device

Blood spattered around the coop maybe from the cloacal or from an injury to the wings, feet, beak, or body. If the blood is spread, it is probably from wing trauma, possibly a damaged growing feather.

Regurgitation

Examine the carrying device for any evidence of adherent small flecks of white material. This may be evidence of regurgitation. Liquid or putty-like castings, or those with blood or excessive mucous, are abnormal. These signs can be seen with Capillaria, Trichomoniasis, T-2 toxicosis, vitamin A deficiency, and/or Candidia.

Other observations to be made on the coop

With a magnifying lens it may be possible to see signs of parasitic mites or lice on the coop. These appear as minute black, red, orange, or grayish-white specks, which are seen to move. Mites hide in cracks and crevices and emerge to feed on the bird at night, so they are best seen with a torch in a dark room or when the electric light is switched on suddenly.

Observation of the birds

The problem with most sick birds is that usually by the time someone realizes that they are ill, they are very ill. The bird should have well-rounded and bright eyes. An eye, which is slightly oval means that the bird is not fully alert. Any bird that spends its time huddled near the bottom of the coop, taking no notice of an observer, is near death.
The plumage of the bird should be sleek and lie flat over the body. If all the body feathers are ruffled, the bird is trying to conserve heat loss, a common sign of sickness.

**Breathing abnormalities**

A bird that is obviously dyspneic (labored breathing) with its mouth open and gasping, may not necessarily have a respiratory condition, but is certainly ill (figure 2.2). “Tail-bobbing” in small birds is a sign of an impaired respiratory system. In both these types of abnormal breathing, a space-occupying lesion (abnormal appearance) of the abdomen may prevent the full expansion and contraction of the posterior air sacs, so that airflow through the lungs is reduced. *Cyanosis* (lack of oxygen) is sometimes indicated by a blue coloration of the beak and legs. If the part of the neck in the region of the crop slightly inflates with each expiration, but breathing is otherwise normal, this may indicate obstruction of the outlet ostia (entrance) of the anterior air sacs, where these connect with the secondary bronchi.

A change in the voice, which becomes harsh, or a change in the pitch, could indicate a problem with the syrinx. Hypovitaminosis A with or without secondary bacterial infection and abscessation involving the tissues of the syrinx could be responsible for these signs. Clicking or asthmatic noises, which may be almost imperceptible unless carefully listened for, can be caused by viral, bacterial, fungal, or yeast infection of the respiratory tract, or by the nematode, *Syngamus trachea*, which affects many species of birds. In the latter case, obstruction of the airflow in the trachea is enough to cause gaping typical of the disease.

A change of voice in a bird always indicates a pathological condition of the syrinx and the prognosis (outcome) is more serious. This contrasts with the situation in the mammal where a change in voice indicates an upper respiratory condition and the outlook is more favorable.

![Figure 2.2. Stretch neck may indicate abnormal breathing](image-url)
Central nervous system signs

Birds may show any of the following signs: torticollis (twisting of the head), opisthotonos (arching of the head and neck backwards), ataxia (difficulty in moving), circling, paralysis, and clonic (contracting and relaxing) spasms or fits. All these may be caused by deficiency of B or E vitamins, infectious disease, poisoning, concussion, cerebral vascular disturbances, and tumors. Most important among the infectious diseases causing nervous signs are Newcastle disease (ND), Marek's disease (MD) and Avian encephalomyelitis (AE), which affect all species. The variant of Paramyxovirus, causes nervous signs in pigeons both domestic and feral.

A flaccid (no tone) paralysis with an inability to hold the neck up ("limber neck") is seen in botulism and lead poisoning (figure 2.3). Folic acid deficiency can also cause paralysis of the neck in turkey poults.

![Figure 2.3. Paralysis indicative of botulism](image)

Handling birds

When handling larger birds, care should be taken to control the feet, which have a powerful grip, and also to watch the beaks of the larger birds, which can cause a severe biting injury. All birds are best cast on a cushion or soft surface before examination. The wings need to be held gently but firmly to the body with no undue pressure placed on the thorax.

Physical examination of the restrained bird

Feathers and plumage

The plumage have a good, even, dense color. Barbules should lock together so that the feathering gives a uniform outline to the body form. In the healthy bird, only the axillae are sparsely covered in feathers. If the areas of skin covering the lumbo-sacral and sternal regions are thinly covered or are covered in an abnormal grayish fluff instead of the usual contour feathers, the cause may be of nutritional or endocrine origin, e.g., thyroid. Progressive feather loss with a typical white, flaky, but thickened skin may be due to ringworm (Trichophyton spp.) infection, particularly if this is seen around the head and neck. A nutritional deficiency may cause
dermatitis and failure of feather growth. Examine new feathers to see if they are short and club-shaped. See if they have a circumferential construction or are curled or deformed. Any of these signs may indicate a viral infection or nutrient deficiency. Self-mutilation may be initiated by parasitic infection.

Mite infestation may lead to invasion of the feather follicle, and damage and loss of the feather. Both mites and lice can cause irritation. A careful search of the plumage will show any lice situated along the feather shaft or on the skin surface. Healthy birds groom themselves to keep infestation in check, whereas sick birds do not. Examination of the skin or of the powdery remains of a feather shaft with a magnifying lens will be necessary to identify mites. Feather picking by an incompatible or dominant cage mate is not uncommon. This may be worse during the breeding season. Older hens may be completely devoid of feathers on their back as a result of continuous mating by aggressive males.

Malformed and curled flight feathers, or those without proper vane formation, are usually the result of faulty nutrition (inadequate essential amino acids or vitamin deficiency), but may also be the result of chewing by lice or other infections. Yellowing of green feathers may be due to a deficiency of the amino acid lysine. Feathers that are frayed, or have the shaft cleanly broken or snapped off, are the result of careless handling or inadequate caging. Molting or feather replacement takes place in most birds at well-defined intervals—once, twice or three times a year. Nutritional or infectious conditions that cause feather abnormalities often have similar effects on the germinative cells of the beak and claws.

Occasionally a developing feather will fail to emerge properly from the feather sheath. The follicle continues to enlarge pushing its way below the surface of the skin and a feather cyst is formed. The cyst is often associated with an inflammatory condition of the skin and causes marked irritation to the bird, so that the bird picks at the cyst and may rupture it.

**The head region**

After detailed examination of the plumage it is best to continue with an examination of the head region starting with the eye (figure 2.4).

![Figure 2.4. Swollen head](image)
The eye

The observer may see a variety of conditions. Keratitis is an inflammation of the cornea. Edema of the eyelids due to a foreign body is relatively common. Matting of the feathers around the eye may be unilateral (one side) or bilateral (both sides). If bilateral, this could be due to lesions blocking the opening of the naso-lacrimal ducts, where they are situated close together in the posterior part of the choanal opening. Swellings just above or below the eye may be evidence of sinusitis of the supraorbital or infraorbital sinuses, which may have progressed to abscess formation. Brown, crusty eruptions around the eyelids and commissures (corners) of the beak may be due to avian pox. AE may cause cataracts (figure 2.5). MD can cause tumors in the pupil or iris. Foaming of the eye is common with many viral and/or mycoplasma infections.

![Figure 2.5. Cataract due to AE](image)

The beak

Examine the beak for any evidence of cracking or splitting, which may be a sign of underlying fractures of the premaxilla or mandible. Care should be taken when examining some birds such as ducks in which the edges of the beaks are quite sharp. Cracking of the horny beak may be traumatic or a sign of vitamin A deficiency or infection. Overgrowth or distortion of the beak may be due to a neoplasms (e.g., osteosarcoma) or trauma to the proliferating epidermal cells. Deficiency of vitamin D, calcium, biotin, and B vitamins may cause abnormal beak formation.

The mouth and oropharynx

A speculum may be necessary to examine the mouth of a conscious powerful bird. A pair of artery forceps can be placed between the two beaks and then opened. There is considerable inter-species variation both in the anatomy and mobility of the avian tongue. Abscesses are seen sometimes on the surface and small pinpoint lesions of Candidiasis may be observed also. Both these conditions may be brought on by vitamin A deficiency. This leads to a hyperkeratosis of the epithelium of the mucus-secreting glands. Abscesses may also be seen anywhere on the mucus membranes of the mouth, particularly around the choanae where they block the naso-lacrimal opening. Closer inspection of the nasal mucus membrane can be carried out by endoscope examination through the choanal space.
Abscesses in the mouth may be bacterial, in origin, or may be early signs of *Trichomoniasis*. This is seen more usually as an extensive cheese-like diphtheritic (false) membrane covering the oropharynx and sides of the mouth. Again, hypovitaminosis A may predispose to this condition. The lesions of *Trichomoniasis*, *Candidiasis*, fowl pox and T-2 toxicosis, look very similar and may occasionally be confused with *Capillaria* infection (figure 2.6). Avian pox lesions may be seen at the commissures of the beak, in all species. Lesions should be scraped and examined on a wet mount to determine if an organism is the cause of the lesion. Microscopic examination of tissue sections of the mouth can be used to differentiate T-2 toxicosis.

![Figure 2.6. Oral lesions due to Trichomoniasis](image)

The glottis is a slit like opening into the larynx and trachea lying on the floor of the mouth, usually just posterior to the root of the tongue. Neoplasms (tumors) and exudative lesions can affect this area resulting in partial obstruction of the airways. Sinusitis of the infraorbital sinuses can lead to a gross swelling, filled with catarrhal (cloudy) exudate, on both sides of the oropharynx. This condition can be caused by mycoplasma and has been seen in a number of species. Examination of the mouth and oropharynx and laboratory culturing of any exudate obtained should be done.

**The neck**

This should be palpated for any swelling, which may indicate a foreign body impacted in the esophagus (e.g., an impaction of the crop), which can occur in most species. A fluid swelling may be due to the condition of "sour crop", when there may be excessive gas present also. Swelling of the esophagus or crop may occur with *Trichomoniasis*, *Capillaria*, or vitamin A deficiency.

*Examination of the body*
The condition of the pectoral (breast) muscles should be assessed by palpation. They should be symmetrical, but one side may have undergone atrophy, in which case the bird's flying ability will be affected. The condition of the pectoral muscles is an important guide to the overall nutritional state of the bird. The sternum can be felt but should not be very prominent.

**The region of the thoracic vertebrae and the syncrosacrum**

These areas should be carefully examined for wounds caused by predators, fighting or trauma. The preen (uropygial or oil) gland should be examined for impaction or neoplastic (cancer) changes.

**The abdomen**

In the larger birds, it may be possible to palpate the tip of the liver beyond the edge of the sternum. Should the liver be easily felt it is probably enlarged. The ease with which the abdominal contents can be palpated will obviously depend on the size of the bird. In younger birds it is almost impossible to carry out safely without putting too much pressure on the air sacs. However, it is possible to distinguish a fairly large, rather irregular neoplasm from a regular, smooth and rounded retained egg in the female. The female often has a history of laying several eggs, then may suddenly stop, and is often noticeably unwell. Occasionally a solitary egg may form and cause obstruction. In older birds, the thick-walled gizzard is easily palpated; a firm and globular mass, with angular margins and its retained grit can be felt to grate between the fingers.

Softer and more fluid enlargements of the abdomen which can become quite pendulant (swinging sac like), sometimes without apparent ill effect, may be due to either ascities or rupture of the abdominal muscles. Ascities (water belly) can be confirmed by very careful paracentesis (withdrawal with a needle and syringe). This is carried out in the midline at the most pendulant part of the swelling. Ascities is often due to neoplasia of the liver or gonads, bacterial infections, or right ventricular hypertrophy. In females, a soft abdominal swelling may be due to an enlarged oviduct caused by salpingitis. This may be accompanied by egg peritonitis and a noticeable illness.

The cloaca should be palpated. It may contain a calculus (stone) of impacted urate crystals or show a prolapse. Digital exploration of the cloaca in a larger bird with a well-lubricated, gloved finger and microscopical examination of the evacuations is helpful. Matting of the feathers around the cloaca together with excoriation (scratch) of the surrounding skin can indicate either an alimentary or urinary problem. If the adherent mass is mainly composed of fecal material, then the problem is probably due to diarrhea. If the secretions are white, and if accompanied by an impacted cloaca, the bird has a kidney problem. Since the urodeum is the posterior part of the cloaca in which the urates from the kidney and ureters collect, an impaction in this region due to a urate calculus will necessarily hold up the evacuation of fecal matter in the anterior part of the cloaca or corprodeum, and the bird will become constipated.

The body temperature of a bird can be taken via the cloaca, but since there is such a great inter-specific variation as well as a normal diurnal variation in individuals, this is not especially helpful in clinical examination. The body temperature of most birds falls within the range 40-42°C (105-107°C).
**The wings**

Examine each wing bone separately for any evidence of fractures, or luxations (dislocations) of the joints. Excessive mobility of the shoulder joint compared with the other side, together with a wing that is slightly dropped at the side could indicate a rupture of the tendon of the supracoracoid muscle (deep pectoral), which can be confirmed only by surgical exploration. Swellings of the bones may be due to old fractures or to tumors or infections. Swellings and suppuration (formation of pus) of the joints may be due to *Salmonella* or *Staph* causing a chronic arthrosynovitis. In young birds, deformation of the bones may indicate metabolic bone disease due to calcium/phosphorous imbalance in the diet.

**The legs and feet**

Each of the bones of the leg should be examined for any evidence of fractures or luxations. In young birds with nutritional deficiencies this does not happen and the gastrocnemius tendon becomes permanently displaced medially. The bird becomes crippled. This condition is called perosis and is caused by deficiency in biotin, folic acid, manganese, or choline.

The feet should be examined for any evidence of abscesses. This condition, known as "bumblefoot", is commonly seen in domestic waterfowl and poultry; the heavier birds are at greater risk. Bumblefoot abscesses (figure 2.7) may extend as far as the hock, and may erode the bones of the foot. Smaller birds may show abscesses on the feet that may be difficult to distinguish from gout due to accumulations of urate crystals. Bumblefoot may be due to bacterial or mycoplasma infections and is usually associated with overused, damp litter. If the suspected tophi are opened and the contents placed on a slide, confirmation that urates are present can be obtained from the following test: the crystals are mixed with a drop of concentrated nitric acid and carefully evaporated to dryness over a Bunsen burner. A drop of ammonia is then added. If urates are present a mauve (light pick) color will develop.

Figure 2.7. Swollen hocks and foot-pads

Sudden severe drops in egg production and/or shell quality can occur with ND, infectious bronchitis, mycoplasma, influenza, infectious coryza, egg drop syndrome, laryngotracheitis, epidemic tremor, heat or cold stress, and mycotoxicosis. Subtle changes in egg production and
egg shell quality (figure 2.8) can occur with vaccination responses, internal parasites, changes in
diet, and additions of certain drugs to the diet. Malformed ovaries (figure 2.9) may be due to a
viral, bacterial, or mycoplasma infections.

Figure 2.8. Runny egg albumin (right) is commonly seen with viral respiratory pathogens

Figure 2.9. Malformed ovary due to a viral infection
3. Laboratory diagnostic procedures

Simple laboratory investigations

Collection of blood samples

There are three main sources from which blood can be obtained: the claw, the jugular vein, and the brachial vein. Usually a 20- to 23-gauge, 5/8- to 1-inch hypodermic needle is found to be satisfactory for collection. Because of the fragile nature of avian veins, hematoma (blood tumor) formation often occurs and pressure should be applied with a swab as soon as the needle is withdrawn. In the larger birds where restraining the wings is a problem, the right jugular vein can be used. The left jugular is much smaller in most species of birds. When using either the brachial or jugular veins, it may be necessary to first pluck a few feathers to see the vein, after which they can be cleansed in the normal way. When blood is collected from a claw, the claw needs to be cut with nail clippers from top to bottom. Cutting the claw from side to side tends to compress the blood vessels and blood does not flow so freely. After clipping the claw, the adjacent, soft tissues of the toe often must be alternatively squeezed and released to obtain sufficient blood. When obtaining a blood sample from the claw, bleeding should be stopped by pressure from a swab, or if necessary, by cauterization using silver nitrate.

Hematology

The hematocrit or packed cell volume (PCV)

With microhaematocrit heparinized centrifuge capillary tubes, determination of PCV is quick and easy. The result provides valuable information. In most birds normal values for PCV can be 30-55%. Values below 27% may represent anemia.

Blood smears

Only one drop of blood is needed for a smear that can provide information on blood parasites and a differential white cell count. Slides can be stained with Leishman's, Wright's, or Giemsa stain. However, avian blood does need a somewhat longer staining period than mammalian blood, at least 5 minutes, and the buffered water used for washing the slide after staining needs to be more acid, pH 5 instead of pH 7, and should be left on the slide for at least 5 minutes. Avian white cells can be more difficult to find than the corresponding mammalian cells. Apart from the fact that the avian red cell is nucleated, the leukocytes in the blood smear are scattered throughout the slide and not aggregated at the edges of the smear as in the case of mammals. There is also much more variation in the appearance of leukocytes in avian blood. Blood smears can show leucocytozoan parasites (figure 3.0).
Total red and white cell counts and hemoglobin values

All these values can be obtained by using standard techniques. As a guide, the values for total red cell counts range from 2.5-4.5 million mm$^3$ with a mean value of 3.5 million mm$^3$. The figures for hemoglobin vary from 11 g to 19 g per 100 ml of blood.

The counting of white cells is more difficult because the nucleated avian red cells cannot be selectively lysed (broken up) to make counting of the white cells easy (figure 3.1). As with the differential white cell count, the total white cell count should be done in a specialist laboratory.
**Clinical biochemical information**

The simplest value to be determined, and the one, which is a useful prognostic guide, is total serum protein. For small birds one drop of serum, obtained after carrying out a PCV in a microhematocrit, can be placed in a hand-held refractometer. Normal values range from 5.2 to 2.5 g/dl, with a mean value of 3.8 g/dl. A value below 2.3 g/dl indicates a poor prognosis.

**Microbiological investigations**

Since bacteria and fungi take an important part in the development of avian disease, the diagnostician should try to establish what potential pathogens (disease causing agents) are present. However, it is easy to make a hurried decision and conclude that a single microorganism is the sole cause of the disease process.

Birds pick up a variety of microorganisms from their contacts such as wild birds, rodents, and human handlers. Birds newly introduced into a flock, as is practiced by bringing in young males in a breeder flock to increase fertility (spiking), may introduce pathogenic organisms into the flock.

Bacteriological swabs can be taken from a variety of sites such as bumblefoot abscesses, suspected cysts, wounds and natural orifices including the trachea. They can also be taken after paracentesis (passage of needle into the cavity to aspirate fluid) of abdominal fluid. In the first instance they should be cultured on blood agar plates at 37°C, and the organism checked for antibiotic sensitivity.

Fecal swabs are best obtained direct from the cloaca, the vent having first been cleaned and sterilized with a quaternary ammonium antiseptic. If this is not possible the swab can be taken from fecal droppings on a clean surface. When a bird is first handled it will often eject fresh fecal matter from the proctodeum and this can be utilized. Fecal swabs should be routinely cultured on blood agar and MacConkey agar plates. If *Salmonellae* are suspected, enriched culture media will be needed and culture is best carried out by a specialized laboratory. *Salmonellae* are found in many species of wild birds and rodents and are easily spread to aviary birds by fecal contamination. *Salmonella typhimurium* is one of the most common specific organisms in this group that can be isolated from birds.

It should be noted that a wide variety of bacteria are normal commensals (beneficial flora) in the gut of many birds and these may be pathogenic only if the bird is subjected to stress. A careful assessment of the bird is necessary before one can be reasonably certain that the organism isolated is causing the disease. To some extent the spectrum of avian gut flora is influenced by the diet of the bird. *Escherichia coli* is a normal inhabitant of the gut of domestic poultry. However, *E. coli* is an important pathogen when found in other areas of the body.

Tracheal swabs can be taken, and a human naso-pharyngeal calcium alginate swab is very useful for this purpose. When *Aspergillus* is suspected, swabs should be cultured on Sabouraud's dextrose agar at 37°C for 36 to 48 hours.
Swabs should be taken from a sample of eggs that have failed to hatch. The surface of the egg should be first sterilized with alcohol, before a small hole is made in the shell and a swab used to sample the contents. Swabs should be cultured on blood agar and *MacConkey* agar, because fecal contamination is a common cause of infection of the egg.

When taking swabs from post-mortem specimens, one should take into account that cultures obtained from birds that have been dead more than 24 hours may not be representative. Some organisms, such as *Proteus*, that are normally present in the gut of birds may rapidly invade other organs after death and overgrow other pathogens on a culture plate (figure 3.2). Antibiotic sensitivity testing (figure 3.3) should always be done after detecting the presence of pathogenic bacteria.

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*Figure 3.2. Swabbing for microorganisms*

*Figure 3.3. Antibiotic sensitivity testing*
Examination of immunosuppression

Immunosuppression is a common occurrence in young poultry, and can be brought on by a number of factors including infectious bursal disease, Marek's disease, chicken anemia virus, reoviruses, mycotoxins, Alcaligenes faecalis, heat or cold stress, nutrient deficiencies, pesticides, and certain antibiotics. Common problems associated with immunosuppression include: vaccine failure, an increase in morbidity, mortality and/or condemnation. Other unnoticed changes can be atrophy of the spleen, bursa and/or thymus.

Common measures of immunosuppression include: weight of bursa of Fabricius and spleen, size of the bursa and thymus, histologic observation of lymphoid tissues in the thymus, bursa, spleen or gland of Harder, and antibody responses to common viral vaccines such as Newcastle disease or infectious bronchitis viruses.

Normal bursal weight divided by body weight ratios x 1,000 for 2-6-week-old birds range from 2-4. Ratios below 1.5 indicate atrophy of the bursa and possible immunosuppression. Measurement of shank, thymus and bursa length (figure 3.4) using a microchrometer is also helpful. Shank length is measured from the footpad to the end of the shank at the hock joint. Thymus diameter is measured in mid-neck area and measured across. Thymus length divided by shank length ratios x 10 for normal birds is between 0.9 and 1.5. Bursa diameter divided by shank diameter x 10 for normal birds is between 1.6 and 2.0. Values below these figures may represent atrophy and immunosuppression.

Examination of stained smears

This is quick, and although not conclusive, it is a useful guide to examine stained smears of pus, feces and exudate. These can be stained with Gram's stain, methylene blue (for bipolar staining of Pasteurella), or where avian tuberculosis is suspected, with Ziehl-Nielsen stain. Liver impression smears can also be stained for acid-fast organisms. Where Chlamydia (Psittacosis) infection is suspected, smears can be stained by a modified Ziehl-Nielsen technique to show the intracytoplasmic inclusion bodies. The modified Ziehl-Nielsen technique is carried out as follows: the slide is flooded with dilute carbolfuchsin stain for 10 minutes but is not heated as in normal Ziehl-Nielsen staining. The slide is then washed and decolorized with 0.5% acetic acid--not acid alcohol which is normally used. Decolorization is carried out only for 20 to 30 seconds until the slide is very faintly pink. Counterstain with methylene blue in the normal manner. The tissue cells may then be seen to contain clusters of very small red intracytoplasmic inclusion bodies. However, because of the risk of zoonotic infection, investigation of this disease is best left to specialized laboratories that have the necessary air extraction safety cabinets.

Routine staining of a sample by Gram's method can help in the interpretation of antibiotic sensitivity testing. The stain will indicate the relative numbers and morphology of Gram-negative and Gram-positive bacteria and also if yeasts are present. This technique may show anaerobic bacteria to be present when there is little or no growth with a routine blood agar culture. If Aspergillosis (figure 3.5) is suspected in a post-mortem preparation, direct culture should be made from the lesion on Sabouraud's dextrose agar at 37 C for 48 hours.
Figure 3.4. Normal (right) and tiny (lower left) bursae of Fabricius

Figure 3.5. Septicemic chick
4. Post-mortem examination

There is no other way in which the diagnostic accuracy is increased than by post-mortem examination. The post-mortem exam subsequent to an ante-mortem diagnosis will help increase the diagnostic ability, and indicate ways in which the diagnostic routine can be improved. The service person should bring a number of live and freshly dead birds to the lab. Live birds should include both sick, but not culls, and healthy appearing birds. Other useful specimens include litter, feed, and/or water samples.

To be of maximum value, post-mortem specimens should be as fresh as possible. However, sometimes a farmer will produce a carcass in which death occurred 24-36 hours previously. In this case the carcass should be thoroughly soaked with cold water and placed in a plastic bag with any excess air expressed. The sealed bag should then be put in a refrigerator so that the body is kept at a temperature just above freezing, but not frozen. The feathering helps to retain body heat, so cooling the body as quickly as possible will help to reduce autolytic changes. If the carcass is deep-frozen the fine cellular structures are ruptured by ice crystals. Such tissues are useless for histopathology. In those specimens that are up to 3 days old and in which no precautions have been taken to prevent autolysis, some useful information may often still be obtained.

A post-mortem exam should always be carried out in a systematic and routine order so that nothing is overlooked or forgotten. Having a check-list in hand is invaluable. The observer should realize that the gross lesions seen during post-mortem provide only a tentative diagnosis. Further laboratory tests will be needed for confirmation in most cases. Therefore, it is wise to have certain equipment ready before starting the procedure.

Some equipment (figure 4.0) that may be required during a post-mortem examination, together with an indication of the laboratory techniques that can be used are as follows:

1. A checklist of organs to be examined.
2. Scalpel and blades.
3. Rat-toothed forceps.
5. Alcohol lamp or Bunsen burner.
6. Bacteriological culture plates, blood agar and MacConkey agar.
7. Bacteriological swabs.
8. Transport media. All living specimens have a better chance of survival if placed in transport media. This can be obtained commercially or prepared using saline, cell culture media, nutrient media or calf serum.
9. Screw-top containers with 10% formol saline that should preferably be buffered. When harvesting solid organs, specimens should not be more than 0.5 cm thick, otherwise the formalin will not fully penetrate the tissue. At least 10 times the volume of preservative as the specimen is required.
10. Screw-top sterile containers to contain tissues for bacteriological culture.
11. Slides can be used for the following:

a. bacteriological staining; if the slide is sterilized by heating in the flame, the bacteriological swab will remain uncontaminated after the smear has been made on the sterile slide.

b. slides can be used to make impression smears from liver, spleen and air sacs. Liver smears can be stained with modified Ziehl-Nielsen or Macchiavello's stain for chlamydia inclusion bodies or with Hematoxylin and eosin for herpes virus inclusion bodies seen in some diseases such as laryngotracheitis or fowl pox.

c. If the bacteriological swab is rolled along the sterile slide instead of smeared across it, it can be stained with Wright's or Leishman's stain and then used for cytological examination.

12. Strong scissors or even bone shears for large birds.

13. Some sterile gauze swabs are sometimes useful if a blood vessel is inadvertently punctured.

14. Sterile syringes and needles for the sterile collection of heart, blood and intestinal contents for culture. If the ingesta are too viscid, a little sterile normal saline injected into the lumen of the gut will make them more fluid. To collect sterile samples from the interior of unopened hollow organs, first sterilize the surface by searing with a hot spatula before inserting the needle.

15. At least one pair of sterile petri dishes to collect tissues for virus isolation.

16. Good lighting possibly combined with a magnifying lens is a great help.

17. A suitable board and dissecting needles for pinning out small birds.

Always wear gloves and a mask. Apart from the risks of chlamydia infection there are many other avian zoonoses including Newcastle disease (ND), Pasteurellosis, Salmonellosis, avian influenza, and Ersipellosis.

If alive, the bird may be killed by the following:

1. Administration of CO₂ gas in an appropriate closed container.

2. Disarticulation (breaking the spinal cord) of the head at the atlanto-occipital joint, using the hands or a large clamping device (figure 4.1).

3. Injection of 10-25cc air directly into the heart.

4. Cutting the jugular vein.

**External examination**

Prior to opening the body, a thorough examination should be carried out looking for any of the external signs. External parasites are often a lot more obvious on the dead body as they move
away from the cooling surface of the skin. The specimen should next be saturated with a quaternary ammonium antiseptic. This reduces the amount of airborne feather debris. Feather dust can carry *Chlamydia* and other organisms and also contaminates the viscera when the carcass is opened.

Plucking the whole body is unnecessary. The removal of feathers along the mid-line in densely feathered species, such as ducks, does help to make it easier to incise the skin cleanly without damaging the underlying viscera.

![Figure 4.0. Necropsy equipment](image)

![Figure 4.1. Cervical Dislocation](image)

**Opening the body**

The initial incision is made through the skin, from the cranial end of the sternum to just in front of the vent. The cut is then extended on each side just along the caudal edge of the sternal plate. By blunt dissection the skin is then eased away from the underlying pectoralis muscle and at the same time the condition of the muscle is observed.
**The pectoralis muscle**

Both sides should be similar and well-rounded. If the two muscles are not symmetrical, this may indicate an old injury or contracted abscess. The muscle should be of normal red color showing no sign of anemia, hyperemia or bruising. The latter may be anything from blue-black to green (within 24 hours) in color depending on how long previously the bruise occurred. Discoloration of abdominal muscles may occur if the bird has been dead for a prolonged period. Bacterial invasion from the gut or gall bladder into the surrounding tissue can be relatively rapid in the uncooled carcass. Incise the pectoralis muscle and look for any evidence of petechial hemorrhages in the muscle which could indicate Warfarin (rodent poison) toxicity or vitamin K deficiency.

**Skin**

Examine the skin for signs of necrotic dermatitis, tumors, exudative feathers or blood sucking parasites.

**Exposing the viscera**

The skin incisions are now deepened through the muscle, and the lateral incisions are now extended to the level of the costochondral junctions, which are either cut with scissors, or, in small birds, dislocated by pressure from the handle of a scalpel. Do not cut through the coracoids or clavicles at this stage, as this may damage the large blood vessels. The sternum now can be lifted upwards away from the underlying viscera. As this is done, examine the underside (anatomically the dorsal aspect) of the sternum together with the general appearance of the organs. If the body cavity is filled with exudate, take swabs for culture. Decide if the color of the tissues looks a normal pink or is hyperemic indicating a possible septicemia. Discoloration, due to hypostatic congestion, on one side only, would indicate the bird had been left for some time lying on that side after death.

The carcass may look anemic (pale). Even if heavy infestation with blood-sucking parasites had been noticed initially, there may also be other less obvious contributing factors. The muscle may look dry, indicating dehydration; or shrunken, indicating cachexia (wasting away).

**Examination of the viscera before removal from the body**

The signs of airsacculitis may be seen and will become more evident as the post-mortem proceeds. During the initial stages of airsacculitis the crystal-like clarity of these delicate sheets of tissue is lost. They become increasingly opaque and thickened as exudate begins to collect between their 2 layers of cells. At first this cloudiness is patchy but later extends through the whole system of air sacs. Yellow caseous (cheezy) material becomes more evident. There may be a varying distribution of discrete disc-like plaques, which show a necrotic center. These indicate *Aspergillus* infection and diagnosis should be confirmed by taking a swab for culture and microscopical examination. An impression smear can also be taken and stained with Gram's stain or lactophenol blue when mycelia and the club-shaped fruiting heads can be seen, particularly at the edge of the specimen. In airsacculitis due to *E. coli*, the thickened parts of the air sac and caseation tend to be more generalized and irregular in shape.
Occasionally at this stage of the post-mortem the organs can be covered with a sheen of urate crystals indicating visceral gout.

**The liver**

If the liver is ruptured and is accompanied by a large blood clot, this could be due to a blow over the sternum. In this case there will usually be signs of bruising of the overlying muscle and skin. The liver may be bile-stained (in those species which have a gall bladder) due to bile diffusion from the gall bladder through the dead tissue of the bladder wall—a process, which takes place within a few hours of death. The liver may be enlarged, and in fatty livers rupture may occur easily without external trauma. Enlargement of the liver is indicated by loss of the normal sharp edges which become rounded. If this is accompanied by faint areas of necrosis, which are seen at the same time as a fibrinous or serous pericarditis and air sacculitis, death may have been due to *Chlamydia* (*Psittacosis*) infection.

An impression smear of the liver stained with either a modified Ziehl-Nielsen stain or Macchiavello's stain, may show the pink intracytoplasmic inclusion bodies. Also in *Chlamydia* infection the spleen will be enlarged or distorted in shape or possibly ruptured. In avian tuberculosis, small white or yellowish pustules up to the size of a pea and possibly not raised above the surrounding surface can occur. Some of the other organs may be covered by these lesions. Avian tuberculosis should not be confused with the pin-head necrotic foci of *Salmonella*, *E. coli*, or *Pasteurella*. A swab stained with Ziehl-Nielsen and Gram's stain may identify the organism. The liver may be a rich golden color and somewhat fatty in the case of septicemic *Salmonella* infection.

In turkeys and game birds, the black, circular lesions of blackhead due to *Histomoniasis* infection may be found. If the liver is mottled with irregular, lighter-colored areas this may be neoplasia. Tumors may be seen with Marek's disease (MD) or lymphoid leukosis. Yellow fatty livers may be seen with fatty liver syndrome or mycotoxicosis.

If at this initial examination of the viscera there are signs of a septicemia (toxicity spread through the blood), a sterile specimen of heart blood should be taken. The surface of the organ is first sterilized by searing with the blade of a hot spatula. A sterile needle attached to a syringe is then inserted into the heart to withdrawal blood. If the bird has not been dead a long time, you may make a smear and look for blood parasites. The blood should also be cultured and stained with Gram's stain.

**Removal and examination of the alimentary canal and spleen**

This should be carried out by cutting the lower esophagus or proventriculus and incising the skin around the vent. The cloaca and the attached bursa of Fabricius should be removed intact and care should be taken not to contaminate the rest of the carcass. The spleen should be attached to the underside of the caudal end of the proventriculus (anatomically the dorsal side).
The spleen

The spleen is globular in most species, but may be triangular in ducks and geese, and is usually about one-quarter to one-third the size of the heart. Never ignore an enlarged or angular-shaped spleen or one that may have ruptured. It may indicate *Chlamydia* infection. The spleen may be slightly enlarged and hyperemic due to a septicemic infection, or as in the case of the liver, mottled with the foci (circular area) of a neoplasia. The signs of tuberculosis, *Pasteurella*, *E. coli* or *Salmonella* septicemia, MD, lymphoid leukosis, and *Aspergillosis* are similar to those seen on the liver.

The lower alimentary canal

Before dissecting the gut, examine the pancreas, which can be seen before the alimentary canal is removed from the abdomen. The pancreas should be examined for evidence of atrophy or neoplasia. Atrophy (shrinking) of the pancreas may occur with enteric reovirus infection. The accompanying duodenum may look congested or distended. Take a sterile sample of the contents, in the same way as harvesting a sterile sample of heart blood.

If the ingesta are too viscid, dilute by injecting sterile saline. Examine the sample for *Coccidia*, by wet mount, or Gram's stain. The Gram's stain will enable assessment of the numbers of Gram-positive and Gram-negative organisms. The latter should not be predominant in healthy birds. Look for signs of intestinal hemorrhage, which could be generalized or patchy throughout the intestine. If this is accompanied by pathological signs in other parts of the body, it may indicate visceral trophic, velogenic Newcastle disease (VVND). However, since the pathological signs of ND vary among species, strains of virus and lesions may be present in any of the viscera. Always look at the pattern of pathological change in the intestine together with other changes in the viscera. A single intestinal hemorrhage may not be due to a bacterial enteritis, but rather caused by terminal venous congestion brought on by right heart failure as a result of toxemia. Hemorrhages may also be associated with bacteria, Mycotoxicosis, and helminths. The intestines should also be examined for necrotic or ulcerative enteritis.

Examine the ceca; these vary in shape and size in different species. They are large and obvious in poultry. In turkeys, chickens and game birds, lesions of blackhead may be seen. The ceca are swollen, the mucosa is extensively ulcerated, and the lumen contains necrotic material. In *Salmonella* infection, the cecal wall may have a white, glistening appearance. After an external examination of the bowel, the whole alimentary canal should be opened to expose the lumen. The interior may be filled with a green fluid without any ingesta, indicating anorexia (not eating). The lower intestine may contain grit from the gizzard, indicating increased peristalsis. The mucosa of the bowel may be congested and swollen or flaccid (limp), and dilated. If the lumen is filled with catarrhal exudate, this could be caused by a parasitic infection. The contents of the bowel and scrapings of the mucosa should be examined for *Coccidia* or *Capillaria* (up to one centimeter long) or helminth eggs. The gut may contain ascarid worms, which may be so numerous as to cause impaction and rupture of the bowel.

In the case of *Salmonella* infection the mucosa may show signs of desquamation (shedding) or show small nodules of necrosis. Foreign bodies are sometimes found in the lumen of the gut and occasionally penetrate the bowel wall.
The proventriculus and ventriculus (or gizzard) should be examined for evidence of the cheesy exudate of *Trichomoniasis* infection, which is more commonly found higher up in the alimentary canal. Hemorrhages at the junction of the proventriculus and gizzard are associated with VVND. Erosion of the gizzard is associated with mycotoxins. Enlarged proventriculi glands and small flaccid gizzard are associated with enteric reovirus infections. Signs of *Aspergillus* infection are occasionally found in the lumen of the bowel. Striations seen in the muscle of the wall of the gizzard may be due to vitamin E deficiency.

Lastly, the cloaca together with the bursa of Fabricius should be examined. The latter should be small and involuted in the adult bird. The cloaca may be impacted with urate crystals forming a crumbling calculus, or it may be filled with blood clot as the result of damage during artificial insemination. The mucosa of the cloaca can show signs of inflammation or neoplastic change. The bursa should be examined for tumors of lymphoid leukosis, edema or hemorrhages associated with infectious bursal disease (IBD) or atrophy associated with IBD, mycotoxins, reovirus, or chicken anemia virus infections.

**Examination of the heart and associated major blood vessels**

The exterior of the heart together with the pericardium will have received attention when the carcass was opened, but must now be examined in greater detail. The pericardial sac (sac around the heart) should be examined for an increase in fluid content, the amount of which is normally imperceptible. If the pericardium is unusually opaque, this may be caused by infiltration with urate crystals. Examine the myocardium, endocardium, and coronary blood vessels for signs of hemorrhages. Occasionally the right atrium may be ruptured as a consequence of massive dilation during circulatory failure brought on by an overwhelming disease. Right ventricular hypertrophy can be seen with ascites associated with a number of conditions, which may cause apoxia (lack of oxygen).

The major blood vessels leaving the heart should be examined. At the same time look for signs of airsacculitis in the cervicle and interclavicular air sacs. If this is present, it may be productive to cut through the head of the humerus and take a swab from the medullary cavity since this is connected to the anterior air sacs. In examination of the brachiocephalic trunk and the carotid arteries leading away from it, the crop must not be damaged. The interior of the major blood vessels, as well as those of the abdominal aorta and renal arteries, may show atheromatous plaques, and these may be so extensive as to apparently occlude (block) the lumen of the vessel.

**Examination of the crop**

Normally the crop wall is thin in small birds and as delicate as tissue paper. However, where there is infection, as with *Candida*, *Capillariasis*, or *Trichomoniasis*, the mucosa of the crop can become hypertrophied and noticeably thick. If the white, caseous exudate of *Candida* is scraped from the mucosa, the surface will look velvet. Occasionally a crop will become impacted, a condition affecting all species. The trapped food will ferment with superimposed bacterial infection and inflammation of the crop. The layman's term of "sour crop" can cover any of the
above conditions (resulting in a distinct odor). Necrosis of the crop can occur, caused by a penetrating sharp object.

**The esophagus and oropharynx**

The esophagus should be opened by making a parallel cut with scissors along each side. If a pair of strong scissors or a pair of bone forceps (in large birds) is inserted with one blade in the mouth, the quadrate bone can be cut and the lower jaw disarticulated. The upper alimentary tract can be examined. A caseous exudate could indicate *Trichomoniasis*, *Candida*, or *Aspergillus*. The signs of these infections can be confused and diagnosis should be confirmed by laboratory examination. *Trichomonads* are sometimes difficult to find under the microscope. Use of potassium hydroxide stain will aid observation of *Candida* or *Trichomonads*. One should be aware that signs of *Trichomoniasis* may be superimposed on an underlying *Chlamydia* infection.

Excessive mucus in this region may be indicative of *Capillaria* infection and the worms are sometimes easily seen in the mucus by naked eye, although microscopical examination may be necessary. Abscesses in the mouth of birds are not uncommon and may be due to an underlying vitamin A deficiency. Plaque-like eruptions may be seen with fowl pox or mycotoxicosis. Hemorrhage into the choanal space or into the oral cavity may be noticed when the mouth is first opened. This may be as a result of trauma.

**The respiratory system**

**The palatine choanal opening**

Mites may be found inhabiting this area. Look for signs of infection in this region. The upper beak should be cut across just in front of the cere and the sinuses examined. They may contain catarrhal (mucous) or caseous (cheezy) exudate or a blood clot.

**The glottis**

There may be signs of inflammatory change along the edges. Open the trachea by making two parallel cuts. The mucosa may be congested or there may be signs of fungal infection. In those birds that feed on invertebrates, and this includes a wide range of species, the nematode *Syngamus trachea* is commonly found particularly in young birds. Occasionally a foreign body is found such as seeds obstructing the trachea. Caseous plaques are not uncommon in the region of the syrinx and may partially occlude the airway.

**The lungs**

The air sac system has received attention in discussing opening the carcass. The lungs should be examined *in situ* (as they lay) and then carefully eased away from the adjoining ribs using the handle of a scalpel. Look for evidence of abscesses or hemorrhage. If any is present examine the adjacent rib. There may be signs of a recent or old fracture.
Hemorrhage into the lung substance may be agonal (unorganized clot) and occur as the result of right heart failure. It may also be the result of inflammatory change. If the lung looks solid, try and float a piece in water. If there is pneumonic change the piece of lung will sink. Nodules may be due to bacterial or fungal infections. Viral induced tumors or fibrinous exudate associated with *E. coli* or *Psittacosis* may be seen.

**The urogenital system**

**The gonads, adrenals, and kidneys**

Look at these organs *in situ* (in the body). It may be possible to strip them out from the "bed" beneath the synsosacrum. This is attempted by gripping the fascia (sheet of fibrous tissue) just cranial to this group of organs, peeling back gently, and easing the organs out with scissors.

Both ovaries and testes vary in size according to the maturity of the bird and the breeding season. In both cases there may be total or partial pigmentation of the gonads—which is normal. In the ovary affected with *Salmonella pullorum* disease the follicles, instead of being globular, may be misshaped and angular. Both male and female gonads can undergo neoplastic (tumor formation) change.

The adrenal, closely associated with the cranial end of the gonad, is normally a pale-pink color, but it may become hyperemic (blood filled) during the course of an infection or it may look white. The kidney may be hyperemic together with the rest of the viscera in septicemic conditions. Alternatively it may be grey, due to cloudy swelling. The kidneys may show any of the signs seen in the liver due to infectious disease mentioned above. The kidney may be pale in color and tubules may be prominent when impacted with urate crystals. This could be an indication of salt poisoning. Occasionally neoplasms are seen in the avian kidney with MD or lymphoid leukosis.

**The nervous system**

**The peripheral nerves**

After the kidney, examine the nerves of the sciatic plexus where these leave the spinal cord and emerge beneath the synsosacrum. These nerves together, with those of the axillae and the intercostal nerves, should be examined for signs of irregular thickening typical of MD, seen in poultry. Thickening of the nerves may also occur in riboflavin (B2) deficiency.

**The brain**

During removal of the skin covering the head, evidence of subcutaneous hemorrhage may be seen. This is significant only if there has been a lot of bleeding. Next find the *foramen magnum* and in small birds insert the blade of a scalpel; in larger birds a pair of strong scissors or bone forceps will be needed. Cut around the cranium on each side and raise the calvarium to expose the brain.
Signs of hemorrhage within the substance of the bone, sometimes quite extensive, are not significant and are caused by blood extravasated (loss) from blood vessels very soon after death. However, an organized blood clots either over or under the meninges or in the substance of the brain is important. This is so even if the blood is not clotted, and may be evidence of concussion, particularly if there is also a matching bruising of the skin, or hemorrhage into the nasal cavities. Fungal colonies associated with Aspergillosis may be present. Necrosis (death of tissue) may be associated with avian encephalomalacia (AE) and edema with encephalomyelitis.

**The skeleton**

Open and look particularly at the joints. Greenish discoloration in the muscles around the joints with discharge of exudate may be a sign of Salmonella infection in pigeons and poultry. The articular cartilage may also show petechia. Urate crystals may be seen in the joints of those birds affected with visceral gout as well as subcutaneous tophi (calcium deposit).

Cut off the head of the femur and sample the medullary cavity for blood-borne bacteria, and examine the blood corpuscles for signs for any cellular disorder. Examine the color of the bone marrow for signs of anemia (figure 4.2) associated with inclusion body hepatitis, Mycotoxicosis, or CAV.

![Figure 4.2. Pale bone marrow seen with chicken anemia virus infection](image)

**Tissues used for routine diagnostic procedures**

**Impression smears**

To make a good impression smear (actually a touch preparation), it may be easier to bring the slide to the tissue. Grasp a small piece of the tissue with forceps so that a freshly cut, well-blotted surface faces upward (figure 4.3). Lower a clean slide to the tissue, touching it lightly. Retract
quickly without sliding the slide across the tissue. Make several "touch preps" on each slide. Impressions are generally more useful when air-dried. If other fixation is necessary (e.g., heat fixation for acid-fast stains or acid alcohol for certain Giemsa preparations), it can be done later. Exudates may be prepared for cytologic evaluation.

![Bipolar rods seen with Pasteurella](image)

**Figure 4.3. Bipolar rods seen with Pasteurella**

*Histopathologic procedures*

Most tissues are fixed in formalin for optimum histology. Ten per cent buffered formalin penetrates only about 0.2 cm in 24 hours. That means that a 0.1 cm strip on the center of a 0.5 cm piece of tissue remains unfixed. Penetration is slower in very bloody, dense tissue (e.g., congested spleen or liver) and more rapid in relatively porous tissue (e.g., lung). Formalin will not penetrate well into the brain through the unopened calvarium or into the marrow of bone unless the bone has been cracked. The biggest problem with submission of fixed tissues is inadequate fixation due to prior severe autolysis or inadequate volume of fixative, allowing continuing decomposition. Initial fixation is achieved with ten times the volume of formalin as volume of tissue. The amount of formalin may be reduced after 12 to 24 hours of fixation in preparation for mailing. Wet formalin-fixed tissue may be stored and shipped in plastic heat-sealed bags. Other fixatives, such as those required for electron microscopic evaluation, are probably not necessary for routine submission. However, if gout (uric acid accumulation) is suspected, a small piece of affected tissue should be placed in a vial with absolute alcohol, as urates are water-soluble and are lost in formalin fixation.

Histology laboratory use may depend on the cost per tissue. If you do not send complete tissues, it is wise to save the rest of the viscera in formalin while awaiting a diagnosis (Tables 4.1 and 4.2). If only grossly visible lesions or limited tissues are submitted, a diagnosis may not be possible. Too often, the limited tissues suggest a diagnosis, which cannot be confirmed because other tissues have already been discarded. In addition, subsequent submission of supplemental tissues may help generate new information regarding patterns of disease. The retained tissues can be disposed of when the final report is received or can be stored for future reference.
Table 4.1. Tissues that should be saved for post-mortem analysis

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brain</td>
<td>Tongue or soft palate</td>
</tr>
<tr>
<td>Trachea and syrinx</td>
<td>Esophagus</td>
</tr>
<tr>
<td>Crop</td>
<td>Proventriculus</td>
</tr>
<tr>
<td>Ventriculus (gizzard)</td>
<td>Duodenum and pancreas</td>
</tr>
<tr>
<td>Intestines</td>
<td>Cloaca and bursa</td>
</tr>
<tr>
<td>Liver</td>
<td>Cecum and colon</td>
</tr>
<tr>
<td>Heart</td>
<td>Thyroids</td>
</tr>
<tr>
<td>Thymus</td>
<td>Lungs</td>
</tr>
<tr>
<td>Adrenals and gonads</td>
<td>Kidneys</td>
</tr>
<tr>
<td>Oviduct</td>
<td>Sciatic nerve and thigh muscle</td>
</tr>
<tr>
<td>Bone marrow</td>
<td>Skin and breast muscle</td>
</tr>
<tr>
<td>Spleen and Bursa</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.2. Tissues that should be submitted to the laboratory for post-mortem analysis

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart</td>
<td>Liver</td>
</tr>
<tr>
<td>Gonad</td>
<td>Piece of intestine</td>
</tr>
<tr>
<td>Lung</td>
<td>Duodenum</td>
</tr>
<tr>
<td>Spleen</td>
<td>Bursa</td>
</tr>
<tr>
<td>Kidney</td>
<td>Pancreas</td>
</tr>
</tbody>
</table>

Tissues for histopathology should not be frozen. Freezing creates crystals and ruptures cells, making histopathology virtually useless.

Toxological and virological procedures

Tissues for toxicological analysis should be frozen. They may be frozen at -20°C (regular deep-freeze) after being wrapped in aluminum foil. Freezing for virus isolation is done on dry ice, in liquid nitrogen, or in a laboratory freezer at less than -60°C. If this cannot be accomplished, the tissues for viral isolation should be sent (by overnight mail) in sterile containers (petri dishes or test tubes) on wet ice.

Most common laboratory procedures

In a 1996 survey of poultry diagnostic laboratories by the Centers for Epidemiology and Animal of the USDA:APHIS:VS, a number of findings were reported. The survey cited annual accessions processed in 1992-1994. The average number of accessions for all laboratories reporting was 615 per year. Other tests, which were done, are listed in Table 4.3. Clinical signs reported, which were referable to the gastrointestinal tract, ranged from 3% to 70% with an average of 30% (figures 4.3 and 4.4). Accessions with signs referable to the respiratory tract ranged from 5% to 95% with an average of 41%. Accessions with clinical signs referable to locations other than the intestinal and respiratory tracts ranged from 2% to 95% with an average of 32%. An average of 68% of all accessions was from commercial flocks, 34% were from backyard flocks and 3% was from unknown source.
**Table 4.3**

<table>
<thead>
<tr>
<th>Service</th>
<th>Number Reporting</th>
<th>Average Annual Accessions</th>
<th>Range of Annual Accessions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Necropsy</td>
<td>36</td>
<td>615</td>
<td>0 to 5,000</td>
</tr>
<tr>
<td>Bacterial isolation</td>
<td>30</td>
<td>398</td>
<td>5 to 4,173</td>
</tr>
<tr>
<td>Virus isolation</td>
<td>27</td>
<td>105</td>
<td>0 to 1,472</td>
</tr>
<tr>
<td>Serology</td>
<td>30</td>
<td>10,927</td>
<td>0 to 250,000</td>
</tr>
<tr>
<td>Parasitology</td>
<td>24</td>
<td>71</td>
<td>0 to 500</td>
</tr>
<tr>
<td>Histology from Field necropsies</td>
<td>26</td>
<td>202</td>
<td>0 to 1,500</td>
</tr>
</tbody>
</table>

Additional services provided for the poultry industry per laboratory and their costs are shown in Table 4.4. Thirty-four laboratories reported a charge for their services. The average cost per accession was $28 with costs ranging from $2 to $70. Services included necropsy, bacteriology, virology, toxicology, and histology. Other services included electron microscopy, serology, Parasitology, and farm visits.

**Table 4.4**

<table>
<thead>
<tr>
<th>Service</th>
<th>Number Reporting</th>
<th>Cost range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteriology</td>
<td>29</td>
<td>$3.00-$24.00</td>
</tr>
<tr>
<td>Virology</td>
<td>20</td>
<td>$10.00-$75.00</td>
</tr>
<tr>
<td>Serology</td>
<td>27</td>
<td>$0.00-$40.00</td>
</tr>
</tbody>
</table>

**Table 4.4 (Continued)**

<table>
<thead>
<tr>
<th>Service</th>
<th>Number Reporting</th>
<th>Cost range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parasitology</td>
<td>27</td>
<td>$2.00-$15.00</td>
</tr>
<tr>
<td>Histology</td>
<td>27</td>
<td>$5.00-$36.00</td>
</tr>
</tbody>
</table>
Table 4.5  List of common poultry diseases, number of laboratories reporting, the primary tests done and the range of tests performed.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Number Reporting</th>
<th>Primary Tests</th>
<th>Range of Tests Performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avian influenza A</td>
<td>39</td>
<td>Agar Gel Precipitation</td>
<td>0 to 90,640</td>
</tr>
<tr>
<td>Velogenic Newcastle disease</td>
<td>26</td>
<td>Virus</td>
<td>0 to 15,691</td>
</tr>
<tr>
<td>Infectious bursal disease</td>
<td>31</td>
<td>Eliza/Histology</td>
<td>None</td>
</tr>
<tr>
<td>Marek’s disease</td>
<td>31</td>
<td>Histology</td>
<td>None</td>
</tr>
<tr>
<td>Mycoplasmosis (MG)</td>
<td>38</td>
<td>Serum Plate Agglutination</td>
<td>14</td>
</tr>
<tr>
<td>Psittacosis-Ornithosis</td>
<td>28</td>
<td>Eliza</td>
<td>16</td>
</tr>
<tr>
<td>Avian infectious bronchitis</td>
<td>30</td>
<td>Eliza</td>
<td>None</td>
</tr>
<tr>
<td>Avian infectious laryngotracheitis</td>
<td>31</td>
<td>Histology</td>
<td>16</td>
</tr>
<tr>
<td>Avian tuberculosis</td>
<td>26</td>
<td>Histology</td>
<td>7</td>
</tr>
<tr>
<td>Duck virus hepatitis</td>
<td>18</td>
<td>Histology</td>
<td>1</td>
</tr>
<tr>
<td>Duck virus enteritis</td>
<td>20</td>
<td>Histology</td>
<td>5</td>
</tr>
<tr>
<td>Fowl cholera</td>
<td>34</td>
<td>Culture</td>
<td>None</td>
</tr>
<tr>
<td><em>Salmonella enteritidis</em></td>
<td>34</td>
<td>Culture</td>
<td>16</td>
</tr>
<tr>
<td><em>Salmonella typhimurium</em></td>
<td>35</td>
<td>Culture</td>
<td>8</td>
</tr>
<tr>
<td>Pullorum-typhoid disease</td>
<td>39</td>
<td>Culture</td>
<td>25</td>
</tr>
<tr>
<td>Infectious coryza</td>
<td>27</td>
<td>Culture</td>
<td>2</td>
</tr>
<tr>
<td>Avian encephalomyelitis</td>
<td>26</td>
<td>Histology</td>
<td>2</td>
</tr>
<tr>
<td>Avian spirochetosis</td>
<td>17</td>
<td>Smear/Histo/Culture</td>
<td>None</td>
</tr>
<tr>
<td>Avian salmonellosis (excluding typhoid and pullorum)</td>
<td>32</td>
<td>Culture</td>
<td>3</td>
</tr>
<tr>
<td>Avian leucosis</td>
<td>27</td>
<td>Histology</td>
<td>1</td>
</tr>
</tbody>
</table>
Table 4.6 Lists each major poultry producing state in the US and the reportable diseases diagnosed in 1995.

<table>
<thead>
<tr>
<th>States</th>
<th>Reportable Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alabama</td>
<td>Laryngotracheitis, <em>S. enteritidis</em>, <em>S. typhimurium</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>Arkansas</td>
<td><em>M. gallisepticum</em>, Ornithosis, Laryngotracheitis, <em>S. enteritidis</em>, <em>S. typhimurium</em>, Coryza, Pullorum-typhoid</td>
</tr>
<tr>
<td>California</td>
<td>Ornithosis, Avian tuberculosis, <em>S. enteritidis</em>, Pullorum-typhoid from another laboratory (private)</td>
</tr>
<tr>
<td>Colorado</td>
<td>Ornithosis, Pullorum-typhoid</td>
</tr>
<tr>
<td>Connecticut</td>
<td>Ornithosis, Laryngotracheitis, Avian tuberculosis, <em>S. enteritidis</em>, <em>S. typhimurium</em>, other Salmonella, Pullorum-typhoid</td>
</tr>
<tr>
<td>Florida</td>
<td><em>M. gallisepticum</em>, Laryngotracheitis, <em>S. enteritidis</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>Georgia</td>
<td>Ornithosis, Laryngotracheitis, <em>S. enteritidis</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>Iowa</td>
<td><em>M. gallisepticum</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>Illinois</td>
<td><em>M. gallisepticum</em>, Ornithosis, <em>S. enteritidis</em>, <em>S. typhimurium</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>Indiana</td>
<td>Ornithosis, Laryngotracheitis, <em>S. enteritidis</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>Kentucky</td>
<td>Pullorum-typhoid, Avian encephalomyelitis</td>
</tr>
<tr>
<td>Maryland</td>
<td>Ornithosis, Laryngotracheitis, Avian tuberculosis, <em>S. enteritidis</em>, <em>S. typhimurium</em>, Pullorum-typhoid, Duck virus enteritis, Avian encephalomyelitis</td>
</tr>
<tr>
<td>Mississippi</td>
<td><em>M. gallisepticum</em>, Laryngotracheitis, Pullorum-typhoid</td>
</tr>
<tr>
<td>North Carolina</td>
<td><em>M. gallisepticum</em>, Ornithosis, Laryngotracheitis, Pullorum-typhoid, Coryza</td>
</tr>
<tr>
<td>Nebraska</td>
<td><em>M. gallisepticum</em>, Ornithosis, <em>S. enteritidis</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>New Hampshire</td>
<td><em>S. enteritidis</em>, <em>S. typhimurium</em></td>
</tr>
<tr>
<td>New York</td>
<td>Laryngotracheitis, Pullorum-typhoid, Duck virus enteritis</td>
</tr>
<tr>
<td>Oregon</td>
<td><em>S. enteritidis</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>South Carolina</td>
<td><em>M. gallisepticum</em>, Ornithosis, Laryngotracheitis, <em>S. enteritidis</em>, Pullorum-typhoid</td>
</tr>
<tr>
<td>Tennessee</td>
<td>Ornithosis, Pullorum-typhoid</td>
</tr>
<tr>
<td>Texas</td>
<td>Laryngotracheitis, Pullorum-typhoid</td>
</tr>
<tr>
<td>Utah</td>
<td><em>M. gallisepticum</em>, Avian tuberculosis, Pullorum-typhoid, Other Salmonella</td>
</tr>
</tbody>
</table>
Figure 4.3. omphalitis

Figure 4.4. Fungal nodule in the lower lobe of the lung
5. Pathology

This chapter will provide an introduction to the study of disease. A disease is any departure from a state of health. Health and disease are expressions of a dynamic relationship among three ecologic factors (agent, host, and environment). An agent is any power, principle, or substance capable of acting upon the host. The host is the animal upon which the agent has an effect. The environment is the surroundings of every living organism. It is the combination of all external conditions and influences affecting the life and development of a host.

Agents may be biologic, virus, bacteria (figure 5.2), etc., chemical (poisons, toxins, etc.), nutrient (vitamin and mineral deficiency or toxicity), and physical (freezing, burning, trauma). Host factors in disease include habits and customs such as litter eating or cannibalism. Environmental factors, which influence diseases: include climate, weather, etc. There is a delicate balance between host, agent, and environment and any disturbance of this balance results in disease.

Hygiene is the whole art of preserving health by whatever means necessary. Hygiene equals preventative medicine. Sanitation is the prevention of disease by eliminating or controlling environmental factors, which are involved in transmission of disease. Sanitation is a specific part of hygiene. Sanitation includes cleanup, disinfection, and disinfestation. Hygiene procedures other than sanitation include medication, vaccination, and optimal management procedures. Specific hygiene practices used to prevent or control diseases of poultry will be provided in subsequent chapters.

Stress is the borderline condition between health and disease. Stress factors include hunger, chilling, overheating, inadequate floor space, thirst or vaccination. Stress can produce a reduction in the immune response and result in disease. Hygiene practices, including optimum management, attempt to remove or reduce stress.

Pathology is the study of alternations and reactions developing in a host when it is exposed to an agent. Pathology is the branch of medicine, which relates specific effects to definite causes. In general, an injurious agent causes a specific injury and promotes a specific reaction in the tissues. When an agent injures cells, a series of events takes place which a trained individual (pathologist) studies. A pathologist studies macroscopic and microscopic changes in cells or tissues in an attempt to diagnosis disease.

An injury to host cells by an agent may upset the biochemical balance. An enzyme or substrate may be altered, causing no product to be produced. A lack of product causes degeneration of the cellular components, which may be seen by an electron or light microscope. This degeneration to a specific-cellular organelle, such as mitochondria, nucleus, cell membrane, endoplasmic reticulum, etc., may be reversible or irreversible. If the degeneration continues unchecked, irreversible degeneration may lead to cell death or necrosis. When many cells die the host starts to show clinical signs, for example depression, diarrhea, etc. Continuation of necrosis may lead to tissue death, visible as necrotic foci or changes in color or consistency of the organ upon post mortem exam. If necrosis of vital tissues continues unchecked, the death of the host may occur.
The host reacts to an injurious agent by producing changes in circulation, which bring in cellular components, which can neutralize agents and repair degenerated cells, or in some cases replace necrotic cells with new healthy cells.

**Inflammation** is a body reaction to an agent. The cardinal signs of inflamed cells are redness, swelling, heat and pain due to increased blood supply and circulation in the damaged area. Inflammation is a beneficial phenomenon. Inflammation is characterized by dilation of vessels and increased permeability of the capillaries. Inflammation brings reactive cells to the damaged area, which is capable of neutralizing agents. These cells include macrophages, which can engulf foreign agents, and antibodies, which can neutralize infectious organisms and other reactive lymphoid cells. Large amounts of elements needed to repair damaged cells, including fibrous components, are also brought to the area after the agent is neutralized and removed by circulation. The onset of inflammation is the secretion of vasoactive compounds by injured cells. Eosinophils and basophils release such substances as histamine, which result in increased circulation and increased capillary permeability.

Inflammation can be characterized by duration and cellular character. **Exudate** is defined as the movement of fluid outside the vessels. Exudate contains the cellular components of inflammation (figure 5.1). **Acute** inflammation is rapid (24 hours or less) and is characterized by edema, heterophills, basophils, and macrophages. **Edema** is the excessive accumulation of water in the intercellular spaces. Edema is the first response of inflammation. **Chronic** inflammation (figure 5.0) occurs over days, weeks or months. It is characterized by a thick colored fluid. It may contain phagocytes, lymphoid cells, antibody, and necrotic debris.

![Figure 5.0. Chronic inflammation in the hock joint caused by *Staph aureus*](image)

If the inflammatory exudate results in destruction of the invading agents, the exudate can be quickly resolved, and restitution of normal structure and function can occur. This is the process of healing. Healing restores the injured area to its previous normal condition. This usually involves three phases. The first, or removal phase, results in the removal of the inflammation products (exudate and dead cells), usually by liquefaction with absorption into the blood and lymph. The second or repair phase results in the proliferation of fibrous tissue. The third or
regeneration phase is the process by which lost cells and tissues are replaced by others of the same kind.

Figure 5.1. Microscopic section showing necrosis and inflammation in the bursa of Fabricius

Figure 5.2. Bacterial inflammation of the brain on the right
6. Causes of avian diseases

A disease is an illness resulting from a disturbance of function in one or more of the body organs or tissues. Diseases may be (1) infectious, that is, due to a parasitic agent, or (2) non-infectious, not due to a parasitic agent. Infectious diseases may be contagious or non-contagious. In a contagious disease, the causative agent may spread from bird to bird. Spread does not occur with a non-contagious disease.

Infectious diseases

Bacteria

Bacteria (figure 6.0) are very small living microorganisms. There are many kinds of bacteria, most of which perform useful functions such as removal of filth and maintenance of soil fertility. Only a few bacteria cause disease. In birds these include Salmonella, Erysipelothrix, Clostridia, Pasteurella, Staphylococci, Streptococci, Hemophilus, Mycobacterium, E. coli, etc. Bacteria contain cell walls, which have both DNA and RNA, and replicate by fission. Bacteria can be grown on artificial media and can be inhibited by antibiotics.

Viruses

Viruses are the smallest microorganisms known, and can multiply only within living cells. They can be seen only with the electron microscope. Viruses are not generally responsive to common antibiotic drug treatments. Expensive antiviral compounds exists for humans and pets. Important viral diseases include Newcastle disease (ND), infectious bronchitis (IB), laryngotracheitis (ILT), fowl pox, infectious bursal disease (IBD), chicken anemia virus (CAV), Avian encephalomyelitis (AE), influenza, Marek's disease (MD), lymphoid leukosis and viral tenosynovitis. Viruses contain either DNA or RNA, and do not have cell walls or cellular organelle, and cannot be isolated on artificial media.

Mycoplasma

These are very small organisms at the limit of vision when viewed with the compound microscope. Many mycoplasma serotypes are infectious for birds. These include Mycoplasma gallisepticum, M. synoviae and M. meleagridia, M. gallinarium, and M. iowae. They contain both DNA and RNA, producing tiny colonies on artificial media and are inhibited by certain antibiotics. They do not contain cell walls.

Protozoa

Protozoa (figure 6.2) are similar to bacteria, but usually much larger. Included are coccidia, Histomonas (blackhead), Hexamita, Plasmodium (malaria-like), Trichomonads, and Toxoplasma. Unicellular organisms with complex life cycles, containing both sexual and asexual reproduction, they do not grow on artificial media, contain both DNA and RNA, and are inhibited by certain antibiotics.
Figure 6.0. Gram positive rod-shaped bacteria

Figure 6.1. Fungal culture

Figure 6.2. Tricomonads in a blood smear
Parasites

Ectoparasites (outside) include arthropods and insects, mites, lice, fleas, gnats, chiggers, and mosquitoes. Endoparasites (inside) are mainly helminths (worms) and consist largely of ascarids, cecal worms, tape worms, and Capillaria.

Fungi

There are two important diseases in this group, Aspergillosis (brooder pneumonia) and thrush (Candida) caused by yeast (figure 6.3). Fungi (figure 6.1) may also produce toxins in feed, resulting in considerable morbidity and suboptimum performance. Fungi contain both DNA and RNA, produce fur-like down or slimy colonies on artificial media, and can be inhibited by certain antibiotics. Fungi have complex sexual and asexual life cycles.

Chlamydia

Chlamydia are obligate intracellular bacteria with a partial cell wall. They cannot replicate on artificial media and are inhibited by certain antibiotics. They contain both DNA and RNA. Chlamydia psittaci produces psittacosis, a severe respiratory disease of many avians.

Non-infectious diseases

Nutritional deficiencies

The ration of poultry must be nutritionally balanced. The nutritional requirements of birds are more completely known than of any other species including man. This knowledge is readily available to all nutritionists, and gross nutritional problems seldom occur with the use of commercial feeds. There is continued need, however, for refinements in the nutritional quality of feeds in order to parallel continued advances in the genetic improvement of birds. There is an
increase in leg problems in the "new" broiler and turkey lines. Leg problems (figure 6.4) are common, due in part to subtle nutritional imbalances currently not understood and the rapid growth rate of these modern breeds.

Figure 6.4. Rickets caused by a nutritional deficiency

**Poisons and toxins**

The major intoxication of birds is mycotoxicosis. Others include botulism, plant toxins such as crotalaria, excess dosages of medicants, excess intakes of salt, ingestion of insecticides or fungicides, and occasionally heavy metal poisoning such as arsenic.

**Management failures**

Management practices cannot be dealt with in detail. Obviously attention must be given to proper temperature and humidity control, ventilation, bird density, excess dust, improper de-beaking, gizzard impaction, light and litter conditions.

**Genetics**

Genetics is a substantial factor in the control of any infectious disease. Genetic selections are important in many areas. Control of such defects as hereditary myopathy (muscle degeneration), spondylopathy (kinky back), and curled toes is obtained by eliminating these birds from the reproductive cycle. Osteodystrophy (tibial dyschondroplasia), a major cause for bone deformities
in both turkeys and chickens, as well as ascities and flip-over disease, are subject to genetic and nutritional control.

**Environmental**

The enclosed, highly concentrated environment of commercial birds with special reference to cage egg layers is abnormal, and may result in physiological disturbances. Cage layer fatigue, fatty liver syndrome, piling up, dehydration, hysteria, ascites, ammonia intoxication, and cannibalism are maladies associated with environmental conditions.

**Transmission of infectious diseases**

**Embryonic (ovarian, shell penetration)**

Organisms causing disease are present in the ovary or uterus and become encased within the egg, or are in the intestinal tract and contaminate and penetrate the egg shell. Ovarian or uterine transmission occurs with *Arizonosis*, chronic respiratory disease (CRD), encephalitis (epidemic tremor), infectious sinusitis, infectious synovitis, lymphoid leukosis, *M. meleagrisidis*, pullorum-typhoid, viral arthritis, viral hepatitis, and CAV. In addition, ovarian transmission of adenoviruses occurs.

Shell penetration occurs with *Salmonellosis* (Arizona and paratyphoid serotypes) and coliform organisms. Egg yolk infection or omphalitis, a common cause for unthrifty chicks, is due to shell penetration by microorganisms including *E. coli*, *Staph aureus*, and *Pseudomonas*.

**Hatchery dissemination**

Infection occurs during the interval between egg pip and chick delivery. Diseases of importance are aspergillosis (brooder pneumonia) and omphalitis (umbilical infection). Other possibilities include *Salmonella*, *E. coli*, and *Pseudomonas* infections.

**Air-borne (aerosol)**

The agent causing the disease is present in the respiratory tract, and is exhaled or coughed into the air. Infection occurs when the agent is inhaled by other birds.

Air-borne infection is not to be confused with wind-borne, which rarely occurs. Air-borne infection occurs among birds in close contact as pen mates, and across narrow partitions as wire barriers or feed alleys. In the absence of air-current, air-borne diseases may not spread more than 10-20 feet. Spread from building to building is usually accomplished by persons, movement of contaminated equipment, and mixing of birds.

**Rapidly air-borne.** CRD, IBV, infectious sinusitis, infectious synovitis, influenza, NDV.
Slowly air-borne. ILTV, fowl cholera (chronic), infectious coryza, Colibacillosis, MDV, and Chlamydia.

Equipment. Feed trucks and cars may transfer disease agents where the etiologic agent occurs on poultry ranges. Diseases of importance are fowl cholera and Ornithosis. Pickup trucks filled with dead birds or used litter can easily spread disease. Any unsterilized equipment that circulates between different flocks is a potential source of disease. Included are bird crates, vaccinating, and insemination equipment and egg flats. Egg flats are a proven source for ND, ILT and mites. The transport of adolescent pullets can be risky. Peak labor loads are often required, and safety precautions are easily relaxed. CRD may be a major risk. Mycoplasma may be transmitted with infected semen.

Feathers. The herpes virus of MD is present on chicken feathers, and every feather that moves in the wind is a potential source of the virus.

Litter and droppings

Litter will commonly be contaminated with numerous infectious agents including Coccidiosis, MDV, IBDV, reoviruses, Salmonella, and the ova (eggs) of internal parasites. Dusty litter can readily transmit viruses, whereas moist litter contains embryonated ova and oocytes (life cycle stage of Protozoa) and bacterial spores (protective covering). To prevent the transmission of infectious agents, litter should always be wet or covered when transported on public highways.

Mixing of birds

The mixing of adolescent and adult birds from different sources is a major source for new outbreaks with many diseases. A number of diseases result in "avian carriers", that is, the etiologic agent continues to be shed from the bird after apparent recovery, and disease transfer may occur when these birds are mixed with susceptible birds. Carrier states include the following: Histomoniasis (blackhead), Coccidiosis, Salmonella diseases (pullorum-typhoid, paratyphoid), fowl cholera, fowl coryza, ILT, CRD, IB, infectious synovitis, M. meleagridis, transmissible enteritis, tuberculosis, and MD. When introduced into vaccinated birds, velogenic viscerotropic Newcastle disease (VVND) may also become a carrier disease.

Contaminated feed or water

The source of mycotoxins and Aspergillosis is commonly contaminated feed. Water contamination is important in the spread of Pasteurellosis, Salmonellosis, infectious coryza, and E. coli.

Fecal contamination

The disease-producing agents are in the droppings. Infection occurs by oral ingestion of the organisms. The following are included in this group:
Salmonella, Coccidiosis, Encephalomyelitis, Enteritis, (hemorrhagic, ulcerative), Erysipelas, fowl cholera, Histomoniasis (blackhead), IBDV, necrotic dermatitis, Ornithosis, tuberculosis, VVND. A virus may be shed from the digestive tract and be spread through fecal contaminations. Most internal parasites such as Ascaridia, cecal worms and Capillaria are spread through fecal contaminations. Adenoviruses and reoviruses are shed from the intestinal tract.

**Vectors**

Disease agents are carried from one flock to a second by a transporting agency. Transfer may be mechanical or involve the life cycle of a parasite.

**Man**

Transport by persons is a major source of outbreaks of ND, IB, ILT, CRD, and infectious sinusitis. Contamination occurs on one's hands, face and clothing. The risk is high when susceptible birds are handled on the same day after handling birds with a respiratory disease. Persons may also transfer the agents of those infectious diseases whose etiological agents are present in the litter. The mechanism of transfer could be by way of foot-gear.

**Free-flying and exotic birds, predators and rodents**

Sparrows transmit lice and mites. Flying birds are a major source of ornithosis, fowl cholera, influenza, and hexamitiasis. Movements of exotic birds, particularly by smuggling, are a major source for outbreaks of VVND. The risk occurs also with game fighting fowl. Any species of wild life, including rodents, are a potential source of Salmonella and Pasteurella. Predators, particularly dogs and cats, transmit disease from one premise to another. Raccoons and wild birds are also reservoirs for bacteria.

**Insects and worms**

Mosquitoes are the major source of fowl pox, west nile, and equine encephalomyelitis viruses. Black flies transmit Leucocytozoan. Beetles (figure 6.7), flies, slugs, mites (figure 6.6), lice, ants, grasshoppers, and others are required for the transmission of tape worms. The cecal worm (figure 6.5) and the earth worm are also important in the transfer of Histomoniasis (blackhead). The lesser meal worm becomes contaminated with Salmonella serotypes present in bird environments, and must be destroyed in decontamination programs.
Artificial insemination

AI is a factor in the transmission of *Salmonellosis*, fowl cholera, *Erysipelas*, influenza and *Mycoplasma meleagris*.
7. Disease prevention and control

Management practices which relate to disease control

Preparation of premises

Following depopulation of pullet, breeder and layer flocks, and at least once a year for broiler flocks, or after an outbreak of disease, you should totally clean out a house. Start by removing all unused feed and old litter and then clean feed and water equipment. Wash down houses with water and detergent. All dust, fecal material, and debris must be removed before disinfection will be effective. Disinfect the facility with a high pressure, hot water machine, and then fumigate. For fumigation, use 35 cc of 37-percent formalin with 17.5 grams of potassium permanganate per 100 cubic feet (2.8 cubic meters) of room space. Place chemicals in a large container, and seal house for up to 24 hours; this will also kill many microorganisms. In most U. S. states the formalin has been prohibited. Suitable safer chemical replacements include: gluteraldehyde, phenolics, sulfate compounds, hydrogen peroxide, and synthetic fuels.

Equipment Handling

- Water Systems - Water lines, nipple drinkers, cups or troughs should be flushed, sanitized and drained prior to either raising or removing from house. Bell drinkers should be removed and disassembled for removal of organic debris to permit proper cleaning and disinfection. Reservoirs should be flushed, sanitized and drained during the house cleaning procedures.

- Feeding Systems - Feed remaining in the pans, feed lines, chains, augers or hoppers should be removed and placed on the floor for removal with the litter. These lines and equipment must be removed or raised prior to removal of litter and floor material.

- Ventilation - Fans, casings, motors, belts, curtains, ventilation pads and louvers should be individually cleaned free of manure, debris, dust and feathers prior to disinfection. Equipment such as thermostats, scales, time clocks, electrical panels, switches and light bulbs, etc may need to be individually wiped, cleaned, sanitized and protected from the more severe effects of cleaning such as high pressure sprayers and disinfectant chemicals and protected from recontamination during the cleaning process.

- Slats - Slats should be scraped of adhering caked manure and debris before being removed from the house and then subjected to high pressure washing and disinfection.

- Egg belt, egg flats, egg buggies and packing machines - Adherent yolk, egg material and shell debris should be removed prior to washing and disinfection of this
- Egg room, storage areas - Mechanical equipment, supply rooms, egg rooms and storage areas should be cleaned of materials, debris, equipment and supplies for proper removal of organic materials and disinfection.
- Floor areas litter and manure - All removable and obstructive equipment such as feeders and watering systems need to be raised or removed prior entry of back hoes, bobcats and other house cleaning equipment into the house. All litter, manure and organic debris should be removed from the house and disposed of by previously approved handling procedures for this material. Approved methods may be land application, composting (shed or under plastic) or possibly burial. Equipment used to clean houses must be cleaned and disinfected prior to leaving farm.
- Dry Cleaning - Once litter and manure material is removed from the house and disposed of the house, air blowers/vacuums should be used to remove dust, cobwebs and other material on ceilings, rafters and other areas. Floor areas should be blown down and broom clean prior to the wash down step.
- Washing - Houses should be washed down with high pressure water with detergent to remove remaining dust and organic debris. Curtains should be exposed to permit correct cleaning and removal of adherent feathers, dust and organic material.
- Exterior of house - A perimeter of 10 feet around the exterior of the house free of uncut grass, materials and obstructions is necessary. Areas of rodent entrances or penetration should be sealed at this time. Roof areas and eaves with holes or nesting areas for wild birds should be addressed at this time.
- House Disinfection - After the washing step the house should be permitted to dry out prior to the spray disinfecting all surfaces in the house with appropriate disinfectant. A reasonable down time after disinfection should be given prior to repopulation and resumption of normal procedures. Main doors should not remain open with out proper screening to prevent reentry of wild avian population.

**Personnel Requirements**
- Personal protective equipment should be used, including boots, coveralls, rain suits (including both pants and jackets with hoods), gloves specific to the materials being handled, face shields when applying disinfectants, and goggles when handling concentrate powders or solutions. Respirators and chemical-resistant suits may be required for some solutions. During all C&D operations, respirators should be available if the personnel are at risk from a disease organism or chemical hazard, if significant amounts of dust are generated, or upon individual request.

Disinfect bulk feed tanks after removal of all feed, with special reference to caked feed. Air the building out for at least 7 days. Allow sunlight to disinfect equipment and inside of house. Place in new litter or rolled paper, feed, and equipment. In between broiler flocks, equipment should be disinfected. At least ten days should be allowed between flocks. Other practices to reduce microorganisms in the litter, by reducing pH include adding phosphoric acid 1.2 lb per 1 m$^2$ or superphosphate 1 kg per 1 m$^2$ or 50 to 100 pounds of aluminum sulfate per 1,000 square feet of
floor space. Some commercial compounds include Sodium bisulfate (Poultry Litter Treatmen®), Acidified clay (Poultry Guard®), and aluminum sulfate (AL Clear®). These compounds will also reduce ammonia levels in the house and at higher levels bind soluble phosphorus in the litter. The chemicals should be lightly moistened to dissolve them. Biological litter treatments include Micro Treat P® and Impact P®. They have a blend of nonpathogenic microbes and enzymes. They can improve litter quality and reduce ammonia.

Microbial and urease inhibitors such as para-formaldehyde flacks and volatile fatty acids are antimicrobial. Some of these include De-Odorase®, YS-50® (Yucca Plant extract), and Ultra Litter® Treatment. Heating the house to 40°C for 24 hours or 50°C for 4 hours can also reduce pathogenic microbes. Inside and outside of poultry houses and hatcheries should be treated with disinfectants, insecticides, and rodenticides. Change litter, clean and disinfect premises following outbreaks of infectious diseases. Coccidia, Marek's disease (MD), infectious bursal disease virus, chicken anemia virus, and reoviruses persist on premises in spite of any known sanitation program. Change the litter following the buildup of severe internal parasitic infections with special reference to *Ascariasis* and *Capillaria*. Used litter moved on highways should be either wet or covered. New litter can be added on the top of old litter (top dressing) in brooding areas in sections of the country where litter is scarce or where disposing of litter is a problem due to runoff contamination of water tables. Mechanical equipment is available (Litter Bug®, etc) to remove only the caked litter and should be used between flocks. In areas where excessive runoff contamination is a problem, possible solutions include feeding litter to ruminants, pelleting for use as garden fertilizer, composting (figure 7.0) and/or fermentation with farm mortalities as prescribed by veterinary regulations.

Water lines can have biofilm build up which can reduce water flow. Biofilm is a microscopic framework of salts and polysaccharides that can attach to the inner surface of pipes. It can act as a shelter for bacteria and viruses. Suitable products to sanitize water and remove biofilms include organic acids (Selko pH® and hydrogen peroxide (CID 2000® and Aquaclean®). They should be left in the water for 24 hours after clean out and then flushed clean with regular water.

![Image 7.0. Composting to dispose of litter](image1.png)  ![Image 7.1. Nipple drinkers to control water](image2.png)
Selection of chicks or poults

Chicks or poults should originate from good genetic breeders and be fed nutritionally balanced mycotoxin-free feed and giving pure water free of heavy metals and pathogenic bacteria (figure 7.3). Chick parent flocks should be pullorum-typhoid clean, and be free from *M. gallisepticum*, *M. synoviae*, and lymphoid leukosis viruses. The breeders should be immune to avian encephalomyelitis (AE), infectious bursal disease virus (IBDV), Newcastle disease (NDV), infectious bronchitis (IBV), and viral arthritis. Turkey breeders should be pullorum-typhoid clean, and be free of *M. gallisepticum*, *M. synoviae*, *M. meleagridis*, and *Salmonella*.

Breeder flocks

Breeder hens should be fed and watered over slates to avoid dirty floors (figure 7.2). Males may be fed over the floor. Eggs should be collected five times per day. Dirty or cracked eggs, floor eggs, eggs with poor shells, double yoked eggs and eggs below 50 grams should be separated and not sent to the hatchery. The remaining eggs should be sprayed with disinfectants 2.5% hydrogen peroxide and 1% quaternary ammonia (quats) or 1% hydrogen peroxide and 1% formalin. Another formula is 1% hydrogen peroxide, 0.05% acetic acid and 1% quats. Nest boxes should be cleaned every 2 weeks and 20 grams of paraformaldehyde pellets added. Automatic nests provide cleaner eggs with fewer cracks. Eggs should be stored at 60°F at 75% relative humidity on the farm. Eggs should be picked up at least every 3 days in refrigerated trucks and taken to the hatchery. A checklist for examining management practices for breeder flocks can be found in the appendix.

Hatchery sanitation
The quality of hatchery sanitation and operation is judged by the quality of chicks or poults produced. The following deficiencies in bird quality may be of hatchery origin. Omphalitis (navel ill, "mushy" chick disease) indicates unsanitary breeder farm or hatchery conditions, dirty delivery boxes, or dirty box pads. Managers of hatchery and/or breeder flocks must initiate vigorous cleanup program. Hatcheries should be monitored for bacterial contaminations every month by placing nutrient agar plates in all areas of the hatchery.

*Aspergillosis* (mold infection)

Accumulation of organic dusts in the ventilating system permits growth of molds, and can result in mold infections in poults or chicks. The hatchery must be kept clean. Determine the source and degree of the problem by exposing plates of Sabouraud dextrose or corn meal agar in various locations. Incubate and note numbers of fungal colonies. The hatchery should not be located in an area, which exhausts organic dust into the atmosphere such as processing or rendering plants. Hatchery waste should be exhausted directly into a truck via a closed system for render. Exhausted air should be vented through an air or water filter.

Dehydration in newly-hatched birds

Dehydration is indicated by shriveled legs or shanks. Chick bodies feel "hard". Dehydration results from low humidity during incubation or hotspots in the hatchery resulting in chicks hatching out at different time intervals in the same machine. Hatched chicks should not spend more than 24 hours in the hatchery.

Weak chicks or poults

Identify by pressing down on the chicks in boxes with the palm of the hand. If strong, the birds will offer considerable resistance to the pressure; if weak, they can be pushed down readily. Weak chicks may result from too high temperature during hatching, inadequate ventilation in the hatchery, over-fumigation at hatching time, rough sexing, or setting of old eggs. Chicks should be examined and culled for obvious anatomical malformations. Red hocks are a sign of machine problems, and unhealed navels are a sign of bacterial contamination or a machine problem. Chicks should also be checked for improper debeaking, feather length sexing or Marek’s disease virus vaccination (using a safe vegetable dye). Spot candle at about the 7th day of embryonation to determine fertility. If there is a substantial difference (a spread of over 7%) between fertility and hatchability, there is a problem.

*Egg sanitation*

Through proper breeder flock care, good egg sanitization, and correct hatchery operations, a hatch of 86-88 percent can be secured with broiler eggs. Operations securing substantially less than these results should examine their sanitation practices.
Clean eggs

Clean eggs begin with breeder flock management. Prevent dirty litter through proper ventilation and repair of faulty waterers. Rear hens and roosters separately until 18 weeks of age in dark out houses with 8 hours of light on restricted skip-a-day pullet diets. Weigh weekly to obtain target weight. Move roosters into the laying facility (1 per 10 hens) 1 week prior to hens. The laying facility should contain feeders and waterers over slates. Roosters and hens should be fed separately. Begin stimulation with increase light and feed at weekly intervals. Provide a sufficient number of nests acceptable to the birds with clean nesting material (1 nest per 4 hens). Semi-trap nests may be used with turkeys. Gather eggs every 2-3 hours. Do not mix floor or soiled eggs with clean eggs. Automatic egg collection systems and nipple drinkers will also result in cleaner eggs and dryer houses. Hens should be stimulated by light and feed to peak in production at around 30 weeks of age. Eggs should be stored at 55-65°F (13-18°C) and at a relative humidity of 80-90%. Hatchability declines when eggs are held longer than 7 days. There are also increased leg problems in progeny chicks. Keep separate hatch records for each breeder flock. Problems occurring in chicks/poults can often be traced to parent breeder flocks.

Figures 7.4 and 7.5. Automatic commerical egg handling systems

Disinfection of eggs

Automated egg collection (figures 7.4, 7.5, 7.8) and egg disinfection by spraying is common and associated with decreased contamination, increased hatchability, and decreased disease problems, and provides for stronger, better quality, and better livability in chicks/poults. Clean, non-sprayed eggs may average up to 31,000 bacteria per shell. Soiled or floor eggs may contain much more. Proper spraying can reduce this by more than 99 percent. Submersion washing usually removes the cuticle and may be detrimental. Wet brush washing may transfer bacteria from egg to egg. Eggs can be subjected to a spray wash with a disinfectant. Drying is accomplished by air. The result is a clean, bacteria-free, dry-hatching egg with cuticle intact. If washing is done, water temperature should exceed 100°F to prevent penetration of disinfectant in the shell. Separate hens from egg storage (figure 7.9)

Small amounts of fecal material can be removed with a clean, disinfected towel. Sandpaper should not be used because it can destroy the protective cuticle of the shell and permit easier access of microorganisms. Spray as soon as possible after collection. Do not wash dirty eggs,
which have caked fecal material. Formalin, because of its irritating properties, is unpopular with personnel. Hydrogen peroxide (2%) and quaternary ammonia (1%) are more popular.

In the hatchery at least 30 eggs per flock should be checked at monthly intervals for shell quality by floating in salt solution. Flocks with poor shell quality will allow greater penetration by bacteria and hatchability will be reduced. Flocks with poor shell quality should be treated with increased oyster shell or aluminosilicates added to the feed. Clinafarm® (Schering-Plough, Inc., Millsboro, DE) is a compound, which can be sprayed on eggs or used to fumigate hatchers or chicks; it is highly fungicidal. A useful management survey sheet for examining hatchery management is the appendix.

**Egg yolk infection**

Weak chick or starve-out mortality is due primarily to the growth of bacteria in the yolk sac prior to hatching. This mortality will begin soon after hatch, peak at the 3rd or 4th day, and decline to normal mortality by the 6th or 7th day. Chicks that do not die may be inferior birds. They are more susceptible to respiratory diseases, and may show an excess (3 to 4 percent) condemnation for septicemia-toxemia at processing. The source of the bacteria will be shell penetration, either prior to incubation or from contamination during incubation due to "exploders", or unsanitary incubator conditions.

Bacteria in yolk sacs are those present on the skin and feathers of birds, and include *Coliforms, Streptococci, Staphylococci, Proteus, Salmonella, Aerobacter, Clostridium* and *Pseudomonas*. Often these are pathogenic only for the egg yolk sac. When a livability problem occurs during the 1st week of brooding, culture yolk sacs from a number of birds during the first 12-24 hours. In normal birds, there will usually be isolated a single species or no bacteria. In problem birds, there will be cultured two or more types of bacteria. Chicks can die at any stage during the grow-out from omphalitis. Inflammatory process (IP) and cellulitis, chronic localized infections causing high condemnation in the processing plant, are partially due to hatchery sanitation.

Yolk sac infection can usually be corrected by improved egg shell quality and sanitation, or better incubator practices. In some cases it may be associated with individual breeder flocks, in which case the problem may be embryonic in origin. Treatment of the breeders with an antibiotic may clear up the problem. Treatment of problem chicks/poults with an antibiotic may reduce mortalities. Recommended: sulfas, nitrofurans*, lincomycin, spectromycin, or quinolones in the water for the first five days after hatching. Gentamicin or Naxal®* injected SQ in the hatchery are beneficial in problem flocks.

**Cleaning and disinfection**

Cleaning must precede disinfection, for disinfection is not possible in the presence of organic debris such as droppings, feed accumulations, dust, and so forth. Cleaning is best accomplished by the use of pressure hoses with or without a detergent. Steam cleaners may be used to decontaminate truck beds and cabs, and equipment moved from farm to farm. Disinfectants include the following:

**Quaternary ammonium compounds**
There are a number of "quats" which vary in composition, and are sold under trade names. They are clear, odorless and detergent in action, and are effective against bacteria and fungi, but less active against viruses. They are inactivated in the presence of soaps, detergents, mineral salts and organic materials. They are used in concentrations of 100-800 ppm for disinfecting hatcheries, poultry houses, equipment, and for sanitizing poultry drinking water. Efficacy is reduced when used with hard water. The latter is subject to some risk, because it may be somewhat toxic to birds even when given at recommended intakes.

Iodophores

These contain iodine, and possess wide germicidal activity, but are corrosive in acid solution. When combined with alcohol or detergents, iodines have good efficacy in presence of organic matter. They have slight residual activity and low toxicity, but stain on contact. Used for disinfection of instruments and other small objects. Used also for egg dipping, hatchery or poultry house infection, and for sanitizing footbaths or poultry drinking water. Organic iodines are available for use in treating poultry suffering from vaccine reactions or diarrhea.

Chlorins (Sodium or potassium hypochlorites)

The active ingredient is chlorine. They have wide germicidal activity but are corrosive. Activity in presence of organic matter is short. They have only slight residual effect. Their activity is reduced by detergents. Soft water must be used when making hypochlorite stock solutions for proportioners, or calcium precipitation will occur. 1.5-percent sodium hypochlorite (1 oz of commercially available bleaching powder per gallon of water) can be used for general use. Recommended for use in sanitizing reasonably clean objects such as water cans or jars or similar equipment. Used also for egg dipping, egg washing and sanitizing of poultry drinking water. Chlorox® at 2-4 oz per gallon can be used to treat birds suffering from vaccine reactions or diarrhea (3 ppm). Up to 5 ppm of chlorine can be tolerated by birds in the drinking water through the growout. Many new drinking water treatments are commercially available which claim to be more toxic to organisms and less toxic to birds than Chlorox®.

Pine oil

These contain limited germicidal range when used alone, but their effectiveness can be improved when combined with detergents, soaps, or ammonium salts. They have good cleaning qualities, are not corrosive, but have strong and lasting odor. They can be used effectively in disinfection of surfaces such as walls and/or floors.

Creosols and Cresylic Acid (coal tar distillates)

They have wide germicidal activity, but are corrosive and toxic at high concentrations. There is high efficacy in the presence of organic matter, and have residual activity. There is heavy odor with them and they may be mixed with detergents and are most active in an acid pH range. They are recommended for general use in disinfection of hatcheries and poultry premises, but
sufficient time must be allowed for noxious and toxic gases to diminish before chicks can be placed. Creosols are for truck drive through baths or foot baths.

Multiple phenolics (synthetic)

They have wide germicidal range. Their range and efficacy are increased by combining several synthetic phenolics. They are of low toxicity and corrosiveness. Penetration is increased with detergents. They are efficient in the presence of organic matter, are effective deodorizers and have long-lasting activity. There is a residual effect on surfaces where they may be sprayed or painted. Efficacy is increased with heating or at low pH. Commercial phenols mixed with detergents are popular for one-step washing and disinfection of equipment.

Formaldehyde

They have wide germicidal activity and are not corrosive. They have slight residual activity and are moderately toxic. They are effective against spores and are used at 5-10 percent solution. They are an excellent disinfectant for dirt floors. They are used for house fumigations. One pint (500 cc) of commercial formalin and one pound (one-half kilogram) of potassium permanganate is sufficient for the fumigation of 1000 cubic feet (28 cubic meters) of air space. For it to be effective, however, the humidity must be very high. A temperature of 80°F (27°C) is required to maintain high humidity. Seal the premise, and allow at least 24 hours for complete fumigation. The U.S. government has all but outlawed its use in hatcheries, because of its carcinogenic potential.

Hydrogen peroxide and Potassium Permanganate (oxidizing agents)

It has wide germicidal activity and is neither corrosive nor carcinogenic. It breaks down harmlessly to hydrogen, oxygen, and water. It is used mainly for cleaning eggs by spray or by aerosol for disinfecting hatcheries. It is not very effective in organic matter. New machines are available for fumigation of H2O2. The substance breaks down quickly and safely into hydrogen, oxygen, and water.

Sulfates

New compounds which are highly effective in organic matter. The most common commercially available one is VerKon S® from Anatec, Inc., of Gainesville, GA. It is the only safe compound, which can kill infectious bursal disease virus in the chicken house. It is caustic and should be handled with care.

If the objects to be disinfected are free from organic matter, and reasonably clean, chlorine, quaternary ammonium compounds, or single and multiple synthetic phenolics can be used. The phenolic compounds must be rinsed off waterers. If the objects to be disinfected are not essentially clean and if a viral, bacterial or protozoan pathogen has contaminated the house,
multiple phenolic disinfectants with detergent should be used on waterers. Multiple synthetic phenolics with or without detergents, or creosol should be used on walls, floors, ceilings and equipment. Rough surfaces require penetration and long lasting disinfectants such as creosol or phenolics with or without detergents.

<table>
<thead>
<tr>
<th>Disinfectant</th>
<th>Examples</th>
<th>Uses</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>Ethyl, isopropyl</td>
<td>Hands, thermometers</td>
<td>Moderately virucidal at 70 - 80 %; ethanol is preferable</td>
</tr>
<tr>
<td>Chlorhexidine</td>
<td>Hibitane, Nolvasan</td>
<td>Many uses including examination tables, cages, other surfaces</td>
<td>Tolerant to the presence of organic compounds, body fluids, etc.; expensive</td>
</tr>
<tr>
<td>Detergent iodophores</td>
<td>Betadine, Wescadine, Redene</td>
<td>Drinking water, food and utensils, dairies, spot disinfection</td>
<td>Action based release of iodine &amp; detergent action; less affected by high protein; expensive</td>
</tr>
<tr>
<td>Ethylene dioxide</td>
<td></td>
<td>For heat sensitive materials</td>
<td>Available as a compressed gas at 10% with 90% CO₂, otherwise toxic and explosive</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>Formalin</td>
<td>Laundry &amp; bedding surfaces; as a vapor for other surfaces</td>
<td>Low power of penetration; irritating hypersensitivity occurs</td>
</tr>
<tr>
<td>Glutaraldehyde</td>
<td>Cidex</td>
<td>Cold sterilization of instruments with lenses</td>
<td>2% solution buffered with sodium bicarbonate; virucidal (10 min., pH 7.5 - 8.5); expensive</td>
</tr>
<tr>
<td>Phenol derivatives</td>
<td>Lysol, Dettol, Staphene, Sudol</td>
<td>Hands, examination tables, cages, other surfaces</td>
<td>2.5% aqueous solution; efficacy dependent on concentration and temperature; high protein decreases effectiveness</td>
</tr>
<tr>
<td>Quaternary ammonium compounds</td>
<td>Zephiran, Roccal, Savlon</td>
<td>Zephiran (benzalkonium chloride) used for cleaning wounds</td>
<td>Not effective against many viruses; high protein decreases effectiveness</td>
</tr>
<tr>
<td>Sodium hypochlorite</td>
<td>Chlorox, Chlorize</td>
<td>Same as detergent iodophores</td>
<td>Highly effective, rapid action; high protein decreases effectiveness; irritating; inexpensive</td>
</tr>
</tbody>
</table>

*This information applies to most viruses; there are some exceptions.

In the choice of a disinfectant, consider the following:

Type of surface to be disinfected.
Cleanliness of surface.
Type of pathogenic organisms, which are to be destroyed.
Time available for disinfection.
Cost
Sanitizing water for poultry

Chlorines, iodines, quaternary ammonium compounds and ultra-violet radiation are used as water sanitizers. The major objective is to control slime molds in watering equipment. Quaternary ammonium compounds should be used with caution as continued use may prove adverse to birds. Chlorine sanitizers are effective at 3-10 ppm. Levels up to 50-100 ppm are tolerated by birds for a day or so. Sodium hypochlorite is effective and inexpensive. One gallon or 4 liters of laundry bleach (Chlorox®) in 17,500 gallons (65,000 liters) of water will give a chlorine content of 3 ppm. Higher levels may be needed for the adequate control of slime. Do not start chicks on a high level of chlorine. Test kits, to measure the level of chlorine in water, are readily available. Iodophores are more effective than chlorine against mold.

Ammonia, breast blisters and ventilation

The amount of ammonia in the air of poultry premises depends upon the amount of fecal material in the litter, moisture, temperature, humidity, floor space and season. Literature reports vary as to the toxic effects of ammonia. Most reports recommend that the concentration of ammonia in the air at bird level, not human, be held to less than 20 ppm, and that concentrations in excess of 25 ppm are detrimental and may be manifested by increased susceptibility to respiratory diseases, diminished growth rate, lowered feed conversion, increased breast blisters and undergrads, and increased air sac and total condemnations. The ammonia problem is a bellwether indicator of the standard of management being practiced. Simple chemical tests using pH litmus paper are available for detecting ammonia concentration in the air. More expensive gas tube methods are also available.

Lifelong exposures of 60-140 ppm significantly reduced growth rates and induced ocular lesions, but did not affect feed conversion, skin color, size of adrenal glands or bursae of Fabricius, or condemnations. One presumes that these birds were otherwise disease free, and the toxic effects of ammonia might be enhanced in the presence of respiratory or other diseases.

Ammonia, breast blisters, ventilation and wet litter are interrelated. Poor ventilation is the most important single cause for breast blisters, and a high blister problem can be reduced by improved ventilation. Fans may be required for adequate ammonia and moisture removal. Ventilation in a house can be checked using low volume smoke emitters (30 sec/4,000ft³) from Mitchell Instrument, Annandale, NJ. Converting a house to tunnel ventilation will greatly increase air flow in a house. In tunnel ventilation, air enters vents on one side of the house and is pulled through the house by fans located on the other end.
Proper adjustment of water height and depth of water in the waterers will reduce water spills. Use of nipple waterers and positive pressure brooders or tunnel ventilation systems help keep houses dryer. Ammonia levels can also be reduced by lowering litter pH with phosphoric acid, propionic acid, or ferrous sulfate (.10/lb per sq ft), or adding commercially available extracts from the yucca plant. Litter moisture in the range of 25-30 percent is desirable from the standpoint of controlling particulates in the air, but increased moisture contents become adverse. Blisters appear in birds in excess of 4 pounds (1.8 kilograms). These birds sit more, and there are secondary leg problems. Well-feathered birds have fewer blisters.

Wet litter and ammonia can be very adverse in adults. The soles of the feet become eroded and bacterial infection may result. Hens will not enter nests because of sore feet, and there may be serious infertility problems in the males. This is due to extension of infection from the feet to the testes. The occurrence of erosions on the soles of the feet in birds is cause for alarm, and the environmental causes should be corrected at once.

Management practices which relate to poultry health

In non-epidemic situations, good management is the most important factor in controlling losses from mortality and decreased egg production. The best managed flocks perform best in the presence of disease agents normally present in poultry house environments. Weather changes are second as they affect bird health. Disease agents are third. Excluding virulent pathogens, many agents of disease are in the environment, and await only a favorable opportunity to show their effects. Observe the following management factors. A checklist is found in the Appendix.

1. Do not mix chicks/poults from different sources. Limit the mixing of hatching eggs from multiple-age breeder flocks. Chicks from multi-age flocks are of different size and have different levels of maternal antibody. Higher mortality will result from mixing birds from old and young breeder flocks. Also, there is often a need to trace the origin of problem chicks to the parent source.

2. Do not mix age groups. Older birds serve as reservoirs of infection for younger ones.

3. Exclude pets from poultry premises, and control entry of visitors. Visitors should be provided with clean outer coats or coveralls and disposable plastic boots. Locate foot disinfection baths at the entry of each building. Iodophores are by a margin the most satisfactory disinfectant. A good policy is to provide a registry of all visitors, including maintenance workers.

4. Provide doors with external locks and internal latches. Post "No Trespassing" signs, and install a locked gate at the entrance to the farm (figure 7.7).
Figure 7.6. Spraying of tires with disinfectant  Figure 7.7. No trespassing signs

5. Other than cockerels, never add additional birds to an adult flock; never return commercial birds from fairs and shows.

Figure 7.8. Automated egg collection  Figure 7.9. Separation of hens from egg storage

6. Sanitize all equipment that circulates between bird premises (figure 7.6). Discard used chick pads.

7. Remove dead birds several times per day. Method of disposal will relate to location and environmental considerations. Standard practices include incinerating, composting, fermentation or freezing (figure 7.10) for use in rendering into feed ingredients.

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8. Broiler and pullet grow-out operations should be on an all-in, all-out basis. Move chicks in, move birds out, clean up and repeat. Allow 10-14 days between lots of birds.

9. Follow approved practices with regard to disinfections, light, bird density, feeder and water spaces, and more. When completed, every detail of management practices can be evaluated on a precise, scientific basis.

10. Keep honest, accurate and usable records.

11. Practice good debeaking (figure 7.11). Pullets should be debeaked at 5 to 7 days of age with a precision debeaker and rechecked again at 12 to 16 weeks of age. The beak should be a nickel's distance from the nares. The bottom beak should be a nickels distance longer than the top beak. Broilers need not be debeaked. Sanitize hands between flocks (figure 7.12).

12. Be on the alert for signs of trouble. Note abnormal feathering, abnormal behavior, decreased activity, decreased feed intake, dust levels, ammonia, abnormal droppings, and caked litter.
13. Have some knowledge of the "normal" mortality. Normal mortality for broilers in the United States is 0.75% for the first week, 0.5% for the second week, 2% for the next 4 weeks and 1% during the last week. Mortality for pullets should be 7% until housing in the laying facility, and then 1% per month. Necropsy (figure 7.13) a proportion of dead birds and look for a general condition, location, size and color of lesions. Kidney lesions, for instance, are associated with many infections and intoxications, and can be an indicator of impending trouble in a flock.

![Figure 7.13. Postmortem exams should be done if mortality rises](image)

14. Feed bulk tanks and fuel deliveries should be external to premises. Never drive unnecessary vehicles on poultry/turkey ranges.

15. With primary breeders, establish two separated gene pools in case of catastrophic losses.

16. Mow grass and weeds around house and remove any debris, which could serve as a shelter for external parasites and rodents. Fill any holes around houses. Keep cats outside house to act as rodent control.

17. Screen wild birds and rodents out of the house. Keep doors closed and locked at all times to prevent unwanted visitors.

18. Add and use fans religiously, foggers and/or evaporative coolers, and adequate brooders to maintain fresh and comfortable air.

19. Keep waterers clean and sanitized.

20. Regularly check all automatic equipment.

21. Do not keep exotic birds or domestic pigs on the same farm as domestic poultry.

22. Keep dogs and cats from inside poultry houses.
23. Practice external parasite control by regular use of approved pesticides inside and outside the house (figure 7.14.)

![Figure 7.14. Rodent bait stations](image)

24. Isolate farms as much as possible and provide buffers between them (fenses, streams, trees, etc.) (figures 7.22 and 7.23).

**Servicing flocks**

There is a necessity in commercial poultry/turkey production for many persons to circulate between flocks. Included are service personnel, vaccinators, debeakers, blood collectors, graders, movers, egg pickup, maintenance and others. A simple management survey sheet for examining broiler flocks can be found in the appendix.

Circulating persons transmit disease in two ways, namely, by their feet and by contamination on their clothing, hands and face. Those diseases whose etiologic agents are in the droppings may be transmitted on shoes or other footgear.

Major risk diseases include ND, *Salmonellosis*, fowl cholera, IB, hemorrhagic enteritis, IBD and transmissible enteritis of turkeys. The risks are reduced by the use of disposable plastic foot gear, which is left on the premises, or by rubber boots which are sterilized prior to and after entry. The agents of respiratory diseases, however, are in the air, not the litter, and contamination occurs on one's hands, face, cap and clothing. Velogenic viscerotropic Newcastle disease (VVND) is both in the litter and in the air. Beyond reasonable doubt, transmission of respiratory diseases by persons is far more important than is the transmission of the non-respiratory group, and circulating persons are a major source for new outbreaks of chronic respiratory disease (CRD), ND, IB, infectious laryngotracheitis (ILT), and avian influenza (AI). Attention to footgear alone is not enough to prevent transmission of these diseases. Be conscious of the following:
a. There is high risk when a person on the same day handles susceptible birds after handling birds in which a respiratory disease is present. Transmission is a near certainty.

b. The risk is minimized if a day is allowed to intervene between flock visits, and the person showers and changes into clean outer clothing.

c. Any person who has not contacted birds for five or more days can enter any poultry premise with little or no risk of disease introduction.

The following recommendations are made regarding the servicing of flocks.

1. The service functions for broilers, replacement birds, and adult birds should be separate.

2. Persons routinely servicing flocks should put on clean clothing daily including caps. Wipe watch bands. Use disposable plastic footgear, or disinfect rubber boots prior to and after entering premises. The daily disinfection of truck cabs is a good procedure. A disinfectant pool may be provided, through which all vehicles must pass.

3. Breeder service personnel should always proceed on the same day with pullet flocks from younger to older birds, then adult layers, than the hatchery. Shower before entering large multi-age facilities or hatcheries and put on clean clothes.

4. All circulating persons should, not on the same day, enter a bird premise after entering a premise in which a respiratory disease is present. Broiler flocks should be visited from younger to older. Again, shower before entering large multi-age farms.

5. If possible, dispense with the use of circulating vaccinating crews. There are often persons with little or no knowledge of disease control, and disease transmission is a risk.

6. Chick groups should be regarded as isolation units, with access denied to persons other than caretakers and service persons, until such time as immunization has been completed against ND and IB.

7. Broiler service personnel should always visit the hatchery first before entering a poultry farm.

8. Always visit the processing plant last.

9. Practice good vaccination techniques and keep vaccines in a dry and clean place (figures 7.17. and 7.18).
Performing a quality control program in the hatchery to assess breeder flock and hatchery performance is essential for maintaining the quality poultry health and performance parameters. A quality control program (QAC) for hatcheries within a complex can best be described as a routine sequence of procedures that assesses (1) breeder flock performance, (2) hatching-egg quality, (3) incubation and hatching, (4) sanitation, and (5) chick quality. A program that does not include all five is incomplete, and emphasizing one area at the expense of others will not do. The program must be administered on a routine basis if it is to be effective and meaningful. Identifying problems within a hatchery often requires a profile of past performance. Therefore an incomplete QAC program often leads to a "back-to-square-one" approach, whenever specific standards are not met or a major problem arises.

The following summarizes a QAC program that can be adapted to any operation with minor variations:

1. Breeder flock performance (see appendix)

   Egg rooms — should be routinely monitored for cleanliness, temperature, humidity, and egg handling. Includes number of times eggs are collected, percent culls and on-the-farm cracks, plus percentage of floor eggs.

   Nests — The litter condition should be checked for quantity, cleanliness, and type. Hardwood shavings are unacceptable. Use soft wood like pine because they have less moisture and fungal spores. Nests and flock should be free of external pests (mites, lice, etc.) Nest material should be changed every 2 weeks and paraformaldehyde pellets (20 gr) added. If automatic nests are used egg collection belts should be regularly disinfected.

   True fertility - periodic check by flock for accurate fertility with 7-10 day candle-out and break-out procedures. Follow up on unhatched eggs when chicks are pulled. It should include infertiles, early, middle, and late embryonic mortality patterns, culls, cracks, and pips. The procedure is important because low fertility can be quickly and easily identified. Fertility checks on unhatched eggs only, without 7-10 day candle-out, may be inaccurate and misleading. Infertile eggs and eggs containing embryos that died early can be difficult to delineate accurately at hatch. Realistically, a 7-point (%) spread between fertility and hatchability is the rule, not the exception. Thus, it is not surprising that 90% fertility leads to an 83% hatch at best. Additionally, as fertility declines, the spread between fertility and hatchability increases. Today it is not uncommon to see the hatchery criticized for low hatchability when, in fact, it is hatching within 7 points of fertility. The problem is low fertility. Approximately 450 eggs should be candled per flock prior to peak and periodically thereafter through 45 weeks. True fertility must be assessed early in production if corrective procedures are to be applied in problem flocks.

2. Hatching egg quality (see appendix)

   Periodic check of temperature and humidity in egg rooms.
Monitor the average period that eggs are held prior to setting. Set eggs from oldest flocks first, as declining shell quality may affect dehydration rate and hatch time. Monitor number of post-farm cracks and/or sweating of eggs. General egg cleanliness. Monitor shell quality as measured by specific gravity.

3. Incubation and hatching

Periodic check of setters and hatchers for proper adjustment and operation should be done. Use independent thermometers-hygrometers for checking temperatures and the relative humidity to avoid hot and/or cold pockets in machines (figure 7.16.).

Monitor the hatching time and periodically cross check with flock age and length of time eggs are held to identify machine variability as opposed to egg-handling or shell quality problems (figures 7.14 and 7.15).

4. Sanitation/microbiological monitoring

Routine microbiological monitoring after clean-up on an off-hatch day is vital. Monitoring should include selective agar plates for both bacterial and fungal isolation. At least 70% of the setters should be negative for fungal and bacterial contamination as verified by plates exposed for 10 minutes. Nutrient agar plates should be incubated at 37° for 18-24 hours and colonies counted. Sabouraud's or corn meal agar plates should be held at room temperature (about 70°F) for 96-120 hours and then colonies counted. Periodically, plates should be sent to a diagnostic laboratory for identification of fungal colonies. Occasionally selective agars for specific bacteria can be exposed and sent to the laboratory for identification. With few exceptions, if counts are
low or negative, it is of limited concern what organisms are present. However, the presence of *Aspergillus* (particularly *A. fumigatus*), regardless of number, may be significant, and it is important that the hatchery manager becomes familiar with this species. Check vaccines, vaccinating equipment and diluents expiration dates and sterility, or verify by microbiological monitoring.

**Figure 7.16. A thoroughly clean hatchery is important to quality chicks**

5. Chick quality

Continually monitor chicks for general vigor, health, and condition. Check vaccinating and sexing personnel for chicks processed per hour and maintain strict limitations that result in maximum broiler performance and livability.

**Figure 7.17. Vaccination room should be neat and orderly**

**Figure 7.18. In Ovo vaccination machine**

Periodically monitor yolk sacs from 10 broiler chicks per breeder flock for bacterial contamination by streaking yolk material on to appropriate plating media.
**Egg breakout: true fertility, embryonic mortality**

*Equipment*

Candling light  
Forceps or sharp pocket knife  
Egg flats  
450 egg sample/flock-incubated 7-10 days

*Procedure*

Candle approximately 450 egg samples at 7-10 days of incubation. Remove clears and those with obviously dead embryos. Place eggs with live embryos back in setter. Mark eggs for breakout at hatch. Open eggs that have been candled-out at large end containing air cell. Determine whether egg is infertile or contains a dead embryo. Break out the remaining untouched eggs at pull (21 days). Determine period of embryonic deaths (early 0-10 days, middle = 10-15, late = 16-21 days), pips, cracks, and percent hatch.

Calculate:

- % infertile  
- % embryonic mortality & pattern  
- % culls  
- % cracks  
- % pips  
- % hatch  
- % spread - fertility/hatchability

*Evaluation - approximate figures*

Early embryonic mortality 1-10 days = 3.5% or less  
Middle embryonic mortality 11-15 days = negligible  
Latter embryonic mortality 16-21 days = 1.5% or less  
Culls = 0.5% or less  
Cracks = 0.5% or less  
Pips = 1.0% or less  
Fertility = 97% plus  
Spread between fertility and hatchability 7 points or less

*Monitoring shell quality by egg specific gravity (SG)(figure 7.19)*

*Equipment*
Six 5-gallon plastic buckets with sealable lids.
Feed grade salt, 25-30 lbs supply.

Figure 7.19. Egg shell monitoring

Hydrometer - with specific gravity range of 1.050 to 1.100.
Graduated cylinder (plastic - 2 liter).
Household chlorox diluted with tap water to 10% solution.
One dozen egg flats.
180 fresh eggs per flock (S.G. must be done on or the day after the eggs are laid. Egg should be cooled to approximately the same temperature as the salt solution).

Procedure

Prepare 4 salt and 2 chlorox solutions as follows:

- **Bucket #1** - 4 gal. water + 3.2 lbs. salt = specific gravity of 1.070
- **Bucket #2** - 4 gal. water + 3.6 lbs. salt = specific gravity of 1.075
- **Bucket #3** - 4 gal. water + 4.0 lbs. salt = specific gravity of 1.080
- **Bucket #4** - 4 gal. water + 4.1 lbs. salt = specific gravity of 1.085
- **Bucket #5** - Chlorox rinse - 2 = 10% (1 part chlorox + 9 parts water)
- **Bucket #6** - Chlorox rinse - 2 = 10% (1 part chlorox + 9 parts water).

Adjust exact specific gravity for each solution using hydrometer. Hydrometer will float on mark of desired specific gravity. Increase specific gravity by addition of salt; decrease by addition of water. Place approximately 1.5 dozen eggs from sample in solution one (specific gravity 1.070). Remove those that float to flat 1.070. Transfer those that sink to next solution (specific gravity 1.075). Repeat procedure c. until all eggs have been either removed when they float or passed to solution 4 (specific gravity 1.085). Calculate percentage of eggs that floated in each solution.
Those that passed through all solutions have the best shell quality as measured by specific gravity.

In general, a specific gravity of 1.080 is considered the cutoff point between poor or low shell quality and good or high shell quality, and can effect hatchability. Approximately 85% of all eggs from hens through market age should have a specific gravity of 1.080 or better for maximum hatchability. Rinse all eggs in two solutions of 10% chlorox prior to setting.

**Microbiological monitoring: hatchery and equipment**

**Equipment**

Media plates (open air and swab sampling)

- Nutrient Agar (NA) or tryptic soy agar (TSA)
  -- Sabouraud’s Agar (S) or corn meal agar (CMA)
  -- Disposable transport swabs (Stewart's media)
  -- Mechanical counter (optional)
  -- Table Top Incubator (optional)
  -- Magnification counter (optional)
  -- Wax marking pencil
  -- Scotch® tape

![Figures 7.20 and 7.21. Microbial monitoring](image)

**Procedure**

Obtain plates from diagnostic laboratory (figures 7.20 and 7.21) or biological supply house, or plates prepared in company laboratory. Incubate 24 hours prior to use to verify sterility. Expose plates (one of each type) in various locations of hatchery being careful not to touch agar, inner part of plate or lid. Do not place where moisture will splash on agar. Label and tape plate securely after exposure.

--Exposure time should be approximately 10 minutes in setters and all other areas except hatchers. Hatchery exposure should be 5 minutes or less.
---Adjust all exposures to 10 minutes using a simple proportion.

Sample selected moist surfaces with transport swabs and ship to diagnostic laboratory. Incubate all plates for 24 hours at 37º (in laboratory incubator or setter). After first counting at 24 hours hold plates at room temperature (approximately 65-75ºF). Make plate counts at 24, 48, 96 or 120 hours.

Adjust counts: (Example)

<table>
<thead>
<tr>
<th>Count</th>
<th>13 colonies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>7 minutes</td>
</tr>
<tr>
<td>7/13</td>
<td>10/X</td>
</tr>
<tr>
<td>7X</td>
<td>130</td>
</tr>
<tr>
<td>X</td>
<td>18.5</td>
</tr>
</tbody>
</table>

Bacterial colonies are generally glistening and smooth. Mold colonies are generally hairy, powder-like, with dull appearance. These colonies tend to grow slower than bacterial colonies, particularly at room temperature. Magnification counter - count approximately 6 squares, take average and multiply by 40. Periodically send set of exposed plates to diagnostic laboratory for identification.

Water quality

Maintaining water quality is essential in rearing healthy poultry. There is great variation in the quality of city and well water in various areas. The following provides a list of items to be knowledgeable about when determining water quality.

WATER QUALITY CHART

<table>
<thead>
<tr>
<th>Average content item</th>
<th>Noteworthy levels (ppm) and effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total dissolved solids</td>
<td>500; over 1,000 is contaminated and 2,000 may cause diarrhea.</td>
</tr>
<tr>
<td>Total hardness (CaCO₃)</td>
<td>100, 0-60; soft. Over 180: very hard.</td>
</tr>
<tr>
<td>Calcium (Ca)</td>
<td>60; seldom a problem in poultry.</td>
</tr>
<tr>
<td>Magnesium (Mg)</td>
<td>14; over 125 has laxative effect.</td>
</tr>
<tr>
<td>Carbon dioxide (CO₂)</td>
<td>40; over 200 in surface water: check for bacteria.</td>
</tr>
<tr>
<td>Chlorides (Cl)</td>
<td>14; over 250: may cause metabolic problem.</td>
</tr>
<tr>
<td>Iron (Fe)</td>
<td>0.2; over 0.3: causes off odor, bad taste, precipitate.</td>
</tr>
<tr>
<td>Sulfate(SO₄)</td>
<td>125; over 250: has laxative effect.</td>
</tr>
<tr>
<td>Substance</td>
<td>Range/Concentration</td>
</tr>
<tr>
<td>----------------------------</td>
<td>--------------------------------------------</td>
</tr>
<tr>
<td>Copper (Cu)</td>
<td>0.002; over 0.6: bitter, causes liver damage.</td>
</tr>
<tr>
<td>Phosphate (PO₄)</td>
<td>0.7; high levels indicate sewage contamination.</td>
</tr>
<tr>
<td>Odor</td>
<td>From organic contamination and hydrogen sulfide.</td>
</tr>
<tr>
<td>PH</td>
<td>6.8-7.5; acid (under 7) alkaline (over 7)</td>
</tr>
<tr>
<td>Sodium (Na)</td>
<td>32; high salt: diuretic; from road runoff or from water softener.</td>
</tr>
<tr>
<td>Nitrates (No₃)</td>
<td>10; over 45: from organic material harmful to human infants. Over 50: harmful to chickens. Over 75: harmful to turkeys.</td>
</tr>
<tr>
<td>Nitrites (NO₂)</td>
<td>0.4; over 4: toxic; polluted by proteinaceous organic material.</td>
</tr>
<tr>
<td>Heavy metals (lead, mercury, arsenic)</td>
<td>0.01 to 0.02: toxic.</td>
</tr>
</tbody>
</table>

In addition, well water should be examined for coliform bacterial count. To high a count, would require chlorine treatment or filtration.

**Disease surveillance**

Some areas have established a voluntary disease surveillance program. The appearance of designated infectious diseases is reported to a central agency. The following steps are taken:

1. The location of the outbreak is entered on a grid map.
2. Concerned persons are notified at once, some by phone.

These include diagnosticians, neighbors, service men, hatcheries, processing plants, salesmen, public health officials, integrated operations. Measures to be taken will have been earlier determined within the cooperating groups. This may include vaccination of threatened flocks, restricted movement of birds, restricted movement of persons, sanitizations, safe disposal of the flock (figures 7.22, 7.23, 7.24). The surveillance diseases would vary with the area, but would include such diseases as ILT, fowl cholera, pullorum, typhoid, VVND (exotic ND), *Mycoplasmas*, infectious coryza, and influenza.
Figure 7.22. Biosecurity means wearing disposable clean garments

Figure 7.23. Isolation of farm to reduce disease spread

Figure 7.24. Isolate farm with fences and no vegetation around building

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8. Disease diagnosis

Tax-supported laboratories were established many years ago in most states in the US for avian disease diagnosis. Diseases were mainly bacterial or protozoal and diagnoses relatively simple. These laboratories provided a great service in the control of pullorum-typhoid and coccidiosis, a hurdle that had to be surmounted before the modern poultry industry could develop. Reduced profits occurred also from such diseases as infectious coryza and tuberculosis. While the incidence of bacterial diseases has declined, the incidence of viral diseases, mycotoxicosis and metabolic diseases such as skeletal and circulatory problems has increased, and many are of recent origin. Viral tenosynovitis, enteric reovirus infections, chicken anemia virus (CAV), J virus of avian leukosis, and inclusion body hepatitis are viral diseases discovered during the last 3 decades. The diagnosis of viral diseases and mycotoxicosis require greater sophistication. The poultry industry has become very large and highly automated, and there is the need that diagnostic services, where deficient, be upgraded. A balanced health service program consists of the following:

--Field laboratories for posting and routine diagnosis
--A central laboratory for microbiological isolations, histological procedures, chemical and immunological assays.
--An extension service for transmission of new information to the industry, and for investigation of field problems.
--Research facilities, when required, for the clarification of unresolved field problems.

Diagnostic procedures

Necropsy and culture

The diagnostician does postmortem exams on birds, considers the case history, and performs routine culture or microscopic examinations (figure 8.0). Often this suffices to establish a diagnosis.

Figure 8.0. Necropsy exam room
Serological testing

Infection with a disease-producing agent usually stimulates the production of a specific antibody, which appears in the blood stream. Detection of the antibody is evidence that the bird is or has been infected with the corresponding disease-producing agent. Serological tests are important in diagnosis, and can usually be performed in the field laboratory. There are many modifications of serological testing. Some of these are as follows:

- Plate or tube agglutinations
- Serum neutralization
- Hemagglutination-inhibition
- Fluorescent antibody
- Agar gel precipitation test
- Enzyme-linked immunosorbent assay (ELISA): kits available from IDEXX, Inc., Portland, Maine; Synbiotics, Inc., Gaithersburg, MD; and Affinitytech in Ark.
- Complement fixation assay (ELISA)

Fluorescent antibody (FA) testing

The presence of disease-producing organisms or their toxic by-products, bacterial or viral, can often be detected directly within tissues or in substances such as feed. Procedures are rapid, and accurate, and require an ultra violet (UV) microscope. Commonly used to detect ILTV.

Bird inoculations or feeding trials

Bird inoculations are often useful in diagnosis of "wet" fowl pox and other viral diseases such as reo or adenoviruses, which may often be non-pathogenic. Feeding trials may be the only way to determine if a given feed is the source of the problem as with a toxin in the feed.

Microbial isolation and identification

Microbial isolation and identification (figures 8.1 and 8.2) is technical and normally performed in a central laboratory. Bacteria, mycoplasma and fungi can be isolated and identified in artificial media in a few days, whereas viruses need a living host (cell culture or embryo) and may take a week or longer to identify.

Figure 8.1. Mycoplasma colonies (fried-egg appearance)
Antigen capture ELISA

Monoclonal antibodies are attached to a microtiter plate. Monoclonal antibodies are highly specific, produced against only a single antigenic determinant (epitope). Antigens from homogenized tissues are captured by the antibody to the plate and a standard ELISA is done. This technique can be used to sero or subtype IBDV depending on the specificity of the reagents used (figure 8.3). It can also be used to detect AIV infections.

Figure 8.2. Embryo inoculations by CAM route for ILT and Fowl pox viruses

Figure 8.3. AC ELISA
Recombinant probe assay

Nucleic acids, complementary to nucleic acids of microorganisms, are labeled and used as probes. These probes will bind to (hybridize) to their complementary sequences and can be detected in tissues or cell cultures (in situ) using a color detection system similar to ELISA.

Polymerase chain reaction (PCR)

Nucleic acids of microorganisms are enzymatically amplified in tissues or cell culture and are then separated in electrophoretic gels, transferred to membranes and detected with labeled recombinant probes. Commercial kits are now available from IDEXX, Inc. for detecting mycoplasma, or subtyping avian leukosis viruses and IBDV’s by PCR. Used also to detect ILTV. New, more sensitive assays, which combine PCR and in situ hybridization, are now being developed to detect organisms which are latent (not replicating) or replicate at a lower rate in the tissues or cell culture (figure 8.4).

![PCR Products](image)

Figure 8.4. PCR test showing IBDV cDNA in an agarose gel

Real Time Polymerase chain reaction (RT-PCR)

New highly sensitive PCR, which can quantitate the amount of PCR product. Procedure uses fluorescent primers and/or probes and a spectophometer to measure the amount of fluorescence. Melting point curves can distinguish organisms that have only one different nucleic acid base. Can determine the HA type of AIV.

Histopathology

Tissues are sliced into thin sections, mounted on glass slides, stained, and examined for microscopic lesions specific for certain diseases. A trained technician is required. Often more than one agent can cause similar microscopic pathology so other tests are combined such as the FA, or immunoperoxidase assay.
**Immunoperoxidase assay**

Monoclonal antibodies against various organisms (sero or subtype specific) are attached to organisms found in tissue sections. Secondary antispecies monoclonal antibodies attached to a substrate are added. An enzyme (peroxidase) is then added and a reaction produces a product which turns color when indicators are added. Stained tissue sections are viewed under a microscope and stained antigen particles are evident in cells. These sections can then be counter stained for routine histopathological observation and viewed with a light microscope. With this technique a correlation between antigen presence and microscopic lesions can be made resulting in a definitive diagnosis (figure 8.5).

**Figure 8.5. Immunoperoxidase assay shows presence of IBDV RNA in the bursa**

**Western immunoblotting**

Structural proteins from isolated microorganisms are separated on electrophoretic gels and then transferred to special paper strips. The strips are incubated with monoclonal antibodies, and protein bands detected after color reactants are added in a test similar to the ELISA. This test can be used to sero or subtype an organism depending on the specificity of the reagents used.

**Chemical assays**

Chemical assays may be required for the detection of poisons as mycotoxins, insecticides, fungicides, plant poisons, drug residues and others. Corn samples are routinely checked in the U. S. for aflatoxin, ochratoxin, and T-2 toxin using commercially available "mini" columns or ELISA systems.

**Bird submissions and case histories**

When in doubt, service personnel should submit problem cases promptly to a diagnostic laboratory. A good procedure is to call the laboratory prior to submission. Select typically affected birds in various phases of the diseases as well as dead birds. A rule of thumb, submit six to eight birds if under three weeks of age, about five birds if under 12 weeks of age, and four...
birds if 12 weeks or older. Transport, if possible, in cardboard boxes, which can be burned. Ensure that birds do not smother on the way to the laboratory, which is a common occurrence. Be prepared to submit a complete case history such as the following:

<table>
<thead>
<tr>
<th>Farmer</th>
<th>Other birds on farm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hatchery</td>
<td>Type of litter</td>
</tr>
<tr>
<td>Number of birds</td>
<td>Egg production prior to the problem</td>
</tr>
<tr>
<td>Age</td>
<td>Present Egg Production</td>
</tr>
<tr>
<td>Breed and strain</td>
<td>Vaccination programs</td>
</tr>
<tr>
<td>When was illness first seen?</td>
<td>Medication history</td>
</tr>
<tr>
<td>Percent affected</td>
<td>Coccidiosis, blackhead control</td>
</tr>
<tr>
<td>Daily mortality rate?</td>
<td>Was onset gradual or sudden?</td>
</tr>
<tr>
<td>Do sick birds recover?</td>
<td>Is there a sex (gender) incidence?</td>
</tr>
<tr>
<td>Has disease previously been seen?</td>
<td>Was spread rapid or slow?</td>
</tr>
</tbody>
</table>

**Disease eradication**

Eradication is defined as the total elimination of organisms causing disease from both the bird and bird environments, and should be the objective whenever possible.

**Eradication of non-egg-transmitted diseases**

Eradication of non-egg transmitted diseases is possible when domestic fowl are the major hosts and the infectious agents do not survive for long periods of time in the environment.

**Vaccinations**

This is possible with laryngotracheitis. Success is dependent upon the cooperation of all segments of the industry within the eradication area.

**Management practices**

Exotic Newcastle disease (ND), *Salmonella pullorum*, *S. gallinarium*, and fowl plague have been eradicated from U.S. commercial flocks by quarantine and slaughter. Transmissible enteritis of turkeys, infectious coryza and fowl cholera, particularly in chickens, can be eradicated through management practices. Tuberculosis has been controlled and eventually eradicated in commercial poultry by annual replacement of a laying flock.

**Use of drugs**

During the past several years, there has been a great decline in the incidence of some parasitic diseases, namely *Ascaridia*, cecal worms, lice, red mites and probably others. The use of anthelmintic and insecticides and improved sanitation and hygiene has been an important factor.

**Vector (invertebrate hosts) control**

This has been effective for the eradication of tapeworms from commercial operations.
Blood testing in disease eradication

The National Poultry Improvement Plan (NPIP) of the USDA is now testing all breeder and multiplier flocks of chickens/turkeys for pullorum-typhoid, *Mycoplasma gallisepticum*, *M. synoviae*, and avian influenza. In addition, these flocks should be tested for lymphoid leucosis, especially the J virus subtype and turkeys for *M. meleagrisidis*. Primary breeders must constantly monitor their birds for the presence of these diseases through blood testing. Antigens are commercially available for the testing of pullorum-typhoid, *M. gallisepticum* and *M. synoviae*. *M. meleagrisidis* antigen is most difficult to prepare, but is produced by some universities.

Normally, 1% of the breeders are tested once as pullets and once during lay using serological procedures. NPIP now plans to test all commercial (non-AIV vaccinated) poultry flocks for AIV using the AGP test. Any positive flocks must be destroyed and the premises quarantined and disinfected. Depopulation is by C02 asphyxiation or the use of chemical foam. Birds and organic matter can be buried in a farm pit or class I land fill with a temporary permit issued by the State Veterinarian.

Turkey breeders should be free from *S. typhimurium* and *S. arizona*. Commercial layer pullets should be tested for *S. enteritis*. Eggs from positive flocks need to be broken and pasteurized. Small or older positive flocks should be slaughtered and the premises cleaned and disinfected. Before starting a flock again, the premises should be examined for Salmonella using swabs dragged through the house over the ground. These swabs are then inoculated in selective enrichment media for propagation of Salmonella.
9. Vaccination procedures and the immune response

This section is written to clarify the basic principles of vaccination. In human medicine, trained persons administer vaccines, in contrast, persons with no medical training may give vaccines to poultry. There can be vaccine misuse with poultry.

The implements of vaccination

The antigen

The "antigen" is any part of a microbial preparation, which is capable of inducing an immune response in the host. Single (monovalent) or multi antigens (polyvalent) are commonly included in live or killed vaccines for the induction of resistance in a host. Antigens are also used in diagnostic tests to detect the presence of antibody in sera.

The antibody

Any living organism seeks to eliminate foreign bodies. Viruses and bacteria within a living host are also foreign bodies, which the hosts seek to eliminate. They cannot be eliminated through the urine or changed chemically. A mechanism for removal is the development of a chemical substance not previously present in the blood known as the "antibody". The antibody can destroy the antigen, or the disease-producing agent. Antibodies are specific proteins (immunoglobulins [Ig]) which are capable of attaching to a specific antigen, which induced them.

A host may be quickly overwhelmed by a disease before the antibody appears. If, however, the host survives sufficiently long enough (normally a week), the presence of the disease producing agent within the body will stimulate the production of an antibody. Antibody production is important in the recovery from disease, and in general recovery does not take place in viral diseases until the antibody appears. The presence of the antibody protects the host against the disease. The antibody is not produced against the viral or bacterial cell as such, but against the proteins, or combination of carbohydrates and proteins (glycoproteins), which these cells contain. If an invading bacterial cell, for instance, contains but one protein, one antibody will be produced. If four proteins are present, four antibodies maybe produced and so on.

Antibodies in birds may be one of four different Igs, termed IgM, IgG, IgA, and IgE. IgM is produced early on, within a few days after antigen stimulation. IgM is involved in precipitation and neutralization of viruses and bacteria in circulation. IgG is produced at 4-7 days after initial stimulation and has a similar function as IgM. IgA is produced both in circulation and in local lymphoid centers for neutralization of mainly viral infections in the respiratory, enteric, and reproductive tracts. IgE is involved in the immune response to parasitic infections. IgE is also important in allergic reactions, which have received little study in birds.

Antibodies may be polyclonal, produced against several antigens, or monoclonal, which are produced against only one antigenic determinant called an "epitope". Monoclonal antibodies are highly specific and have use in diagnostic assays for differentiation of closely related organisms. Monoclonal antibodies are produced from hybridoma cells, which are formed in cell culture.
during fusion of an antibody producing mouse spleen cell and a myeloma cell. The myeloma cells are cancer cells with an unlimited life span usually obtained from special lines of mice. The hybridoma cells, once screened and expanded, will continue to produce only highly specific Ig's.

**The purposes of vaccination**

The purpose of vaccinations in birds is the stimulation of the immune response to overcome the disease. A host rendered resistant to an infection by vaccination is immunized. Immunity is the result of the formation of antibody, activated lymphoid cells, or macrophages, which are induced to the immunizing antigen. Immunity may result from recover from a natural outbreak of disease or the use of a vaccine.

Vaccines are prepared from the organisms causing the disease. In the development of a vaccine, the fully virulent organism has been "attenuated" or weakened, or the organism may be inactivated or killed (figure 9.0). Most viral vaccines consist of living attenuated agents, whereas bacterial vaccines are usually, but not always, inactivated. An exception would be for fowl cholera (FC) or mycoplasma.

Recombinant fowl pox (FP) vaccines have been produced that contain Newcastle disease (ND) virus, infectious bronchitis (IB) virus, Marek's disease (MD) virus, avian influenza, and/or infectious bursal disease (IBD) viral antigenic determinants. A portion of the viral gene, which code for the antigenic determinants, is introduced into a replicating, avirulent virus, such as FP. When the pox virus replicates it will also replicate the inserted viral gene, resulting in the production of antigen. The antigen induces the immune response without the production of whole particles and unwanted vaccine reactions.

New live bacterial vaccines are being produced by chemically altering the genetic make up of the bacteria. This may cause the bacteria to replicate at lower temperatures than normal. These so-called "temperature sensitive mutants" will replicate only in the upper part of the respiratory tract of the bird, which is at a lower temperature. These "mutants" will induce local immunity without causing disease in the lower respiratory tract. Temperature sensitive mutant vaccines have been made against mycoplasma, turkey rhinotracheitis bacteria, and fowl cholera.

![Figure 9.0. Vaccination of breeder pullets with an inactivated vaccine](image-url)
Live, conventionally produced, vaccines to produce immunity must multiply within the body. They may produce a mild disease from which the host rapidly recovers. Antibody and cellular immunity is stimulated and a state of immunity develops. Some reaction to the vaccine normally occurs, and, in general, the greater the reaction, the greater the immune response. Care must be taken at times of stress, or severe reactions may cause increased morbidity and mortality. Inactivated vaccines contain adjuvants, which can induce a local tissue reaction, thereby increasing the immune response. Adjuvants may contain aluminum hydroxide, oil emulsion, or lipid microsomes (lyposomes). Adjuvants serve to slowly release antigen, which continually stimulates the immune response.

There is always an interval of time between the administration of a vaccine and the appearance of the immune response. This varies with the individual immunized and the disease, but usually takes from 7 to 10 days. The subject, therefore, never becomes immunized immediately by active immunity. Immunity may be "active" or "passive". In active immunity, the subject actively produces the antibody in consequence of recovering from a disease or following vaccination. The antibody in the blood stream can be replaced as their loss takes place.

In passive immunity, the antibody is introduced into the host by (1) injection, (2) maternal transfer, or (3) egg yolk transfer. Passive immunity imparts an immediate, although temporary, state of immunity against an infection and protects the very young against disease organisms during the first few weeks of life. The egg yolk transfer of IgG antibodies from the hen to the chick aids the chick in fighting off infectious disease organisms. However, this parental immunity can be detrimental causing neutralization (interference) with immunization of vaccines given at an early age.

**Parental immunity**

Parental immunity and passive immunity are the same in that they refer to the transfer of antibodies from the hen to the chick via the egg yolk. The transfer is near quantitative, that is, about one-third to one-half the concentration of antibody will be present in the egg yolk as is present in the blood of the hen. Many antibodies might be transferred as for instance against NDV, IBDV, and reoviruses. The antibody content in the egg yolk at time of hatching can vary from nil to high for any disease depending upon the antibody content in the blood of the hen.

After hatching, the egg yolk is absorbed and its antibody content transferred to the blood of the chick. The chick is said to possess passive, or "parental", immunity. The parental immunity remains near constant for 3-4 days, or until the yolk is absorbed. Thereafter, it declines as the antibodies are lost. The half-life of IgG antibody is about 4 days. The amount of antibody half-lives will depend upon the amount of antibody, which was transferred. A small amount vanishes quickly, whereas a larger amount takes more time to disappear. Most of the parental immunity has vanished by the 3rd week. Experiments have shown that the level (titer) of antibody passed to the offspring at hatching is about 1/2 of that in the hen and is strain and line dependent.

Parental immunity protects the young against attacks of some diseases as, for instance, IBDV. Parental immunity, however, does not fully protect chicks against attacks of viral respiratory diseases such as IBV and NDV. The reason is as follows: The parental antibodies are in the blood. Naturally-contracted respiratory viral agents, however, are inhaled and contact their host
cells without coming into contact with the parental antibodies. Since these viruses cannot be destroyed (or neutralized) by the parental antibody, there is no hindrance to infection, and parental antibodies do not protect young birds against the viral respiratory diseases. Parental antibody in circulation does, however, prevent a viremia (virus in the blood) and the spread of IBV and NDV in the blood.

**Mechanism of immunity**

The immune response is controlled by two central lymphoid glands, the bursa of Fabricius, and the thymus. The bursa of Fabricius is a small gland near the vent, while the thymus consists of several lobes and is located along both sides of the neck. The immune response develops rapidly during embryonation. Soon after hatching, however, specific cells in the bursa known as "B" cells or "B" lymphocytes, and corresponding cells in the thymus termed "T" cells migrate to other tissues as the spleen, bone marrow, cecal tonsils, Harderian gland (located in back of the eye), and others. These are the cells involved in immune responses. Upon stimulus, the mature B cells form plasma cells, which respond with antibody production. This is termed "humoral" or circulating immunity. Some T cells may suppress B cells, called suppressor cells, and others may stimulate T cells to produce antibody, called helper cells.

The T cell’s immune response is induced through the action of cells and is termed "cell-mediated" immunity (CMI). The mechanism of CMI is complicated. One manifestation of CMI is phagocytosis, ingestion of organisms or tumor cell cells, by phagocytic cells. The T cells may receive processed antigen from macrophages or dendritic cells. Dendritic cells are specialized antigen presenting cells that reside in several lymphoid tissues including the spleen. Macrophages or other monocytes may produce monokines, which stimulate or suppress T and B cells. The T cells may also secrete lymphokines, which effect B cells or other important lymphoid cells. Important lymphokines include interferon, which inhibits viral replication, and interleukins. Interleukins are important controllers, stimulators, and suppressors of the immune response.

Vaccination, or an infection with a disease causing organism, may stimulate either or both immune systems. Humoral immunity is particularly important in the case of viral diseases, and the virus may be neutralized or inactivated upon the appearance of the antibody. Exceptions are MD, FP, and lymphoid leukemia. Thereafter, after a temporary further rise, the antibody content in the blood slowly reduces, and after 4-6 months may decrease to zero. The individual is then again susceptible to the disease. A second attack of the disease, however, is usually clinically less severe because of the "anamnestic" or memory response. Following reinfection, there may be very rapid regeneration of antibody.

Bacterial agents, on the other hand, are not destroyed upon the appearance of the antibody, although antibody production aids in the body defense. Bacterial agents are ingested and destroyed by phagocytic cells. The phagocytosis, however, is usually incomplete, and even though clinical recovery may take place from the disease, the organisms causing the disease often remain in the host for life. The host may become a "carrier". The continued presence of the organisms provides continued stimulation for antibody production, and serological tests can often be used to detect infected individuals or flocks. Vaccines are more effective in the control of viral than bacterial diseases. Drugs are often of great value in the control of bacterial diseases,
but other than the control of secondary bacterial complications, are too expensive for the control of viral diseases in poultry. Cellular immunity is important in the resistance to local infections in the gut with protozoan or helminth organisms.

In order to maintain a continued state of immunity, with the exception of such diseases as MD, Coccidiosis, and FP, the host must be re-vaccinated at 2-4 month intervals. The protection period following use of bacterial vaccines is usually less.

**Host response to vaccines**

Vaccines properly applied will induce a state of immunity persisting for 3-6 weeks, with partial protection for longer periods. The following factors affect the response to vaccines:

*Immunological competence*

The young do not respond well to vaccines. The capacity to fully respond to vaccines is termed "immunological competence". Chicks/poults generally do not become fully immunological competent until the 6th week of age, although they develop partial competence during the 12th day of embryonation and can be immunized as early as the 18th day of embryonation by *in ovo* immunization.

*Parental immunity*

The presence of parental antibodies may seriously interfere with vaccine responses. The reason is that parental antibodies, if present, may destroy or neutralize vaccines, living or inactivated, and the duration of protection following vaccination is shortened. When chicks with parental immunity are vaccinated at day-old, they are protected against the disease, except for MDV, for no longer than 1 month. If vaccination is delayed until the 7th day, the period of immunity is a little longer and may last for 4-6 weeks. The longest and best immunity is secured when birds are vaccinated at the 6th week or later. In this case, some immunity may persist up to 3 months.

For best results, therefore, vaccinations should be delayed until the 6th week or later. This is often possible with such diseases as FP, FC, coryza, and avian encephalomyelitis (AE). Outbreaks of IB, NDV, MD, and IBDV, most often occur, however, in young chicks, and vaccinations have to be given in the hatchery. When chicks are vaccinated before the 6th week, and particularly before the 3rd week, there is often the need for re-vaccination, because of immaturity of the immune response and neutralization of vaccine virus by maternal immunity.

*Immunosuppression*

Certain infectious organisms such as IBDV, chicken anemia virus (CAV), MDV, reoviruses, and mycotoxins, or nutritional deficiencies may reduce the immune response in birds by interfering with either cellular or humoral immunity. Immunosuppressed birds have poor vaccine responses and are more susceptible to infectious agents. This dysfunction can cause an increase in septicemia-toxemia and air sacculitis condemnation in the processing plant.
The titer of vaccines

The titer of vaccines corresponds to the number of vaccine particles, living or dead, per unit volume. The correct dose and titer has been determined for every vaccine. This is the responsibility of the vaccine manufacturer and is regularly monitored by the federal government, universities and private laboratories. Titer, however, can be lost through improper use, handling or storage. Pay attention to the following:

--Use vaccines according to the manufacturer's directions.

--Note the expiration date on vaccine packet. Do not use expired vaccine. Titer drops slowly even under optimum holding conditions.

--Refrigerate at all times other than actual use. Most viral vaccines consist of living organisms, and will die if not refrigerated. Lack of proper holding is a common cause for vaccination "failures".

--Dilute the vaccine according to directions. If a 5000 dose vial is used to vaccinate 10,000 birds, the titer is reduced by one-half. Diluting the vaccine by one-half dose to reduce vaccine reactions does not generally significantly reduce vaccine efficacy. However, greater reduction in vaccine titers may produce poor immunity.

--Unless immediate freezing is possible, destroy diluted and unused vaccine at end of day.

Administration of avian vaccines

Avian vaccines are administered in the following ways:

--Water
--Eye drop or intra-nasal
--In ovo (embryo)-Machine is available for renting from Embrex, Inc., Research Triangle Park, NC
--Feed
--Injection
--Coarse or fine spray
--Wing web puncture

Administration by spray, water, or on feed are mass methods of vaccine application and are preferred when effective because of reduced labor costs. In all cases where chicks/poults are vaccinated against respiratory diseases and an option is possible, administration by spray is the most effective, followed by eye-drop. Water administration of vaccines may be unsatisfactory, especially when nipple waterers are used. Vaccines normally contain a vegetable dye. Vaccines
are metered through a proportioner generally 1 oz per gallon. Waterers should not be lowered until the dye is seen at the end of the water line. All vaccine should be consumed within 2 hours. Vaccination is normally done between 7 and 10 days of age for best results. Birds should be walked in the house every half-hour, and feed lines run to encourage all birds to drink water. If ILT vaccine is given by water to broilers it is normally given once in the morning and again in the afternoon, or on two successive days, to allow all birds to consume some vaccine. The mouth and tongue of 20 birds per house should be checked for dye color to determine the efficacy of vaccination by water.

Vaccination by coarse spray is technical and persons must be knowledgeable. Effective equipment must be used properly. The vaccine may be destroyed by excess heating as emission takes place from the nozzle. Contrary to previous recommendations, the spray must be directed at the birds, not the room space. One type of portable sprayer is the ULVA Fan® from MicroWest in Houston, TX (figure 9.1). It delivers particles from 40-60 microns and works well for delivering ND and IB vaccines. The SprayMaster® from Merial, Inc. (Gainesville, GA.) or InterVet, Inc. (Millsboro, DE.) delivers 80 micro size particles and works well for ILT, reovirus and IBD vaccines. With either product, 2 vaccinators should be used per house. All curtains should be raised and fans turned off and two passes, up and back, in a house should be made for thorough coverage.

More severe reactions will occur with spray than drinking water, especially if the birds are of poor quality, are stressed, or have concurrent infections. However, coverage and efficacy of spray is superior to water and there are fewer rolling reactions as are common with the water route. Vaccinators should wear a ventilated mask when applying NDV vaccine, because conjunctivitis or a flu-like disease may arise from NDV infectious. Vaccine reactions usually appear by the third day and reach their peak by the seventh day. If reactions persist past the tenth day, antibiotics, vitamins, or chlorox should be given in the water to prevent secondary infections and weakened birds.

Figure 9.1. Spray vaccination of layers in cages

Coarse spray cabinet in the hatchery (figure 9.2) is available for application of IB, ND, IBD and reoviruses. Vaccines against AE, mycoplasmas, coccidiosis, and hemorrhagic enteritis are given
in the water. Water or spray options are available for FC, IBD, coccidiosis, and reoviruses. Injections are required for MD, erysipelas, and infectious coryza. Some attenuated strains of reoviruses and IBD must also be injected (figure 9.3).

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**Figure 9.2. Course spray cabinet vaccination in the hatchery**

Inactivated vaccines, prepared with an adjuvant, must be given by injection, and are often costly, but have the following advantages:

--These vaccines can be prepared with very high titers. Vaccine particles and antibody units neutralize or inactivate each other. In consequence of high titer, parental (or active) antibodies are removed from the blood stream. There is an excess of vaccine particles, however, and vaccinations can be made effective in birds possessing parental (or active) immunity.

--In consequence of the adjuvant, the vaccine particles are presented to the immune system over a period of time. This improves the vaccine response.

**Adjuvants:**

Substances or chemical formulations used to enhance the immune response to inactivated vaccines. They act by retaining the immunogen at the injection site, as by a depot effect, and thus delaying its release; the antigenic stimulation is prolonged and consequently increased. Some adjuvants may also stimulate macrophages, lymphocytes and other cells involved in the immune response. Salts of metals, such as those of aluminum, oil emulsions (Freund's adjuvants), and synthetic lipid vesicles (liposomes) are some of the adjuvants used.

--The vaccine reaction on the part of the host is mild and local or nil. Inactivated vaccines except for NDV or IBDV are given to pullets later than 6 weeks of age, which have received several live priming vaccines, which induce memory cells. These memory cells are needed to produce an optimum response to the killed vaccine. Killed NDV or IBDV vaccines may be given at day of age by SQ route simultaneously with live vaccine by coarse spray in broilers in areas of severe...
challenge. When chicks are vaccinated at 1-7 days against IB, ND or IBD, a booster vaccination will normally be required in 2 weeks.

The best immune response occurs when the inactivated vaccine is given intramuscular in the breast muscle. However, this can lead to abscesses, which can cause trimming and down grading in the processing plant. Other muscles can be used including the thigh or vent area. Often inactivated vaccines are given SQ in the skin behind the neck, pointing the needle away from the head, in order to avoid abscess formation. Any birds dying within one month of receiving a killed vaccine should be observed for the presence of deposited oil at the sight of injection. If none is present, the vaccination crews should be consulted and deficiencies discussed. Care must be taken that individuals not inject themselves with oil emulsions, because serious abscesses may result.

Precautions need to be taken when vaccines are given in drinking water. Be conscious of the following:

--The equipment must be free from disinfectants. As little as 1ppm chlorine or quaternary ammonium compound may inactivate or reduce the titer of vaccines. The vaccine water must be free from organic matter such as feces and debris. Rusty iron is adverse to the titer of the vaccine.

--Add one pound (500 grams) of powdered skim milk to 50 gallons (190 liters) of water, or one packet (90 grams) per 10 gallons (37 liters). The milk protects the vaccine against residues of disinfectants and the presence of feces or other organic matter.

--Every bird must get a dose of vaccine. Normally water is withheld for a period of time before vaccine is given, and all birds will be thirsty. All birds must drink. If water is withheld too long, birds may fight, and splash vaccine out of waterers, or contaminate it with droppings. During cooler months, 2 hours is normally sufficient, whereas during warmer months 1 hour of water deprivation is satisfactory.

--Consider the length of the water line from the vaccine barrel. The first birds to drink may get only pipe line water without vaccine. Check the mouth and tongue of a small number of birds for the presence of dye, which is contained in most live vaccines.

--Respiratory disease vaccines must wash over the mucosal surface of the bird’s throat in order to induce local immunity. Vaccine entering the gut can be inactivated by low pH and/or proteolytic enzymes. The vaccine will reach the respiratory tract with bell, cup, trough waterers, or nipple waterers. Therefore, water administration of IBV, NDV, or ILT vaccines is less efficient than by coarse spray.
**Facts and tips for successful vaccination**

(See following "Vaccination Schedule for Chicks" for full name of diseases mentioned in this section.)

**Facts**

--The vaccination schedule is only a guide. It must be tailored to meet your specific needs.

--Strict sanitation and isolation are essential for a satisfactory vaccination program. Vaccination is no substitute for effective management.

--Vaccinating chicks less than 10 days of age cannot be depended upon to produce a uniform or lasting immunity, even in the absence of parental immunity.

--Do not use outdated vaccines. An old product may not have adequate potency. Do not mix live or inactivate vaccines together from different manufactures; their titers of diluents may be incompatible.

--Each vaccine is designed for a specific method of application. Use only the recommended method.

--Immunity from live ND-IB combination vaccines will give dependable protection for only 2-3 months in adult birds. These vaccines will not interfere with each other.

--Live ILT should not be used in broilers if there is no history of the disease on the farm. Use of live ILT vaccine is usually controlled by the State Veterinarian and is limted to pullet replacements. ILT and FP are vaccines that can be used during an outbreak to halt the spread of infection, because the viruses spread slowly through a flock and immunity to vaccination is rapid (4 days). Water vaccination using ILT products are not as reliable and are usually given twice during the same day or on two successive days.

--Flock immunity against AE should be checked by challenge of fertile egg soon after the flock is in production.

--In problem flocks requiring FP vaccination of baby chicks, the flock should be re-vaccinated after reaching 8 weeks of age or older to assure lasting immunity.

--Force molted flocks should be re-vaccinated against IBV, NDV and ILT, because outbreaks are common after this stressful event. Vaccines should be given several days after the birds are placed back on feed.

**Tips**

--Know the disease history of your farm. Obtain a laboratory diagnosis of all disease problems.

--Do not vaccinate sick birds (except for ILT and FP).
--Record all vaccination dates, manufacturer's lot numbers, and other pertinent information on your Started-Pullet Program chart.

--Do not vaccinate for ILT, FP, or FC unless they have been a problem on your farm.

--If there is a history of FC on the farm, consult a poultry pathologist to outline a control program.

--Acquaint yourself with the advantages and limitations of each vaccine and select the one which meets your needs.

--Protect vaccines from heat and direct sunlight, which may inactivate live vaccines or cause the emulsions in inactivated vaccines to destabilize.

--Follow the manufacturer's instructions.

--Be sure each bird gets its proper dose of vaccine.

--Use a full 500 doses for 500 birds. Do not stretch the vaccine. Exceptions are made when using MDV vaccines in broilers and when administering ND, IBV or IBD vaccines by coarse spray. Dilutions of 1/2 to 1/4 are common with these vaccines.

![Figure 9.3. Day-one administration of MDV vaccine by SQ route](image)

--Do a careful job; do not rush. The time saved may be costly.

--When using the drinking-water method, be sure the water is free of sanitizers. Live viral vaccines are destroyed by these chemicals. Always add powdered milk to live vaccines when given by water route. If the water has a large amount of scale due to salts (hard water) acetic acid should be given to clear the lines, followed by powdered milk to neutralize the acid.

--Most vaccines are living, disease-producing agents. Handle them with care.
--After vaccinating, burn or disinfect all opened containers to prevent accidental spread to other poultry. (Do not break seal on container until ready to vaccinate).

--Birds may need to be given vitamin-electrolyte (stress packs) immediately before or after use of live vaccines to reduce reactions.

--If vaccine reactions continue after 7-10 days, antibiotics or antiprotozoal drugs should be given by water to reduce the reaction depending on which vaccine is given.

**Vaccination schedule for broiler chicks, pullets, breeders and layers**

<table>
<thead>
<tr>
<th>Age</th>
<th>Disease</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>01MD (HVT+SBI or HVT +301B and IBD, or SC at day one in the neck or <em>In Ovo</em> (18 days of embryonation)</td>
<td>HVT + Rispens) (1/3 to full dose)*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ND-IB Combination ND-bronchitis (ND-IB)** Method of administration coarse spray (figure 9.5), or 7-10 days ND-IB and/or IBD DW or CS 18 to 21 days</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>ND-IB, IBD</td>
<td>DW or CS (booster)</td>
</tr>
<tr>
<td>14</td>
<td>LT</td>
<td>DW or CS</td>
</tr>
<tr>
<td>4</td>
<td>Wormer (piperazine)</td>
<td>DW</td>
</tr>
<tr>
<td></td>
<td>Coccivac day 1</td>
<td>CS or gel (figure 9.5)</td>
</tr>
<tr>
<td></td>
<td>Fowl pox (mixed with MD vaccine)</td>
<td></td>
</tr>
</tbody>
</table>

**Pullets**

<table>
<thead>
<tr>
<th>Age</th>
<th>Disease</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>HVT+SB or Rispens Full dose</td>
<td>SQ</td>
</tr>
<tr>
<td>1</td>
<td>Salmonella (commercial egg strains)</td>
<td>CS (figure 9.4)</td>
</tr>
<tr>
<td>7-10</td>
<td>Coccivac®</td>
<td>DW or feed</td>
</tr>
<tr>
<td>10</td>
<td>Reovirus</td>
<td>SQ (subcutaneous)</td>
</tr>
</tbody>
</table>
Vaccination is primarily for breeder flocks. May be given up to 4 weeks prior to start of production by the DW route.

<table>
<thead>
<tr>
<th>10</th>
<th>AE</th>
<th>Eyedrop</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>LT</td>
<td>FP vaccination earlier than 6-10 weeks of age will not assure lasting immunity. Use wing web (WW) route.</td>
</tr>
<tr>
<td>12</td>
<td>FC</td>
<td>Live or killed by SQ and may require two injections.</td>
</tr>
<tr>
<td>18</td>
<td>Reovirus</td>
<td>Killed reovirus and IBD vaccines usually contain ND-IB.</td>
</tr>
</tbody>
</table>

*Optimal, HVT=Herpes Virus of turkeys, DW=drinking water, CS=Coarse Spray, and SQ=subcutaneous.

**ND-IB vaccines can be administered to chicks by the spray method. Vaccination by spray **MUST** be started during chick-rearing period. Do not start it after 16 weeks of age. First two ND vaccines should be B1 or cloned Lasota, followed by Lasota. IB vaccines should contain Mass+Conn or Mass+Ark 99 or high passage Holland. At forty-weeks with killed IBD+reovirus by SQ if titers are low or nonuniform, 4 and 22 weeks for wormer.

### Months

2  

ND-IB Combination ND-IB live vaccination must be repeated at 60-70 days intervals in both breeder and layer flocks during grow-out and through production to maintain protective immunity if killed vaccine not given at 18 weeks. Sometimes Holland serotypes (low passage) are used in older birds for increased protection.

![Day old spray vaccination for Salmonella](image-url)
**Immunosuppression**

The term "immunosuppression" refers to circumstances that interfere with the immune response. Immunosuppression and immunodepression are terms usually used synonymously. However, suppression is normally thought to be permanent. The mechanism of antibody production (humoral immunity) has been briefly described, and involves migration of the B cells from the bursa of Fabricius. CMI involves migration of T cells from the thymus. Damage may occur with both of these immune systems as follows:

**IBD**

IBD is due to a virus. The virus is widely distributed in poultry premises and chicks often become infected during the 1st week. The target organs for the virus are the bursa of Fabricius and other organs containing B lymphoid cells. When infection occurs at an early age, the virus may partially or completely destroy the B cells, and the bird loses in part its capacity to produce antibodies and defend itself against disease. There is increase susceptibility to respiratory disease organisms, inclusion body hepatitis, gangrenous dermatitis, coccidiosis, salmonellosis, coliforms, coccidiosis, MD, infectious coryza, and possibly others. In addition, there is impaired response to vaccines, and in the past IBD is thought to have been a major factor in poor vaccination responses.

Infection during the first week is detrimental. Infection at 15 days has less effect because many of the B cells have migrated. Infection at 21 days or later has a temporary effect. The first two weeks, therefore are critical, and during this period chicks require protection against infection with IBD. Effective vaccination programs have been developed. Many broiler companies have their breeder vaccination examined by challenge of progeny at two-weeks-of-age. Flocks with at least 50% of their chickens resistant to challenge are considered well protected. For details, see section on IBD.

**Mycotoxins**

Mycotoxins are also immunosuppressants. Aflatoxin, ochratoxin, T-2 toxins and fumonisins are all immunosuppressive. In this case, CMI, controlled by the thymus, is damaged. Damage occurs after the T cells have migrated and can be very substantial. The precise mechanism is unknown, but there is increased susceptibility to a number of diseases. Resistance to bacterial diseases is decreased.

**MD**

MDV increases susceptibility to coccidiosis and interferes with the action of coccidiostats. MD also markedly increases susceptibility to chronic respiratory disease--CRD (*Mycoplasma gallisepticum*). Presumably there are other effects. The mechanism is unknown.

*Mycoplasma meleagridis*

This egg-transmitted disease of turkeys has recently been shown to be immunosuppressive. The mechanism and degree of immunosuppression has not been established.
Others

Infections with certain reoviruses and adenoviruses, CAV, and *Bordetella avium* have also been shown to be immunosuppressive. Immunosuppression may also have an environmental or nutritional basis. Continued high temperatures, and particularly low and high temperatures, extremes as sometimes occur between night and day, are reported to be an immunosuppressive. Starvation and deficiencies of Vitamins E and C and certain amino acids are also immunosuppressive.

*Immunostimulation*

Immunostimulation can be achieved. The following effects are known:

Vitamin E (alpha-tocopherol)

Vitamin E supplementation (in addition to normal requirement) is reported to be an immunostimulant. When the diet of experimental chicks was supplemented with 150 ppm of vitamin E from day-old to the 5th week, there was increased resistance to ND, and in breeders, increased transfer of parental antibodies. Vitamin E also partially neutralizes the endotoxins of some bacteria.

This area of research, however, is new and knowledge is incomplete. The only claim made at the present time is that vitamin E supplementation significantly increases resistance to infection with coliform organisms in experimental birds.

Macrophage activating factor

Fort Dodge Animal Health (Overland Park, KS.) has marketed a compound (ACMAN®) sold with their HVT diluent. The substance is claimed to nonspecifically stimulate the immune response for greater immunity to a variety of vaccines used at day of age and to result in reduced morbidity and mortality associated with several disease agents.

Other immunostimulants that have shown efficacy in young chicks include spray dried plasma, egg yolk antibodies, and conjugated linolic acid. They can be added to the feed.

*Stress and resistance to disease*

Stress may be defined as the reaction to menacing threats or destructive situations. Agents, which are capable of inducing stress, are termed "stressors". These may be environmental or infectious. Important environmental stressors are temperature extremes, handling, fear, food or water deprivation, debeaking, crowding, dusty air, vaccination, ammonia fumes, moving, or social stresses such as the introduction of birds into an established social order. Stressors of an infectious nature include pathogenic microorganisms, and internal and external parasites.
The reaction to stressors is complicated. Stress is first perceived by the bird in the brain. The pituitary gland secretes a substance known as "adreno-cortico-tropic hormone" (ACTH). The ACTH is carried in the blood to the adrenal glands (near the kidneys). The adrenals respond by secreting a substance known as "corticosterone". Corticosterone sets up an alarm reaction in the bird, mobilizing against the stress. This has been termed the "flight or fight" syndrome. Physiological changes take place. There is increased blood pressure and heartbeat, dilation of blood vessels, and increased alertness. Unless the stress is lethal, however, the bird soon returns to normal. Antibiotics are incorrectly thought of as anti-stress agents. Anti-stress agents act by reducing the level of corticosterone in the blood. Most antibiotics act only against bacterial agents. Continued elevation of corticosterone, however has far-reaching effects. There is increased feed intake, decreased growth rate and decreased antibody production. There is decreased resistance to infectious microorganisms.

On the other hand, birds with low stress threshold (continued lowered corticosterone) also show altered effects. They are larger, grow faster, are less active, less adaptive, have less fat, lay larger but fewer eggs and show increased antibody production. They have increased resistance to viral diseases but lowered resistance to bacterial diseases. Increased resistance to viral diseases may be very great, in instances as much as a ten fold increase.

Wild birds to be able to survive must be able to react to stress, for they are subject to such environmental hazards as temperature extremes, storms, deprivation of feed or water, and must be alert against predators. Domestic birds are not subject to these risks and do not need the same alarm reaction to stress. Potential applications are as follows:

--Administer tranquilizers or sedatives. Reserpine has attracted the most attention. This hormone relieves stresses resulting from transportation, debeaking, and vaccination reactions which otherwise may prove fatal. Reserpine also increases tolerance to heat. The drug is used to combat aortic rupture in turkeys. It cannot be given continually for there are toxic effects on the thyroid.

--Reduce stress as much as possible. One of the most effective ways to reduce stress is to darken the house. Birds can be grown in complete darkness. In darkened houses, the birds are less active and more resources become available for growth. Along with lowered corticosterone levels, these birds have increased resistance to viral and mycoplasma diseases. They respond better to vaccinations.

Controlled lighting is a new area in poultry production. Alteration of lighting and feed times during the early life for broilers permit maximum growth rates combined with minimum expenditure on feed and reduced incidence of leg problems. There is better feed conversion, more economy and reduced risks from pecking and cannibalism. Broilers are placed on a step down (decreased photo period during the first 2 weeks of age, followed by a step up (increased lighting) over the next two weeks. Rearing pullets in reduced photoperiod of 8 hours a day in dark-out houses, during seasons of increasing light, also brings birds into production with more uniform body weight, which results in higher more sustained egg production peaks.
Poultry disease groupings

The following disease groupings are to be regarded as generally but not rigidly correct. There is a good deal of overlapping.

Non-infectious causes for poor bird health (all ages)

--Ascites
--Ammonia
--Hysteria
--Aneurysms
--Impaction
--Botulism
--Mycotoxicosis
--Cage layer fatigue
--Nutrient deficiencies
--Cannibalism
--Osteodystrophy syndrome
--Drug or chemical toxicities
--Scabby hip dermatitis
--Fatty liver syndrome
--Sudden death syndrome
--Heat stroke
--Green muscle disease

Infectious diseases, which may be seen during the 1st week

--Arizonosis
--Salmonellosis
--Aspergillosis
--Avian Encephalomyelitis
--Omphalitis
--Yolk sac infection

Infectious Diseases, which may be seen during the 1st to 6th week

--Arizonosis
--Infectious bronchitis (figure 9.6)
--Aspergillosis
--Infectious bursal disease
--Avian encephalomyelitis
--Infectious sinusitis
--Botulism
--Marek’s disease
--Coccidiosis (figure 9.5)
--Mycoplasma meleagridis
--M. synoviae
--Necrotic dermatitis
--Chicken anemia virus
--Necrotic enteritis
--Chronic respiratory disease

Figure 9.5. Day one consumption of cocci vaccine

Figure 9.6. Course spray vaccination for ND, IB and/or IBD at day of age.
10. Antibiotics and Probiotics for disease control

Vaccination can offer a method of preventing poultry disease agents from becoming established. In contrast, drugs and antibiotics are often used in the poultry industry to alleviate the clinical signs from diseases. The drugs make up an unassociated list of chemicals. A great many are specific for a certain disease or for a group of similar diseases. New ones may come on the market, others may be taken off by the Federal Government, whereas others are in experimentation.

Drugs are used for three reasons:

--To aid in promoting growth and better feed conversion.
--To treat infectious diseases.
--To help prevent diseases from becoming established in the flock.

Drugs in disease control

How drugs are administered

Drugs may be administered as follows:

--through the feed
--through the drinking water
--through injections

Water solutions and suspensions

The preparations used to treat birds have varying properties, making some of them acceptable for administration in the drinking water or by injections, whereas others are not.

--Some form solutions. These are suited for drinking water. They may also be used as feed additives. The water-soluble forms are more expensive than the insoluble forms used as feed additives.

--Some form suspensions. Drugs for water application must mix uniformly. When the drug goes into solution the mix is uniform. However, soluble forms of some drugs are not available. Some drugs go into suspension; that is, they do not dissolve but float in the water and do not settle out. These also may be used as feed additives.

--Some do not form solutions or suspensions. These, for the most part, are the insoluble drugs, and their use is confined to administration in the feed.

--Some do not pass from the intestines to the bloodstream. This property may or may not be an advantage. If the microorganism involved is one that localizes in the intestinal tract, it is advantageous that the drug not be absorbed, but remain in the intestines to act on the organism. However, if the drug must reach the
bloodstream to be effective, its ability to pass the intestinal wall becomes important.

**Classification of Drugs**

Examples of drugs and some of their effects when used for disease control are shown below. The list is not complete, and some drugs that are available in other countries, but have yet to be approved for general use in the United States. Many are sold under other trade names in various countries.

**Sulfonamides**

Sulfonamides represent a group of synthetic drugs that inhibit the use of para-amino-benzoic acid (PABA) by the bird, a chemical necessary for the synthesis of folic acid. This in turn reduces cell multiplication. Since bacteria have a higher need for folic acid than the bird, their cell division is entirely inhibited. Sulfonamides are bacteriostatic rather than bacteriocidal.

Most drugs in this group produce toxicity; thus, they must be used in prescribed low doses. Sulfonamides are especially active against *Salmonella* organisms. The sulfonamide group includes sulfachloropyrazine (Esb3®, sulfadimethoxine (and ormetoprim), sulfaethoxypyridazine (SAE), sulmethazine (Sulmet®), sulfathiazole, and sulfaquinoxaline (SQ®).

Most sulfonamides depress egg production at high levels, particularly sulfaquinoxaline. They may also produce secondary hemorrhagic anemia. Sulfadimethoxine is usually supplemented with Ormetoprim to augment the action of the sulfonamide. Sulfathiazole is a weak sulfonamide and is used for treating infectious coryza. Some sulfa drugs have a four-week withdrawal period in chickens and therefore, have a limited use in broilers, which are normally processed at six-weeks-of-age.

**Nitrofurans**

The list is limited to furazolidone (nf-180®, Furox®) and nitrofurazone (nfz®, Amifur®). These drugs are used for gram negative bacteria and have some coccidiostatic properties. They also have growth promotant activity. The Food and Drug Administration (FDA) has recently removed these compounds from the list of approved compounds that can be used in United States poultry flocks.

**Coccidial-specific drugs**

In this group are the coccidiostats. However, some also are effective against poultry pathogens (bacteria) other than coccidiosis. Furthermore, some drugs from other groups used to treat birds afflicted with certain diseases, also have anticoccidial properties in some degree. These drugs are slowly being replaced by vaccination.
For additional drugs of this type, consult the manufacturer or your pathology specialist. This book does not indorse any specific drug over another. Some common coccidial-specific drugs are:

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amprolium</td>
<td>Amprol®</td>
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<tr>
<td>Clopidol</td>
<td>Coyden®</td>
</tr>
<tr>
<td>Decoquinate</td>
<td>Deccox®</td>
</tr>
<tr>
<td>Halofuginone</td>
<td>Stenorol®</td>
</tr>
<tr>
<td>Lasalocid</td>
<td>Avatec®</td>
</tr>
<tr>
<td>Maduramicin</td>
<td>Cygro®</td>
</tr>
<tr>
<td>Monensin sodium</td>
<td>Coban®</td>
</tr>
<tr>
<td>Narasin</td>
<td>Monteban®</td>
</tr>
<tr>
<td>Narasin+Nicarbazin</td>
<td>Maxiban®</td>
</tr>
<tr>
<td>Nequinate</td>
<td>Statyl®</td>
</tr>
<tr>
<td>Nicarbazin</td>
<td>Nicarb®</td>
</tr>
<tr>
<td>Robenidine</td>
<td>Robenz®</td>
</tr>
<tr>
<td>Salinomycin</td>
<td>Bio-Cox®</td>
</tr>
<tr>
<td>Sulfadiazine</td>
<td>Several brands</td>
</tr>
<tr>
<td>Zoalene</td>
<td>Zoalmix®</td>
</tr>
</tbody>
</table>

**Anthelmintics**

Anthelmintics, for controlling worms, have a purging action, and with some degree of variability, remove worms from the intestinal tract. A partial list is as follows:

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butynorate</td>
<td>Tinostat®</td>
</tr>
<tr>
<td>Cambendazole</td>
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</tr>
<tr>
<td>Coumaphos</td>
<td>Meldane®</td>
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<tr>
<td>Fenbendazole</td>
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<tr>
<td>Hygromycin B</td>
<td>Hygromix®</td>
</tr>
<tr>
<td>Levamisole</td>
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<tr>
<td>Mebendazole</td>
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</tr>
<tr>
<td>Phenothiazine</td>
<td>Several brands</td>
</tr>
<tr>
<td>Piperazine</td>
<td>Several brands</td>
</tr>
<tr>
<td>Tetramisole</td>
<td></td>
</tr>
</tbody>
</table>

**Drug treatment**

**Potency of drug treatment**

For any drug to be effective, it must locate at the point of infection in a concentration high enough to combat the invasion of the microorganisms. The amount of the drug in the blood
serum is one indication of concentration; another is the quantity in the urine; another is the amount in the intestinal contents.

Drugs differ in their ability to impart concentrations in the blood, urine, intestinal contents, and body fluids. The dosages recommended by the manufacturer have been worked out to obtain body concentrations that are adequate to reduce the infection. These recommendations should be followed carefully.

Once a dose of a drug is administered, the concentration in the body soon reaches a maximum level. This maximum will be reached in the blood in 3-4 hours. Then the elimination of the drug begins and concentration drops off rapidly. In many instances the drug is depleted in 24 hours. If the drug is effective only at maximum concentrations, the period during which it will affect microorganisms is short. To administer less of the drug than the recommended amount will result in body concentrations under those required to effect a kill; to over-administer is costly and of little value.

Overdose is sometimes detrimental. With certain drugs an overdose may produce detrimental effects. These effects are twofold.

--- Injurious to the bird. They may cause a toxic reaction or otherwise affect some physiological function of the bird.

--- Form residues in the tissues. Some drugs are not broken down during the process of digestion and metabolism and are not eliminated, and as some drugs are toxic or poisonous, their accumulation in the tissues gradually produces a more drastic reaction. Some are deposited in egg yolk and albumen.

Drug retention and humans

Some drugs are not altered during the process of digestion and metabolism and are not expelled from the body of the chicken in the feces and urine after a chemical breakdown. They soon accumulate to an extent that they may be injurious to humans when the poultry meat or the eggs are eaten.

The FDA has established levels of tolerance of the drugs in the tissues or eggs. Above these levels the safety of the meat or eggs for human food is questioned, and in many instances the government may condemn the produce as unfit for consumption and destroy it. Drug manufacturers are aware of this problem and write their directions for administration carefully.

Tolerance levels and administration levels

Because certain drugs may be retained in the tissues and accumulate to a detrimental level, feed regulatory agencies have set up standards showing the amount of each drug that may be used for all methods of administration (feed, water, injection, and so forth). Feed manufacturers and poultrymen must abide by these directives, and in most instances feed manufacturers must furnish a tag with the feed to show the name and amount of the drug added.
Combinations of some drugs may be cumulative

Some drugs, similar in chemical makeup, supplement the retention potential of other drugs. As a consequence, regulatory agencies will allow only certain drugs to be fed simultaneously; others may not be fed at the same time.

Length of drug treatment

To be effective, any drug must remain at a high concentration in the body from 3-5 days. This is difficult, as the period of maximum concentration is of short duration after a single administration of the drug. Administration in the feed or water on a continuing basis is the only acceptable method, but this may prove costly. Low-level feeding of a drug to alleviate the disease condition usually is of little benefit. Second administrations of some drugs and antibiotics usually are less effective than the first. Either procedure may foster the appearance of drug-resistant organisms. Drugs are eliminated in the urine much more rapidly after the second administration than after the first.

Injecting a drug produces only temporary effects. Although the activity reaches a high level quickly, it dissipates more rapidly. Continuous injections every day or two require too much labor and are uneconomical. The only economical time for injection of drugs in broilers is at hatch, using automated machines to deliver antibiotics mixed with vaccines for reduced first week mortality due to bacteria. In some countries, where labor is cheap, breeders or layers can be economically injected with antibiotics in the field. Some drugs used for injection are dissolved in water. These are absorbed very rapidly by the system, but their effective duration is short.

Antibiotics

An antibiotic is a substance that can inhibit the growth of or kill another microorganism. There are hundreds of such antibiotics, but through biological testing, only a few are of value against certain poultry conditions.

How antibiotics work

Antibiotics are used for disease control in poultry. Usually they are specific for those diseases caused by bacteria or related organisms. The beneficial effects of antibiotics are due to their ability to disrupt various phases of cellular metabolism. An antibiotic will prevent bacterial multiplication, provided enough is present to attack all bacteria. If the amount of antibiotic is small and the number of bacteria is large, the antibiotic will not produce its full effect. Under this situation, bacterial resistance to drugs can occur. This resistance may be passed on to humans or other animals, making the drugs less effective. The FDA, therefore, has put the Food Animal industry under increase pressure to reduce antibiotic usage.

Antibiotics may also destroy beneficial bacteria, which make up the intestinal flora. This can result in the increase in pathogenic microbes such as Salmonella or yeast. Therefore, continued
use of broad spectrum antibiotics should be avoided. Most antibiotics are given in the feed. In some instances, however, they are added to the water so that they may reach the digestive tract and the bloodstream faster, for some birds will not eat during the course of a severe disease outbreak, but will drink. In other cases, certain antibiotics may be injected.

**Facts about some antibiotics**

*The following is a partial list of antibiotics*

**Bacitracin (Zinc Bacitracin®)**
- Form: Feed and water
- Intestinal absorption, none.
- Used primarily for treatment of necrotic enteritis and improved feed conversion.

**Ceftiofur* (Naxal®)**
- Form: Injection.
- Intestinal absorption, poor.
- Used for controlling bacteria and mycoplasma

**Chlortetracycline (Aureomycin®)**
- Form: Feed and water.
- Never used for an injection.
- May be potentiated.
- Intestinal absorption, medium.
- Slightly anticoccidial. Used for controlling bacteria.
- Levels over 100 g per ton (2,000 lb) are not to be fed to laying chickens.
- Increases growth rate.
- At high levels do not feed continuously to young chicks for more than 5 days.

**Enteroflaxin (Baytril®)* or Danoflaxin (water)***
- Form: Injectable, water.
- Intestinal absorption, medium.
- Used for controlling bacteria and mycoplasma.

**Erythromycin**
- Form: Feed, water, injectable.
- Intestinal absorption, low.
- Used for controlling bacteria.

**Gentamicin**
- Form: Injectable.
- Absorption poor.
- Used at day-one for controlling bacteria.
Lincomycin (Lincomix®) - Spectinomycin - LS 50 or LS 100®
Form: Feed, water.
Absorption poor. Used for controlling bacteria.

Neomycin sulfate (Neomycin®)
Form: Feed, water.
Intestinal absorption, none.
Used for controlling bacterial enteritis

Oxytetracycline (Terramycin®)
Form: Feed, water, injectable.
When injected, may cause skin irritation.
Do not feed to laying hens at levels higher than 200g per ton (2,000 lb).
In feed of low calcium content do not feed for more than 5 days.
Slightly anticoccidial. Used for controlling bacteria.
Intestinal absorption, low to medium.

Penicillin
Form: Feed, water, injectable.
Generally used as an injection.
Intestinal absorption, low.
Aids in growth promotion.
Generally not used anymore because of wide spread bacterial resistance.

Streptomycin
Form: Feed, water, injectable.
Large injections somewhat toxic.
Intestinal absorption, none.
Used for bacterial enteritis.

Tilmicosin
Form: Water
Intestinal absorption, low.
Used for controlling mycoplasma.

Tylosin (Tylan®)
Form: Feed, water, injectable.
Intestinal absorption, low.
Used for controlling mycoplasma.
Low-level antibiotic feeding

Some antibiotics are added to feed continuously at a low level to improve growth and feed conversion. This supplementation is not to be confused with therapeutic feeding of high levels. Because of the possibility of resistance to antibiotics being transferrable to humans, this procedure has not been approved in many countries. But this probability is very low in the case of those antibiotics that are not absorbed from the intestinal tract. Some of these include bacitracin, chlortetracycline, flavomycin, and virginamycin.

Resistance to antibiotics

When antibiotics are administered over a long period of time, particularly at a low level, certain species of bacteria become resistant, and finally the resistance becomes so great that the antibiotic is ineffective. In most instances resistance develops only to those antibiotics that are absorbed from the intestinal tract. One antibiotic not absorbed is bacitracin. Therefore, bacteria causing a systemic infection will not become resistant to bacitracin.

Antibiotic sensitivity test

Some antibiotics used in the poultry industry produce major effects in treating specific diseases; others are less valuable, and some are ineffective. In some instances the organisms have become resistant to the antibiotic, producing a change in the value of the drug. The laboratory employs a technique known as a sensitivity test to determine which antibiotics will be effective in treating a disease. The organisms are cultured and grown on media in which various antibiotics have been incorporated at prescribed concentrations. If an antibiotic is to be effective in treating a disease, the antibiotic in the medium will prevent reproduction of the organisms and produce a clear circular zone of inhibition, which can be measured. This test shows antibiotics that will be ineffective.

Antibiotic potential

Certain antibiotics are used to treat diseases localizing in the intestinal tract. In these cases the antibiotic is administered in the feed or the drinking water, and soon reaches the portion of the digestive tract affected by the disease. If the amount in the tract is identical with that consumed; there is no loss. However, many diseases are systemic, and for the antibiotic to be effective it must leave the digestive tract, be taken into the bloodstream, and be transported to the point of infection. During this process some of the capabilities of the antibiotic are lost. To prevent most of this loss, certain antibiotics are given by injection. The loss of the antibiotic when given orally is not the same for all antibiotics. This variation is probably due to the fact that all are not equally absorbed from the intestinal tract.

Oxytetracycline (Terramycin®) and chlortetracycline (Aureomycin®) are examples of two commonly used antibiotics that show this difference. They are of equal importance in treating intestinal disorders, but more than twice as much chlortetracycline is absorbed from the intestinal
tract as oxytetracycline. In order to get the same amount of the antibiotic into the blood, more than twice as much oxytetracycline as chlortetracycline must be fed.

**Increasing activity of an antibiotic**

Calcium from the feed forms an insoluble salt when combined with oxytetracycline and chlortetracycline in the intestinal tract. This salt is insoluble and cannot be absorbed into the bloodstream. If the calcium in that ration is reduced, the absorption is increased, as a small quantity of insoluble salts is produced. The amount of each antibiotic absorbed may be increased more than twofold by reducing calcium in the ration.

**Terephthalic acid (TPA) and potentiation**

This drug produces its effect by reducing elimination of the antibiotic in the urine. It increases the response to chlortetracycline four times, and two times to oxytetracycline.

*Caution: The use of terephthalic acid is illegal in some countries.*

**Potentiation cumulative**

Potentiation from reducing the calcium in the feed and from TPA is additive; thus the value of chlortetracycline may be increased eight times when both methods of potentiation are used together. The cost of feeding these antibiotics is materially reduced, and it is possible to use high-potentiated levels economically.

**How to potentiate a feed**

--Remove the added calcium from the formula. This method is not always the most practical because the feed will be low in calcium. It should not be fed to young chicks for over 5 days. A rachitic condition may develop if fed longer.

--Remove the added calcium from the formula and replace it with 39 lb (13.6 kg) of sodium sulfate per ton (2,000 lb) of feed. The sodium sulfate removes the soluble calcium from the intestinal tract by forming calcium sulfate rather than uniting with the oxytetracycline.

--Add TPA at the rate of 0.4%. This means 8 lb (3.6 kg) per ton (2,000 lb) of feed.

**Withdrawal period for drugs and antibiotics**

In order to protect the consumer of poultry and eggs, and still provide effective drugs in the United States, the FDA has set very precise tolerances for drug residues. Failure to properly withdraw a drug before slaughter can result in illegal residues. The preslaughter withdrawal time for many drugs is given in number of days that must pass between the last treatment with the drug, and the day on which the chicken may be shipped for slaughter. The following list is not complete, but shows the withdrawal periods for many common drugs. For a more complete list,
and more information in the United States write: Bureau of Veterinary Medicine, Food and Drug Administration, United States Department of Health, Education, and Welfare, Rockville, Maryland 20857. Although the following list is in effect at this writing, the list and withdrawal periods change often. Secure the latest information from the Rockville, MD address or a current Feed Additive Compendium.

<table>
<thead>
<tr>
<th>Active Ingredient</th>
<th>Withdrawal Days for Chickens*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amprolium</td>
<td>5</td>
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<tr>
<td>Bacitracin</td>
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<tr>
<td>Bambermycins</td>
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</tr>
<tr>
<td>Chlortetracycline</td>
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<tr>
<td>Clopidol</td>
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</tr>
<tr>
<td>Dihydrostreptomycin sulfate and streptomycin</td>
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</tr>
<tr>
<td>Sulfate (injectable)</td>
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<tr>
<td>Erythromycin</td>
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<tr>
<td>Furazolidone</td>
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<tr>
<td>Gentamicin</td>
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<td>Novobiocin</td>
<td>4</td>
</tr>
<tr>
<td>Oxytetracycline</td>
<td>3 (feed)</td>
</tr>
<tr>
<td>Roxarsone</td>
<td>5</td>
</tr>
<tr>
<td>Salinomycin</td>
<td>0</td>
</tr>
<tr>
<td>Sodium sulfachloropyrazine monohydrate</td>
<td>4</td>
</tr>
<tr>
<td>Spencinomycin dihydrochloride pentahydrate</td>
<td>5 (water)</td>
</tr>
<tr>
<td>Streptomycin sulfate</td>
<td>4 (water)</td>
</tr>
<tr>
<td>Sulfadimethoxine</td>
<td>5 (water)</td>
</tr>
<tr>
<td>Sulfanitran and aklomide</td>
<td>5</td>
</tr>
<tr>
<td>Sulfadoxin</td>
<td>10</td>
</tr>
<tr>
<td>Tylosin</td>
<td>5 (feed)</td>
</tr>
<tr>
<td>Zoalene</td>
<td>5</td>
</tr>
</tbody>
</table>

*Withdrawal time for turkeys may vary.
Drugs and drug administrations

Drugs and vaccines used in poultry disease control

Some of the Drugs and vaccines used in the U.S. for the maintenance of poultry health include the following:

**Anthelmintics:** Butnorate (Polystat®, Tinostat®), coumaphos (Meldane®), hygromycin B, phenothiazine, piperazine, Methrydine,** Meretin,** Levamisole (Ripercol®), Tetramisole,** Mebendazole,** (Mebenvet®), Cambendazole* (Ascapilla®), Fenbendazole (Panacure®).

**Antibiotics:** Amidacin (Amiglyde®), Amoxicillin (Amoxi®), Ampicillin (PolyPlex®), Bacitracin (Baciferm®), Bambermycins, chlortetracycline (Aureomycin®), erythromycin, lincomycin, neomycin, novobiocin (Albamix®), nystatin (Myco-20®), oxytetracycline (Terramycin), penicillin, spectinomycin (Spectam®), streptomycin, tylosin, gentamicin (Garisol®), ceftifur** (Naxal®), Eneroflaxin (Baytril®), Chloramphenicol (Chloromycetin®), Sulfadimethoxine (Rofenaid®, Agribon®), sulfadiazine, sulfamethazine (Sulmet®), sulfathiazole, tylosin (tylan), tilmicosin.

**Anticoccidials:** Aklomide (Aklomix®), amprolium (Amprol®, Amprol plus®), buquinolate (Bonaid®), clodid (Coyden®), decoquinate (Deccox®), Lasalocid (Avatec®), monensin (Coban®), nicarbazin, nitrofurazone (nfz®), nitromide-sulfamitran (Unistat®), robenidine (Robenz®), sulfonamides, Tinostat®, zoaline (Zoamin®), Salinomycin (Bio-Cox®), Narasin (Monteban®), Halofuginone (Stenorol®), Maduramicin (Cygro®), Narasin and Nicarbasin (Maxiban®), Semiduramicin (Aviax®).

**Antihistomonads:** Carbarsone (Carbo-O-Sep®), dimetridazole (Emtrymix®), furazolidone, nitarsone (Histostat®), ipronidazole (Ipropan®), Metronidazole (Flagyl®).

**Insecticides:** Carbaryl (Seven®), coumaphos (Co-Ral®), Korlan®, malathion, nicotine sulfate, Rabon®, Ivermectin (Ivomec®).

**Antimycotics:** Amphotericin B (Fungizone®), Gentian Violet, Nystatin (Mycostatin®).

**Others:** Furazolidone (nf-180®), roxarsone, and arsanilic acid.

**Vaccines:** Avian encephalomyelitis (AE), Chicken anemia virus (CAV), Coccidiosis, E. coli, Egg drop syndrome,* Enteric reoviruses, Erysipelas, Fowl cholera (FC), Fowl pox (FP), Hemorrhagic enteritis, Infectious coryza, Infectious bronchitis (IB), Infectious bursal disease (IBD), Influenza (AI), Laryngotracheitis (ILT), Newcastle disease (ND), Marek's disease (MD), Mycoplasma gallisepticum, M. synoviae, Paramyxovirus, Salmonellosis, Swollen head syndrome*, Viral arthritis (VA), Turkey rhinotracheitis,

*Not available in United States. **Products not approved in United States.
Drugs and chemicals useful in disease and parasite control in poultry

Generic

name/company/form/route/species/dosage/frequency/duration/notes

Amprol or Corid - Merck
Solution (9.6%).
A soluble powder containing 132 g/kg.
Water.
Food.
Most.
2 ml/gallon.
See maker's instructions.
5 days or longer.
For treatment of coccidiosis.

Chlorotetracycline
Aureomycin Soluble Powder (Cyanamide).
Terramycin Soluble Powder (Phibro).
A green-tinted soluble powder containing 55 g per kg.
A soluble powder containing 55 g/kg. A level measure holding 200 mg is supplied with the pack.
Drinking water, food.
Terramycin Soluble Powder 5 mg/g.
2.5-7.5 mg/30 ml, 50-150 mg/500 ml (pint), 1 mg/oz (30 g) of food.
Palatability may be increased by adding honey to the water. Rapidly loses its potency in solution.
Therefore, it must be changed at least every 24 hours. Use this as the only source of drinking water. Antibacterial.

Dimetriadazole*
Emtryl. Antiprotozoal.
Soluble powder containing 40% Dimetriadazole 400 mg/g powder.
Oral in drinking water.
1 oz (30 g) of soluble powder/10 gallons (45 liters).

Erythromycin
Erythrocin Injectable
Erythrocin i.v.
A sterile water miscible solution containing 200 mg/ml.
A sterile powder for reconstitution 1 g vial add 20 ml water to produce 5%w/v/50mg/ml i.m., s.c.
Inhalation therapy by nebulization.
10-25 mg/kg once daily.
1 ml of reconstituted injection in 10 ml normal saline 15-minute treatment t.i.d. or q.i.d.
Can cause a severe reaction when injected IM. Useful for the treatment of chronic respiratory infection, air sacculitis, sinusitis, and some enteric infections including Campylobacter.
**Flagyl-S**
Suspension containing 200mg/5ml in a 125-ml bottle, 50 mg/kg once daily.
In humans used against a number of anaerobic infections.
Not as toxic as Dimetridazole. Toxic for finches. Antiprotozoal.

**Gentamicin**
(Gentocin-Schering)
Injectable (50 mg/ml).
I.M. Antibacterial.
Chickens.
5 mg/kg.
t.i.d. 5 to 10 days.
The dosages maintain therapeutic blood level.

**Oxytetracycline**
Long-acting (LA 200 - Phibro).
Injectable.
I.M.
Most.
200 mg/kg.
s.i.d. for 3-5 days.
Has worked well in treating chlamydiosis in breeding birds to control outbreak and while getting birds to eat form of CTC or OTC to finish treatment.

**Procaine penicillin G and benzathine penicillin**
Injectable.
I.M.
Turkey.
11/100 mg/kg of each drugs
i.d. or every two days.
Provides therapeutic blood levels for one to two days. Care must be used in the treatment of small birds because of potential procaine overdose.

**Lincomycin and Spectinomycin (LS-50 - Upjohn)**
Soluble powder (16.7 gm lincomycin and 33.3 gm spectinomycin/2.55 oz).
Water.
Most. Antibacterial.
1/8-1/4 level tsp/pint 10-14 days for chronic respiratory disease when *Mycoplasma* is suspected.
Therapy may be extended if necessary. May also be effective for mild enteric infections. Sugar may be added to improve acceptance.

**Neomycin**
Biosol-M-Upjohn solution with methscopolamine bromide.
Water.
Bacterial enteritis.
1-3 days.
Contains anticholinergic; care must be taken not to overdose.
**Nitrofurazone***
Soluble powder (9.3%).
Water.
1 tsp/gallon-1/2 tsp/gallon - put in water 7-10 days.
Excellent for the treatment of gram-negative especially *E. coli* enteric infections. Will slow the spread of salmonellosis in a flock. Effective for many strains of coccidia.

**Metronidazole***
Flagyl
Tablets containing 200 mg Metronidazole.
Oral.
Pigeons, 1/10th of a tablet for 5 days.
Active against *Trichomoniasis* and *Giardiasis*.

**Nystatin**
Nystan Oral Suspension.
Mycostatin-20.
A suspension containing 100,000 units/in a 30 ml dropper.
A powder containing 44 g/kg.
Oral.
In feed.
2-7 ml/kg, b.i.d. or t.i.d. for 7-14 days.
2.25 kg/20 ton.
Not absorbed from the gut, therefore very safe. Not active against *Aspergillus*. Useful for treating candidiasis.
Suitable only for treating large flocks for fungi.

**Piperazine**
Suspension.
Oral.
Poultry.
100-500 mg/kg.
Once; repeat in 10-14 days.
For ascarids in poultry.

**Pyrethrin**
Spray. External parsite (Lice and mites).
Topical.
Most.
Lightly mist feathers.
Repeat as necessary.
For external parasites, especially lice, which are resistant to carbaryl. When treating lice, spray must be applied in axillary area with wing extended.

**Streptomycin**
Injectable.
I.M. antibacterial.
Large birds.
30 mg/kg.
b.i.d. or t.i.d.

*Spectinomycin*
(Spectam®)
Water-soluble solution.
Water.
Antibacterial.
20 cc/gallon.
5-10 days.
For gram-negative enteric infections. Good for flock treatment.

*Spectinomycin*
Spectam Injectable.
Spectam® soluble.
A sterile solution for injection containing 100 mg/ml.
i.m., s.c., intra-sinus.
Drinking water, food.
Drinking water.
10-45 mg/kg poultry daily.
100-200 mg/150 ml or 100-200 mg/kg body weight daily.
100 g/20 gallons or 90 liters.
A wide range of activity similar to Gentamicin. Very effective against Salmonella. Not absorbed from the gut. Useful for "sour crop" and diarrhea. Add one-quarter teaspoonful of honey to increase palatability. Suitable for treatment of large numbers of birds.

*Tilmicosin*
Water soluble (Elanco).
Well distributed in the tissues. A safe antibiotic with a narrow range of activity against Gram-negative bacteria, *Pasteurella multocida*, and Mycoplasma. Useful for upper-respiratory infection

*Tylosin*
Tylan 50® Injectable (Elanco).
A sterile 50% injectable solution in propylene glycol containing 50 mg/ml.
i.m.
10-30 mg/kg t.i.d. or q.i.d.
Highly lipid-soluble and well distributed in the tissues. A safe antibiotic with a narrow range of activity against Gram-negative bacteria and Mycoplasma. Useful for upper-respiratory infection.

*Not approved for use in United States.*
Selected Pharmaceutical Houses and Products for Avian Use:

This list is partial and no endorsements are made by this CD.


**American Cyanamid Co.**, P.O. Box 400, Princeton, NJ 08540 (Robenz-Robendine HC1, Aureomycin-chlortetracycline soluble powder, Polyotic-tetracycline soluble powder, Ripercol-L® injectable solution-levamisole, Sodium sulfamethazine soluble powder-sodium sulfamethazine solution 12.5%, Cygro®-maduramicin).

**Elanco Products Company**, P.O. Box 1750, Indianapolis, IN 46206 (Tylan® plus vitamins-tylosin powder, Hygromix-Hygromycin, Coban-Monensin, Tylan 50 injection-tylosin, Tylan 200-tylosin nebulize, Monteban®-Narsin).


**Novis International**, 20 Research Park Drive, St. Charles, Missouri 63304. Feed additives


**A.H. Robins Company**, Pharmaceutical Division, 1407 Cummins Drive, Richmond, VA 23220 (Sainomycin).


**Rhone-Poulenc, Inc.**, Hess and Clark Division, Ashland, OH 44805 (Meldane®-Coumaphos-Bacifern®-Zinc Bacitracin).

**Schering-Plough Animal Health Corporation**, 2000 Galloping Hill Road, Kenilworth, NJ 07033 (Gentocin® solution-gentamicin), vaccines.
Probiotics

A relatively new concept is the use of probiotics for the purpose of controlling pathogenic microorganisms. Probiotics are mixtures of live bacteria and/or yeasts, which are administered to poultry to provide a source of beneficial microorganisms. These beneficial microorganisms (lactobacillus, Bacillus subtilis, enterococcus, Bifido bacterium, etc.) work by the principle of competitive exclusion. They out compete pathogenic microorganisms like Salmonella for nutrients in the intestinal tract. They prevent the colonization of the intestinal tract with unwanted pathogenic bacteria. They may produce a biofilm as a barrier to pathogenic microorganisms. Probiotics can prevent adhesions by bacteria to the epithelial cells of the gut wall, stimulate antibody production by gut wall stimulation, and synthesize protective substances like short chain fatty acids and antibacterial agents.

Probiotics are becoming common in commercial pullet replacement flocks to reduce the incidence of Salmonella spp., especially S. enteriditis. However, they also have shown efficacy in controlling other Salmonella spp. and clostridium, which may cause necrotic and ulcerative enteritis, and E. coli. There is also much new interest for their use in broilers due to the introduction of the Federal Government’s MEGAREG and HACCP programs. These programs mandate the reduction of Salmonella from the processing plants. E. coli and Camphylobacter will no doubt be added to future lists.

A typical, practical program using probiotics in egg layers is as follows:

1) spray chicks in the boxes at the hatchery and again at reception in the growing house;

2) add a feed grade probiotic from 1 day to 6 weeks;

3) avoid the use of antibiotics as they may decrease the activity of the probiotic;

4) a water soluble probiotic may be administered for the first 5-7 days;

5) add lactose to the ration at the rate of 5% for the first 2 weeks of life (whey or dried skim milk are examples) to promote the growth of beneficial microorganisms;

6) give a water soluble probiotic for 3 days (figure 10.0) or feed grade probiotic for
5-7 days following a stressful period such as beak trimming, moving, vaccinations, or after cessation of antibiotic therapy.

**Prebiotics**

Prebiotics (a mixture of organic acids) can also be used to simulate beneficial bacteria. A new product (Biomin®) has a mixture of pro and prebiotics as well as immunostimulants. It can be given by spray, drinking water, or in the feed at an early age. Probiotics are indigestible food ingredients, usually carbohydrates, which beneficially affect the host by selectively stimulating the growth and/or activity of one, or a limited number of bacteria in the colon. Examples of prebiotics are oligofructose, fructooligosaccharides, mannan, oligosaccharides and inulin.

Examples of beneficial bacteria that are simulated by them include bifidobacteria and lactobacilli.

**Synbiotics**

The use of synergistic combinations of pro and prebiotics.

![Figure 10.0. Adding probiotic to the water (top and bottom photos).](image)

*Figure 10.0. Adding probiotic to the water (top and bottom photos).*
11. Bacterial diseases

Infection of poultry with salmonella is called salmonellosis. Most pathogenic salmonella sp. have been eradicated from commercial poultry in major poultry producing countries. However, salmonella sp. that may cause disease in humans continue to plague the industry.

*Salmonellosis (pullorum, bacillary white diarrhea)*

**Species of bird**—All galliform (chickens, turkeys, quail, pheasants, partridges, peafowl, etc.) birds

**Action**—Acute in young birds and chronic in older flocks.

**Age of bird**—all.

**Etiology**—*Salmonella pullorum* is a non-motile, non-spore or capsule forming gram-negative rod-shaped bacteria.

**Mode of transmission**

1. Vertically spread through egg (transovarian) or on egg (by fecal contamination), or by feed and water contamination, contaminated incubators, exploding eggs, and bird to bird (horizontal) transmission.

2. It is species specific (occurs in birds only).

**Clinical signs**

1. Young birds have a pasted vent (figure 11.1).

2. White diarrhea (figure 11.0), huddled up, lameness, somnolence (sleepy), labored breathing and blindness can occur.

3. Mortality peaks at 7-10 days with up to 100% mortality and/or morbidity.

4. Adults are usually subclinical, or a drop in egg production, fertility or hatchability may occur. Occasionally see depression, anorexia (won’t eat), diarrhea and dehydration.
Figures 11.0 and 11.1. Undigested feed in litter (left) and pasted vent (right) are common with diarrhea

Postmortem lesions

1. Young birds have a red streaked liver, enlarged spleen, and gray nodules in peritoneum.

2. White cecal plug, misshapen yolk and omphalitis (swollen navel) can occur.

3. White areas on gizzard, liver, heart, lungs, omphalitis, swollen joints, kidneys swollen and urate filled may be seen.

4. Adults have misshapen ovaries, testicular abscesses or may be atrophic, swollen joints, nodular myocarditis (inflammation of the cardiac muscle) or pericarditis (inflammation of the sac around the heart).

5. There is no definitive diagnosis based on signs or lesions.

Diagnosis

1. Since there are no definitive lesions or signs, you must culture organism on Salmonella-Shigella agar, brilliant green, MacConkey's or Triple sugar iron agar.

2. Culture salmonella and use antibiotic sensitivity discs.

3. It is a reportable disease.

4. Antibody positive breeders are destroyed.

5. It simulates typhoid, paratyphoid, and colibacillosis.

Prevention

1. Testing of breeder flocks by serum agglutination or enzyme linked immunosorbent assay (ELISA).
2. Spraying and/or fumigation of eggs with formaldehyde. Place paraformaldehyde pellets in nests. Spray eggs in house with 2.5% hydrogen peroxide and 1.0% quaternary ammonia.

3. Pellet feed to kill bacteria, more difficult to heat inactivate bacteria in feed high in energy (5% fat), NF-180® (0.011%) (Neomycin 35 g/t). Heating process during pelleting kills bacteria.

4. National Poultry Improvement Plan (NPIP) monitors hatcheries for Salmonella in the U.S.

5. Irradiation of feed to kill bacteria.

Treatment (for young birds only)

1. NF-180® Furazolidone* (feed) (0.055%), Sulfonamide (0.5) in starter mash, 50-100 g/T. (Furox®),* Furacin in water,* Neomycin 70-140 g/ton, and sulfa drugs in water for young birds will treat the signs.

*Removed from approved drugs by FDA.

Special note

It has been eradicated from commercial U.S. flocks, but is still common in backyard flocks and commercial flocks in third world countries. A few isolated outbreaks of pullorum have recently occurred in broiler breeder and parent stocks in the U.S.

Fowl typhoid

Species of bird—Galliforms.

Action—Acute to chronic.

Age of bird--Young adults or mature.

Etiology—Salmonella gallinarum produces no spore or capsule, and is a rod-shaped gram negative bacterium.

Mode of transmission

1. It is spread through the egg. The incubation period is 4 to 5 days.

2. Contaminated feed, H₂O, and fomites (trucks, workers, and equipment, etc.) will transmit the organism. Fomites are inanimate objects which can carry infectious organisms and spread disease.
3. The organism is species specific (occurs in birds only).
4. Horizontally spread from bird to bird.

**Clinical signs**

1. Birds have elevated temperature 109-111°F (44-45°C), pale combs and wattles, shrunken combs, greenish-yellow diarrhea, depression, and/or anemia.

**Postmortem lesions**

1. Birds have bronze liver and grey foci in lungs and gizzard.

2. Congested breast muscle, enteritis often with ulceration, mottled swollen spleens and kidney, and thin watery blood are often seen.

3. Necrotic foci on the liver and heart, misshapend and ruptured ovaries, and peritonitis can occur.

**Diagnosis**

1. Laboratory (Necropsy, serology, microbial isolation and identification) monitoring is needed for a definitive diagnosis.

2. Culture salmonella and determine antibiotic sensitivity.

3. It simulates paratyphoid, pullorum, and cholera.

**Prevention**

1. Sanitation includes spraying of the eggs.

2. Control vectors

3. Test breeders for positive serum

4. Destroy positive breeders

5. Pellet feeds

6. Use chlorine in the water
Treatment

Neomycin or sulfaquinoxaline

Special Note

It has been eradicated from most U.S. commercial poultry flocks, but is still common in backyard flocks. There is only an occasional occurrence in commercial flocks in the U.S.

Paratyphoid

Species of bird--Turkeys and many higher forms of animals.

Action--Acute to chronic.

Age of bird--All, usually young.

Etiology--Salmonella typhimurium and other species; at least 33 Salmonella shown to infect birds. All are serologically related.

It is a facultative anaerobe, with motile flagella, and it produces endotoxins.

Mode of transmission

1. Fecal contamination of eggs is the most common mode of transmission. Transovarian transmission is rare.

2. Contaminated feed and water spreads the organism.

3. Biological vectors include mice, insects, dogs, cats and birds.

4. Fomites include trucks, litter, dust, etc.

5. It is not species-specific and therefore it is difficult to eradicate because of many natural, wild biological vectors, which cannot be eliminated.

Clinical signs

Young

1. Young birds tremble (cry), whereas birds older than 1 month are usually subclinical.

2. Young birds act cold, gasp, and are anorexic (do not eat).

3. Watery diarrhea, blindness, conjunctivitis, weakness, and lameness can occur.
Adults

Subclinical.

**Postmortem lesions**

1. Congested organs, caseous ceca, necrotic foci in heart and air sacs and pneumonia (figure 11.2) may be seen.

2. Unabsorbed yolks, pericarditis, arthritis, hemorrhagic enteritis in duodenum, and hemorrhagic streaks in liver can occur.

![Image](image_url)

**Figure 11.2. Pneumonia seen with lungs on the right is common with Salmonellosis**

**Diagnosis**

1. Serologic plate agglutination or indirect hemagglutination test can monitor antibodies in the bird.

2. Culture salmonella from lesions or feces on tetrathionate broth base with brilliant green or selenite. No definitive sign or lesion.

**Prevention**

1. Fumigation of hatching eggs with formaldehyde on the farm. Collect hatching eggs 5 times per day.

2. Clean eggs, test breeders for positive serum using ELISA.

3. Inject chicks *in ovo* or at day 1 with antibiotics (Gentamicin (0.2 mg), Naxal, or Spectinomycin).
4. Irradiation of meat where available.

5. Chlorinate water in the processing plant.

6. Commercial inactivated vaccine available for pullets against *S. enteriditis*.

7. Dipping poultry carcass in trisodium phosphate to prevent attachment of bacteria to the carcass.

8. Pro and prebiotics given by feed or water at any early age of life.

9. Commercial recombinant Salmonella vaccine available for use at day of age by spray cabinet for broilers.

10. Live *S. typhimurium* vaccine given to commercial egg pullets or birds in lay by water or spray or killed *S. enteriditis* vaccine for commercial by IM to egg pullets against *S. enteriditis*.

**Treatment**

1. NF-180® (50-200 g/T), Neomycin, gentamicin, and sulfas can be used.

2. Competitive exclusion of Salmonella from the intestinal tract with lactobacillus or other probiotic cultures.

3. Break out and pasteurize all eggs from *S. enteriditis*-infected flocks.

**Special note**

It has a public health significance and is a common zoonosis (disease transmitted from animals to humans). *S. enteritidis* infects eggs in northeastern United States and *S. typhimurium* may contaminate broiler meat any where in the world. It is the most common salmonella infection in poultry. It commonly infects dogs, cats, and livestock. One in 2000 eggs in the U.S. are contaminated with *S. enteritidis*. USDA requires testing of all layer flocks for *S. enteritidis*. U.S. strain of *S. enteritidis* is nonpathogenic in avians. European isolate has a plasmid for virulence and may produce significant morbidity and mortality in poultry. US government has legislated the MEGA REG regulations. HACCP is under these guidelines and its aim is to eliminate or reduce critical points in the field and processing plant, where pathogenic organisms can contaminate poultry meat and eggs.
**Arizonosis**

Species of bird—Turkeys and chickens (Broilers).

**Action**—Acute.

**Age of bird**—Young.

**Etiology**—*Salmonella arizona* is gram-negative and has flagella.

**Mode of transmission**

1. It spreads the same as other salmonella.
2. Vectors include birds and reptiles.
3. Transovarian spread can happen.

**Clinical signs**

1. Signs are the same as for other salmonella (figure 11.3).
2. Opaque eyes (blindness), tremors, convulsions, and twisted necks may be seen.

![Figure 11.3. Depression and soiled vent feathers](image)

**Postmortem lesions**

1. Lesions are the same as for *Salmonella pullorum*, which include bacteria septicemia; peritonitis, retained yolk sacs, emaciation, hepatitis (figures 11.4 and 11.5).

2. Congested (filled with blood) duodenum, mottled (white necrotic spots) liver, caseous plugs in ceca, and caseous air sacs can be seen.
**Diagnosis**

1. You must culture the organisms on brilliant green agar from lesions, egg yolk, and the like for a definitive diagnosis.

2. Agglutination or ELISA test using sera from breeders.

3. It simulates pullorum, *E. coli*, and typhoid.

**Prevention**

1. Prevention is the same as for salmonella. Bacterin for turkey breeders prevents egg transmission.

2. Egg and hatchery sanitation are important.

3. Test and slaughter serologically positive breeders.

4. Vector control helps control spread of the organism.

**Treatment**

1. Rofenaid® (SQ and Ormetoprin) in the water and furazolidone in the feed are effective treatments.

2. Gentamicin and spectinomycin by SQ injection at day of age.

**Special note**

Not as common as paratyphoid.
**Colibacillosis, coli granuloma, mushy chick disease, cellulitis**

It has nick names because it causes granulomas in adults and inflamed egg yolk and ascites in chicks.

**Species of bird**--All.

**Action**--Coli Granuloma is chronic in mature birds, and may be acute in chicks occurring as omphalitis.

**Age of bird**--All.

**Etiology**--*Escherichia coli* are gram-negative, non-acid-fast, non-spore-forming bacilli, and many strains have flagella.

**Mode of transmission**

1. E. coli may be a primary or secondary invader.

2. It is the most common bacterial pathogen in poultry and second most common of all pathogens of poultry.

3. Fecal contamination of eggs, transovarian transmission, and contaminated water and feed can occur commonly.

4. Aerosol spread may occur.

**Clinical signs**

1. Low performance in older birds or high mortality in younger birds, high embryonic mortality, respiratory distress, and enteritis (diarrhea) can be evident.

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**Figures 11.6 and 11.7. Cellulitis**
**Postmortem lesions**

1. Cauliflower-like nodules on viscera (granuloma) and omphalitis in chicks--discolored and misshapen yolk (mushy chick) are seen (figure 11.8). These lesions are characteristic.

2. Airsacculitis, salpingitis (inflammation of the oviduct), enteritis, synovitis, arthritis, pericarditis, peritonitis, panophthalmitis, or swollen head syndrome are common.

3. Cellulitis, an inflammation of the cellular or connective tissue, can be caused by *E. coli*. The soft tissue is heavily necrotic and may be gangrenous. It can result in extensive processing plant condemnation. It is sometimes called infectious process or IP (figures 11.6 and 11.7).

![Granulomas seen with E. coli](image)

**Figure 11.8. Granulomas seen with E. coli**

**Diagnosis**

1. Laboratory isolation of *E. coli* from lesions, yolk, and/or blood on MacConkey's or methylene blue agar (EMB). Colonies are pink on MacConkey's and dark with metallic sheen. It simulates Salmonella, Staph, tuberculosis, fowl cholera, Marek's disease, and aspergillosis.

**Prevention**

1. Use mycoplasma free stock to prevent interaction with E. coli.

2. Pellet feed to kill bacteria.

3. Bacterin for breeders or turkeys (serotype 02, 078) egg and hatchery sanitation to reduce organisms.
4. Chlorinate water (3 to 5 ppm) and use nipple drinkers to reduce transmission in water.

**Treatment**

1. NF-180®,* Chlortetracycline (CTC) (400 g/ton) (Aueromycin®), Oxytetracycline (OTC), Quinolones* (Danofloxin®, Enterofloxin® or Baytril®), Tylosin (Tylan®), Sulfadimethoxine and Ormetroprim, Neoterramycin®, LS50® (2 g/gal) can be used.

2. Gentamicin or Naxal® can be given by subcutaneous injection at 1 day of age.

3. Chlorox® in water at 2 to 4 oz/gal for 1 to 3 weeks or Quinolone in water for 3 to 5 days to treat clinical signs.

**Special note**

*E. coli* is a normal contaminant in the intestines and may complicate mycoplasma, IBV, LT, and/or NDV in the air sacs, heart, liver, and lungs causing airsacculitis and/or chronic respiratory disease (CRD). Several serotypes make vaccination difficult. *E. coli* is a very important cause of economic losses in poultry due to mortality, drops in weight gain, hatchability, and an important cause of Septicemia-toxemia (sept-tox), airsacculitis, and IP condemnations in processing plant. Sept-tox is the leading infectious cause of condemnation in the broiler processing plant in the U.S., airsacculitis is the second, and IP the third.

*Not approved for use in the United States.

**Turkey coryza (turkey rhinotracheitis)** — Swollen sinuses and tracheal inflammation.

**Species of bird**—Turkeys and broilers (rare).

**Action**—Acute to chronic.

**Age of bird**—More than 3 weeks.

**Etiology**—*Bordetella avium* is a gram-negative, nonfermentative, motile, and strictly aerobic bacillus.

**Mode of transmission**

1. It is highly contagious and is spread by aerosol. The incubation period is 7-10 days.

2. The organism can contaminate feces or water for up to 6 months.
Clinical signs

1. Stunting, huddling, decreased consumption of food and water, respiratory distress, sneezing, dry cough, and nasal discharge (tenacious brown) can occur.

2. Loss of voice and submaxillary edema may be seen. Turkeys may have almond-shaped eyes and mortality to 50%. Mortality is low in broilers.

Postmortem lesions

1. Mucous in trachea and turbinates, and the trachea is softened and distorted.

2. Pneumonia, edema of the interstitial tissues of head and neck, airsacculitis (figure 11.9), pericarditis, and bursal atrophy may be evident.

![Figure 11.9. Airsacculitis](image)

Diagnosis

1. Culture bacteria from trachea on MacConkey's agar. Colonies are clear and pinpoint and may develop a brownish raised center.

2. Microagglutination or ELISA test for determination of antibody in unvaccinated flocks.

3. It simulates mycoplasmosis, avian influenza (AI), ornithosis, Newcastle disease (ND), and swollen head syndrome.

4. Rhinotracheitis in turkeys is characteristic for this disease.

Prevention

1. Sanitation and biosecurity reduces the problem.
2. Bacterin prepared from whole bacterial cells can be given to turkeys-breeders at 6 weeks. Live vaccine is a temperature (cold) sensitive mutant (replicates only in upper respiratory tract) given by spray at 1-day and drinking water at 14 days.

**Treatment**

Aerosol spray of Oxytetracycline HCL (10 \( \mu \text{m} \) size particles).

**Special note**

It causes immunosuppression, which may lead to an outbreak of cholera in turkeys or poor vaccination response to NDV, *E. coli* or *Pasteurella multocida*. Coryza means head cold. In Europe, turkey rhinotracheitis is caused by a pneumovirus.

**Infectious coryza**

**Species of bird**--Chickens in cages. It is more common in tropical areas and where multi-age pullet farms are kept. Coryza means head cold.

**Action**--Acute to chronic.

**Age of bird**--15-30 weeks.

**Etiology**—*Haemophilus paragallinarum* is a gram-negative, polar-staining, non-motile bacterium, and appears as short rods or coccobacilli.

**Mode of transmission**

Fecal, aerosol.

**Clinical signs**

1. Strong odor (rotten eggs) given off by the organism.

2. Watery eyes, facial edema (figures 11.10 and 11.11), diarrhea, anorexia, and high cull rate (20%) may be evident.

3. Nasal discharge, swollen infraorbital sinus, labored breathing, drop in egg production and shell quality can occur.
Figure 11.10. and 11.11. Swollen sinuses

Postmortem lesions

1. Oral or tracheal lesions, catarrhal inflammation of nasal passages and sinuses may be seen (figure 11.12.

2. Congested lungs, facial swelling, swollen wattles, pneumonia, airsacculitis, and conjunctivitis may be evident.

Figure 11.12. Necrotic material in the infraorbital sinus characteristic for coryza.

Diagnosis

1. Respiratory signs, odor and isolation of organisms are important. The organism is a polar-staining, facultative anaerobic gram-negative rod. Brain heart infusion and NAD yields tiny dew-drop colonies.

2. Serologic tests include agar gel precipitin and hemagglutination-inhibition.

3. It simulates many respiratory problems, fowl pox (FP), vitamin A deficiency, fowl cholera (FC), and mycoplasma infections.

Prevention
1. Bacterin (containing serogroups A,B,C) at 10-12 and 16-18 weeks and one age per farm can help prevent the disease. Destroy all clinically ill birds to contain spread of the organism.

2. Live vaccine using homologous field strain can be given by water in tropical areas where bacterin is not effective.

**Treatment**

Sulfadimethoxine and Ormetropim, Gallimycin® (185 g/ton), and Sulfathiazole (30 g/5 gal) are effective.

**Special note**

It is found in Southern U.S. and Third World Countries (multi-age farms) and is common in backyard flocks. Several serotypes (A-C) make successful vaccination more difficult.

**Streptococcosis**

**Species of bird** --All

**Action**--Acute to chronic.

**Age of bird**--All.

**Etiology**--*Streptococcus sp.* include *zoopneumoniae, gallinarum, avian, faecalis, and durans*. It is a gram-positive, spherical, non-motile, non-spore-forming, facultative anaerobe, and occurs in short chains.

**Mode of transmission**

1. The organism can be spread by oral, aerosol, and transovarian transmission and with soiled eggs.

2. Secondary infections through wounds, contaminated hatchery, and wet litter are also important.

**Clinical signs**

1. Signs include depression, lethargy, lassitude, pale combs and wattles, tremors, drop in egg production, lameness, and reduced body weight.

**Postmortem lesions**
1. Lesions include splenomegaly, hepatomegaly (swollen liver), enlarged kidneys, peritonitis, omphalitis (swollen navel), tenosynovitis, arthritis, salpingitis, pericarditis (figure 11.13) and myocarditis.

![Pericarditis](image)

**Figure 11.13. Pericarditis**

**Diagnosis**

1. Laboratory isolation of organism from yolk, blood, and lesions on blood agar. Colonies are small and usually grayish in 24 hrs. They may be mucoid to rough.

2. It simulates *staphylococcus, Mycoplasma synoviae, Salmonella*, and *E. coli*.

3. No diagnostic sign or lesion exists.

**Prevention**

1. Prevention methods include egg and hatchery sanitation, remove sharp objects from the house, and use clean, dry litter.

**Treatment**

Penicillin, erythromycin, novobiocin, nitrofurans, tetracyclines.
Staphylococcosis

Species of bird--All.

Action--Acute to chronic.

Age of bird--All.

Etiology --Staphylococcus aureus is a gram-positive coccoid-shaped, ubiquitous organism and is found in clusters.

Mode of transmission

1. Transmission modes include transovarian, soiled eggs, secondary infection through wound, contaminated hatchery, and wet litter, which cause ammonia burns. Staph readily contaminates burnt skin.

Clinical signs

1. Signs include down on hocks, swollen feet (bumblefoot) or hocks, high mortality in baby chicks (omphalitis), and gangrenous dermatitis.

2. Morbidity and mortality are low with this disease.

Postmortem lesions

1. Lesions may include exudate on heart, liver, and yolk in chicks, puss in joints (figure 11.16), breast blister (figure 11.15), and foot pad in older birds (bumblefoot) (figure 11.14).

2. Osteomyelitis (focal yellow areas of caseous exudate in the bones) and septicemia (congestion of liver, spleen, kidney and lungs) may occur.

Figure 11.14. Bumble foot

Figure 11.15. Breast blister
Figure 11.16. Abscess in joint

**Diagnosis**

1. Laboratory isolation of coagulase positive organisms.
2. *Staph isolation* on blood agar produces white to orange smooth colonies.
3. Type bacteria using phages (bacterial virus).
4. It simulates *Mycoplasma synoviae, viral tenosynovitis, Salmonella, E. coli, Pasteurella, and Streptococcus*.
5. Swollen, localized abscesses are characteristic for this disease.

**Prevention**

1. Fumigate eggs and incubator, remove sharp objects from house, use clean dry litter, and use nipple-drinkers for a dryer house to reduce the bacteria.
2. Pro and prebiotics given by feed or water at any early age of life.

**Treatment**

1. NF-180®, Gallimycin® (185 g/ton), Albamix® (Novobiocin) (200-350 g/ton), Penicillin, Lincomycin, and Streptomycin are effective.

**Special note**

This organism leads to trimming and downgrading in the processing plant. It is a common contaminant of skin, and secondary infections occur in the joints following viral infection or stress. *Staph* is a leading cause of arthritis and synovitis in poultry. Arthritis and synovitis are a leading cause of condemnation (parts) in the U.S. broiler processing plant. *Staphylococcosis* is the second most common bacterial disease of chickens.
Ulcerative enteritis

Species of bird--Quail, broilers, chickens pullets, and turkeys

Action--Acute to chronic.

Age of bird--Young (6-14 weeks).

Enteritis--*Clostridium colinum* is spore-forming, gram-positive, aerobic, and non-motile.

Mode of transmission

1. Vectors, feces, soil, and litter containing the bacteria.

Clinical signs

1. High mortality in quail, watery diarrhea, ruffled feathers, dull, listless, increased thirst, emaciation, and atrophy of pectoral muscles can occur.

Postmortem lesions

1. Yellow irregular ulcers (figures 11.17 and 11.18) on small intestine and ceca, and hemorrhagic enteritis are seen.

2. Congested lungs, enlarged hemorrhagic necrotic spleen, light yellow mottling of liver, and crop filled with water may occur.

![Figures 11.17 and 11.18. Yellow ulcers in the intestine with ulcerative enteritis.](image)

Diagnosis

1. Gross lesions (ulcers on intestine and ceca) (figures 11.17 and 11.18) and bacterial isolation on tryptose - phosphate agar with yeast extract are a definitive diagnosis.

2. Fluorescent antibody test will detect bacteria in organs.

3. It simulates coccidiosis, necrotic enteritis, and histomoniasis.
**Prevention**

1. Improved sanitation, add 500 lbs of salt/house or sulfate compounds to the soil to kill spores, raise birds on wire, and/or feed Bacitracin at 50-100 g/ton to prevent the disease.

**Treatment**

1. NF-180 50-100g/T, streptomycin (60 g/ton), and chlortetracycline, vitamins and minerals in water, and/or Lincomycin 2 g/ton (Lincomix®) will reduce the signs.

2. Remove dead birds, and feed Bacitracin (200 g/ton) (Baciferm®).

**Special note**

It often accompanies coccidiosis in broilers. Clostridium infections are becoming more common, due to less use of anticoccidial drugs and growth promotants that can also inhibit these bacteria.

**Necrotic enteritis**

**Species of bird**--Broilers, commercial layer pullets.

**Action**--Chronic.

**Age of bird**--3-6 weeks.

**Etiology** --*Clostridium perfringens* produces type A and C alpha toxin and type C beta toxin. It is called creepers because chickens are ataxic (can’t move).

**Mode of transmission**

1. Soil, dust, litter, and feces spread the organism.

**Clinical signs**

1. Ataxia, intoxication, diarrhea, depression, ruffled feathers, and reluctance to move may be seen.

**Postmortem lesions**

1. Dehydration (darkened skin), emaciation (no breast muscle), congested liver, cooked (ruffled up) intestinal mucosa--primarily jejunum and ileum can be seen. Intestines often are distended and filled with gas (figures 11.19 and 11.20). There is water in the crop.

**Diagnosis**

1. Diagnosis is based on the gross lesions (ruffled intestinal mucosa), clinical signs and bacterial isolation on blood agar plate. Colonies are surrounded by an inner zone of complete hemolysis and an outer zone of discoloration and incomplete hemolysis.

2. It simulates coccidiosis and ulcerative enteritis.

**Prevention**

1. Bacitracin 50 g/ton feed continuously, improved sanitation, Coban® in feed, Lincomycin in feed or water and rear birds on wire to prevent the disease. Salt added to the soil to kill the organism.

2. Use of pro and prebiotics in feed or water at an early age.

**Treatment**

1. Bacitracin (200 g/T) in the feed and vitamins and minerals in the water to reduce the disease.

2. Lincomycin, oxytetracycline, penicillin, and tylosin can also be used to treat the disease.

**Special note**

It may cause malabsorption syndrome leading to vitamin or mineral deficiency.

*Botulism (limberneck or western duck sickness)*

It causes paralysis of the neck and frequently occurs in duck populations of the Mid Western U.S.
Species of bird--All (common in broilers, pullets, wild water fowl)

Action--Peracute.

Age of bird--Any age.

Etiology--Clostridium botulinum is gram-positive and spore-forming rod, anaerobic, and a soil-borne pathogen.

Mode of transmission

1. The organism produces a type A neurotoxin. There are 8 antigenically distinct toxigenic groupings of this type A toxin.

2. It is spread by cannibalism and eating insects. There is a 1 to 2 day incubation period.

Clinical signs

1. Signs include loose feathers, sleepiness, neck, wing, eyelid and/or leg paralysis, diarrhea, soiled vent, and high mortality (figure 11.21). Dead birds decompose rapidly.

Figure 11.21. Neck paralysis is characteristic for botulism.

Postmortem lesions

1. Few to no gross lesions are seen.

2. Maggots can be seen in the upper digestive tract.

Diagnosis

1. Diagnosis includes rapid central nervous system signs (paralysis) with no gross lesions.
2 Mouse bioassay includes inoculating mice with serum to determine if toxin is present. If the toxin is present, the mice will show clinical signs.

3. Fluorescent antibody detection of organism.

4. It simulates Marek's disease (MD) and drug and chemical toxicities.

**Prevention**

1. Pick up and destroy dead birds at regular intervals (twice a day), control flies, remove litter, and disinfect house.

**Treatment**

There is none for commercial poultry. Bacitracin, streptomycin, and vitamin and mineral therapy are somewhat effective for expensive birds.

**Special note**

In commercial poultry it is a filth disease caused by poor management and sanitation. Outbreaks in wild ducks in western U.S. can cause mortality in tens of thousands of bird. Large numbers of ducks produce high volumes of organic fecal material. This material produces an anaerobic environment, which favors growth of clostridium that is washed into the area from eroded soil. Flies and maggots feeding on carcasses spread the toxin.

**Gangrenous (necrotic) dermatitis**

**Species of bird**--Broilers and turkeys.

**Action**--Chronic debilitating.

**Age of bird**--3-7-week-old broilers.

**Etiology**--*Clostridium septicum*, *E. coli* and *S. aureus*.

**Mode of transmission**

1. Transmission is by contact with infected wet, caked litter.

2. The disease often occurs in immunosuppressed birds.
Clinical signs

1. Signs include loss of feathers, low mortality, dead birds decompose quickly, pale combs and wattles.

2. Depression, incoordination, inappetence, leg weakness, and ataxia (can’t move) can also be seen.

Postmortem lesions

1. Lesions are reddish, greenish, necrotic skin usually devoid of feathers, overlying wings, breast, abdomen, or legs (figures 11.22 and 11.23).

2. Gas may be given off upon palpation of skin, and blood-tinged edema (watery fluid).

3. It may affect muscle under skin causing discoloration.

4. Anemia (paleness due to lack of red blood cells), retained yolk sacs, discrete white foci in the liver may also be seen.

Figures 11.22 and 11.23. Necrotic discoloration of skin with gangrenous dermatitis.

Diagnosis

1. Diagnosis is by post-mortem lesions, which include congestion, hemorrhage and necrosis of skin with intra lesion bacteria under the microscope.

2. Bacteria can be isolated anaerobically on 2.5% blood agar.

3. It simulates erysipelas.
Prevention

1. Clean out house and add new litter to prevent the disease.

2. Medication in starter feed Flavomycin, (Bambermycin®), Virginamycin (Stafac®), Bacitracin, 3-Nitro®, NF-180®, CTC can reduce bacteria.

3. Proper vaccination against IBDV, CAV, and MDV, prevent mycotoxin formation in the feed, and eliminate ALV in the breeders to prevent immunosuppression.

Treatment

1. Erythromycin, penicillin, furoxone® and Albamix® in the feed to treat signs.

2. NF-180®, chlortetracycline, oxytetracycline, and copper sulfate can be added to the water to reduce morbidity.

Special note

It causes increased condemnation. Immune depression increases incidence and severity of dermatitis. Immunodepression may be due to a prior infection with IBDV, MDV, ALV, or CAV infection(s), or mycotoxin ingestion.

Ornithosis (turkeys) parrot fever (man) Psittacosis (Pet Birds)

It causes fever in man and Psittacine (parrots) birds.

Species of bird--All.

Action--Chronic.

Age of bird --All.

Etiology--Chlamydia psittaci is an obligate intracellular bacterium. Elementary body (EB) is a small, dense, spherical body, which is the infectious form. Reticulate body (RB) is intracellular and metabolically active. It is larger than EB and multiplies by binary fission. The organism has a complex development cycle, which results in a change from the EB to the RB in the host.

Mode of transmission

1. It is spread by feces and airborne.

2. Processing plant exposure may occur for humans.
Clinical signs

1. Signs include fever and sleepy depending on the strain. Toxigenic strain causes high morbidity and mortality, whereas the non-toxic produces a mild respiratory or gastrointestinal disease.

2. There may be a drop in egg production and green colored droppings.

Postmortem lesions

1. There can be a fibrinous pericarditis, airsacculitis, and hepatitis (figure 11.24).

2. Large congested spleen, pneumonia, congested kidneys, liver, swollen necrotic vent, and brain hemorrhage (parrots) can be seen.

![Figure 11.24. fibrenous exudate over liver](image)

Diagnosis

1. Eliminate CRD due to mycoplasma, ND or infectious bronchitis (IB) and *E. coli*.

2. Laboratory isolation of organism in chicken embryos or mice. Serology (complement fixation test), or latex agglutination test can be done. Recovered birds shed organism up to 42 days.

3. Giemsa stain from tissue impression smears reveals red EB's or bluish-green RBs.

4. Antigen capture ELISA will stain chlamydia smear from swabs of vent or cleft (Abbot or Kodak, Labs.).

5. It simulates CRD, colibacillosis, influenza, and cholera.

6. Fibrinous pericarditis, airsacculitis, and congested spleen in turkeys is diagnostic.

Prevention

1. Destroy dead birds, prevent wild and free-flying bird exposure, and quarantine infected flocks.
Treatment

1. CTC 400 g/ton (3 weeks) and Doxycycline (Vibramycin®)* are effective drugs.

Special note

Primarily turkeys on range and aviary birds are affected. It has public health (can cause disease in humans) importance and is a reportable disease. It can produce high fever, flu-like illness and mortality in humans; causes abortion in cattle. Cats and pigeons spread organisms. Veterinarians, pet bird owners and processing plant employees are a high-risk population.

*Not approved in the United States for use in commercial poultry.

Avian tuberculosis (TB)

Species of bird--All.

Action--Chronic.

Age of bird--Older (over 2 years of age), rare in commercial flocks

Etiology --Mycobacterium avian is acid-fast, bacillary in character, and club-like. Curved and crooked forms of the bacterium can also occur.

Mode of transmission

It is spread by older carrier birds, or contaminated fecal material, soil, litter, aerosol, and cannibalism.

Clinical signs

1. Pale, regressed combs, wattles, and depression can be seen.

2. Emaciation, anemia, unthrifty, icterus (yellow skin), and lameness may occur.

Postmortem lesions

Internal yellow or gray-white pearl-shaped nodules in lungs, liver, spleen, intestines, and bone marrow may be seen (figure 11.25).
Diagnosis

1. Gross lesions (nodules in lungs and bone) with the TB skin test of wattles using antigen from state diagnostic laboratory are diagnostic.

2. Acid-fast stain of Tubercle bacilli is also important.

3. Histopathologic observations of tubercule with acid-fast bacilli can be done.

4. Serology tests include ELISA and rapid agglutination test.

5. It simulates lymphoid leukosis and coligranuloma.

Prevention

1. Separate by age and remove skin-test-positive birds.

2. Isolate birds from hogs and quarantine affected flocks.

Treatment

1. Depopulation of commercial flocks is important.

2. Exotic birds can be given isoniazid (30 mg/Kg), ethambutol (30 mg/Kg), and rifampicillin (45 mg/Kg) for 18 months.

Special note

It has public health significance. Some cases of *M. avium* induced disease can occur in man. Humans with AIDS and other immune suppression diseases are very susceptible to this organism. It can spread to swine and cattle causing a positive skin test. Backyard flocks, zoo, and aviary birds over 2 years of age are commonly infected.
**Erysipelas (red skin).** It causes red lesions of the skin.

**Species of bird**—Turkeys, ducks, geese, chickens, quail, peafowl.

**Action**—Acute to chronic.

**Age of bird**—Growing (4-7 months).

**Etiology**—*Erysipelothrix rhusiopathiae* is a gram-positive bacillus. It does not form spores, is non-acid-fast, and non-motile.

**Mode of transmission**

1. It can be transmitted by a break in skin or mucous membranes, or fighting in males.

2. It is a soil-born organism, and can be spread by cannibalism or by a biting fly.

3. Contaminated fish meal is also a source of infection.

**Clinical signs**

1. Signs include swollen snoods (turkeys) and hocks (figure 11.26). The incubation period is 2 to 3 days.

2. Diarrhea, emaciation, weakness, anemia, skin hemorrhage and necrosis can be seen.

3. Fever, cyanotic toes and head, drop in egg production and/or fertility, and embryonic mortality can occur.

**Postmortem lesions**

1. Enlarged, friable, purple-black spleen, breast muscle hemorrhage, oral mucous, hemorrhage in muscles, spleen, lungs, fat and small intestine, and endocarditis may be seen.

2. Fibrinopurulent exudate in the joints, thickening of walls of proventriculus, ulceration of gizzard, and yellow nodules in the ceca can occur.
**Swollen snood**

**Figure 11.26 Swollen snood with *Erysipelas***

**Diagnosis**

1. Laboratory isolation from lesions is important and can show smooth colonies colorless to a bluish gray, of pin-point size with smooth edges.

2. Hemorrhagic, swollen red spleen and snood lesions are diagnostic.

3. It simulates cholera, *Salmonella*, gangrenous dermatitis, aspergillosis, and *E. coli*.

**Prevention**

1. Vaccinate birds twice with a bacterin (whole cell culture in aluminum hydroxide at 4-week intervals), once at 10-12 weeks and again at 14-16 weeks.

2. Debeak and desnood poults at day one to prevent fighting.

3. Hogs should not be reared near poultry and rotate the turkey range to reduce bacteria.

**Treatment**

1. Gallimycin and penicillin can reduce signs.

2. Disinfect premises with aerosol phenols or iodine.

**Special note**

It occurs more in males. It is necessary to wear gloves when performing a necropsy exam with turkeys, since it can infect humans, and causes diamond skin disease in hogs. It will also cause dermatitis in sheep, fish, mice and chipmunks.

**Campylobacteriosis**

**Species of bird**--Chickens, turkeys, ducks, pigeons, game birds, quail, puffins, gulls, and geese.

**Action**--Acute to chronic.
Etiology--*Campylobacter jejuni*, *C. coli* and *C. lardis* are microaerophile, gram-negative, spiral and uniflagellate bacteria.

Mode of transmission

Contaminated feed and water, litter, cannibalism, houseflies, cockroaches, wild free-flying birds, mice, and exploding eggs in hatchery can spread the organism.

Clinical signs

There is a 24-72 hour incubation period. Depression, soiled vent, and diarrhea can be seen.

Postmortem lesions

1. Distention (enlargement) of the intestinal tract, accumulation of mucus, watery fluid and hemorrhages in intestines can be seen.

2. Focal hepatic necrosis may also be present.

Diagnosis

1. Isolation of organism from feces, intestinal tract, bile, blood or liver is needed. Incubate cultures for 48-72 hr at 43° in a microaerobic atmosphere. Colonies are flat, translucent and gray with a tendency to coalesce or be raised, opaque and brown gray with discrete margins. Selective media include brucella agar, blood agar with various antibiotics including bacitracin, novobiocin, and colistin.

2. It simulates coccidiosis.

Prevention

Biosecurity and restrict wild birds and vermin to prevent the disease.

Treatment

Kanamycin*, gentamicin, furazolidone*, and doxycyline* are effective.

Special note

Humans are very susceptible to the organism. Food-borne contamination of uncooked poultry is a significant problem. Up to 2 million cases of enteritis a year may be due to this bacteria.

*Not approved for use in United States.
**Fowl cholera**

**Species of bird**--Turkeys, chickens, ducks, geese, birds of prey.

**Action**--Peracute to chronic.

**Age of bird**--Young adults.

**Etiology**--*Pasteurella multocida* is gram-negative, non-spore-forming rod, bipolar bacteria with capsule, and some contain pili. Variation in pathogenicity occurs between isolates.

**Mode of transmission**

1. Spread is by rodents, cats, dogs, insects, horses, cattle, sheep, and pigs.
2. Respiratory spread and cannibalism are important.
3. Fomites include equipment and feedbags.

**Clinical signs**

1. Peracute death without signs can occur.
2. Acute disease causes high fever, thirst, cyanotic (figure 11.28), anorexia, and ruffled feathers.
3. Chronic disease causes torticollis (retraction of the head and neck backwards), emaciation, severe mortality and enlargement of wattles, combs, legs, footpads, and wing joints.
4. Swollen sinuses and hocks (figure 11.27) dehydration, respiratory distress, swollen joints, drop in egg production, fertility, and hatchability can also occur.
**Postmortem lesions**

1. Peracute disease produces no lesions.

2. Hemorrhage on heart and fat (figure 11.28), conjunctivitis, subepicardial and subserosa hemorrhage, congested breast, and septicemia can occur with chronic form.

3. Free yolk in abdominal cavity, liver-yellow brown streaks, "egg yolk" peritonitis, consolidation of lungs (figure 11.29), mucous in the mouth and nasal passages, and caseous exudate in palatine cleft (upper part of the mouth) may also be seen.

**Diagnosis**

1. Laboratory isolation of organism with or without oxygen.

2. Culture Pasteurella on blood agar or meat infusion media or liver impression smear stained with Wright's stain. The stain will yield bipolar rods, which are diagnostic.

3. Colonies are 1 mm in diameter and smooth, but often with concentric rings. Colonies have a musty odor.

4. It simulates typhoid and *E. coli*.

5. Peracute septicemic disease in pullets and large swollen necrotic liver are a presumptive diagnosis.

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**Figure 11.28. Enlarged necrotic liver**  **Figure 11.29. Nodules in lung**

**Prevention**

Vaccinate with bacterin at 10 and 16 weeks, or use live CU (Clemson University) strain or temperature sensitive mutant vaccine of mild or intermediate virulence by wing web in pullets, or one live and one killed. Use live vaccine by water in turkeys. Destroy infected carriers. Sixteen serotypes are known. Some cross protection occurs between serotypes. Serotypes 1, 3, 4 are most common and are found in most commercial vaccines.
Treatment

OTC (Terramycin®) (100-200 g/ton), Erythromycin (Gallimycin®), tilmicosin (250ml of drug/800 liters of drinking water, Sulfadinoxaline and Ormetropin (Rofenaid®) (0.125%+0.0075%), and Sulfamethazine (0.49%) are effective.

Special note

It is a stress disease occurring with seasonal change and at point of lay. Disease occurs in swine and cattle; cats spread the disease. Several serotypes make vaccination difficult.

Avian mycoplasmosis airsacculitis

Species of bird--All.

Action--Chronic.

Age of bird--All.

Etiology--M.G.=M. gallisepticum, MB=M. gallinarum, MS=M. synoviae, MI=M. iowae, and MM=M. meleagridis. Coccoid to coccibacilliform bodies appear singly, in pairs or in clusters. Slender rods, filaments and ring forms have been reported in Giemsa-stained films. Mycoplasma have no cell wall, very thin, and are weakly gram-negative. They grow on artificial media and are inhibited by certain antibiotics.

Mode of transmission

It spreads vertically through egg or laterally by aerosol.

Clinical signs

1. It has an incubation period of 6 to 21 days. Signs include foaming of the eye, conjunctivitis, labored breathing, nasal discharge, sneeze and cough, rales (a rattling sound produce by air moving through a trachea that is congested with mucous, swollen sinus, anorexia, weight loss, increase in condemnation, lowered egg production, and poor shell quality.

Postmortem lesions
1. Lesions include mucous in turbinates, sinus (figure 11.30), trachea, bronchi, cloudy air sac, and salpingitis.

![Swollen sinus](image)

**Figure 11.30. Swollen sinus**

**Diagnosis**

1. Serologic tests include plate agglutination or hemagglutination-inhibition test, and/or ELISA. All breeders and layers are tested twice, once as pullets and once during egg production for antibodies against Mycoplasma according to the NPIP. Breeders tested within four weeks of receiving many inactivated vaccines may falsely test positive for antibody due to a cross-reaction with a substance in the vaccine. They should be retested several weeks later.

2. Isolate organism from air sacs or lesions in Frey's medium, supplemented with 10-15% swine or horse serum and thallus acetate or penicillin to repress other bacteria. Incubate cultures for 5-7 days at 37°C. Colonies are small, circular and somewhat flat with a more dense central elevation. Giesma stain of colonies reveals small coccoid organisms. *Mycoplasma sp.* can be differentiated from one another by various biochemical tests.

3. It simulates many respiratory diseases, such as *E. coli*, cholera, ND, IB, infectious laryngotracheitis (ILT), AI, and coryza.

4. PCR test kit for mycoplasma detection from tracheal or fecal swabs.

**Prevention**

1. Egg-dipping with Tylan® using a warm egg and cool solution (4°C) for 20 minutes to kill organism.

2. Inject chicks with antibiotic or use medicated starter for first 10 days.

3. Depopulate infected broiler breeders, use clean (mycoplasma-free) stock only, or MG+MS bacterin for egg laying breeders. All pullet and adult breeder flocks are tested for antibodies against MG and MS according to the NPIP plan. Use F strain, 6/85, or temperature-sensitive mutant (TS11) live MG vaccines for commercial egg layers only.
The F and TS11 vaccines may be shed vertically or horizontally resulting in respiratory disease. The 6/85 vaccine does not produce an antibody response and can be given by fine spray. The F and TS 11 strains can be given in the drinking water.

4. Egg-heating for 14 hours at 46°C will inactivate mycoplasma, but hatchability will be reduced from 1 to 2%.

5. One age flock per farm, and all in, all out-rearing of birds can prevent disease.

Treatment

1. Tylosin (800-1,000 g/ton), LS 50®, (2 gm/gal), Quinolones (Danoflaxin®, Endoflaxin®, Baytril®) by water route to reduce morbidity.

Special note

It may be complicated by IBV+NDV+ILT and/or _E. coli_ leading to chronic respiratory disease (CRD). Airsacculitis is the second leading cause of condemnation in the U.S. processing plant. 95% of breeder flocks in the US are free of mycoplasma. Multi-aged commercial layer flocks have a high incidence of mycoplasma positive flocks.

**Mycoplasma complicated chronic respiratory disease (CRD)**

Species of bird—All.

Action—Chronic.

Age of bird—All.

Etiology—MG, MS, MB, MM, MI, or IBV, and/or _E. coli_ and/or NDV and/or ILT.

Mode of transmission

1. It is spread through egg and contact with infected litter, feed or water.

2. _E. coli_ is a secondary invader of mycoplasma-infected birds.

3. Aerosol spread occurs.

4. Severe ND, IBV or ILT reactions may occur after vaccination, especially if given by spray to chicks previously infected with mycoplasma or _E. coli_.

Clinical signs

1. Signs include cough, tail bobbing when breathing, emaciation, rales, sneezes, open mouth breathing, poor growth, decreased feed consumption, and lowered egg production and shell quality.
Postmortem lesions

2. Yellow fibrin on heart, liver (figure 11.31) and viscera organs, caseous air sacs, mucous in trachea, and green livers can be seen.

![Figure 11.31. Fibrin over heart and liver](image1)

![Figure 11.32. airsacculitis](image2)

Diagnosis

1. Laboratory isolation and identification of *E. coli* or mycoplasma from lesions. Detection of mycoplasma colonies using fluorescent antibody test, recombinant probe and hybridization, or antigen capture ELISA.

2. Serology includes ELISA, plate agglutination and hemagglutination-inhibition, (HI) testing of sera for antibodies against mycoplasma.

3. Cheesy exudate in air sacs is a presumptive diagnosis.

Prevention

1. Hatch stock free of mycoplasma infection, pellet feed to kill *E. coli*. Vaccination for breeders against *E. coli*, MG, MS, NDV, IBV, ILT, and infectious bursal disease (IBD) to prevent the disease.

2. Vaccinate progeny against ND, IB, ILT and IBD, and hatch and place mycoplasma infected stock separate from mycoplasma negative stock to reduce organism spread.

3. Treat all mycoplasma positive flocks with antibiotics to reduce shed into eggs.

4. Medicate all mycoplasma-positive broilers for first 7 to 10 days in the feed or water.
**Treatment**

1. Effective drugs include tylosin, LS 50®, and Quinolones.

**Special note**

*E. coli* is a normal contaminant of intestines, but is a primary pathogen in respiratory tract. Birds compromised by stress of immunosuppression due to an early infection with IBD commonly develop CRD. It is a major cause of condemnation in the processing plant.

**Mycoplasma infectious synovitis**

**Species of bird**—Chickens and turkeys.

**Action**—Acute to chronic.

**Age of bird**—All.

**Etiology**—*M. synoviae*.

**Mode of transmission**

1. Spread through egg and laterally by aerosol.

**Clinical signs**

1. Signs include breast blisters, pale or shrunken combs, lameness, ruffled feathers, and greenish droppings with large amounts of urates.

2. Birds sit on hocks, are dehydrated, listless, have hot swollen hocks, wing joints, and foot pads, and/or lowered egg production and shell quality.

**Postmortem lesions**

1. Lesions include viscous creamy to gray exudate in yolk, synovial membranes, tendon sheaths, joints, and keel bone over breast, and hepatosplenomegaly.

2. Honey colored fluid on breasts and joints (figure 11.32), and swollen, mottled and pale kidneys may be present.
**Diagnosis**

1. ELISA test for antibodies is important.

2. Isolate organism from medium on agar. Identification of the organism using Giemsa stain of culture or antigen capture ELISA with monoclonal antibody, nucleic acid probe, or fluorescent antibody testing.

3. It simulates bacterial or viral arthritis.

4. Honey (yellow) colored fluid from swollen joints is a presumptive diagnosis.

**Prevention**

1. Prevention includes serologic testing of breeder flocks twice during their life, vaccination of pullets with killed bacterin or temperature sensitive mutant live vaccine by water, one age per farm, and all in, all out farms.

**Treatment**

1. Effective drugs include tylosin, tilmicosin, LS 50® at 2 g/gal, and Quinolones.

**Special note**

It may cause disease in swine.

*Mycoplasma turkey venereal disease.* It spreads through artificial insemination of turkeys with contaminated semen.

**Species of bird**—Turkey.
Action--Chronic.

Age of bird--Breeders become infected, often subclinically, and pass organism to poults, which show lameness and feather deformities.

Etiology--MM, MI.

Mode of transmission

Spread by mating via semen, or transovarian to progeny, and laterally among poults by aerosol.

Clinical signs

1. Signs include high embryonic mortality, stunted growth, airsacculitis, and lameness (figure 11.33) (bowing and twisting of legs and shortening of the tarseometatarsal bone). Hock joint swelling, deformation of cervical vertebrae, and feather deformities may occur.

Postmortem lesions

1. Lesions include skeletal deformities especially of the neck and legs and airsacculitis.

![Figure 11.33. Enlarged foot pad seen with mycoplasma](image)

Diagnosis

1. Diagnosis is by serology (ELISA or HI test) and/or isolation of organism from lesions.

2. It simulates other mycoplasma infections and nutritional deficiencies.

Prevention

1. Prevention is by dipping eggs in Tylan, or heat eggs to kill mycoplasma, depopulate infected breeders, hatch only MM or MI clean flocks, and/or inject poults or eggs with antibiotics, and/or treat in the feed or water.

Treatment

1. Medications include Tylosin, Tilmicosin, or LS-50® in water, Quinolones, gentamycin, nalidixic acid*, tiamulin*, or spiramycin*.
*Not approved in United States.

*Mycoplasma infectious sinusitis*

Species of bird--All.

Action--Chronic.

Age of bird--All.

Etiology--MG, MM, MS.

Mode of transmission

Spread through egg or contact with infected birds by aerosol.

Clinical signs

1. Signs include nasal discharge, emaciation, increased condemnation, sneeze and cough, swollen infraorbital sinus (above the eye), off feed, foaming of eye, and/or airsacculitis.

Postmortem lesions

Caseous sinus and airsacculitis can be seen.

Diagnosis

1. Serologic tests include plate agglutination, HI, or ELISA test.

2. Isolate organism from sinus and identify it with antigen capture ELISA test and monoclonal antibody; fluorescent antibody or recombinant probe tests, and nucleic acid hybridization assays.

3. It simulates many diseases including coryza, NDV, E. coli, and IBV.

Prevention

1. Depopulate infected stock, hatch clean stock only, and vaccination of pullets for MG with live or killed vaccine to prevent the disease.

Treatment

Drugs, which can be used, include tylosin, LS 50® at (2 g/gal), Quinolones, and spiramycin*.

*Not approved for us in the U.S.

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12. Viral diseases

*Marek's disease (range paralysis)*

It was named for a Hungarian pathologist (Marek) and because it caused paralysis in chickens, which used to be reared on the range (range paralysis).

*Species of bird*—Chickens (all breeds), pheasants, and quail occasionally. It occurs worldwide in commercial flocks.

*Action*—Chronic. It takes 4-6 weeks for tumors to form.

*Age of bird*—It occurs usually under 16 weeks, but birds can die of Marek's disease (MD) near the onset of egg production.

Etiology—It is caused by a cell-associated Herpes virus containing double-stranded DNA. It has hexagonal naked particles or nucleocapsids of 85 or 100 nM. Enveloped particles of 150-400 nM are occasionally seen in the feather follicle epithelium. There are 3 serotypes. Serotype 1 viruses can be oncogenic (causes tumors). Viruses are subcellular microbes that cannot be isolated on artificial media and are inhibited with only a few very expensive antibiotics.

*Mode of transmission*

1. It is spread by contaminated litter, dust, down, or air-borne (bird to bird).

2. Feather (dust or dander) epithelium contains virus.

3. There is an incubation period of 2 weeks for virus shed and 3-6 weeks for clinical signs.

*Clinical signs*

1. It causes increased susceptibility (immunosuppression) to other diseases.

2. Signs include weak, pale, off feed, diarrhea, poor performance, culls, and blindness (figure 12.0).

3. There is paralysis or perisis (partial paralysis). There can be unilateral or bilateral paralysis of wings and legs, mortality, tumors, and central nervous system signs (tremors). One leg stretches forward and the other backwards due to leg paralysis.
Postmortem lesions

1. The peripheral nerves are often enlarged (vagus, sacral, sciatic, and brachial) with a loss of striations (figure 12.3). They can also have gray or yellow discoloration and be edematous (fluid filled).

2. The bursae are sometimes enlarged with tumors, but most often are atrophic.

3. There are enlarged organs (gonads, spleen, heart, lungs, liver, kidneys, proventriculus, intestines, etc.) with focal to nodular white or gray tumors (figure 12.2).

4. The skin has nodular, palpable tumors (figure 12.1).

5. The skeletal muscles have tumors. The eye (iris and pupil) may have diffuse depigmentation, diffuse bluish fading or diffuse gray opacity of the iris (figure 12.0). The pupil may be irregular and tiny.

6. The bone is never involved and the blood and fat sometimes have tumors.
**Diagnosis**

1. The diagnosis is by observation of gross tumors in immature birds.
2. Ocular or skin leukosis and nerve involvement are diagnostic for MD.
3. Histopathologically, small to medium lymphocytes comprise tumors.
4. Lymphoid cells occur in peripheral nerves.
5. It simulates riboflavin deficiency, lymphoid leukosis, reticuloendotheliosis, and colibaccillosis.

**Prevention**

1. Vaccinate (HVT--serotype 3 and SB1--serotype 2) chicks at day-old SQ in hatchery. Most injections are done at 18 days of embryonation (*in ovo*).
2. HVT and SB1 are used for broiler breeders and white leghorns at full dosage.
3. HVT and SB1 at 1/3-1/4 dosage for broilers.
4. SBI may cause immunosuppression and increase leukosis in some Leghorn strains.
5. HVT = Herpes virus of turkeys. SB1 = S (susceptible strain) and B1 (pen B1). New vaccine Respns is a serotype 1 vaccine. HVT + SB1 or HVT + Respns vaccine can be used.
6. Attenuated Rispens strain (Serotype 1) may be used in combination with HVT.

**Treatment**

None.

**Special note**

It is immunosuppressive. Tumors are a leading cause of condemnation in broilers and MD is the leading cause of tumors in US broilers. There has been an increase in the incidence of MD is broilers in the US since the change to dry cups and nipple drinkers. These systems make the house drier and dustier, which provides an environment where the virus is more easily spread.
Lymphoid leukosis (big liver disease, lymphomatosis, visceral lymphoma)

It is called big liver because the liver is usually enlarged with nodular tumors.

**Species of bird**--Chickens (some exotic birds).

**Action**--Chronic, it takes 14-30 weeks for disease to occur.

**Age of bird**--It occurs usually in 16-week-old birds or older except for osteopetrosis. Osteopetrosis occurs in broiler-age birds.

**Etiology**--RNA virus in the family of retroviruses. Ten viral subgroups (A-J) are known. Avian leukosis viruses (ALVs) are common around the world, because they are transmitted through the eggs of common commercial breeders. J virus is a recombinant virus.

**Mode of transmission**

1. It spreads through the egg (transovarian). It is a ubiquitous organism in commercial poultry flocks.
2. There is some lateral transmission with congenitally infected chickens.

**Clinical signs**

1. Signs include paleness, emaciation, weakness, and inappetence.
2. Tumors are external. The abdomen is enlarged and feathers are sometimes spotted with urates and bile.
3. The feed/gain ratio is decreased.
4. Increased culls and decreased egg production occurs.

**Postmortem lesions**

1. Peripheral nerves are never involved.
2. Bursae are always enlarged and may contain nodular tumors.
3. Visceral tumors are soft, smooth, and glistening. Tumors may be nodular, miliary or diffuse. Tumors are common in gonads, lungs, liver, spleen, heart, kidney, mesentery, and bone marrow.
4. The skin is sometimes affected, and skeletal muscles often contain tumors.
5. Eyes are never affected.
7. The blood often contains tumors (especially with J virus).
8. Fat tumors are common (Liposarcoma).

**Diagnosis**

1. Tumors in adult birds are characteristic for this disease (figures 12.4 and 12.5).
2. Liposarcoma or myeloid tumor (J virus) is also characteristic.
3. The CoFal (complement fixation for avian leukosis virus) or ELISA tests are confirmatory. Histopathology reveals large lymphoblasts in tumors. Lymphoblasts have an ovoid nucleus and a finer, more delicate chromatin network. PCR test used to select out J virus positive broiler breeders and grand parent stocks.
4. CoFal and ELISA test use specific serum to determine the presence of the organism in blood, tissues, serum, vaginal swab or egg albumin. PCR test used for J virus.
5. It simulates *E. coli*, Aspergillus, MD, tuberculosis, erythroblastosis, and myelobastosis.
6. The tumors are grossly distinct from those caused by other ALVs. However, the definitive diagnosis is based on virological isolation or histologic observation of the tumor cells. The myeloid cells can be differentiated from other tumor cell types by a trained histopathologist.

![Figure 12.4. Liver tumor](image1.png)  
![Figure 12.5. Bone tumor under sternum](image2.png)

**Prevention**
Select birds that are leukosis virus negative using serologic methods to prevent spread of the disease.

**Treatment**

None.

**Special note**

It is immunosuppressive and a major cause of condemnation in adult broiler breeders and layers. Tumors are a common cause of condemnation in layer processing plants.

**Hemangioma (Hemangioblastoma, Hemangioendothelioma)**

**Species of bird**--Chickens, less common in commercial flocks than lymphoid leukosis.

**Age of birds**--6-9 months of age.

**Etiology** - RNA virus in the family retrovirus.

**Mode of transmission**

It spreads transovarian or laterally among hatchmates.

**Clinical Signs**

1. Anemia, weakness, and poor egg production are seen.

2. Feathers near tumors are bloodstained.

**Postmortem lesions**

1. Hemangiomas (blood blisters) on the skin and blood clots in visceral tumors occur.

2. The disease often occurs in birds with erythroblastosis and myeloblastosis.

![Figure 12.6. Blood tumor over the liver](image)

**Diagnosis**
1. The presence of blood tumors is characteristic of the disease (figure 12.6).

2. Histologic observation of tumors reveals solid masses varying from gray to pink. The endothelium may proliferate into dense masses (hemangioendothelioma), leaving more clefts for blood channels. Skin tumors are more encapsulated and have more trabeculae than visceral tumors.

3. It simulates wounds, bleeding of feather follicles and cannibalism.

**Prevention**

Select breeding stock, which are not infected with the virus.

**Treatment**

None.

**Osteopetrosis (marblebone, thickleg disease, osteoperiostitis)**

**Species of bird**--Chickens; it is more common than other rare forms of leukosis.

**Action**--Chronic.

**Age of birds**--8-12 weeks of age.

**Etiology**--RNA virus belonging to family retroviridae; belongs to subgroup B of lymphoid leukosis viruses.

**Mode of transmission**

It is spread transovarian or laterally among hatchmates.

**Clinical signs**

1. Incubation period of 1 month.

2. Long bones of limbs show a uniform or irregular thickening of the diaphysial or metaphysical regions.

3. Characteristic sign is a stunted, pale bird, with "boot-like" shanks. The birds walk with a stilted gait or limp.

**Postmortem lesions**
1. Bone alterations occur in the diaphysis of the pelvis, shoulder girdle and ribs (figure 12.7).

2. Distinct pale yellow foci occur against the gray-white translucent normal bone.

3. The periosteum is thickened and abnormal bone is spongy and is easily cut.

4. Osteopetrosis and lymphoid leukosis frequently occur together in the same bird.

5. There can be atrophy of the spleen, bursa, and thymus.

![Figure 12.7. Bone tumors on the right seen with osteopetrosis.](image)

**Diagnosis**


2. Spongy bone converges centripetally toward the center of the shaft.

3. There is an increase in size and irregularity of Haversian canals and increase in number and size of lacunae. Osteocytes are more numerous, large, and eosinophilic. New bone is basophilic and fibrous.

4. It simulates rickets and osteoperosis.

**Prevention**

Select breeders free of the virus.

**Treatment**

None.

**Squamous cell carcinoma (elephant skin, skin leukosis)**
Species of bird--Broilers.

Action--Chronic.

Etiology--Unknown virus.

Mode of transmission

Spread transovarian or laterally among hatchmates.

Clinical signs

Usually no signs.

Postmortem lesions

1. Lesions are usually seen only in the processing plant after the birds have been scalded and the feathers are picked.

2. Large scabby lesions occur on the surface of the skin. They are more numerous over the thigh and legs, but also occur on the breast and back.

3. Some scabs are removed by mechanical processing; revealing large irregular crater-like ulcers (figures 12.8 and 12.9).

4. Bluish to greenish discoloration of the skin around the tumors is common.

5. Only one localized tumor is cut out in the processing plant and the bird is down-graded. Multiple tumors result in condemnation of the entire carcass.

Figures 12.8 and 12.9. Crater-like skin tumors seen with squamous cell carcinoma.

Diagnosis
1. Gross and microscopic observations of skin tumors in broilers are necessary.
2. Microscopic tumors have disorganized basal cells and keratinized cells.
3. It simulates bacterial folliculitis, MD, and Favus.

**Prevention**

1. Select breeders free of virus.

**Treatment**

None.

**Reticuloendotheliosis (acute reticulum cell neoplasia, runting disease syndrome)**

**Species of bird**--Turkeys, chickens, pheasants, quail.

**Action**--Chronic.

**Age of bird**--All.

**Etiology**--RNA retrovirus similar to avian leukosis/sarcoma group. Some viruses are defective and require a helper virus to complete their replication cycle.

**Mode of transmission**

1. It is spread transovarian from infected hens to offspring.
2. The virus can spread laterally by contaminated feces and litter (although contact infection rarely results in disease).

**Clinical signs**

1. Acute reticulum cell neoplasia results in enlarged abdomen, weakness, and death.
2. Runting disease syndrome is seen as stunted, pale birds with abnormal feather development, and lameness.
3. Chronic neoplasia, chicken bursal lymphoma, turkey lymphoma, and multis syndromes cause weakness, paleness, and anorexia.

**Postmortem lesions**
1. Acute reticulum cell neoplasia causes large livers and spleens with infiltrative focal or diffuse lesions. Lesions are also common in the pancreas, gonads, heart, and kidney.

2. Runting disease syndrome causes acute hemorrhagic or chronic ulcerative proventriculitis, atrophy of the thymus and bursa, enlarged peripheral nerves, enteritis, anemia, and necrosis of liver and spleen.

3. Chicken bursal lymphoma causes tumors in the liver and bursa of Fabricius.

4. Chicken non-bursal lymphoma produces tumors of the heart, liver and spleen, atrophied thymus, and enlarged peripheral nerves.

5. Turkey lymphoma results in tumors in the liver and other visceral organs.

**Diagnosis**

1. Gross and microscopic observations of tumors are necessary.

2. Tumors consist of large vesicular mononuclear cells of the reticuloendothelial system. Areas of necrosis are associated with tumors.

3. Detection of virus using fluorescent antibody test with antibodies can be done.

4. It simulates avian leukosis and MD.

**Prevention**

Use ELISA for selection of breeders free of the virus.

**Treatment**

No effective treatment.

**Newcastle disease**

Named for town in England where it was first seen.

**Species of bird**--All. It is one of the most common respiratory diseases of poultry. It occurs worldwide.

**Action**--Acute and chronic.

**Age of bird**--Any.
**Etiology**—Single-stranded, nonsegmented, enveloped, RNA virus belonging to paramyxoviruses. Three pathotypes or strains exist. The lentogenic cause mild disease; the mesogenic produce moderate disease; and the velogenic produce severe morbidity and mortality.

**Mode of transmission**

1. It is spread airborne by inhalation or ingestion of virus.

2. Free-flying birds in United States are commonly infected with lentogenic viruses and can readily spread them.

3. Exotic birds from tropical areas are commonly infected with velogenic viruses.

**Clinical signs**

(The incubation period is 2-15 days.)

1. Watery eyes and a plug in the eye are seen with lentogenic strains.

2. Coughing, gasping and some mortality are seen with mesogenic strain.

3. It affects egg production and quality (brown broiler eggs turn to white eggs). It may produce torticollis (figures 12.10 and 12.11), paralysis, and bloody diarrhea. High morbidity and mortality occur with visceral tropic velogenic (VVND). VVND (exotic) rarely occurs in commercial poultry in the United States. The most recent outbreak of VVND in 2003 in California and several nearby states (Nevada, Utah, Texas, and Arizona) have resulted in destruction of 3 million commercial birds. Many more back-yard birds were also destroyed. However, outbreaks in pet bird populations in the U.S. are more frequent.

Figures 12.10 and 12.11. Central nervous signs common with Newcastle disease.
Postmortem lesions

1. Lesions include cloudy air sacs (figure 12.15), congestion, conjunctivitis (figure 12.14), and edema of lungs, abdominal yolk, edema of bronchi and parabronchi, and mucous in trachea and nasal turbinates (figures 12.12 and 12.13).

2. Internal hemorrhage may be seen in VVND (lungs, intestines, gizzard, proventriculus, and cecal pouches) (figure 12.9).

Diagnosis

1. The clinical disease (respiratory and central nervous signs) and gross and microscopic lesions in trachea, nasal turbinates and lungs are helpful in the diagnosis.

2. Isolate and identify virus from trachea of clinically ill birds in cell culture or chicken embryos for definitive diagnosis.

3. The HI or ELISA test for measuring a rise in antibody titer is helpful.

4. It simulates IB, Coryza, chronic respiratory disease (CRD), infectious laryngotracheitis (ILT), and Paramyxovirus II and III.
5. PCR test for detection of virus.

**Prevention**

1. Vaccinate by coarse spray cabinet or eye drop (Bioinjector®) at 1 day, or by water, or coarse spray at 7 days of age. In VVND endemic areas, an inactivated vaccine may also be given at 1 and/or 14 days of age.

2. Always revaccinate by water or spray at 14-21 days later in the wintertime, when the incidence of respiratory disease is increased.

3. Breeders or layers are vaccinated by water or coarse spray at 60-90 day intervals throughout the growing period.

4. You can use an inactivated NDV vaccine at 18-22 weeks for breeders or layers, or continue live vaccination throughout lay.

5. The B1 type, B1 strain (Lento) is used for the 1st and 2nd vaccination, and the Lasota (Meso) or cloned Lasota is used thereafter.

6. It is usually combined with infectious bronchitis (IB) vaccine.

7. First commercially licensed recombinant vaccine for use in poultry contained Hemagglutinin gene inserted into fowl pox vector for injection at day-of-age.

8. Biosecurity is important to control the disease.

**Treatment**

A broad-spectrum antibiotic can be given to control secondary invaders, especially *E. coli* from causing CRD. Add chlorine to drinking water a 5 ppm to reduce reactions.

**Special note**

It is a very common viral disease of poultry worldwide. Most poultry are vaccinated several times against this virus. Paralysis, incoordination, central nervous signs after first respiratory signs, are diagnostic for NDV. It can be complicated by Mycoplasma or *E. coli* resulting in CRD and severe air sacculitis. All imported birds are quarantined to control importation of VVND for 45 days. Domestic lento or mesogenic NDV cause mild respiratory disease. VVND is a reportable disease.
Other Paramyxovirus infections

Species of bird--Paramyxovirus (PVM) II; chickens and turkeys, PMV III; turkeys, pigeons, Pscittacine birds, PMV VI; turkeys.

Action--Acute to chronic.

Age of bird--All.

Etiology--Paramyxoviruses are single-stranded, nonenveloped RNA viruses. PMV I, (NDV), PMV II and PMV III are all serologically distinct.

Mode of transmission

Spread aerosol as with NDV.

Clinical signs

1. PMV II produces a mild respiratory disease, sinusitis, elevated mortality, low egg production, and reduces fertility and hatchability.

2. PMV III produces a mild respiratory disease with lower egg production and poor egg shell quality.

3. PMV VI produces a mild respiratory disease and egg production losses.

Postmortem lesions

All PMV's produce mucous in the nasal turbinates, sinuses, trachea, and edematous bronchi, parabronchi and lungs.

Diagnosis

1. The clinical disease, and gross and microscopic lesions are helpful aids in the diagnosis.
2. Isolation and identification of virus in chicken embryos or cell culture is necessary for a definitive diagnosis.

3. Serological analysis of antibodies against PMV's using ELISA or hemagglutination inhibition is also helpful.

4. It simulates NDV, IB, Coryza, chronic respiratory disease (CRD), and infectious laryngotracheitis (ILT).

Prevention

An inactivated vaccine against PMV IV is available for use in turkeys or pigeons only.

Treatment

Antibiotics in feed and water for secondary invaders are helpful.

Infectious Bronchitis

Species of bird--chickens; common in commercial layers.

Action--acute or chronic.

Age of bird--any, however, young is most susceptible.

Etiology--Coronavirus is pleomorphic, enveloped with spikes, and has a single strand of RNA.

Mode of transmission

1. It is very contagious and spreads rapidly by aerosol.

2. Contaminated feces, litter, and fomites also spread the virus.

Clinical signs

1. Sneezing and watery eyes are seen early on, followed by depression, coughing, and nasal discharge (figures 12.16 and 12.7).

2. Poor egg shell quality, watery albumen, ruffled feathers, and wet droppings are seen in laying birds (figure 12.18).

3. A drop in egg production and weight gain, tracheal rales, gasping, and urate diarrhea are also seen.
Postmortem lesions

1. Exudate in trachea, nasal turbinates, air sacs thickened or frothy, and pneumonia can be seen.

2. Swollen pale kidneys with urates, misshapen (hypoglandular) ova and oviduct, and yolk in abdominal cavity may also occur.

Diagnosis

1. Eliminate CRD, Coryza, ILT, NDV, and other paramyxoviruses.

2. Virus neutralization, HI or ELISA tests for measuring antibody are helpful.

3. Virus isolation in embryos or chicken kidney cell cultures is necessary for a definitive diagnosis.

4. Curling, stunting, and death of embryos can be seen in inoculated embryonating eggs.

5. It simulates many respiratory diseases (NDV, paramyxoviruses, ILT, CRD, and Coryza).
6. Respiratory signs and lesions with kidney lesions give a presumptive diagnosis.

**Prevention**

1. Vaccinate with multiple serotypes (Mass, Conn, Ark 99, Holland or Florida) for broad-spectrum protection. Mass and Conn serotypes are most commonly used. New Ga (Georgia) isolate has caused problems in surrounding states. PCR test is used to rapidly differentiate serotypes.

2. The vaccine is usually mixed with ND vaccine and given by coarse spray, drinking water or eye drop.

3. Inactivated vaccine can be given for breeders or layers at 18-22 weeks of age by SQ injection.

4. Sanitation, hygiene, and biosecurity are also important.

**Treatment**

Antibiotics for killing secondary bacterial invaders in feed or water are helpful.

**Special note**

It is the most contagious viral respiratory disease in poultry. Many serotypes make successful vaccination difficult. It can be complicated by Mycoplasma or *E. coli*. It is a very common cause of egg production losses in commercial layers.

**Infectious laryngotracheitis (ILT or trach)**

**Species of bird**--Chickens, pheasants.

**Action**--Acute to chronic.

**Age of bird**--All (usually older than 4 weeks). It is very common in force molted layer flocks.

**Etiology**--Herpes virus is a cuboidal, enveloped DNA virus with icosahedral symmetry.

**Mode of transmission**

1. Older carrier birds are a common source of infection.

2. Aerosol, fomites, and ingestion of contaminated litter are also common means for viral spread.

**Clinical signs**
(Incubation period of 6-12 days. There are many pathotypes of the virus. Some are very mild and others can cause severe morbidity and mortality.)

1. Mortality (1-10%), morbidity (90-100%), drop in egg production (1-20%), watery eyes early on, and then nasal discharge, gasping, tracheal rales and stretching necks are common signs. Slinging of blood from the nose causes blood stains along the sides of walls (figure 12.19).

![Figure 12.19. Common signs and lesions seen with ILT](image)

2. Most birds recover in 10-14 days if infection is not complicated by immunosuppression or a secondary bacterial or mycoplasma infection.

**Postmortem lesions**

1. Mucous in trachea is seen first, followed later by necrotic tissue then blood (figures 12.20, 12.21, 12.22).

2. Inflammation of bronchi and lungs, foamy air sacs, edema and congestion of the conjunctiva and infraorbital sinuses are commonly seen.

![Figures 12.20, 12.21, 12.22. Blood in the trachea is diagnostic for ILT.](image)
**Diagnosis**

1. Laboratory tests include microscopic observation of intranuclear inclusion bodies in the lesions (usually trachea).

2. Blood in the trachea is an important lesion.

3. Isolation of the virus on the chorioallantoic membrane (CAM) of embryos is important. Plaques (lesions on the CAM) have inclusion bodies.

4. It simulates many respiratory diseases (CRD, NDV, paramyxovirus, IBV, and Coryza).

**Prevention**

1. Vaccinate broilers with live attenuated product by-water (embryo-derived), spray or eye drop (cell culture derived) at 2-4 weeks of age only in endemic areas. State veterinarian controls use of vaccine in broiler flocks in the U.S.

2. Revaccination of breeder and layer pullets at 10-14 weeks, by eye drop with mild cell culture vaccine or by wing web with new recombinant fowl pox vector vaccine, is common throughout the US. Revaccinate force molted hens. The vaccine virus may cause a latent infection, which can appear later causing morbidity.

3. Don't import mature birds. Spiking of breeder flocks with additional males to improve fertility often introduces ILT virus into a flock.

**Treatment**

Antibiotics for secondary invaders are helpful.

**Special note**

Vaccinate broilers in endemic areas and quarantine affected flocks. The virus moves slowly, so birds can be successfully vaccinated on a farm to reduce the spread of the disease. An outbreak of this disease must be reported to the state veterinary diagnostic laboratory.

**Fowl pox (avian diphtheria)**

It is named pox because of the scabby lesions on the skin and diphtheria because of the pseudo (false) membrane, which forms in the mouth.

Species of bird--All.

Action--Chronic.
Age of bird—Any.

Etiology—Pox viruses are single, linear, double stranded DNA viruses. There are many avian pox viruses (fowl—chicken, turkey, pigeon, canary, quail, sparrow, parrot and starling). A substantial degree of host specificity exists for these pox viruses.

Mode of transmission

1. Mosquito bites and mechanical transmission of virus to lacerated skin or eye are common routes of viral spread.

2. Wild birds are a reservoir for the viruses.

Clinical signs

(Incubation period is from 4-10 days. They are two forms of the disease; the cutaneous or dry and the diphtheritic or wet.)

1. The dry form shows as a pimple or scab on skin (mainly comb, wattles, eyelids and other unfeathered portions of the body) (figure 12.23).

![Figure 12.23. Dry Pox](image)

![Figure 12.24. Wet Pox](image)

2. The wet-mucous form shows diphtheritic cankers or yellow lesions in mouth, esophagus or trachea.

3. Eye involvement (blindness), off feed, lowered egg production, facial swelling, and an increase in culls can be seen.

Postmortem lesions

1. Cankers or false membranes in mouth (wet) are seen as slightly elevated white opaque nodules. Nodules increase in size and coalesce (come together) to form yellow, cheesy, and necrotic membranes.

2. Grey or black papular eruptions on unfeathered portions of skin (dry) are due to epithelial hyperplasia (figure 12.25).
3. Head, face, and feet are most commonly affected, but may spread to feathered portions of the body (figure 12.24).

Figures 12.25 and 12.26. Dry and wet pox lesions in a breeder hen (left) and turkey (right)

**Diagnosis**

1. Eliminate bacterial dermatitis.

2. Virus isolation on CAM will produce plagues, which will reveal intracytoplasmic inclusion bodies.

3. Gross lesions will reveal inclusion bodies.

4. It simulates in the dry form staph-dermatitis and T-2 toxin, and *Candida albicans*, vitamin A deficiency, and trichomoniasis in the wet pox form.

5. Presence of scales on skin and cankers in the mouth in the fall of the year is an important characteristic of the disease.

**Prevention**

1. Vaccinate broilers at 1 day during summer in Southeastern U.S. or year round in tropical areas with live vaccine. Mix diluted (1 to 3) vaccine with HVT and give subcutaneously in the hatchery. Recombinant pox vaccine can have NDV, ILTV, or AE genes.

2. Vaccinate replacement pullets 10-12 weeks (wing web) and again at 16-18 weeks to provide long term immunity. Examine site of injection 1 week later for vaccine reaction (scab).
3. Turkeys are vaccinated in the thigh to prevent spread of vaccine lesions to face and eye, because turkeys sleep with head under wings.

4. Control mosquitoes with insecticides and by getting rid of all standing water (pond, etc.), where mosquitoes may breed.

**Treatment**

Remove scabs on expensive show birds or pets. Use a tincture of iodine to treat lesions.

**Special note**

It causes extreme problems in pen reared quail. The chicken vaccine is pathogenic in quail, so use the quail product. Canaries in aviaries are also very susceptible. Very common in summer and early fall when mosquito population is near its peak.

**Avian influenza**

**Species of bird**--All.

**Action**--Acute to chronic.

**Age of bird**--All.

**Etiology**--Orthomyxovirus is a single-stranded RNA virus containing 8 segments, and 35 serotypes. Types B and C occur in humans only. Type A occurs in humans, swine, horses, birds, mink, seals and whales. Type A viruses are divided into subtypes according to the antigenic nature of hemagglutinin (HA) and neuraminidase (N). The HA is a viral protein which can attach to the cell surface and the N an enzyme which can brake the virus-cell attachment. Viruses are identified by HA, N, species of animal and year isolated. There are 15 HA and 9 N subtypes. H5 and H7 are the most pathogenic types. Pathogenic and antigenic changes are common by antigenic drift (mutation) and antigenic shift (reassortment of segments).

**Mode of transmission**

1. It is spread by aerosol via the respiratory tract and breathing contaminated feces.

2. It can also spread by wild water fowl and contaminated fomites.

**Clinical signs**

(The incubation period is hours to days, depending on age, sex, and species affected, concurrent infections and pathogenicity of virus.)
1. Respiratory distress, coughing, sneezing, rales, depression, sinusitis, emaciation, off feed, nervous disorder, and diarrhea may be seen.

2. Rapid mortality occurs with virulent stains (fowl plague) (figure 12.27). Multiple pathotypes can occur.

Figure 12.27. Rapid mortality

Figure 12.28. Hemorrhagic wattles

3. A drop in egg production and shell quality, watery eyes, excessive lacrimation, edema of head and face, and cyanosis may be observed (figure 12.28).

Postmortem lesions

1. Lesions include mucous in trachea, air sacculitis, swollen head or wattles, egg peritonitis, sinusitis (figure 12.30), watery lungs, and fibrinous enteritis.

2. Pericarditis, necrosis of skin and GI tract; hemorrhages on wattles, combs and legs; necrotic foci on liver, spleen, kidney and lungs, and hemorrhages at junction of proventriculus and gizzard can be seen with fowl plaque (figure 12.29).

Figure 12.29. hemorrhages in cecal tonsils

Figure 12.30. lesions in turbinates

Diagnosis
1. The AGP and ELISA tests can be used for determining the presence of antibody in the sera.

2. Isolation and identification of virus from trachea or vent in embryonating eggs or tissue culture is needed. Test hemagglutinating ability of the virus mixed with chicken sera containing antibodies against influenza. The virus can be sub-typed using specific sera against all known HA and N.

3. It simulates mycoplasmosis, turkey coryza, VVND, ILT, ornithosis, and fowl chlorea.

4. PCR test to detect virus.

**Prevention**

1. Killed vaccines are available in limited areas for turkeys in the mid west and layer flocks in the NE only.

2. Quarantine, depopulation and eradication of virulent form is mandated under law in the U.S.

3. Strict biosecurity is needed.

4. Control of live bird market in large north eastern and western U.S. cities is important to prevent the spread of the virus.

5. Recombinant AI vaccine available in Mexico contains H gene cloned into fowl pox vector.

**Treatment**

Broad-spectrum antibiotics are helpful to control secondary bacteria.

**Special note**

It is a reportable disease, highly contagious, and spread by waterfowl. It caused depopulation of 17 million birds in 1983 and 1984 in the northeastern United States at a cost of $60 million. Outbreaks in Virginia in 2003 have resulted in depopulation of a number of commercial flocks. The virus is highly unstable and can alter patho and antigenic types quickly by passage in various animal species. Migratory birds spread and transmit human and swine influenza viruses across continents. In 1995, a number of fowl plaque outbreaks were reported in Mexico. In 1996, various live and killed vaccines have helped control the disease in Mexico.

Ratites (ostriches, emus and rheas) are sometimes infected. In 1998 and in 2003 virus spread from chickens into human population in Hong Kong and the Neatherlands, causing death of a number of people. The spread of disease was stopped with the eradication of several million birds. In 2004-5, the disease spread throughout SE Asia, Japan, China, British Columbia,
Delaware, Pa., NJ., MD., and Texas resulting in the death or slaughter of 100 million birds (1/3 of world’s population). In 2006, the virus has spread eastward to Europe, India, and Israel, infecting and killing both wild free flying and commercial birds. So far, 100 people have died from bird flu.

Avian encephalomyelitis, epidemic tremor (AE)

Species of bird--Chickens, pheasants, quail and turkey.

Action--Peracute.

Age of birds--1-3 weeks and adults.

Etiology--Picornavirus is a small RNA virus lacking an envelope.

Mode of transmission

1. Spread vertically through egg for 42 days after infection and by contaminated feces and fomites.

Clinical signs

(Incubation period of 1-7 days.)

1. Signs include trembling of the head, lateral recumbency (birds down on their sides), ataxia (figure 12.31), and dull eyes.

2. In chicks drowsiness, lack of coordination, unsteady gait, and mortality (5-10%) can occur. In adults, a drop in egg production and cataracts (opacity of lens) can be seen.

Figure 12.31. Lateral recumbency characteristic of AE in chicks.

Postmortem lesions
1. Brain lesions include disseminated, nonpurulent encephalomyelitis (figure 12.32).

2. Pale gizzard muscle and bluish opacity of the lens may sometimes be seen (figure 12.33).

![Figure 12.32. Edema in brain in young birds](image1) ![Figure 12.33. Cataract in adults with AE](image2)

**Diagnosis**

1. Diagnosis is by virus isolation by inoculating embryos at 5-7 days and observing the hatched chicks for clinical signs.

2. Histopathology of brain lesions, and virus neutralization test, or ELISA to measure antibodies is helpful.

3. It simulates IBD, vitamins E and B₁ deficiency, NDV, and MD.

4. Lateral recumbency in chicks or cataracts in adults are a presumptive diagnosis for AE.

**Prevention**

1. Buy chicks only from clean or immune parents. Vaccinate pullets (1-20%) at 10-16 weeks by wing-web or eye drop. Vaccine spreads rapidly through pullet flocks so all birds do not have to be vaccinated. New recombinant pox vaccine that contains AE immunogen is available.

**Treatment**

None.

**Special note**

The hen sheds vaccine virus for 42 days after infection which can cause disease in the progeny, so don't vaccinate within 42 days of lay.
Quail bronchitis

Species of bird--Bobwhite quail.

Action--Acute to chronic.

Age of bird--Mostly young.

Etiology--Adenovirus is an unenveloped, icosahedral double-stranded DNA virus, which replicates in the nucleus forming basophilic inclusion bodies.

Mode of transmission

1. It is spread by airborne route.

Clinical signs

1. There is a 2-7 day incubation period. Signs include coughing, sneezing, huddling, depression, lacrimation, conjunctivitis, and neurologic signs (tremors and paralysis).

Postmortem lesions

1. Mucous in the trachea or bronchi, cloudy air sacs and corneas, and sinusitis are seen.
2. Foci in liver, spleen swollen and mottled, lungs are reddened and consolidated, and atrophy of bursa of Fabricius can be seen.

Diagnosis

1. Isolation of virus from trachea in embryonated eggs is important.
2. Neutralization of virus with specific sera is also helpful.
3. It simulates Aspergillosis and Newcastle Disease.

Prevention

Biosecurity and sanitation are helpful to control the disease.

Treatment

Broad-spectrum antibiotics for secondary invaders are also helpful.

Special note

It may cause immunosuppression.
Inclusion body hepatitis (IBH)

Species of bird--Chickens, broiler breeder pullets, leghorn pullets.

Action--Acute to chronic.

Age of bird--2-20 weeks.

Etiology--Adenovirus is an unenveloped, icosahedral, double-stranded DNA virus. It replicates in the nucleus forming basophilic inclusion bodies.

Mode of transmission

1. Infected litter and fomites, and transovarian transmission are important means for viral spread.

Clinical signs

1. Signs include depression, ruffled feathers, off feed, and poor feed conversion.

2. Pale comb and wattles, gangrenous dermatitis, high mortality (up to 50%) and/or anemia are common secondary findings.

Postmortem lesions

1. Lesions include fatty and/or hemorrhagic liver (figure 12.35), pale bone marrow (anemia), and hemorrhages in the skeletal and heart muscles and dermatitis (figure 12.34).

2. Atrophy of spleen and bursa due to early IBDV or chick anemia virus (CAV) infections are also a common finding.

Diagnosis
1. Microscopic observations of liver cells will reveal diagnostic intranuclear inclusion bodies.

2. It simulates aflatoxicosis.

**Prevention**

1. Avoid stress and vaccinate against IBD to help control the disease.

**Treatment**

Antibiotics are recommended to reduce secondary invaders which may cause dermatitis and anemia.

**Special note**

Prior exposure to IBDV or CAV depresses the immune response and makes birds more susceptible to IBH.

**Hemorrhagic enteritis (HE)**

**Species of bird**—Turkeys (most common) and chickens.

**Action**—Acute.

**Age of bird**—4 weeks or older.

**Etiology**—Adenovirus is an unenveloped, icosahedral, double-stranded DNA virus. It replicates in the nucleus forming basophillic inclusion bodies (viral factories seen under light microscope).

**Mode of transmission**

Spreads orally from infectious feces or litter.

**Clinical signs**

(Incubation period is less than 24 hours.)

1. Depression, bloody droppings, mortality (10-80%), dark red to brownish blood on skin, feathers, and around vents can be seen.

**Postmortem lesions**

1. The skin and flesh are pale, anemic, and dark in color.
2. Jejunal mucosa is red and highly congested (figure 12.36), spleens are enlarged, friable and mottled, lungs are congested and vascular organs are pale, and the livers are enlarged with hemorrhage.

![Figure 12.36. Hemorrhagic enteritis](image)

**Diagnosis**

1. Clinical signs, and gross and microscopic pathology are useful.

2. Intranuclear inclusion bodies in the reticuloendothelial (RE) system and intestine are diagnostic.

3. Spleens show RE hyperplasia and inclusion bodies. ELISA or AGP tests can be used to detect antibody.

4. It simulates leukosis, reticuloendotheliosis, fungi, toxicities, and coccidiosis.

**Prevention**

Use of a live vaccine in drinking water at 4 and 6 weeks of age can prevent the diseases.

**Treatment**

None.

**Infectious bursal disease (IBD) (gumboro)(chicken aids virus)**

It is named for a town in Delaware (Gumboro), where it was first seen in the 1960's.

**Species of bird**--Chickens and turkeys.

**Action**--Acute.

**Age of bird.** It occurs only in young birds (2-16 weeks), which have a functioning bursa of Fabricius. The bursa atrophies with the onset of sexual maturity.
**Etiology**--Birnavirus contains two segments of double-stranded RNA. The virus is a single-shelled, nonenveloped virion with icosahedral symmetry and diameter ranging from 55-65 nm. It is highly stable and resistant to many physical and chemical agents, and therefore impossible to eradicate.

**Mode of transmission**

1. It spreads by contaminated feces, water and feed.
2. It is a highly contagious and hardy agent. Other vectors can harbor the virus including the lesser meal worms and rats.

**Clinical signs**

(Clinical disease seen in chickens. Incubation period is 2-3 days.)

1. Elevated body temperature, (111°), watery urate diarrhea, anorexia, depression, ruffled feathers, head trembles, sleepiness, and lameness can occur (figure 12.37).
2. It is subclinical in turkeys.
3. Morbidity approaches 80% in white leghorns and 50% in broilers.
4. Hypervirulent strains occur outside the U.S. and can cause up to 100% morbidity and 80% mortality in leghorns. Normal mortality is not more than 40% in leghorns and 20% in broilers.

![Figure 12.37. Urate droppings seen with acute IBD](image)
Postmortem lesions

1. The bursa is enlarged (2-4 times), hemorrhagic and/or edematous early (3-5 days) in the course of the infection (figures 12.30 and 12.39).

2. Other lesions include an increase in kidney urates, a swollen necrotic spleen, and increased mucous in the intestine.

3. Later in the infection the bursa is atrophic (7 days), 1/4-1/2 normal size. Thymus may also be atrophic. The bursa remains atrophic through the life of the bird, whereas the thymus can regenerate.

4. Muscle hemorrhage, rickets, dehydration, hemorrhages at the junction of the proventriculus and gizzard may also been seen especially with the hypervirulent strains.

Diagnosis

1. Edematous involvement of the bursa of Fabricius in young birds is diagnostic.

2. Eliminate AE, aflatoxicosis and coccidia.

3. Serology is of limited value because most chickens are routinely vaccinated against IBDV.

4. It simulates AE, aflatoxin, coccidiosis, and rickets.
Prevention

1. Vaccinate parents at 2 and 6 weeks with live vaccine, and at 10 and 18 weeks with killed vaccine containing both standard and variant subtypes. Occasional boosting of breeders at 40 weeks is done with live or killed vaccine. Intermediate vaccines have replaced mild vaccines, because they can override moderate amounts of maternal immunity.

2. Vaccinate progeny between 1 and/or 14-21 days with attenuated vaccine on problem farms by spray or drinking water. Some vaccine is being given in ovo at 18 days of embryonation mixed with MD vaccine.

3. There are two serotypes of IBDV. Serotype 1 viruses are pathogenic and occur only in chickens, whereas serotype 2 viruses are not pathogenic and can infect chickens and turkeys. Serotype 1 viruses are divided into 6 subtypes using cross virus neutralization assays. Subtype 6 viruses are called "variant", because they are not adequately protected against by the standard viruses in subtypes 1-5. Other untyped variants are continually developing in the U.S. due to mutations in the viruses. Live and killed variant vaccines are available and used extensively in the U.S.

4. Verkon® from Duravet, Inc. is the only USDA approved disinfectant, which can kill IBDV.

Treatment

Vitamins and minerals and/or sugar in drinking water to prevent dehydration, replace lost electrolytes and provide an energy boost.

Special note

It is immunosuppressive and very common throughout the world. Infection prior to 2 weeks causes subclinical immune depression (more common). Infection after to 2 weeks causes clinical disease and transient immune depression. Serologic variant viruses in the field may cause vaccine failures by producing rapid atrophy of the bursae even in birds with maternal immunity against serologic standard viruses. Hypervirulent viruses can be controlled using two standard intermediate vaccines during the first 3 weeks of life.

Viral arthritis (VA) (viral tenosynovitis)

Species of bird--Broilers, broiler breeders, and turkeys.

Action--Chronic to debilitating.

Age of bird--3-30 weeks. Broilers develop problems from 5-8 weeks, whereas breeder pullets and turkeys are affected at 10-18 weeks of age.
Etiology--Reovirus (respiratory, enteric, orphan). It is a nonenveloped, icosahedral, double capsid, and double stranded RNA virus. It contains 10 discrete molecular species. It is resistant to many chemical agents.

Mode of transmission

1. It is spread laterally from bird to bird or vertically from hen to offspring.
2. Fecal-oral or via respiratory tract transmission can also occur.

Clinical signs

(Incubation period of 9-13 days.)

1. Birds are down on the hocks and have stunted growth, are reluctant to move, and have an uneven gait.
2. Swollen tendons above and below the hocks and a ruptured gastrocnemius tendon are also seen. The tendon is fibrous tissue that connects muscle to bone.

Figures 12.40 and 41. Ruptured tendons seen with VA

Postmortem lesions

1. Bilateral swelling of tendons (digital flexor and metatarsal extensor) is diagnostic (figure 12.40).
2. There is an increase in clear, thick fluid in joints, and swelling of footpad, straw color, or blood-tinged exudate in lesions can be seen.
3. Ruptured tendons, hemorrhaging of synovial membrane, erosion of cartilage of distal tibiotarsus can also occur (figures 12.42 and 43.). The condyles (joint capsule) may also be involved.

**Figures 12.42 and 43. Ruptured tendons are diagnostic for VA.**

**Diagnosis**

1. Isolation of virus from lesions on CAM of inoculated embryos and serologic test for VA antibodies (VN or ELISA) are of limited use, because birds are routinely infected with nonpathogenic reoviruses.

2. Histopathology, reveals edema, coagulation necrosis, heterophil accumulation and perivascular infiltration. Hypertrophy and hyperplasia of synovial cells, infiltration of lymphocytes, macrophages, and reticular cells are also evident.

3. VA causes excessive or clear to straw colored fluid; *Mycoplasma synovitis* causes honey colored fluid, and Staphylococcus produces thick white (pus) fluid in swollen joints.

4. Gross and microscopic lesions are diagnostic.

5. Ruptured gastrocnemius tendon is characteristic.

**Prevention**

1. Vaccination of parent stocks no later than 20 weeks of age with the S1133 virus strain.

2. Usually given at 2 weeks (SQ or water) and between 6-10 weeks (water) with live vaccine. Killed vaccine virus is given at 18 weeks and possibly again 40 weeks by SQ injection.
3. Attenuated live vaccine diluted by 1/3 at 1 day with a full dose of HVT for broilers in problem areas or by coarse spray at day of age with a full dose. Boosting at 7-10 days by spray or water is sometimes done. A full dose of reovirus vaccine can interfere with HVT vaccine. It will not interfere with SB1 or Rispens.

**Treatment**

None. Use albamycin to prevent secondary staph infections.

**Special note**

It can cause downgrading and trimming in broilers and culling in breeders. Reovirus that causes viral tenosynovitis is related to malabsorption syndrome virus. Vaccination is highly effective.

*Pale chick or bird syndrome (infectious proventriculitis, malabsorption, stunting, or runting syndrome, helicopter disease).*

Birds have a variety of signs and lesions indicative of vitamin and mineral deficiencies. A syndrome is a collection of signs and lesions.

**Species of bird**--Broilers and broiler breeder pullets, leghorns and turkeys.

**Action**--Acute to chronic.

**Age of bird**--Young (2-20 weeks).

**Etiology**--Reovirus related to tenosynovitis virus.

**Mode of transmission**

1. Spread by vertical or horizontal routes and fecal contamination.

**Clinical signs**

(Incubation period of 7-14 days.)

1. Stunting (stunting or runting syndrome) (figure 12.43), abnormal feathering (helicopter disease) (figure 12.44), pale comb, wattles and legs in broilers and turkeys (pale bird syndrome) are seen.
2. Higher early mortality, weak legs, CNS signs (tremors, incoordination) and passage of undigested food in feces can occur.

3. Delayed and poor egg production peaks may occur in layers and breeders.

**Postmortem lesions**

1. Enteritis can cause undigested feed in intestines and pale intestines hemorrhages around heart may also be seen.

2. Anemia, seen as decreased pigmentation, and atrophy of the pancreas and bursa of Fabricius can occur.

3. An enlarged proventriculus with glandular enlargement and a loss of the normal structural architecture (infectious proventriculitis) may be evident (figure 12.46).

4. Brittle bones and femoral head necrosis with rickets may occur (figure 12.47).

5. Hydropericardium (water around the heart), a small flaccid (flabby) gizzard, and encephalomalacia can often arise.
Diagnosis

1. The clinical signs and gross lesions (particularly proventricular hyperplasia and atrophied pancreas) are characteristic.

2. It simulates nutritional deficiency, mycotoxins, IBD, and coccidiosis.

Prevention

A live VA (S1133) vaccine at 1-2 and 6-8 weeks by injection, coarse spray or water and inactivated malabsorption and VA vaccine at 18-20 weeks for breeders and layers will help control diseases. Malabsorption isolates (2408, 1733, Maryland or CO₈) are used in inactivated vaccines. These isolates are in a different serologic subtype from the S1133 virus. Some broiler flocks are vaccinated at 1-7 days with VA vaccine.

Treatment

1. It responds to antibiotics plus vitamin-mineral supplementation.

2. Vinegar in water (1%) kills viruses by reducing intestinal pH, and reduces spread of virus and some clinical signs.

Special note

Reovirus produces malabsorption and maldigestion syndrome, which leads to secondary vitamin or mineral deficiency. Reoviruses are also immunodepressive. Vaccination is highly effective against this disease. The disease comes and goes and often disappears without a change in vaccination program.

Hemorrhagic enteritis (HE) [related infections: marble spleen disease (MSD) and avian adenovirus II spleenomegaly (AAS)].

Species of bird--Turkeys (HE), pheasants (MSD) and chickens (AAS).

Action--Acute.

Age of bird--HE occurs at 4-14 weeks; MSD, 3-8 months; and AAS in all age chickens.

Etiology--The Adenovirus is a nonenveloped, icosahedral symmetry, DNA virus with diameter of 70-90 nm.

Mode of transmission

It spreads laterally by the oral route or vertically through the cloaca.
**Clinical signs**

1. Signs include paleness, depression, mortality (to 60%), bloody diarrhea, and asphyxia (can’t breath) in AAS.

**Postmortem lesions**

1. Lesions include hemorrhages in the gut, liver, proventriculus and gizzard, and anemia.

2. An enlarged mottled spleen and free blood in the intestines can be seen and with MSD edema and congestion of lungs are also evident.

**Diagnosis**

1. Agar gel precipitin (AGP) test and ELISA can be used to measure antibody.

2. Intranuclear inclusion bodies occur in the reticuloendothelial (RE) cells of the spleen.

3. It simulates coccidiosis, VVND, bacteria, and fungi.

**Prevention**

Commercial vaccines are available for use at 4 weeks in the water for HE and MSD.

**Treatment**

None.

**Special note**

HE can cause a transient low-level immunodepression. There are three serologically distinct avian adenoviruses, which can be differentiated by the AGP test. Serotype I viruses cause IBHV, quail bronchitis, HE and MSDV. Serotype I produces AAS and serotype III virus produces egg drop syndrome.

**Egg drop syndrome (EDS 76)**

**Species of bird**—Laying chickens, ducks, guinea fowl.

**Action**—Acute to chronic.

**Age of bird**—Adults.
**Etiology**--Avian adenovirus is 70-75 nm in length.

**Mode of transmission**

1. It can spread vertically through the embryonated egg, by fecal-oral routes, and by contaminated water.

2. Wild ducks, geese, gulls, owls, storks, swans, and egrets can transmit the virus mechanically.

**Clinical signs**

(There is a 7-9 day incubation period)

1. Signs include loss of color in pigmented eggs, thin-shelled, soft-shelled or shell-less eggs and a reduction in production up to 40%.

2. Watery albumin and a reduction in the size of the egg, inappetence, dullness, and diarrhea may occur.

**Postmortem lesions**

1. Lesions include inactive ovaries and atrophied oviducts, uterine edema, exudate in the shell gland, flaccid ovules, and a mild splenomegaly (enlarged spleen).

**Diagnosis**

1. Clinical signs, gross lesions, and inclusion bodies in the epithelial cells of the shell gland are diagnostic.

2. Isolation and identification of hemagglutination virus in embryonated duck eggs is also important.

3. Immunofluorescent staining of fluids with specific conjugated antisera, and the Hemagglutination-inhibition test and ELISA to check for antibody in blood are also helpful.

4. It simulates IB and mycoplasma.

**Prevention**

1. Biosecurity is important.

2. A killed vaccine can be given during the pullet-rearing phase to help prevent the disease.

**Treatment**

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Special note

It occurs world-wide, however, the disease has not been reported in chicks in the US. The virus has been isolated from migratory ducks in the US.

Duck viral enteritis (DVE) (duck plague)

Species of bird--Ducks, geese, swans.

Action--Acute.

Age of bird--All.

Etiology--Herpes virus.

Mode of transmission

1. It is transmitted directly from bird to bird and by bloodsucking arthropods.

2. Indirectly spread by contact with the contaminated environment or water.

Clinical signs

1. Signs include sudden, high persistent mortality, and a drop in egg production (25-40%).

2. Photophobia can occur as half-closed, pasted eyelids. Inappetence, extreme thirst, droopiness, ataxia, ruffled feathers, nasal discharge, soiled vents, and watery diarrhea may also be evident.

3. Birds may be unable to stand, and have droopy outstretched wings, and head down.

4. Tremors of the head, neck and body, loss of weight, blue beaks, blood stained vent, and mortality (5-100%) may be seen.

Postmortem lesions

1. Lesions include vascular damage (tissue hemorrhages) and free blood in the body cavity.

2. Eruptions in the mouth (figure 12.49) and GI tract and degeneration of lymphoid organs (thymus and bursa) may be seen.

Figure 12.48. Enlarged necrotic ceca (left)
Figure 12.49 Diptherite oral cavity (right)

3. Petechial hemorrhage may be seen on the myocardium, on liver, pancreas, intestine, lungs, and kidney.

4. Surface hemorrhages and yellow-white crusty plaques on the mucosa of the oral cavity, esophagus, ceca, and cloaca may be evident (figure 12.48).

**Diagnosis**

1. Clinical signs and gross lesions in all age ducks are diagnostic.

2. It simulates influenza, duck viral hepatitis, and fowl cholera.

**Prevention**

An inactivated vaccine for breeders and a live attenuated vaccine by injection for ducklings can be used.

**Treatment**

None.

**Special note**

It is immunosuppressive.

**Duck virus hepatitis (DVH)**

**Species of bird**--Ducklings.
**Action**--Acute.

**Age of bird**--1-4 weeks of age.

**Etiology**--3 virus types.

1. DVH type 1--picornavirus, 20-40 nM-heat stable.
2. DVH type 2--astrovirus, 28-30 nM.
3. DVH type 3--picornavirus unrelated to DVH-1.

**Mode of transmission**

It spreads via airborne and fecal-oral routes.

**Clinical signs**

(There is a 3-4 day incubation period.)

1. In DVH-1 birds fall on their sides and kick spasmodically with rapid death. Morbidity can reach 100% and mortality can reach 95%.
2. In DVH-2 convulsions, opisthotonos (arching of head and neck), and death are evident.
3. In DVH-3 outstretched legs and opisthotonosis occurs.

**Postmortem lesions**

1. DVH-1 produces enlarged liver with punctuate ecchymotic (paint brush) hemorrhages, an enlarged, mottled spleen, and the kidney may be swollen and congested.
2. DVH-2 produces a liver which is pink and has hemorrhages, spleen enlarged, kidneys enlarged, and small hemorrhages in the intestinal wall.
3. DVH-3 lesions are similar to DVH-1.

**Diagnosis**

1. A rapid onset of clinical signs in ducklings with liver lesions is characteristic.
2. Isolation and identification of organism in duck embryonated eggs, and staining of virus with specific fluorescent conjugated antisera is diagnostic.
3. It simulates *Chlamydia psittaci*, Salmonellosis, aflatoxicosis, and avian influenza.
**Prevention**

A live attenuated vaccine is available for use in breeders or ducklings by drinking water or by injection against the DVH-1 type only.

**Treatment**

None.

**Rotavirus infections**

**Species of bird**--Chickens, turkeys, ducks, pheasants, guinea fowl, and pigeons.

**Action**--Acute to chronic.

**Age of bird**--All.

**Etiology**--Rotavirus is a 70 nM-double stranded RNA with 11 segments.

**Mode of Transmission**

Virus is spread by fecal-oral route.

**Clinical Disease**

(There is a 2-5 day incubation period.)

1. Subclinical (no signs) to severe diarrhea may occur.

2. Other signs include dehydration, poor weight gain, increased mortality (4-7%), restlessness, litter eating, and watery droppings.

**Postmortem lesions**

1. Lesions include abnormal amounts of fluid and gas in the intestinal tract, dehydration, Inflammed vents, vent picking, and litter in the gizzard.

**Diagnosis**

1. Diagnosis is by detection of the virus in the feces using electron microscope, and isolation of the virus in cell culture and staining with specific fluorescent conjugated antisera.
2. It simulates coccidiosis, pale bird syndrome, toxic enteritis, astrovirus, coronavirus, and enteric bacteria.

**Prevention**

1. Biosecurity is important.
2. No vaccine is available.

**Treatment**

Vitamins, minerals, and electrolytes for treating fluid loss are helpful.

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**Chicken anemia virus (CAV) Blue wing disease**

It is called blue wing disease because birds may show a bluish discoloration at the tips of the wings.

**Species of bird**—Chickens.

**Action**—Acute to chronic.

**Age of bird**—Young, 12-28 days.

**Etiology**—Circo virus is 19-24 nm in diameter, isometric symmetry, which contains a single strand of DNA.

**Mode of transmission**

Virus spread horizontally and vertically.

**Clinical signs**

(There is an 8-10 day incubation period.)

1. Signs include anemia, weight depression and up to 30% mortality.

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**Figure 12.50. Wing necrosis with CAV**
**Postmortem lesions**

1. Lesions include thymic atrophy, the bone marrow is yellow or pink (figure 12.51), bursal atrophy, swelling and mottling of the liver, and hemorrhages in the proventriculus and muscles.

2. Bluish discoloration of the wing (Blue wing disease) can occur (figure 12.52).

![Figure 12.51. Pale blood (upper)  Figure 12.52. Aplastic anemia in the bone marrow (lower)](image)

**Diagnosis**

1. An aplastic anemia (figure 12.51) with gross lesions (swollen liver) is characteristic. Isolation of the virus from liver in MSB1 cells, identification with fluorescent conjugated antisera, and PCR test using thymic tissue is diagnostic.

2. Serologic tests include virus neutralization in MSB1 cells and ELISA.

3. It simulates mycotoxins, IBD, sulfonamide toxicity, and osteopetrosis.

**Prevention**

1. Chicks derived from immune (exposed) parent stock are immune.

2. Live vaccine available for breeder pullets given during the rearing period.

**Treatment**

None.

**Special note**

It is immunosuppressive. Ninety % of parent stock in the United States have antibody against CAV and will produce immune progeny. Combination of CAV and IBDV causes severe immunosuppression. Vaccination controls clinical disease, but subclinical immunosuppressive disease can still exit.
Avian pneumovirus infections (swollen head syndrome of chickens and turkeys)

**Species of bird**--Chickens and turkeys.

**Action**--Chronic.

**Age of bird**--All.

**Etiology**--Paramyxovirus family (pneumovirus) is a non-hemagglutinating, nonsegmented, enveloped single stranded RNA virus with helical capsid symmetry (80-200 nm).

**Mode of transmission**

1. Virus spreads by airborne and mechanical routes (feed, water, and equipment).

**Clinical signs**

1. Signs include snicking, rales, sneezing, nasal discharge, foamy conjunctivitis, and swelling of the infraorbital sinuses.

2. Submandibular edema, mortality 0-10%, torticollis, and cerebral disorientation may occur.

3. A drop in egg production and morbidity approaching 100% of the flock may also be seen.

**Postmortem lesions**

Yellow edema and/or hemorrhaging in nasal turbinates, trachea, and subcutaneous layer of skin around head can be evident.

**Diagnosis**

1. Viral isolation from the trachea, lungs or nasal exudate in embryonating turkey eggs, or chicken organ cultures is diagnostic.

2. Serologic tests include virus neutralization, ELISA, or Agar gel precipitation.

3. It simulates many respiratory diseases including influenza, bronchitis, mycoplasma, *E. coli*, and infectious coryza.

**Prevention**

Live vaccines given by injection for broilers at 1 day of age and/or killed vaccines for turkeys of breeders are available in Europe, Middle East, and Latin America.
**Treatment**

1. Antibiotics for secondary invaders, fresh air in the house, and reduce stocking density are helpful.

**Special note**

Immunosuppression plays a role, and *E. coli* is a common secondary invader. In the United States, Pneumovirus is recognized only as a pathogen in turkeys, whereas in Europe this virus causes disease equally in chickens and turkeys. Swollen heads are typically seen following a severe reaction to NDV-IBV vaccination in stressed immunosuppressed birds. In the United States, turkey rhinotracheitis is caused by bacteria (*Bordetella avium*).

**Turkey viral hepatitis**

**Species of birds**--Turkey.

**Action**--Acute, highly contagious, and typically subclinical.

**Age of bird**--All.

**Etiology**--Unclassified virus.

**Mode of transmission**

1. Spread by fecal-oral route and possibly vertical.

**Clinical signs**

(There is a 1-7 day incubation period.)

1. It is often subclinical.

2. Depression, sudden death, morbidity to 100%, and mortality to 25% can occur.

3. Breeder flocks can show reduced production, fertility, and hatchability.

**Postmortem lesions**

1. Lesions include catarrhal enteritis, bronchopneumonia, peritonitis, or air sacculitis.

2. Focal gray, depressed areas on the liver, and circular gray-pink areas on pancreas may be seen.

**Diagnosis**

1. Clinical signs and gross lesions in the liver and pancreas of turkeys are characteristic.
2. Isolation of the virus from the liver in the yolk sac of embryonating eggs is diagnostic. Reinfection of day-old poult to reproduce the disease is also important.

3. It simulates Histomoniasis and bacterial hepatitis.

**Prevention**

Biosecurity is important.

**Treatment**

None.

*Poult Enteritis Mortality Syndrome (PEMS)*

**Species of birds**--Turkey.

**Action**--Acute, highly contagious

**Age of bird**—young (7 to 28 days of age).

**Etiology**--Unclassified virus (possibly a reovirus, adenovirus, cornavirus, or astrovirus). Several bacteria and protozoa have also been associated with the disease.

**Mode of transmission**

1. Spread by fecal-oral route.

**Clinical signs**

(There is a 1-7 day incubation period.)

1. Depression, sudden death, and morbidity to 100% and mortality to 50% can occur (figure 12.53).

2. Dehydration, diarrhea, and stunting.

**Postmortem lesions**

1. Lesions include enteritis and peritonitis and atrophy of the bursa and thymus.

**Diagnosis**

1. Clinical signs and gross lesions in the intestine of young turkeys are characteristic.
2. Isolation of the virus from the liver in the yolk sac of embryonating eggs is helpful and re-inoculate day-old poult's to reproduce the disease.

**Prevention**

Biosecurity is important. Depopulation of infected flocks for two consecutive growing cycles is also helpful.

![Figure 12.53. Severe prostration seen with PEMS](image)

**Treatment**

None.

**Special Note**

Birds are severely immunosuppressed.
13. Fungal (mycotic) diseases

Aspergillosis (brooder pneumonia)

It has its name because the disease results in pneumonia in young chicks, which are reared under brooders.

Species of bird -- All.

Action -- Acute in young chicks and chronic in adults.

Age of bird -- Mainly seen in young birds.

Etiology -- *Aspergillus fumigatus*, *A. flavus*, and *A. niger*. Fungus has conidiophores which are smooth, colorless to light green near the vesicle. The conidophore enlarges to form a flask-shaped vesicle. Conidiophore contains globose vesicles, phialides and radiate chains of conidia. Fungi can be isolated on artificial media and are inhibited by some antibiotics.

Mode of transmission

1. It spreads by aerosol of spores, which are common in the hatchery.

2. Spreads less commonly by contaminated dust and litter in the house.

Clinical signs

1. Signs include respiratory distress (dyspnea and gasping), central nervous dysfunction (tremors, ataxia, and torticollis), somnolence (sleepy), inappetence, and emaciation (very thin).

2. Conjunctivitis, high mortality, and cloudy eyes can be seen.

Postmortem lesions

Yellowish-green or whitish, caseous (cheezy) nodules and/or green, fur-like down in mouth, palate, lungs (figure 13.0), trachea (figure 13.1), syrinx, viscera, air sacs, brain and eyes may be seen.
Diagnosis

1. Fungus can be identified microscopically (20% KOH stain) from culture or special stain of tissues (Hyphae, Mycelia, Conidiophores).

2. Isolation of culture in 48 hours on Sabouraud dextrose agar is diagnostic. Lactophenol cotton blue staining of colony to see conidophores (figure 13.2).

3. It simulates colibacillosis, MD, lymphoid leukosis, and tuberculosis.

4. Nodules in the lungs and fungal fur-like down in the air sacs are diagnostic.

Prevention

1. Hatchery sanitation includes regular fumigation of eggs, machines and air ducts and regular (monthly) plating of hatchery with media to examine for the presence of fungi.

2. Use clean dry litter and dry cups or nipples to reduce water spills.

3. Place aerosol of thiabenidazole or Clinafarm® (Schering-Plough Inc., Millsboro, Delaware) pellets in the hatchery to kill the fungus. Clinafarm can also be mixed with quaternary ammonia compounds and spread on surfaces to kill fungi or used in evaporative cooling pads to prevent fungal growth.

Treatment

1. Clinafarm® by aerosol treatment of birds.

2. Nystatin (Myco-20®), Miconazole*, Hamycin*, Amphotericin B are used on expensive birds. These drugs are too expensive for commercial poultry. Use Quats (Roccal®),
Chlorox®, and/or copper sulfate in water to help reduce spread of organism and reduce clinical signs.

**Special note**

Debilitated humans can develop a mycotic pneumonia from the organism. It is the most common mycotic infection of poultry. Young chicks are very susceptible, since the cilia in their respiratory tract (which can prevent fungal infection) do not function optimally until several weeks of age.

*Not approved for use in the United States.

![Figure 13.2. Fungal fruiting bodies](image)

**Thrush (crop mycosis) yeast infection**

**Species of bird**—All; cage birds and turkeys are more susceptible.

**Action**—Chronic.

**Age of bird**—All; young are more susceptible.

**Etiology**—*Candida albicans* is a yeast, which contains oval budding cells with septate hyphae and occasionally spherical, swollen cells with thickened membranes called chlamydospores.

**Mode of transmission**

1. It is spread via contaminated feces, liter, and dirty waterers.

**Clinical signs**
1. Signs include emaciation, stunting, listlessness, increased thirst, depression, and rough appearing feathers.

**Postmortem lesions**

1. Whitish postules or nodules in mouth, crop (figure 13.3) and esophagus, and a white towel appearance in the upper digestive tract are common.

![Figure 13.3. Whitish lesions in the upper digestive tract](image)

2. Ulceration of the mouth and esophagus, hemorrhagic mucosa in the proventriculus (figure 13.4), and focal necrosis of the liver can occur.

**Diagnosis**

1. Isolate the organism from lesions on Sabouraud's Dextrose agar. Colonies are whitish, creamy, and highly convex after 48 hours.

2. The hyphae and spores can be seen in a fresh smear (figure 13.5).

![Figure 13.4. Crop mycosis](image)  ![Figure 13.5. Yeast structural components](image)
3. Slimy white lesions in the upper digestive tract are diagnostic.

**Prevention**

1. Clean up water spills, don't give bird’s sugar water for more than 1 day, and use water sanitizers to prevent build up of yeast.

**Treatment**

1. Mycostatin, copper sulfate in water, and nystatin (50 g/ton) in the feed can treat the disease.

2. It simulates trichomoniasis, Vitamin A deficiency, Wet Pox, and T-2 toxicosis.

**Special note**

It is common in pet birds on prolonged antibiotic therapy or prolonged use of sugar water in poultry. Humans are susceptible to the infection. It is more common in backyard birds where poor sanitation is practiced. It is occasionally seen in commercial poultry.

**Histoplasmosis**

**Species of Bird**--Zoo birds, pigeons, mammals, and humans.

**Action**--Chronic.

**Etiology**--*Histoplasma capsulatum*; there are two types:

1) Spherical, minutely spring microconidia, and 2) spherical macroconidia with evenly spaced finger-like projections are seen.

**Clinical signs**

Signs include coughing, fever, inappetence, and emaciation.

**Postmortem lesions**

Nodules in respiratory tract can be seen.

**Diagnosis**
1. Culture the organism on Sabouraud's medium. Colonies appear as white to brownish after 2 weeks incubation. Under the microscope the organism appears as segmented branched hyphae which give rise to chlamydospires, often in chains with large round cells.

2. Skin test with histoplasmin antigen will yield an enlarged area after 24 hours at the site of injection in previously exposed individuals.

3. Histopathologic observations of the gross lesions will reveal yeast forms.

4. It simulates tuberculosis and aspergillosis.

**Prevention**

Sanitation and hygiene will aid in the prevention.

**Treatment**

Nystatin is an effective treatment.

**Special note**

This disease has a Public Health Significance. It can cause a chronic respiratory disease in humans, which become infected with contact of poultry litter containing the organisms. Poultry are generally resistance to this infection. It is common in pigeon houses. Areas bordering the Missouri, Ohio, and Mississippi Rivers are endemic.
14. Mycotoxins

Mycotoxins are toxic metabolic byproducts of fungal growth on grains. There are over 100 known mycotoxins. High moisture content of grains can lead to fungal growth and toxin production. Fungi can produce toxins before or after grain harvest. Drought and insect damage to grain can increase the susceptibility of grain to fungal growth.

Aflatoxicosis

Species of bird -- Ducks, turkeys, broilers, pullets, commercial layers, and broiler breeders are affected in decreasing order of susceptibility.

Action--It can be acute to chronic dependent upon dose, duration, of exposure and age of the bird. Acute disease seen in young, which are given high amounts (1,000 ppb of B1) of the toxin. Chronic disease seen in older birds older than 5 weeks, which are given low levels (ppb) for several weeks. It is the most common mycotoxin in poultry in the SE USA and corn is the most common source of the toxin.

Age of bird--All, but young are most susceptible.

Etiology--Consumption of feed containing grains (usually corn) which contain toxins causes the disease (figure 14.0). Afla comes from Aspergillus flavus ("A" for Aspergillus and "fla" for flavus). Aflatoxins are a group of related poisons produced by several fungi: Aspergillus flavus, A. parasiticus and Penicillium puberulum.

![Moldy corn](image)

Figure 14.0. Moldy corn commonly produces mycotoxins in feed

Mode of transmission

1. Consumption of high moisture grains containing toxin produces the disease.
2. Most common and toxic aflatoxin is aflatoxin B1 followed by B2, G1 and G2. Aflatoxicol, aflatoxitem, and cyclopiazonic acid are other toxic metabolites produced by these fungi on moldy corn. Soybeans are generally resistant to *A. flavus* infection.

**Clinical signs**

1. Signs include sleepiness, depression, paleness, and reduced egg production, fertility, and hatchability.

2. Depressed growth, feed conversion and increased bruising and downgrading can occur.

**Postmortem lesions**

1. Lesions include scattered hemorrhage in the muscles, skin and intestinal tract, fluid around the heart, enlarged pale kidneys, and pale, enlarged fatty livers with hemorrhage (figure 14.1) and anemia.

![Figure 14.1. Yellow fatty and hemorrhagic livers (lower) are common with aflatoxicosis.](image)

**Diagnosis**

1. The clinical history, and gross and microscopic lesions are important. Microscopically, the liver shows fatty change, swollen hepatocytes, and bile duct hyperplasia.

2. Use an ultra violet (UV) (black) light to test corn for the presence of cogic acid (blue green fluorescence) producing fungi (figure 14.2).
3. Feed analysis using column chromatography is the only method for a definite diagnosis. Test kits are available for aflatoxin B1 using ELISA methodology or minicolumns.

4. It simulates infectious bursal disease (IBD), ochratoxin, fatty liver syndrome, and malabsorption syndrome.

**Prevention**

1. Stop moldy feed with a mold inhibitor such as propionic acid (Mold Curb®, Mold Ban®, and Antitox®).

2. Quality control of feeds is necessary. Check grains for B1 (especially southeastern U.S. corn with greater than 16% moisture).

3. Dilute contaminated feed with noncontaminated grain or treat with ammonia, which inactivates the toxin.

4. Feed low moisture corn (below 14%). Do not add moldy corn to the finished feed. Feed contaminated feed to older pullets, which are less susceptible.

5. Two feed bins at farm will reduce grain and feed storage time, which reduces fungal growth and toxin formation.

6. Aluminosilicates in the feed such as zeolites will bind and inactive aflatoxin.

7. MTB-110 in the feed binds many common mycotoxins. This compound contains a glucoman-based ingredient from Alltech, Inc.

8. Pellet feed to kill the fungi.

9. Gentian Violet* kills fungi and binds aflatoxin.
**Treatment**

Increasing the protein content of feed by 1%, increasing vitamin and mineral content of feed, and adding Gentian violet to feed have a sparing effect on aflatoxin-induced disease.

**Special note**

The longer grain is stored, especially under warm, moist conditions, the more fungi grow and can produce toxins. It is a feed storage problem. Aflatoxins are immunodepressive and carcinogenic (cause cancer). It is common in southeastern US grain (corn) and tropical areas around the world. One ppm of AF B1 can produce morbidity and mortality; 500-1,000 ppb can reduce weight gain and feed efficiency; and 200-500 ppb can produce immune depression. Hogs and horses are very susceptible to aflatoxin. Humans may be exposed to aflatoxins through peanuts.

*Not approved for use in U.S. feeds.*

**Ochratoxin**

**Species of Bird**--Broilers and turkeys.

**Action**--Acute to chronic action depending on the level of toxin (high levels produce acute disease, low--chronic); the duration of exposure (long-term--chronic, short-term--acute); and age of bird (young birds have acute, old--chronic).

**Age of bird**--All ages with young more susceptible.

**Etiology**--Consumption of feed containing grain (corn, wheat, and barley) contaminated with toxin produced by Aspergillus ochraceous or Penicilllin viridicatum.

**Mode of transmission**

Consumption of feed containing toxin causes the disease.

**Clinical signs**

1. Signs include diarrhea, depressed growth, reduced pigmentation, soiled eggs, tremors, hypotension, and bradycardia (slowness of the heart rate).

2. Reduced egg production, fertility and hatchability, egg size, and shell quality can occur.
Postmortem lesions

1. Fatty liver with hemorrhage, enlarged pale kidneys with urates (figure 14.3), heart necrosis, and urate deposits on the liver, spleen, and pericardium can be seen.

![Image](image1.png)

Figure 14.3. Enlarged pale kidneys common with ochratoxicosis.

Diagnosis

1. The clinical signs and lesions are helpful, but the only definitive diagnosis is from feed analysis for the presence of toxin.

2. It simulates aflatoxicosis, visceral gout, infectious bronchitis (IB), IBD, citrinin toxicity, and malabsorption syndrome.

Prevention

1. Dry feed ingredients thoroughly and sanitize feed bin and feeders with Chlorox®.

2. Use mold inhibitors in the feed, don't use moldy grains, and reduce feed storage time to prevent the growth of fungi and toxin production.

Treatment

The same treatment as for aflatoxin including increase the protein, vitamin, mineral, and energy content of the diet.

Special note

It is a feed storage problem. Ochratoxin is less common than aflatoxin, but is more toxic (ppb) and is immunodepressive. It affects both humoral and cellular immunity.
**Fusarochromanone**

**Species of bird**--All domestic poultry (turkeys, broilers, ducks).

**Action**--Chronic.

**Age of bird**--Young growing birds.

**Etiology**--Consumption of feed containing grain contaminated with toxin produced by *Fusarium moniliforme* and *F. roseum*.

**Mode of transmission**

Consumption of contaminated grain.

**Clinical signs**

Signs include lameness (dyschondroplasia) and the long bones contain a mass of abnormal cartilage, which results in the bending and swelling of the bone. Diarrhea may also be seen.

**Postmortem lesions**

An abnormal plug of cartilage in the head of the long bone causes bending in the bone. An unmineralized, avascular cartilage material is present. Enteritis can be evident.

**Diagnosis**

1. Gross and microscopic lesions with analysis of the feed for the toxin.

2. It simulates other leg weakness (perosis, rickets, tibial dyschondroplasia, etc.).

**Prevention**

The prevention is the same as other mycotoxins.

**Treatment**

The treatment is the same as other mycotoxins.
Trichothecenes

Species of bird--Chickens, turkeys, layers and breeders.

Action--Acute to chronic depending on dosage of toxin (higher--acute), age of bird (young birds have acute, older have chronic), and duration of exposure (longer periods cause chronic toxicosis).

Age of bird--All.

Etiology--Consumption of feed containing grains infected with fungi producing trichothecenes toxins cause the disease. The following trichothecenes: T-2, diacetoxyescirphenol (DAS) and deoxynivalenal (DON or Vomitoxin) can be produced by Fusarium trichinctum, F. calonectria, F. gibberella, F. cephalosporium, and F. trichoderma.

Mode of transmission

1. Feed contaminated with trichothecenes containing toxin, which are very caustic.

2. It can occur in corn, sorghum, barley, safflower seed, oats, and brewers grain.

Clinical signs

1. Signs include off feed, impaired growth, uneven and poorly formed feathers, and emesis (vomiting).

2. Thin shelled eggs, reduced egg production, swelling of the face, caustic injury to skin, cyanotic (blue-colored) combs and wattles, seizures, and tremors may be seen.

Figure 14.4. Caustic injury to the mouth

Figure 14.5. Caustic injury on skin
**Postmortem lesions**

1. Whitish to yellow, focal nodules in base of mouth, near the salivary ducts and tongue can occur (figure 14.4).

2. An inflamed GI tract (figure 14.7), atrophy of bursa and thymus, necrosis of gizzard and proventriculus (figure 14.6), dermatitis on the toes (figure 14.5), pale or yellow bone marrow, yellow hemorrhagic liver, and gout can be seen.

**Diagnosis**

1. Feed analysis for toxins and feed refusal in many farm animals is diagnostic (figure 14.8).

2. It simulates wet pox, vitamin A deficiency, thrush, trichomoniasis, ochra and aflatoxicosis, IBD, and visceral gout.

**Prevention**

1. The prevention is the same as for other mycotoxins including; mold inhibitors, dry grain, and don't use moldy grains.

**Treatment**

The treatment is the same as for other mycotoxins.
Special note

It is more common in northwestern U.S. grain and is immunodepressive. T-2 toxin is the most common and pathogenic of these compounds.
15. Protozoal diseases

Coccidiosis (coci)

(Note: 9 species of *Eimeria* occur in the chicken and 6 are important; 7 species occur in the turkey and 4 are important.)

**Species of bird**—All. However, all avian *Eimeria*, except for dispersa, are species specific and tissue trophic.

**Action**—Acute to chronic.

**Age of bird**—Any.

**Etiology**—*Eimeria*; oocyst contains 4 sporocysts. Each sporocyst contains 2 sporozoites. The organism undergoes two rounds of asexual reproduction (schizogony) and 1 round of sexual reproduction (gametogony).

**Mode of transmission**

1. Direct transmission by consumption of sporulated oocysts in the fecal material.

2. Only birds reared on moist, contaminated, used litter have access to sporulated oocysts.

3. Oocysts need 48 hours to sporulate (sporogony). Oxygen and moisture are needed for sporulation.

**Clinical signs**

1. Signs include watery and/or bloody droppings, mortality (1-50%), and morbidity (0-100%).

2. Culls appear as pale birds with anemia, depression, poor weight gain and feed conversion, and a drop in egg production.

**Postmortem lesions**

Enteritis characteristic of *Eimeria* species is seen. The intestinal tract can be enlarged and have necrotic and/or hemorrhagic foci, undigested feed, and gas.

**Diagnosis**

1. Intestinal scraping examined for oocysts. The site and degree of lesions, size and shape of oocysts, and schizonts are used to speciate *Eimeria*.

2. It simulates infectious bursal disease (IBD) or bacterial enteritis in chickens and hemorrhagic enteritis in turkeys.
**Prevention**

1. Coccidiostats are continuously fed to broilers in the feed. Drug resistance of certain species against certain drugs is common.

2. CocciVac® vaccine (containing oocysts of *E. acervulina*, *E. maxima*, *E. mivati*, and *E. tenella*, can be given to pullet replacements or turkeys (CocciVac T®) in the water at 5-7 days. It is produced by Schering Plough Animal Health, Inc., Millsboro, Del. Immunocox® also from Schering contains oocysts of *E. acervulina*, *E. maxima*, *E. necatrix*, and *E. tenella*. It is produced in a gel form and is consumed by day old chicks. Advent® is a new live oocysts vaccine from Novus, Inc., St. Louis, Mo. for spray cabinets.

3. CocciVac® can be dropped in the eye at 1 day to broilers by spray machine or sprayed on feed at 3-5 days to roasters or males reared to 8 weeks for de-boning. CocciVac® will immunize birds within 2 weeks, but requires sporulation on warm moist litter. It will cause some weight depression.

4. Rear birds on wire to prevent exposure to sporulated oocysts, keep litter dry, change litter, and/or use poultry litter treatment.

5. Coccidiostats can be given in the feed for the first 8 weeks to pullets.

   **a. chickens**

   1. Nicarbazin (Merck) 0.0125% (cold weather product)
   2. Amprolium (Merck) 0.025% (used more for treatment)
   3. Zoalene (Fort Dodge) 0.004-0.0125%
   4. Clopidal (Al Lab) 0.0125-0.025%
   5. Decoquinate (Rhone-Poulenc) 0.003%
   6. Monensin (Elanco) 0.01-0.012%
   7. Robenidine (Cyanamid) 0.0033%
   8. Lasalocid (Hoffman-LaRoche) 0.0075%
   9. Salinomycin (AgriBio) 0.004-0.0066% (no withdrawal period)
   10. Halofuginone (Hoechst-Roussel) 3 ppm
   11. Narasin (Elanco) 54-72 g/T
12.  Madurimicin (Cyanamide) 5-6 ppm
13.  Narasin and nicarbazin (Elanco) 54-90 g/T
14.  Semduramicin (Pfizer) 25 ppm

**b. turkeys**

1.  Monensin
2.  Butynorate (Fort Dodge) 0.375%

**Treatment**

1.  SQ® Sulfadiazine (feed) (0.05%) or Amprol® Plus (0.024%) (water).
2.  Sulfadimethoxine and Ormetoprin (water).
3.  Sulfadimethoxine (0.1%) (water).
4.  Sulfachloropyrazine monohydrate (0.03%) (water).

**Special note**

It is the most commonly diagnosed and most costly disease of chickens. It is less common and less severe in turkeys, but occurs more frequently in confinement rearing than with turkeys reared on range.

**Coccidiosis**

**Life cycle**

1.  Oocysts are thick-walled zygotes shed in the feces. Sporulation of the oocyst occurs after 48 hours. Infective oocysts contain 4 sporocysts, which in turn contain 2 sporozoites.
2.  Ingested oocyst is crushed in the gizzard and sporozoites are released by action of enzymes in small intestine. Sporozoites enter epithelial cells where development begins.
3.  The life cycle is self-limiting; once oocysts are produced, they pass out feces and must be ingested to start cycle again.
4.  Two generations of asexual development called schizogony or morogony, lead to a sexual phase. The asexual development proceeds by binary fission resulting in massive amounts of schizonts (first-generation and second-generation), which eventually are released as merozoites causing rupturing of the cell and tissue damage.
5. During sexual development microgametes seek out and unite with macrogametes. The resulting zygote matures into an oocyst, which is released from the intestinal mucosa and is shed in the feces.

6. The entire process takes 4-7 days depending on the species of *Eimeria*. The site of the intestinal tract is also species specific.

7. Chicken coccidian that are deep tissue invaders, such as *E. maxima*, *E. necatrix*, and *E. tenella*, cause severe necrosis, hemorrhage of the intestinal mucosa, and bloody diarrhea and may result in death.

8. All avian *Eimeria* with the exception of *E. dispersa* infect only one species. *E. dispersa* may infect and cause disease in turkeys, quail, and pheasants.

9. Control--anticoccidial drugs are either static (interrupt life cycle) or cidal (cause death of the parasite). Some drugs are more effective against certain species. Drug resistance, passed on to the progeny through sexual reproduction, is common. *Eimeria* species develop resistance to certain drugs at different rates. Altering drugs in the same growout (shuttling) or after several growouts (rotation) is a common practice to control drug resistance. The drugs are synthetic, like inophorous antibiotics, or chemical. Common inophorous antibiotics include Coban (monsen), Avatec, Aviax, Sacox (semduramicin), and Biocox (salinomycin). Chemical drugs include Robenz, Nicarb, Stenorl, and Clinacox. Shuttle programs should be from chemical to synthetic or vice versa, not chemical to chemical or synthetic to synthetic.

10. Certain drugs have different withdrawal periods. Drugs, which allow some cycling of the parasite, are commonly used in pullets to allow immunity to develop in 4-6 weeks. This allows the drug to be withdrawn after immunity is developed.

11. Vaccination--the use of vaccines by eye drop at 1 day or in the drinking water or sprayed on the feed during the first week of age is a system of controlled exposure. Sporulated oocysts containing drug sensitive strains of important *Eimeria* are in the vaccine. These organisms are allowed to complete their life cycle. The litter is moistened from 5-7 days after vaccination to allow for sporulation of shed oocysts and reinfection with the parasite. After 3 complete life cycles (3 weeks), the bird is usually solidly immune to the parasite. If the litter is too moist, a second round of infection may cause severe diarrhea and paleness. If this occurs, birds should be given Amprolium and/or vitamins and minerals in the water. Vaccination is normally used in turkeys, pullets, roasters, and broilers, which have at least 8 weeks of rearing. This gives adequate time for the birds to make up for any temporary depression in growth, which may occur after vaccination. Vaccination by water is less consistent, because the oocysts tend to settle out and distribution is not uniform. In Europe, a vaccine made from attenuated (less pathogenic *Eimeria*) is available to be given in the feed. Vaccination is often done in broilers in the spring or summer, when the incidence of coccidiosis is less. Alternating vaccination with
anticoccidial drugs is also common after several broiler growouts to prevent drug resistance.

**Coccidia species of chickens**

1. *Eimeria acervulina*—light infections produce whitish round lesions sometimes in ladder-like streaks (figure 15.0). Heavy infections produce plaques coalescing, thickened intestinal wall in the duodenum. The oocysts are small, ovoid and schizonts are 10.3 microns in size. The parasite locates in the epithelial tissues. It is very common in poultry houses.

![Figure 15.0. E. acervulina necrotic foci in duodenum](image)

2. *E. brunetti*—produces coagulation necrosis, and mucoid, bloody enteritis in the lower intestine. Oocysts are large ovoid and schizonts 30 microns. Second-generation schizonts are subepithelial. It is a less common species of *Eimeria*.

3. *E. maxima*—produces thickened walls, mucoid, blood-tinged exudate and petechial in the mid-intestine (figure 15.1). The oocysts are large, ovoid and quite large, and schizonts are 9.4 microns. Gametocytes are subepithelial. It is one of the more common and pathogenic of chicken coccidia.

![Figure 15.1. Mucoid, blood-tinged exudate and petechial in the mid-intestine.](image)
4. *E. mitis*—produces no discrete lesions in the intestinal tract and some mucoid exudate in the lower intestine. Oocysts are tiny and sub-spherical and schizonts are 15 microns. The parasite occurs in epithelial cells. This species plays a minor role in causing disease in chickens.

5. *E. mivati*—light infections produce rounded plaques of oocysts in the upper intestinal tract, whereas heavy infections produce thickened walls with coalescing plaques. Oocysts are small ellipsoid to broad ovoid (figures 15.2 and 15.3). The parasites are in the epithelial cells.

6. *E. necatrix*—produces ballooning, white spots (schizonts), petechia, and mucoid blood-filled exudate in the mid-intestinal tract (figures 15.4 and 15.5). The oocysts are large and oblong ovoid. Second-generation schizonts are subepithelial. It often develops resistance to commonly used drugs.
Figures 15.4 and 15.5. *E. necatrix* may cause bloody mucoid lesions in midintestine

7. *E. praecox*—produces mucoid exudate in the upper digestive tract or no lesions. It produces large ovoid oocysts and schizonts in the epithelium. It is of little economic importance.

8. *E. tenella*—initially produces hemorrhage in the lumen of the cecum. Later it produces thickening and whitish mucosa and/or cores of clotted blood and necrotic mucosa (figures 15.6 and 15.7). The oocysts are large and ovoid. Second-generation schizonts are subepithelial. It is one of the most pathogenic and common of the chicken coccidia (figure 15.8).

Figures 15.6 and 15.7. Enlarged bloody ceca seen with *E. tenella* in chickens

9. *E. hagani*—this species is of doubtful validity. It produces pinhead hemorrhages in the duodenum. The oocysts are broadly ovoid and schizonts are epithelial.
Figure 15.8. Clinically ill bird

**Coccidia species of turkeys**

1. *E. adenoeides*—produces liquid feces with mucous and flecks of blood and loose whitish core in the cecum and large intestine. It produces large ellipsoidal oocysts. The species is highly pathogenic and very common.

2. *E. dispersa*—produces a cream-colored serosal surface, dilation of intestine, and yellowish mucoid feces. It produces medium-sized broadly oval oocysts. It is only mildly pathogenic.

3. *E. gallopavonis*—produces edema, ulceration of mucosal ileum, yellow exudate, and flecks of blood in feces. Oocysts are large and ellipsoidal. It is highly pathogenic and common in turkey houses.

4. *E. innocua*—apathogenic; oocysts are small and sup-spherical.

5. *E. meleagridis*—produces cream-colored ceca, formation of caseous plug and a few petechial hemorrhages. Oocysts are small and ovoid. It is not very pathogenic and only of minor importance.

6. *E. meleagrimitis*—produces spotty congestion and petechiae from duodenum to ileum, and dilation of jejunum and casts. Oocysts are small and ovoid. It is highly pathogenic.

8. *E. subrotunda*—apathogenic; oocysts are small and sub-spherical.
**Enterohepatitis (blackhead) histomonosis**

It is called blackhead, because affected birds have a dark discolored head.

**Species of bird**--Turkeys and chickens (pullet replacements and broilers), quail, pheasants, grouse, chukar partridges, guinea fowl.

**Action**--Acute to chronic.

**Age of bird**--Young.

**Etiology**--*Histomonas meleagridis* is a highly pleomorphic amoeboid protozoa with a stout flagellum and pseudopodia.

**Mode of transmission**

Chicken cecal worm (*Heterakis gallinae*) engulfs and packages *Histomonas* oocysts in its egg. Earthworm also consumes *Histomonas* oocysts. Birds become infected by eating cecal or earthworms, or ceca worm eggs, which contains oocysts. Birds may also become infected by cloacal drinking, direct aspiration of the oocysts by the vent lips, from contaminated litter. Oocysts can also develop (sporulate) in worms and histomonads can enter the tissues of the worm.

**Clinical Signs**

(It has a 7-12 day incubation period)

1. Signs include drowsiness, dropping of wings, sulfur (yellow) colored diarrhea, stilted gait, closed eyes, head down, and anorexia in turkeys.
2. High mortality to 100% in turkeys, morbidity to 100%, a cyanotic head, and bloody cecal diarrhea in chickens can be seen.

3. Mostly commonly seen between 3 to 6 weeks of age.

**Postmortem lesions**

1. The ceca have an ulcerated (cheesy core) or hemorrhagic exudate (figure 15.11).

2. Crater-like liver lesions (bulls-eye) and an enlarged green colored liver can also be seen.

![Figure 15.11. Liver and cecal lesions seen in blackhead in turkeys.](image)

**Diagnosis**

1. One must eliminate coccidiosis from the diagnosis.

2. It simulates cecal coccidiosis.

3. Clinical signs, gross lesions (intestine and liver lesions), and histopathologic observations of (diarrhea). Life cycle stages near the lesions are diagnostic.

**Prevention**

1. Control helminths (worms) by changing the litter, using poultry litter treatments, and/or antihelmintic drugs in the feed or water.

2. Raise chickens and turkeys separately to prevent introduction of cecal worm.

3. Feed Hygromix® continuously to turkeys in confined areas to control the cecal worm.

**Treatment**

Dimetridazol (Enheptin®) (0.015%) or (Emtryl®)*, Carbasone (Carbasep®) (0.025%), Ipronidazole (Ipropan®) (0.00625%), Nitarsone (0.01875%) (Histastat®) Furazolidone (Furox®)
(.011%), and Roxarsone® (3-Nitro) are effective antiprotazoal drugs. Wormers include albendazole and tetramisol.

*Removed from the approved list by the FDA.

**Special note**

It has reached near epidemic proportions of late in pullet (chicken) replacements of late.

**Trichomoniasis**

**Species of bird**--Chickens, turkeys, pigeons (canker in squabs), doves, falcons (frounce).

**Action**--Chronic.

**Age of Bird** - Growing.

**Etiology**--*Trichomonas gallinae* is a pear-shaped protozoan containing 4 flagella.

**Mode of transmission**

1. It is transmitted by consumption of insects, contaminated feed or water. Also, spread by wild birds.

2. Crop milk can transmit the organism from adult pigeon and doves to young by regurgitation of semi-digested feed during feeding.

**Clinical signs**

1. No specific signs occur, but include lowered feed consumption, spitting up of feed, high mortality, listless, ruffled feathers, and emaciation.

2. A large crop filled with fluid, difficulty in swallowing, stretching of neck, drooling greenish to yellowish fluid, diarrhea (yellow and watery), and a drop in egg production can occur.

**Postmortem lesions**

1. Cone-shaped lesions in the upper digestive tract (mouth, esophagus and crop, pharynx, liver) proventriculus can occur (figure 15.12).

2. A buildup of caseous material may partially or totally occlude (block) the lumen of the esophagus.
Figure 15.12. Cone-shaped lesions in the upper digestive tract

**Diagnosis**

1. The clinical signs and gross lesions (mouth cankers) are characteristic.
2. A histopathologist observes trichomonads in the lesions (figure 15.13).
3. Identify the organism from a scraping for a definitive diagnosis.

4. It simulates thrush, T-2 Toxin, Wet Pox, Vitamin A deficiency, and *Chilomastix gallinarum*.

**Prevention**

Eliminate the insect or wild birds carriers.

**Treatment**
Dimetridazole (Emtryl®) (0.05%), Ipronidizole (Ipropan®), Nitrasone (Histostat-50®) are effective treatments.

**Cryptosporidiosis**

**Species of bird**--Chickens (broilers), turkey, quail, and ducks.

**Action**--Acute to chronic.

**Age of bird**--Age resistance is apparent (birds more than 3 weeks old are not susceptible).

**Etiology**

1. Chickens--*Cryptosporidium baileyi* are small coccidian parasites that do not have sporocytes surrounding the 4 sporozoites. The sporocytes lie naked within the oocyst wall. The life cycle is divided into six major developmental stages: excystation (release of infectious sporozoites, gametogony (formation of male and female gametes), fertilization (union of gametes), oocyst wall formation (to produce an environmentally resistant form), and sporogony (the formation of infective sporozoites within the oocyst wall).

   The life cycle differs from other *Eimeria* species. The intracellular stages are confined to the microvillus region of the host cell and the oocysts, which sporulate within the host cells, are infective when released in the feces. Two types of oocysts are formed, the thin-walled oocyst ruptures to release sporozoites, which can penetrate adjacent cells and reinitiate the infection. The thick-walled oocysts pass in the feces and can transmit infection to other hosts. Also, cryptosporidium can invade the mucosal epithelium of a variety of tissues including the intestines, respiratory tract, cloaca, eyelids and bursa of Fabricius. Cryptosporidium in chickens mainly causes a respiratory disease.

2. Turkeys--*Cryptosporidium baileyi* and *C. meleagridis*, in addition to causing respiratory disease, the species can invade the small intestine causing diarrhea and unthriftness.

3. Quail--both respiratory and intestinal disease occurs. The organisms have not yet been speciated.

**Clinical signs**

(It has a 3-5 day incubation period)

1. Respiratory signs include open mouth breathing, gasping, sneezing, and gurgling.

2. Intestinal signs include swelling of the sinuses, diarrhea, and poor weight gain and feed conversion.

3. Morbidity to 40% and mortality to 10% can occur with the respiratory disease.
**Postmortem lesions**

1. Respiratory--mucous in the trachea, sinuses, turbinates, thickened frothy exudate in air sacs, and pneumonia.

2. Intestinal--thickened mucosa lining of middle and lower small intestine, which becomes pale and distended with cloudy mucoid fluid and gas bubbles.

**Diagnosis**

1. Clinical signs and gross lesions, and the demonstration of life cycle stages associated with lesions in the tissues, are diagnostic.

2. It simulates coccidiosis, yeast, and viral or bacterial respiratory disease.

**Prevention**

1. Organisms are resistant to most chemical disinfectants.

2. No known treatment exists for controlling this organism.

3. It is common in immunodepressed birds and often seen together with IBD and/or reovirus infections.

4. Causes diarrhea in humans through consumption of contaminated water.

**Leucocytozoonosis** (Also called avian malaria, because it is transmitted by the bite of an insect.)

**Species of bird**--Turkeys, chickens, ducks, geese, pheasants, ruffled grouse.

**Action**--Acute.

**Age of bird**--All, but young is most susceptible.

**Etiology**--Leucocytozoan is an intracellular protozoan transmitted by the bite of invertebrate host. Life cycle includes sporogony and schizogony (merogony) in the tissue cells and gametogony in erythrocytes or leukocytes in the insects. Elongated gametocytes with pale cytoplasmic horns exist in erythrocytes. Gametes are found only in leucocytes. Intracellular schizogonous forms are found in the liver. *L. simondi* is found in ducks and geese, and *L. smithi* is found in turkeys.

**Mode of transmission**

1. It is transmitted by a black fly bite. These flies are common in swampy, low-lying costal areas of the Southern US.
2. Sporozoites are in the salivary glands of the black fly.

3. Wild birds and insects are a reservoir for infection.

**Clinical signs**

1. Signs include depression, somnolence, off feed, muscular incoordination, high mortality, anemia, increased thirst, reduced mating, and respiratory distress.

2. Vomiting, decreased egg production, egg weight and hatchability, and green diarrhea may be seen.

**Postmortem lesions**

1. Hemorrhagic liver and enlargement of liver and spleen, congestion of the lungs, spleen, and small intestine can occur.

2. Fluid can occur in the body cavity, and the blood is thin and clotting retarded.

**Diagnosis**

1. Laboratory blood and tissue smears are examined for the presence of parasite (figures 15.14 and 15.15).

 Figures 15.14 and 15.15. Leucocytozoan (dark crescent shaped) parasite in blood.

2. Wright's Giemsa Stain of RBC's and lymphocytes show various life-cycle stages.

**Prevention**

1. Control flies with Carbamate (Celatom®) granules distributed by large scale aerial or treatment of grounds.

2. Eliminate carriers by spraying repellent within the houses to discourage entrance of flies.
3. Clopidol fed continuously at 0.0125-0.0250% also reduces some infection.

**Treatment**

1. Clopidol (Coyden\textsuperscript{®}), Ipropan\textsuperscript{®}, Emtryl\textsuperscript{®} and Pyrimethamine (1 ppm) and sulfadimethoxine (10 ppm) are effective treatments.

**Special note**

It is immunosuppressive and common in turkeys on range in the southeastern United States.
16. Toxicity

Toxins are poisons produced by a variety of substances. Some toxins are beneficial nutrients and/or drugs at selected use levels, however, when they are given in excessive levels they are poisonous.

Salt (NaCl) toxicity

All higher forms of life require salt to survive.

Species of bird--All.

Action--Acute to chronic.

Age of bird - All, usually young.

Etiology--Consumption of feed or water with too high a level of NaCl. Finished feed normally contains 0.85% after salt is added. City drinking water contains 0.12% salt. This equals 8500ppm for feed and 1200 ppm for water.

Mode of transmission

1. Ingestion of food or water with high salt. Above 1% salt in the diet, causes clinical disease and above 10% results in feed refusal.

2. Some waterfowl have salt glands to excrete excess Na+.

Clinical signs

1. Signs include increased thirst, dehydration, crop filled with water, dyspnea, depression, watery diarrhea (squirts), stunting, off feed, nervous signs, and rough dirty, wet feathers or down.

Postmortem lesions

1. Lesions include ascities (water belly) (figure 16.0), excess fluid in lungs, atrophied liver, enteritis, hydropericardium (figure 16.1), right side cardiac (biventricular) hypertrophy, nephritis, cystic dilation of seminiferous tubules and the gonads, and myocardial and skeletal hemorrhage.
**Diagnosis**

1. Diagnosis is by feed analysis. You can taste the salt in the feed or water (over 2%).
2. Occurs after a new feed shipment in multiple farms.
3. Clinical signs and gross lesions are suggestive of salt toxicity.
4. Ultrastructure changes in the heart muscle include myofibrillar disarrangement and disruption of intercalated discs.
5. It simulates crotalaria toxicity and ascites.

**Prevention**

Spot check of feed or water will help prevent this disease. It may require filtration of well water.

**Treatment**

Fresh feed or water will stop the disease and reverse clinical signs within 24 hours.

**Special note**

It is a feed milling error or drinking water from well containing to high a level of minerals. The normal salt level is 0.85% of diet.
**Crotalaria toxicity**

Crotalaria is a toxic weed found in the southeastern US.

**Species of bird**—All.

**Action**—Acute to chronic.

**Age of bird**—All.

**Etiology**—Ingestion of food with *Crotalaria spectabilis* (seed, leaf or stems).

![Figure 16.2. Water belly](image1) ![Figure 16.3. Crotalaria weed seeds.](image2)

**Mode of transmission**

Ingestion of food with Crotalaria (figure 16.3) plant matter (above 0.3% of diet containing alkaloid toxin).

**Clinical signs**

1. Signs include increased thirst, dull, inactive, watery diarrhea, bright yellow-green urates, off feed, and watery discharge from nose and mouth.

**Postmortem lesions**

1. Lesions include atrophied liver, ascites (figure 16.2), subcutaneous edema, hydropericardium, lung edema, nephritis, hepatitis, and myocardial and skeletal hemorrhage.

**Diagnosis**

1. Diagnosis is by feed analysis. You can see black mitten-shaped seeds in feed.
2. Clinical signs, gross and microscopic lesions are helpful.

3. Microscopic examination of the liver reveals bile duct hyperplasia.

4. It simulates salt toxicity, ascites due to metabolic causes, aflatoxicosis, and vitamin E or selenium deficiency (exudative diathesis). The seeds are confused with soybean hulls.

**Prevention**

Spot check of feed.

**Treatment**

Fresh feed.

**Special note**

It is highly toxic to swine and cattle. The weed is common in soybean fields in southeast U.S. and is picked up by mechanical pickers. Soybeans are downgraded if crotalaria are present. Crotalaria can be separated from soybeans by a screening process, but this adds to cost of processing.

**Ionophore toxicity**

**Species of bird**--All; turkeys are most sensitive.

**Action**--Acute.

**Age of bird**--All; however, adults are more susceptible.

**Etiology**--Ionophore antibiotics are common anticoccidial and antibacterial feed additives. These drugs move alkali metallic cations, such as Na+, across cell membranes.

**Mode of transmission**

Ingestion of food with high levels of these compounds. Toxic levels cause potassium to leave and calcium to enter the cells, particularly myocytes, resulting in cell death.

**Clinical signs**

1. Signs include birds down on their sides with partial paralysis, and legs extended, anorexia, weakness, and drop in egg production, dyspnea, and dehydration (figure 16.3).

2. Mortality is variable, but may exceed 70%.
Figure 16.3. Paralysis due to ionophore toxicity

**Postmortem lesions**

1. Lesions include opaque fibrin plaques on the epicardium, hemorrhage in coronary fat, and decreased liver weight.

2. Pallor and atrophy of muscle fibers of the legs and back can also be seen.

**Diagnosis**

1. Diagnosis is by feed analysis. It occurs after a new feed shipment on multiple farms.

2. Clinical signs, gross and microscopic lesions are suggestive.

3. Scattered areas of hyalinization with heart and muscle necrosis, and myofiber degeneration, and necrosis are important lesions.

4. It simulates Marek's disease (MD), nitrofuran toxicity, selenium deficiency (myopathy), *Avian encephalomyelitis* (AE), and *Cassia occidentalis* (Coffee bean) toxicity.

**Prevention**

Spot check of feed will help prevent the disease.

**Treatment**

Fresh feed will discontinue clinical signs.

**Special note**

It is highly toxic to horses. About 75% of the broilers and 50% of turkey diets containing ionophores. Common ionophores include Coban® (monensin), Avatec® (lasalocid), Bio-Cox® (Salinomycin), and Monteban® (narasin).
*Nitrofuran toxicity*

Common antibiotic and anticoccidial compounds.

**Species of bird**--Chickens, turkeys, ducks.

**Action**--Acute to chronic.

**Age of bird**--All (young).

**Etiology**--Nitrofurazone (NFZ) toxicity causes dose-related biventricular cardiomyopathy, prominent dilation of ventricles, and thinning of the right or left ventricle.

**Mode of transmission**

Ingestion of food or water with too high a level of drug causes the disease.

**Clinical signs**

1. Incoordination, loud vocalization, opisthotonos (arching of the head and spine in a backwards direction), aimless running, flying, partial paralysis, ascities, and convulsions can be seen.

2. Birds are commonly down on their sides.

**Postmortem lesions**

1. Catarrhal enteritis, lung edema, congestion or kidneys of the liver, cardiac enlargement, and ascites can be observed.

**Diagnosis**

1. Feed analysis is diagnostic.

2. Clinical signs, gross and microscopic lesions are helpful.

3. Microscopic heart lesions include edema, thinning of myocardial muscle fibers, and increased connective tissue.

4. It simulates coban toxicity; salt toxicity, and ascites, which may cause right ventricular hypertrophy.

**Prevention**

A spot check of feed or water is helpful.
**Treatment**

Fresh feed or water will reverse the disease.

**Special note**

It is a drug residue problem. Nitrofurans are commonly added to the water to treat bacterial septicemia. Nitrofurans are a class of broad-spectrum antibacterial compounds. However, they have been withdrawn for use in the United States by FDA.

**Sulfonamide toxicity**

Common antibiotic and anticoccidial compound. Long withdrawal time makes it not useable for broilers over 3 weeks of age.

**Species of bird** -- All.

**Action**-- Acute to chronic.

**Age of bird** -- All; adults are highly susceptible resulting in an acute drop in egg production.

**Mode of transmission**

1. Ingestion of food or water with too high a level of drug.

2. Sulfa drugs are difficult to mix in the feed accurately. Allowance should be made when adding sulfa to the water when temperatures are high. Birds may over consume water and the drug during hot weather.

**Clinical signs**

1. Signs include paleness, depression, weight loss, and a drop in egg production and shell quality (figure 16.4).

![Figure 16.4. Poor shell quality](image)

2. Secondary bacterial infections may follow due to lymphoid necrosis.
Postmortem lesions

1. Hemorrhages in the skin (head, comb, face, wattles), muscles and internal organs can be seen.

2. Hemorrhagic enteritis, hemorrhages in the proventriculus and gizzard, pink to yellow bone marrow, and a swollen pale liver and / or kidneys may occur (figure 16.5). The spleen is enlarged and hemorrhagic, and there can be atrophy of the thymus and bursa.

Figure 16.5. Kidney lesions (urates) due to sulfur toxicity

Diagnosis

1. Sample feed and water for a definitive diagnosis.

2. Clinical signs and lesions are helpful.

3. Microscopically, areas of caseation necrosis surrounded by giant cells in the liver, spleen, lungs, and kidneys can be seen.

4. It simulates mycotoxicosis, fowl plague, fowl cholera, and visceral trophic velogenic Newcastle disease (VVND).

Prevention

Check feed and water.
Treatment

Fresh feed and water.

Special note

It is a drug residue problem. There is a 4-week withdrawal period for broilers or turkeys. Sulfonamides are widely used as growth promotants, anticoccidial, and/or antibacterial compounds which can be added to the feed or water. Tri or ormetroprim compounds are commonly added to potentiate the sulphonamides by increasing their absorption in the blood. Immunosuppression may follow.

*Organic arsenical toxicity*

Common growth promotant.

**Species of bird**--All.

**Action**--Acute.

**Age of bird**--All.

**Etiology**--Phenylarsonic acids (roxarsone® or 3-nitro, 4-hydroxy phenylarsonic acid) are commonly added to improved feed efficiency. Para-ureidabenzenearsonic acid (carbarsone) and dimetridazole (Nitrazol®, Emtryl®) are used for prevention and control of various protozoal diseases.

**Mode of transmission**

Accidental overdose in feed or water.

**Clinical signs**

1. Peripheral neuropathy causes ataxia and incoordination, stunting, depression and lameness.

**Postmortem lesions**

A small body weight and empty digestive tract are evident.

**Diagnosis**

1. Clinical signs following new feed shipment or initiation of drug treatment in the water.
2. Microscopically, peripheral nerves may show loss of myelin, fragmentation of axons, and proliferation of neurilemmal cells.

3. It simulates Newcastle disease, MD, AE, and Ionophore toxicity.

4. Diagnosis is by feed analysis.

**Prevention**

The correct level of the drug in feed or water will stop clinical signs.

**Treatment**

Fresh feed or water will stop the signs.

**Special note**

Most companies do not feed arsenicals in the US now.

**Quaternary ammonium or chlorine toxicity**

Common water sanitizers.

**Species of bird**--All.

**Action**--Acute.

**Age of bird**--All.

**Mode of transmission**

1. Water sanitizer levels are too high. Grower problem.

2. Sanitizers are commonly used to remove bacteria, fungi, or algae from water lines.

**Clinical signs**

1. Birds do not drink, reduced growth, persistent swallowing, continual spitting, facial swelling (figure 16.6), and ocular or nasal discharge can be seen.

**Postmortem lesions**

1. Ulcers on tongue from epithelial irritation are evident.
2. A pseudomembrane on the mucosae of the mouth, pharynx esophagus, crop, proventriculus, liver, and/or gizzard can be seen.

**Diagnosis**

1. Water analysis of the sanitizers is needed. Occurs on only 1 farm, where misuse of compounds was practiced.

2. It simulates T-2 toxin, Wet Pox, thrush, vitamin A deficiency, and trichomoniasis.

**Prevention**

Use lower level of the chemicals in the water.

**Treatment**

Fresh water will reverse the disease.

*Figure 16.6. Facial lesions due to quat toxicity*
17. Nutritional diseases

Nutritious food and water are essential for growth and development, reproduction, and livability of domestic fowl. More than 36 nutrients are essential and must be in the diet in approximate concentrations and balance to maximize the ability of poultry to express their genetic potential. Whenever a deficiency of an essential nutrient occurs, characteristic signs develop. These are preceded or accompanied by nonspecific signs such as retarded uneven growth, rough-feather development, decreased egg production, and lowered hatchability. This makes it difficult to recognize a nutritional deficiency, since nonspecific signs may be brought about by a number of causes, including infectious diseases and toxicants.

Quantitative nutrient requirements of the chickens and turkeys are well established. All diets are formulated by Nutritionists using requirements of the National Research Council. However, diets are formulated on a "least cost" basis and therefore, small deviations may result in a deficiency. All diets are mixed in automated, computerized feed mills (figure 17.0) by trained individuals. However, despite scientifically formulated, computerized mixed diets, deficiencies may arise. Some causes of nutritional deficiencies include: 1) milling error, usually the result of human error or malfunction of equipment; 2) feed shipment error where the wrong diet is shipped to a farm, since feed mills typically mix at least six different diets, often in the same day; 3) a feed additive or drug ties up the nutrient by binding and preventing the absorption and utilization of nutrient(s); 4) a malabsorption or mal-digestion syndrome brought on by the destruction of the gastrointestinal tract by an organism or toxin; 5) variation in ingredient quality, which may occur when grains are grown in soils deficient in an essential nutrient; 6) improper storage of feed, usually when the feed becomes too hot or moist and results in the destruction of label nutrients; 7) variation in bioavailability, which can occur in mineral sources that differ in their ability to be digested and/or absorbed; and 8) variation in feed intake, which can occur during extreme temperature shifts.

Deficiencies normally take from 7 to 14 days to produce clinical signs in birds. Young chicks derive the bulk of their nutritional content during the first few days from the absorption of egg yolk obtained during embryonation. Therefore, nutritional deficiencies, which occur during the first week or two are generally due to deficient diets in the breeders. Deficiencies in breeder diets may increase embryonic mortality and cause malformations in hatched chicks.

Food substances of nutritional importance in poultry are proteins, carbohydrates, fats, vitamins, essential inorganic elements, and water.

Proteins and amino acids

Proteins are composed of approximately 20 nutritionally important amino acids. The protein requirement represents the collective need for 10 absolutely essential amino acids (arginine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, valine), 2 amino acids (cysteine and tyrosine) that can be synthesized from amino acids, 2 amino acids essential for the young chick (glycine or serine, proline), plus additional amino acids to satisfy the nitrogen requirement for synthesis of nonessential amino acids, purines, pyrimidines, and other nitrogenous compounds.
Practical ingredients are usually limiting in one or more amino acids. In rations composed of corn and soybean meals as sources of protein, methionine supplementation is usually necessary. Lysine may be slightly deficient in such diets for starting broilers or turkeys unless alternate lysine-rich protein sources or feed-grade lysine are included. Diets based on cereal grains and other protein concentrates such as cottonseed meal, safflower meal, or peanut meal may require both lysine and methionine supplementation. Other amino acids such as threonine, tryptophan, arginine, and isoleucine can become limiting when unusual protein sources are used or when the dietary protein level is reduced. Most of these amino acids are available from commercial suppliers for supplementation in poultry feeds.

In contrast to specific signs that may occur in vitamin or mineral deficiencies, the effects of essential amino acid deficiencies are nonspecific such as the following: reduced growth, reduced feed consumption, decreased egg production, and egg size, and loss of body weight in adults. Marginal amino acid deficiencies often result in increased food intake, or maintenance of food intake with concomitant reduction of body weight gain and lean tissue growth, resulting in markedly increased body fat. Severe deficiencies also result in altered body composition. Some amino acids have additional effects. Methionine deficiency may exacerbate choline or vitamin B12 deficiencies owing to its role in methyl group metabolism. Lysine deficiency causes impaired pigmentation of Bronze turkey pouls, the biochemical basis of which is unknown. Arginine deficiency tends to cause the wing feathers to curl upward, giving the chick a distinct ruffled appearance. Several other amino acids have been reported to affect feather growth and structure.

When animals are provided with dietary protein in excess of their requirements, the surplus protein is catabolized and the nitrogen released is converted to uric acid. A large excess or protein may cause hyperuricemia and articular gout, particularly in birds that are genetically susceptible.

**Carbohydrates**

This food component is the primary source of metabolizable energy in practical poultry diets. Commercial corn-soybean diets contain high amounts of metabolizable energy.

**Fats**

Fats are important as concentrated sources of energy and the essential nutrients, linoleic acid, and arachidonic acid. Linoleic acid cannot be synthesized, but can be converted to arachidonic acid, by poultry or other monogastric animals. Both fatty acids are important constituents of cell organelles, membranes, and adipose tissue and have additional physiologic roles as precursors of prostaglandins. Lack of fatty acids in the diet of chicks results in suboptimal growth and enlarged fatty livers. Essential fatty acid deficiency in laying hens results in lowered egg production, egg size, and hatchability. Reduced concentrations of arachidonic acid and increased concentrations of eicosatrienoic acid in tissue and egg lipids is a sign of essential fatty acid deficiency.

Unsaturated fatty acids may undergo oxidative rancidity, with multiple effects. Essential fatty acids are destroyed; aldehydes that are formed may react with free amino groups in proteins, reducing amino acid availability; and the active peroxides generated during rancidification may
destroy activities of vitamins A, D, E, and water-soluble vitamins such as biotin. The addition of synthetic antioxidants to poultry feeds provides protection of vitamins and other essential nutrients.

**Vitamins**

The term "vitamin" refers to a heterogeneous group of fat-soluble and water-soluble compounds essential in nutrition that bear no structural or necessary functional relationship to each other. All recognized vitamins with the exception of vitamin C are dietary essentials for poultry. Although amounts of various vitamins needed in poultry diets range from parts per million to parts per billion, each is required for normal metabolism and health.

A deficiency of a single vitamin in the diet results in breakdown of the metabolic process in which that particular vitamin is concerned. This causes a vitamin deficiency, which in some instances exhibits characteristic changes. In several instances a single disease may result from a deficiency of any one of several nutrients. Perosis, for example, occurs in young chicks or poults when the diet is deficient in manganese or any one of the following vitamins: choline, nicotinic acid, pyridoxine, biotin, or folic acid. Analysis of the diet may be the only way to determine whether a specific nutritional deficiency is responsible for the condition.

Vitamins A and D and riboflavin are most likely to be deficient if special attention is not given to provide them when feed is formulated. However, because of continued extraction and purification of many common ingredients and the tendency to omit animal proteins and high-fiber ingredients such as alfalfa meal and wheat mill by-products from diets, amounts of several other vitamins have decreased to sometimes deficient levels. These are vitamins E, B_{12}, and K; pantothenic acid, nicotinic acid, biotin, and choline. Poultry rations are formulated to contain more than adequate amounts of all vitamins, providing margins of safety to compensate for possible losses during feed processing, transportation, storage, and variations in feed composition and environmental conditions.

![Figure 17.0. Modern Feed Mill](image)
**Vitamin A deficiency (nutritional roup)**

**Species of bird**--All.

Vitamin A is needed for integrity of epithelial lining of the alimentary, urinary, genital, and respiratory systems. Deficiency of this vitamin will interfere with growth, optimal vision, and integrity of mucous membranes.

**Age of bird**--All birds.

**Mode of transmission**

1. Vitamin A deficiency may occur when the vitamin is oxidized by rancid fat.

2. Neomycin, a common antibiotic, decreases absorption of vitamin A.

**Clinical signs**

1. Caseous eye (no odor), emaciation, weak, ruffled feathers, incoordination, eyelids stuck together (Sicca), watery discharge from throat (Roup), nostrils or eyes can be seen in young birds.

2. In adults, egg production and hatchability is decreased.

**Postmortem lesions**

1. Lesions include (white postules) in the throat, nasal passages, esophagus, pharynx, and crop.

2. Urates (whitish crystals) in kidney (swollen) may lead to gout.

3. Oral lesions are caused by blockage of mucous glands.

4. Blood spots in eggs can also occur.

**Diagnosis**

1. Gross (mouth and eye lesions) and microscopic lesions are characteristic.

2. Microscopic lesions include replacement of original epithelium with stratified squamous keratinizing epithelium.

3. Examine the levels of vitamin A in the diet for a definitive diagnosis.

**Prevention**

1. Check the diet at regular intervals for adequate vitamin level.

2. Add an antioxidant in the feed and remove neomycin.

**Treatment**

Restore normal level to the diet.

**Special note**

Hypervitaminosis A may adversely effect skeletal growth.

**Vitamin E deficiency -- crazy chick disease -- encephalomalacia**

(Vitamin E is required for reproduction and normal integrity of central nervous and muscular system. Vitamin E is also an effective antioxidant. It is an important protector of essential fatty acids, Vitamin A and D₃.)

**Species of bird**--Young poultry.

**Age of bird**--Young chicks, usually between 15th and 30th day of life.

**Mode of transmission**

1. Vitamin E is heat labile. A deficiency of selenium will result in a deficiency of Vitamin E.

2. Selenium levels in the soil in the Southeastern U.S. are very variable, which may result in suboptimal levels in cereal grains.

**Clinical signs**

Incoordination (figure 17.1), tremors, rapid contractions, and relaxation of the legs results in the name crazy chick disease.
**Postmortem lesions**

The cerebellum is softened and edematous, which may progress to hemorrhage and/or necrosis.

**Diagnosis**

1. Histopathology reveals diagnostic lesions in the brain, which includes ischemic necrosis, demyelination, and neuronal degeneration.

2. It simulates *Avian encephalomyelitis* (AE), Newcastle disease virus (NDV), and B₁ deficiency.

**Prevention**

Proper storage of premixes and finished feed and proper amount of selenium in feed will prevent the disease.

**Treatment**

Add the dietary ingredient to reduce clinical signs.

**Special note**

Selenium is involved in Vitamin E metabolism. A deficiency of selenium will cause a deficiency of E. A deficiency in vitamin E will also result in testicular degeneration in adult males and increased embryonic mortality.

*Exudative diathesis= subcutaneous edema = weeping dermatitis = water under the skin*

**Species of bird**--All.

**Age of bird**--All.

**Mode of transmission**
Produced by a Vitamin E and/or selenium deficiency in diet.

**Clinical signs**

1. Muscles are pale, chicks stand with their legs far apart, and a weeping dermatitis appearing as a green-blue lesion can be seen.

**Postmortem lesions**

1. Greenish-blue exudate under the skin, and subcutaneous edema (figure 17.2) usually around breast or leg are evident.

![Figure 17.2. Subcutaneous edema with Vitamin E or selenium deficiency](image)

**Diagnosis**

1. Gross lesions are adequate for the diagnosis.

2. It simulates gangrenous dermatitis.

**Prevention**

Diet.

**Treatment**

Restore the normal levels to the diet.

**Special note**

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Selenium is involved in vitamin E metabolism. Selenium is involved in proper development of skin and muscles and is a mineral involved in coenzyme formation. There is a wide variability in the amount of selenium in soils in the southeastern United States.

**Muscular dystrophy**

(Vitamin E and/or selenium deficiency)

**Species of bird**—All.

**Age of bird**—Young.

**Mode of transmission**

Deficiency in the diet.

**Clinical signs**

Weak, unthrifty signs occur.

**Postmortem lesions**

1. Lightly colored streaks of easily distinguished affected bundles of muscle fibers in the breast (myopathy) (figure 17.3), gizzard, and heart muscles can occur.

![Figure 17.3. Myopathy seen with Vitamin E or selenium deficiency](image)

**Diagnosis**

1. Gross and microscopic muscle lesions are characteristic.

2. Microscopic lesions include hyaline degeneration, followed by disruption of muscle fibers.

**Prevention**
Restore proper levels in the diet.

**Vitamin D deficiency—rickets (young) and osteomalacia (adults)**

(Vitamin D₃ is needed for proper metabolism of calcium (CA) and phosphorus (P), in formation of normal skeleton, hard beaks and claws, and strong eggshells.)

**Species of bird**—All.

**Age of bird**—Rickets occurs in young birds and osteomalacia in mature birds.

**Mode of transmission**

A deficiency in the diet caused by variations in Ca and P content of animal byproducts and variation in P bioavailability in various mineral sources. A P deficiency is common in cage-layers, which don't have access to P in the feces.

**Clinical signs**

1. Soft brittle bones (rickets) occur in young birds (figures 17.4 and 17.5).
2. In adults with cage layer fatigue, there are thin-shelled or shell-less eggs, white areas on brown eggs or a drop in egg production and hatchability. Hens may also be crippled.
3. In young birds with rickets, there is retarded growth.

![Figures 17.4 and 17.5. Rickets in poults (left) and chicks (right)](image)

**Postmortem lesions**

1. Beaded ribs (figure 17.6), deviated sternum, softening of the beak, claws and keel bone, skeletal distortions, and poor shell quality (figure 17.7) can occur.
Diagnosis

1. Postmortem lesions and histopathology (widening of epiphyseal plate, hypertrophy and softening of the bone, and irregular patterns of cartilage and bone development in the primary and secondary spongiosa) are diagnostic.

   It simulates other deforming leg weaknesses in poultry.

Prevention

Quality control of the feed.

Treatment

Restore proper levels to the diet.

Special note

Vitamin D₃ is needed for absorption of Ca and P from intestine and deposition of calcium in bone. Ca and P are needed for bone growth. Deficiency in Ca or P, or imbalance of either will result in bone and egg-shell malformations. Excess amounts of Vitamin D may cause renal damage and pimpling of egg shells.

*Thiamin (B₁) deficiency (polyneuritis)*

(B₁ is needed for coenzyme formation involved in proper nervous system functioning.)

Species of bird--All.

Age of bird--All; it takes 3 weeks for the deficiency to develop.
**Mode of transmission**

Vitamin B\textsubscript{1} (Thiamine) deficiency or Amprol excess can be caused by moldy feed or oxidation by rancid fat. Thiamine is converted in the body to an active form, thiamin pyrophosphate, which is an important cofactor in oxidative decarboxylation reactions and aldehyde exchanges in carbohydrate metabolism.

**Clinical signs**

1. Nervousness, anorexia, ruffled feathers, leg weakness, and an unsteady gait can occur.

2. Paralysis (convulsions with head retraction) called polyneuritis (figure 17.8) and star gazing (retracted head due to paralysis of the anterior muscles of the neck) can occur in young birds.

![Figure 17.8. Polyneuritis seen with Vitamin B\textsubscript{1} deficiency.](image)

3. Adults may have a blue comb, a decrease in respiration rate and lowered body temperature.

**Postmortem lesions**

1. Hypertrophy of adrenal glands, cortex and medulla from edema accumulation, and atrophy of the gonads, stomach, and intestinal walls are evident.

**Diagnosis**

1. Clinical signs and histopathology are helpful.


3. It simulates AE, NDV, and Vitamin E deficiency.
**Prevention**

1. Quality control of the feed.
2. A proper drug level will help prevent the disease.

**Treatment**

Alter the drug level or $B_1$ level in the feed.

**Special note**

Amprol®, a common anticoccidial medication, can tie up Thiamine. Since most grains are high in $B_1$, it does not need to be added to the diet.

**Vitamin K**

(Vitamin K is required for the synthesis of prothrombin. Prothrombin is an enzyme produced in the liver, which stimulates blood clotting. Deficiencies can result in clotting difficulties.)

**Species of bird**--All.

**Age of bird**--All.

**Mode of transmission**

Deficiency in the diet. High levels of sulfaquinoxaline may increase the incidence and severity of the condition.

**Clinical signs**

1. It occurs 2-3 weeks after the deficiency occurs.
2. Hemorrhaging and anemia in young birds may be seen.
3. It can cause increased embryonic mortality in breeders and dead embryos can be hemorrhagic.

**Postmortem lesions**

1. Lesions include hemorrhages on the breast, legs, wings and/or in the abdominal cavity and a hypoplastic bone marrow.

**Diagnosis**
Signs, lesions and determination of the prothrombin time are useful.

**Preventions**

Correct the deficiency by altering the diet.

**Biotin deficiency (nutritional dermatitis)**

(Biotin is a cofactor in carboxylation and decarboxylation reactions involving fixation of carbon dioxide. The reactions have important roles in anabolic processes and in nitrogen metabolism. Biotin is involved in a coenzyme formation that is needed for proper formation of skin and feathers may be involved in fatty liver and kidney syndrome and acute death syndrome. Hatched embryos have webbing between the third and fourth toes.)

**Species of bird** -- Young poultry.

**Age of bird**--Young birds can have dermatitis and adults will produce eggs with high embryonic mortality.

**Mode of transmission**

Bioavailability of biotin in grains is extremely variable and can result in a deficiency.

**Clinical signs**

1. Signs include skin sores (crusty lesions) on the toes, foot pad and beak, feather loss in young birds, and poor hatchability in adults (figures 17.9 and 17.10).

![Figures 17.9 and 17.10. Crusty lesions on mouth (left) and toes (right)](image)

**Postmortem lesions**

1. Fatty livers and kidneys with heart attacks characterized by blood clot in the abdominal cavity can be seen.

**Diagnosis**
1. Postmortem lesions (sterile dermatitis) and histopathology are characteristic.

2. It simulates T-2 Toxin, bumblefoot, and bacterial dermatitis.

**Prevention**

Dietary alteration will relieve signs.

**Treatment**

Restore proper amount of the vitamin in the diet.

**Niacin deficiency**

(Nicotinic acid or niacin is the vitamin component of two important coenzymes (NAD and NADP). These enzymes are involved in carbohydrate, fat, and protein metabolism. They are especially important in metabolic reactions that furnish energy.)

**Species of bird**--Young.

**Age of bird**--Young and adults.

**Mode of transmission**

Deficiency in the diet.

**Clinical signs**

1. Signs include reduced feed consumption and body weight, and lameness in young birds.

2. Reduced hatchability can occur in adults.

3. Inflammation of the mouth, diarrhea, and reduced feathering may also occur.

**Postmortem lesions**

Enlargement of the hock joint and bowing of the legs is similar to perosis, however, the tendon rarely slips from its condyles as in perosis.

**Diagnosis**

Analysis of feed for nutrient is diagnostic.

**Prevention**

Quality control of feed.
**Treatment**

Restore proper levels of the nutrient to the diet.

**Folic acid (folacin)**

(Folic acid is part of the enzyme system concerned in single-carbon metabolism. It is involved in synthesis of purines and methyl groups of such important metabolites as choline, methionine, and thymine. Folic acid, therefore, is required for cell multiplication. A choline, manganese or folic acid deficiency may result in perosis.)

**Species of bird**--All.

**Age of bird**--All.

**Mode of transmission**

Deficiency in the diet causes the disease.

**Clinical signs**

1. Perosis is characterized by a slipped tendon, which causes flattening and enlargement of hocks and long bones (figure 17.11). This causes long bones to shorten and thicken and birds become lame.

2. Poor growth and feathering, anemia (figure 17.12), and high embryonic mortality may also occur.

![Figure 17.11. Perosis with a deficiency of several important nutrients.](image)

**Postmortem lesions**

A slipped gastrocnemius tendon (without hemorrhage) is characteristic for the disease.

**Diagnosis**

1. Gross lesions and analysis of diet are diagnostic.
2. It simulates tibial dyschondroplasia and other leg weaknesses.

Figure 17.12. Pale (anemic) bone marrow

**Prevention**

Quality of the diet.

**Treatment**

Restore normal level of the nutrient to the diet.

**Special note**

Perosis is extremely common in broilers and turkeys resulting in trimming in the processing plant. Anywhere from 1% to 10% of the flock can be affected. Manganese is an activator of several enzymes and is required for normal growth and reproduction and prevention of perosis. Choline is present in acetylcholine in body phospholipids and acts as a methyl source in synthesis of methyl-containing compounds such as methionine, creatine, and N-methyl nicotinamide.

**Vitamin B₂ deficiency (curled toe paralysis)**

Vitamin B₂ (riboflavin) deficiency. Vitamin B₂ is a cofactor in many enzyme systems. Many are associated with oxidation-reduction reactions, which are involved in cell respiration.

**Species of bird**--All.

**Mode of transmission**
Most grains are deficient in B₂; therefore, it must be added in the premix.

**Clinical signs**

1. Curled toes, poor growth, weakness, and emaciation are seen in young birds. Leg muscles are atrophied and flabby. The skin is dry and harsh.

2. Poor hatchability and egg production can occur in adults.

3. Dead embryos have "clubbed" down feathers.

4. Poults have severe dermatitis of the feet and shanks and incrustations on the corners of the mouth.

**Postmortem lesions**

1. Enlarged sciatic and brachial nerve, leg muscles are atrophied and flabby in young birds.

2. Adults have enlarged fatty (yellow) livers.

![Enlarged sciatic nerve](image)

**Figure 17.13. Enlarged sciatic nerve**

**Diagnosis**

1. Post-mortem lesions (curled toes) are characteristic.

2. It simulates Marek's disease (MD), since both have enlarged peripheral nerves.

3. Histologically, nerves are edematous with B₂ deficiency and contain lymphocytes with MD.
**Prevention**

Quality control of the diet.

**Treatment**

Increased B₂ in diet.

**Special note**

Vitamin B₂ is needed for proper functioning of the peripheral nervous system.

**Pantothenic acid**

(Pantothenic acid is a component of coenzyme A, which is involved in the formation of citric acid, synthesis and oxidation of fatty acids, oxidation of ketoacids resulting from deamination of amino acids, acetylation of choline and many other reactions.)

**Species of bird**--All.

**Mode of transmission**

A deficiency in the feed causes the disease.

**Clinical signs**

1. Dermatitis, broken feathers, perosis, poor growth, and mortality can be seen.

2. Sicca (contracting of the eyelids), poor vision, and low hatchability of the eggs can be evident.

**Postmortem lesions**

1. Pus-like lesions occur at the corner of the mouth (figure 17.15) and gray-white exudate in proventriculus, hypertrophy of the liver, atrophy of the spleen, and kidneys are enlarged.

2. Perosis is seen as enlargement of the hock joints, bowing of legs, and slipping of tendon (figure 17.14). The tibia may also rotate out of the condyles.

**Diagnosis**

Feed analysis is needed.

**Prevention**
Quality control of feed will be helpful.

**Treatment**

Adjust the nutrient level in the feed.

*Figure 17.14. Perosis with deficiency of several nutrients*

*Figure 17.15. Oral lesions*
18. External parasites

Introduction

Most external parasites, with the exception of flies, mosquitoes and rodents, are arthropods that live on or in the skin. Poultry ectoparasites are members of the animal phylum Arthropoda, characterized by externally segmented bodies, jointed appendages, and continuous exoskeletons.

Lice (figure 18.0), flies, bugs, and fleas are members of the Class Insecta, characterized by possession of a body divided into three regions (head, thorax, abdomen), one pair of antennae attached to the head, three pairs of legs attached to the thorax, and tracheae (air tubes) for breathing. Some adult insects have wings.

Insects undergo metamorphosis, whereby immature stages may appear totally different from adults, and do not show the characteristics given for the Class Insecta; for example, some fly maggots possess no legs, antennae, or obvious body divisions. Lice, on the other hand, are easily recognized as insects regardless of stage.

Mites are members of the class Arachnida, order Acarina, characterized by fused body divisions, no antennae, and four pairs of legs (the first motile stage, larva, has three pairs). Ticks are very large mites, contrasting sharply with most mites, which are much smaller than most insects. Acarina never possess wings (figure 18.1).

Detection

Poultry infested with parasites exhibit irritation and react by scratching and preening. Any unexplained production drop or increase in feed conversion is cause to look for external parasites. Lice and northern fowl mites can be found by examining the skin after parting the feathers. Mites are more easily seen with a magnifying lens. To monitor birds in a production facility, 20-50 birds should be checked a minimum of two times a month. Birds should be checked at random and should be chosen from all parts of the house. The vent, head, and legs should be closely examined. If parasites are found and cannot be identified at first glance, specimens should be sent to a laboratory to have the specific identity determined.

Bloodsucking parasites (bed bugs and chicken mites) that come to the birds only to feed are more difficult to detect. It is necessary to examine bedding, roosts, walls, cracks and crevices, and beneath manure clods. Nest material, dust and other material collected in the house can be spread out on a white pan and examined. The arthropods can be seen crawling on the pan. Night-time examination of birds may detect parasites that feed on them at night.
Figure 18.0. Feather louse

Figure 18.1. Mite eggs around feathers

**General pesticide control procedures**

The synthetic and natural pyrethroid insecticides, organophosphorus, and carbamate are the main ectoparasite and fly control chemicals used for direct application to poultry, litter, or buildings. In general, chemical insecticides and disinfectants should not be mixed for application together. Among the botanical insecticides, pyrethrum remains very effective against flies and is a main ingredient of mist and aerosol fly sprays, particularly with synergists.

The chlorinated hydrocarbon insecticides are banned from use on poultry or in poultry houses because of residues in eggs and meat. Under no circumstances should DDT, benzene
hexachloride, toxaphene, chlordane, aldrin, dieldrin, endrin, or heptachlor be used on poultry houses or on poultry feed or feed ingredients.

Insecticides are available as wettable powders (WP), emulsifiable concentrates (EC), and water dispersible liquids (WDL), all of which are intended to be applied as a spray or mist. Insecticides are also available as dusts and as baits. These low-assay products are prepared, premixed, and ready to use. Care should be taken to ensure that feed and water are not contaminated and that all label directions are strictly adhered to so that tolerances are not exceeded.

Tolerances and residues

There are residue tolerances for pesticides used for control of poultry pests, and time that must elapse between application and slaughter or sale of eggs, to meet legal requirements (Table 18.1).

Table 18.1. Generic or common and trade names of insecticides with specific regulations regarding use with poultry.

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<th>Common name</th>
<th>Name</th>
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<td>Union Carbide</td>
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aM=meat, BY=by-products, F=fat, E=eggs.
bND=no documentation for drug withdrawal listed.
Pesticides are listed as having a zero degree tolerance unless special documentation is approved.
Sulfur and lime sulfur require no tolerances. Fly spray ingredients (Pyrethrin and piperonyl butoxide) may be used on poultry or in poultry houses with no interval required between treatment and slaughter or gathering of eggs.

Insecticides should be not used on poultry or in poultry houses without carefully reading all precautions. In all treatment of poultry, contamination of feed and water must be avoided. All eggs should be gathered before starting to treat with insecticides. Off-flavors in eggs can be caused by direct contamination of eggshells. Ventilation should be supplied during dusting, spraying, or misting.

Pesticide residues may occur in eggs or meat from contaminants in feed, water, litter, or soil. Contaminated carcasses may be seized and destroyed after processing. Residue levels in eggs approximate levels in feed, and contaminated eggs continue to be produced long after pesticide ingestion ceases. Because the Environmental Protection Agency (EPA) and Food and Drug Administration (FDA) are concerned with contamination of foods by pesticides, industrial chemicals (PCBs), and toxins (aflatoxin), regular collections are made of poultry, meat, and eggs from market shelves for laboratory analysis of residues.

**Application**

The key to successful application of an insecticide, and resulting control that meets expectations, is to apply the insecticide directly where the pest is located. If birds are being sprayed, the treatment must thoroughly cover the entire bird and the bird should be wet to the skin. If buildings are being treated, the sites where the pests are located must be treated if control is to be adequate. Methods for caged layers include high-pressure sprays (125 pounds per square inch [psi]) from outside the cages. Other equipment can be used, but if the birds are not treated to ensure the wetting of the skin and feathers, control will not be adequate.

**Dusting.** For conventional houses, apply to litter; attempt to cover the area evenly including under roosts, feeders, and nest boxes.

**Spraying.** The usual cylindric compressed-air sprayers are satisfactory for treating roosts and walls, as are knapsack sprayers (continuously pumped during spraying) that give a continuous spray. Sprayers powered by an electric or gasoline motor that delivers pressures of 125 psi, and use of a spray gun with a solid-stream nozzle are much more rapid and efficient. When spraying houses, high-pressure and large-volume output are most desirable to drive spray into all cracks and crevices.

**Misting.** Electric mist machines (foggers) are efficient, rapid, and often laborsaving. Mist machines are concentrate applicators and do not use the same mixtures as ordinary sprayers. Generally they use 5-10 times the concentration and 1/3-1/10 the volume. In all fog work, the
container should be shaken frequently during spraying to keep insecticide from settling. Mist machines can be used efficiently to dispense fly spray.

**Insects**

**Lice**

Lice are common external parasites of birds. They belong in the order Mallophaga, the chewing lice, and are characterized by chewing-type mandibles located ventrally on the head, incomplete metamorphosis, no wings, dorsoventrally flattened body, and short antennae with 3-5 segments. More than 40 species have been reported from domesticated fowl. Lice will transfer from one bird species to another if these hosts are in close contact. Pediculosis (lice infestation) of birds is diagnosed by finding the straw-colored lice on skin or feathers of birds. Lice of domestic birds vary in size from less than 1 mm to over 6 mm in length.

Mallophaga up to 10 mm long occur on wild birds. Lice spend the entire life cycle on the host. Eggs are attached to the feathers, often in clusters, and require 4-7 days to hatch. The entire life cycle takes about 3 weeks for completion, including 4-5 days for incubation and three nymphal instars of 3 days each. One pair of lice may produce 120,000 descendants within a few months. Their normal life span is several months, but away from the birds they can remain alive only 5 or 6 days. Bird lice eat feather products and may consume blood by puncturing soft quills near the bases and gnawing through the covering layers of the skin itself.

**Control.** Galliform wild or domestic birds should never be allowed to contact poultry flocks. Lice tend to increase during autumn and winter, so flocks should be examined for lice on a regular basis (two times/month minimum) and treated if needed. Birds should be treated two times on a 7-10-day interval. Only the mature and immature forms will be controlled as none of the available chemicals are ovicidal (eggs are not killed). Re-treatment is necessary to control the lice that will hatch after the initial treatment. In houses, the egg-laden feathers will be a source of re-infestation and when the house is depopulated, a thorough cleanup should be completed. Spraying of birds is the most practical means. Care should be taken when spraying to ensure that the whole bird is treated, as it is common for lice to move to the neck from the vent when populations are large. In caged-layer flocks it is important that the birds are checked on a regular basis.

**Darkling beetle or lesser mealworm**

Darkling beetles infest poultry houses around the world. The beetles live in the litter where they feed on feed, manure, and dead or moribund birds. The life cycle of darkling beetles requires from 1-3 months for the development of the larvae, and the adults can live for 2 years. Beetles are small (0.5 cm) and can be easily seen under feeders, waterers or along the walls of the house. The larvae are worm-like and will avoid light.
Beetles within a poultry house can number up to 1000/m². They are important to the poultry industry as possible disease vectors, by damage to insulation and as pests. Only adults and late instar larvae-seeking population sites and tunnel in the insulation. Their climbing activity takes place at night. Beetles can be found throughout the poultry house; eggs, larvae, pupae, and adults are in litter and soil. Due to the beetles' ability to utilize many niches in the poultry house, control is difficult to achieve with any single approach.

**Control.** Stored grains and feeds should not be allowed to become infested with insects. Infested material should be fumigated. Control of lesser mealworms and beetles requires an integrated attack that utilizes all approaches to manage the population. Any control strategy must take into account that there will be a number of eggs, larvae, pupae, and adults in the soil and walls of a building. These life stages will not come into immediate contact with an insecticide, and if the insecticide does not have a long residual life, control of the beetles will be shortened. The best approach is to clean out after each flock. Another is to utilize carefully timed insecticide treatments. Houses should be treated with an insecticide immediately after the flock is removed. Poultry litter treatments that reduce the Ph will also kill these and other insects in the litter. Clean up feed and water spills.

The darkling beetle is not tolerant of temperatures below 40°F. If the air temperature is less than 40°F, the house should be opened up at clean-out to allow the temperature of the litter to drop as low as possible. By using cultural controls, low temperature, clean-out schedules, and chemicals, beetle populations can be managed and maintained below damaging levels. Other chemicals which can be added to litter in broiler houses include boric acid and Safecide® from Schering Plough Animal Health and Tempo SC Ultra® (Betacyflutrin a fifth generation pyrethroid) from Bayer Inc., Watkinsville, GA. Other products include permethrins, tetrachlorinphos, and dichlorvos.

**Flies, mosquitoes, midges**

The order Diptera includes families whose members suck blood from birds and mammals. All dipterans have two wings in the adult stage (except degenerate wingless forms) and pass through a complete metamorphosis including a maggot-like larva and a puparium resting stage. Adult mouthparts are of the piercing-sucking or sponging types. The intermittent nature of their feeding and extensive flight range, render adult flies ideal vectors of disease. Certain species develop in poultry manure and may become so numerous as to create a health and public relations problem. Pyrethroids (first-fifth generation) are excellent for removing this parasites.

**Mosquitoes**

Although mosquitoes are not as important to poultry as to human beings and other mammals, many species feed on poultry and transmit disease, including fowl pox. Some 140 species have been described from North America; a number of these are known to suck avian blood.

Most species are about 5 mm in length, and wings are characteristically veined and scaled. Legs and abdomen are long and slender, and the female is provided with elongated mouthparts for
piercing the skin. The male does not suck blood but feeds on plant juices, nectar, and other fluids. Mosquitoes deposit eggs on pools of water, moist soil, or surfaces subject to flooding. Larval and pupal stages develop in water, with adults emerging from pupal cases to mate and then seek a host. In warm weather the life cycle is completed in about 7-14 days. Adults are most active on dull, quiet days, especially toward evening and at night.

Poultry production facilities that utilize lagoons can have problems with mosquitoes breeding in the lagoon. Lagoons should have steep banks that are free of vegetation along the shoreline and should be relatively deep to provide the proper environment for anaerobic decomposition of the waste. If mosquito breeding is a problem and the lagoon requires chemical treatment, Dursban® would be the chemical of choice.

Mosquitoes may attack poultry in dense numbers. Mosquitoes carry and transmit viral agents of eastern equine encephalomyelitis (EEE), St. Louis encephalitis (SLE), and western equine encephalomyelitis (WEE). Fowl poxvirus is transmitted by *Aedes stimulants*, *A. aegypti*, *A. vexans*, and many species of culicoides-biting midges. *A. stimulant* may harbor the virus for 2 days, whereas *A. vexans* may infect birds up to 39 days after contacting the virus of fowl pox and pigeon pox.

West Nile fever is a viral disease of some birds, animals, and humans. It is carried by wild birds and can cause a flu like syndrome and even death in dibilated humans. The virus can cause disease and death in some species of wild birds. Chickens are not very susceptible to the virus, but can act as carriers. The virus is transmitted by the bite of some species of mosquitoes.

**Control.**

The best approach is prevention of mosquito development. The farm should be surveyed for all water areas that may produce mosquitoes, including swamps, ponds, stagnant pools, and water-filled containers of all types. Mosquito production can be stopped by removal of such containers, covering cisterns and water barrels, clearing pool and pond edges of emergent vegetation, performing drainage operations, and filling low areas that collect water.

For housed poultry, mosquitoes, landing on surfaces inside or outside the house, may be killed by residual insecticide deposits of the type that are recommended for fly control. Poultry in open houses or on range are most difficult to protect from mosquitoes. Pyrethrum fly sprays can be fogged in houses or on ranges to obtain quick kill of mosquitoes in an outbreak, but control will not last more than a few hours. Residual sprays of carbaryl, malathion, propoxpur, or stirofos can be applied to exterior surfaces of buildings or outdoors to vegetation from which poultry are excluded. If needed, breeding areas can be treated with larvacides using temphos Abate®, or Bti (*Bacillus thuringiensis* var. *israelenis*), a biological control agent (bacteria which kills the larvae) that has been shown to be effective.

**Blackflies**

Blackflies (family Simuliidae) are also known as turkey or buffalo gnats. They are similar in size to mosquitoes but are dark, short, chunky, and humpbacked, with short legs; wing venation is
distinctive. More than 20 species have been reported to attack domestic poultry in North America. Blackflies usually suck blood during the day and may cause serious damage to human beings and livestock; in dense numbers on poultry they may cause a severe anemia. They also transmit certain blood protozoans belonging to the genus *Leucocytozoon*.

Blackfly production sources are restricted to running water such as creeks, streams, or irrigation supply and drainage systems. Eggs are laid on rocks, sticks, or floating vegetation, or are dropped into streams. They may hatch in a few days, but some remain through summer or even until the following spring. Larvae attach to stones or other objects and reach the pupal stage after 3-10 weeks. The pupal stage also occurs under water, lasting from a few days to 1 week or more. Adults of some species emerge in spring, others during summer or early fall. Overwintering occurs in the egg or larval stage. Most temperate-zone species have one generation a year.

**Control.** Control is difficult because these pests develop in streams, often some distance from the poultry farm, where insecticide treatment may be harmful to fish. Successful reduction of larval and subsequent adult blackfly populations, without fish kills, were obtained in infested streams treated monthly by helicopters using 2% temphos granules. Area-wide control programs have been developed using biological control agents such as *Bacillus thuringiensis* var. *israelensis* (Bti). These programs involve weekly treatment of all breeding areas in defined geographical areas. Measures recommended for mosquito control, as well as cautions on watershed contamination by pesticides, are also pertinent to blackfly control.

**Housefly and its relatives**

Non-biting flies produced on poultry farms are a health and sanitation problem to the poultry producer and neighbors. Public pressure against poultry enterprises can force producers to move or go out of business if flies, odors, or blowing feathers are not controlled. Modern poultry farms produce a tremendous amount of manure, which must be managed to ensure that it is not attracting flies for breeding or causing an odor problem.

Location of poultry houses and manure disposal areas needs to be carefully planned to prevent filth fly problems from developing. The poultry industry has an important role in community responsibility to control flies in suburban and urban areas. Poultry producers have met financial disaster as new residential developments have invaded formerly suburban locations where they had built their facilities. In many regions, state and county legislative action has strengthened public health codes, and local ordinances have resulted whereby poultry farms can be closed because of unabated fly sources found on their property.

Flies lay eggs in manure (some sarcophagids deposit living larvae), in most spilled feed, or on dead-bird carcasses. In hot weather the housefly can complete its life cycle in 8 days, but in colder weather it may require over 6 weeks. Larvae (maggots) develop in moist manure and then move to drier areas for pupation. The housefly does not diapause, and survives northern winters by slow development in warm indoor locations such as enclosed poultry houses and dairy barns and in towns and cities. Other filth flies survive northern winters by hibernation. Insecticide formulations (organocarabamtes, pyrethroids, and carbamates) can be used as dusts or sprays. Water dispersable liquids, wettable powders (WP), and emulsifiable concentrates are available. WP formulations will give a longer lasting residue than the other formulations. Dirt, type of
surface, and amount of sunlight on the surface will have an effect on how long the product remains active. Dust applications are easy to apply and ready to use, but are subject to wind drifts and must be stored in large containers. Sprays are easy to store and use for spot treatment, but require mixing and spray equipment.

**Baits**

Commercial baits are formulated as granules and should be placed in pans or in protected areas. Bait can be placed in fly traps. To increase effectiveness of dry baits such as methomyl, one part field-grade molasses may be diluted with three parts water in a 5-gallon can and covered with a removable window screen lid on which the dry bait is placed. Some commercial baits add a fly attractant such as Muscamone®, which increases their effectiveness.

**Larvicides**

Control of fly larvae in the manure is done with a larvicide, which can be applied as a liquid, dry, or in the bird feed. Penetration of the manure with a liquid is difficult, and it is adding water to manure, making it more difficult to dry to reduce breeding. Larviciding manure is also devastating to the predators and parasites living in the manure, causing a further imbalance of the fly larvae and predators and parasites. Larvicide treatment should only be done on a spot basis, where large numbers of larvae are seen. One exception to this rule is the larvicide cyromazine, which is toxic to fly larvae but not to the predators and parasites. Another product, Larvadex®, can be fed to cage layers. The product passes out harmlessly in the feces and kills developing larvae.

**Biological control**

One can use larvae from carpenter wasp in the litter. These larvae will consume fly larvae and the wasps are generally not a problem for humans.

**Management practices**

Use of composting bins for poultry litter will generate enough heat to control fly development. Use of dry cups in the house and automatic feeders will keep the litter dry and free of feed. Use of pits and lagoons in cage layer houses will keep feces from building up in the house. Use of slats under feeders and waters will keep the house litter dry. This will also allow for easy treatment of feces with chemicals, in confined areas such as under the pits.
Mites

The common free-living ectoparasitic mites of poultry belong to the family Dermanyssimite. Mites possess well-sclerotized, free dorsal and ventral plates; claws and caruncles on the tarsi; one lateroventral stigma near each third coxa; and small chelicerae on long, sheathed bases. They are bloodsuckers and can run rapidly on skin and feathers (figures 18.2 and 18.3).

Chicken mite

The chicken mite (*Dermanyssus gallinae*), also called red mite, roost mite, or poultry mite, is found worldwide. It is particularly serious in warmer parts of the temperate zone in older poultry houses with roosts. The mite is rare in modern large commercial caged-layer operations but is seen frequently in modern broiler breeder farms. It can be identified by the shape of the dorsal plate and by the long whip-like chelicerae that appear to be stylets. The adult female measures about 0.7mm x 0.4mm, varying in color from gray to deep red, depending on its blood content. The life cycle may be completed in as few as 7 days. Adult females lay eggs in surroundings of the host’s 12-24 hours after their first blood meal. Eggs hatch in 48-72 hours when warm. The 6-legged larvae molt in 24-48 hours without feeding, becoming first-stage bloodsucking nymphs; they then molt to second-stage nymphs in another 24-48 hours and soon afterward molt to the adult stage. Chicken mites have lived up to 34 weeks without food.

Chickens are the most common hosts, but these mites may occur on turkeys, pigeons, canaries, and several species of wild birds. Human beings may also be attacked, and invasions of human dwellings (apartments, hospitals, doctors' offices) by mites from outdoor pigeon nests are frequently seen. English sparrows may transmit this parasite because of the habit of lining their nests with chicken feathers. These mites may not only produce anemia, thereby seriously
lowering production and increasing feed consumption, but actually kill birds, particularly chicks and setting or laying hens. Birds in production may refuse to lay in infested nests.

An increase in feed consumption accompanied by lower production is a sign that poultry houses should be examined for mites. These mites often can be found by looking under loose clods of manure, under slats in a breeder house, in nests or in cracks and crevices of posts and roof bracing. They are evident as tiny red to blackish dots, often clustered together. Inspection during the night is usually necessary to find mites on birds. Occasionally these mites may be found on the shanks of both hens and roosters, but care must be taken to differentiate them from northern fowl mites that also appear on the legs.

**Northern fowl mite**

The northern fowl mite (NFM) (*Ornithonyssus sylviarum*) is a parasite of all major poultry production areas of the United States. It is common in almost all types of production facilities. It has been reported in domesticated poultry, English sparrows, numerous wild birds, rats, and human beings.

This mite is confused with the chicken mite, but can be distinguished by its easily visible chelicerae and the shape of dorsal and anal plates. Unlike the chicken mite, the northern fowl mite can be found on birds in the day as well as at night, since it breeds continuously. In heavy infestations, feathers are blackened and skin is scabbed and cracked around the vent; when birds are handled, mites quickly crawl over the examiner's hands and arms. Parting the feathers reveals mites, their eggs, cast-off skins, and excrement on the body surface and feathers. Poultry producers often diagnose NFM infestation by seeing mites crawling on eggs.

The life cycle of the northern fowl mite is completed in less than 1 week on the birds. Eggs are laid on the feathers and hatch in 1 day. The larval instar and two nymphal instars develop in less than 4 days. In the north, mite densities increase in winter and usually drop to low numbers by summer. Occasionally, however, infestations are found in summer. This contrasts with the chicken mite, which is a pest during warm weather in northern areas but inactive in cold houses during winter. Mites may survive 3-4 weeks in absence of avian hosts.

The NFM is introduced into laying hen flocks from four main sources: infested hatcheries and contract-started pullet farms; trucks and crates used to carry old birds or infested pullets; personnel, equipment, or egg flats and crates; and wild birds. Sparrows, pigeons, etc. that nest in or near poultry houses are suspected to transmit mites.

These mites suck blood, and the resulting scabs may injure the appearance of dressed poultry. Of greater concern is the economic importance of this mite to egg production from infested caged layers. Among broiler breeder laying hens, NFM-infested birds produced 7.7 eggs/hen less than NFM-free birds and feed costs were increased from $.01 to $.06/dozen eggs produced.

**Control.** All mites can be controlled by the same insecticides, which are applied to birds, litter, nests, and walls. Initial strategy should be focused on monitoring all birds and facilities. Proper monitoring will reduce the spread of ectoparasites from farm to farm on service personnel, flats, repair personnel, replacement birds, and live-haul equipment.
All egg flats and cases should be checked if they are coming off an infested farm. Operations that use plastic flats on racks should be sure that they are washed with hot water and detergent prior to being redelivered to another farm. Operations that use fiber flats and cardboard cases should inspect them prior to sending them back to a farm.

Birds can be treated with any of the registered insecticides. All flocks should be treated twice on a 5-7-day interval for NFM, and longer for other parasites. With lice and NFM, the birds should be treated to ensure that the skin is wet, since this is where the pests reside. The most efficient method to use in most poultry facilities is a solid-stream spray at 40-125 psi. Care should be taken to ensure that the birds are wet to the skin or control will be less than desired. Permethrin EC® spray is the most effective chemical registered, lasting up to 9 weeks after treatment when applied at .05%. Red mites can be controlled by treating both the birds and the facility. If the red mites persist in a house, the house should be re-treated at cleanout.

Chigger infestations are sporadic and localized; a heavily infested site may adjoin a habitat that appears similar to it in all respects but is free of these mites. Chiggers affect poultry in the southern states, and birds that are housed outside on the ground. Larval mites of the family Trombiculidae are called chiggers. Nymphs and adults are free-living, usually in or on soil. Although over 700 species are known, only a few attack poultry. The larval chigger is 6-legged and possesses a single dorsal plate bearing a pair of sensillae and 4-6 setae. The legs are 7-segmented and bear 2 claws and an empodial bristle. Unfed chigger larvae are 0.1-0.45mm in diameter, hence hardly visible unless engorged, when they appear as minute red dots.

Adults occur on the ground, especially along fence rows or in undisturbed wooded or bushy areas. Larvae attach to the skin, often in groups, and inject a highly irritating substance into the wound, thereafter feeding on liquefied host tissue but not blood. Itching vesicles or even abscesses surrounded by a zone of hyperemia and edema may form at the points of attachments. Apparently, a toxemia may occur, as indicated by the mortality that follows infestation of chicks, especially quail.

The most important poultry chigger in the United States is *Neoschongastia americana* which is a serious pest of turkeys and wild birds and a minor pest of chickens all across the South (particularly Georgia, the Carolinas, Texas, Alabama, Arkansas, Missouri, and Kentucky) as well as Nebraska. This chigger also occurs in Central America and the West Indies.

**Ticks**

Ticks are large mites belonging to the super-family *Ixodoidea* of the *Acarina*. They are characterized by having a pair of oval or kidney-shaped stigmata posterior or lateral to the coxae; the hypostome is modified as a piercing organ provided with re-curved teeth, and there is a pit-like sensory organ on the tarsi of the first pair of legs (Haller's organ). Unengorged adults of most common ticks are 2-4 mm long, but fully engorged females may reach more than 10 mm. However, unengorged tick larvae are similar in size to adult mites.

Ticks inhabiting poultry houses belong to the family *Argasidae*. They have no scutum (dorsal shield) and except for larvae feed intermittently in all stages. The integument is leathery,
winkled, and granulated in appearance. The capitulum (head) is ventrally placed near the anterior margin of the body. Many hard-bodied ticks in the family Ixodidae will feed on range poultry. These ticks possess a scutum in all stages and feed only once in each stage, remaining on the host for several days. The scutum usually appears shiny, and the capitulum is terminal at the anterior of the tick.

Losses caused by ticks are threefold: loss of host blood, which may cause death; reduced production associated with anemia but also possibly due to tick-produced toxic substances; and transmission of diseases such as avian spirochetosis, tularemia, piroplasmosis, anaplasmosis, dirofilariasis, certain rickettsial diseases (notably Rocky Mountain spotted fever), and many viruses, including encephalitis.

Fowl ticks (chicken ticks, blue bugs, tampons, adobe ticks) are distributed mainly in states along the Gulf of Mexico and the U.S.-Mexican border. They are also established in many other tropical and temperate areas of the world. Although primarily parasites of birds, they may be found on mammals. In North America they commonly have been reported from the following hosts: chickens, turkeys, ducks, geese, guinea fowl, pigeons, canaries, doves, hawks, magpies, owls, quail, sparrows, thrushes, vultures, ostriches, and wild turkeys, as well as, rarely, from cattle, dogs, and human beings.

Mature blood-engorged females measure about 10mm x 6mm. Unfed ticks are relatively easily recognized by their flattened ovoid shape and tan to reddish brown color. Females may lay a total of 500-875 eggs in four or five separate batches but require a blood meal before laying each batch of eggs. Eggs are laid in sheltered crevices, including bark of trees. They hatch in from 6-10 days in warm weather to 3 months during cool periods. Larvae (seed ticks) become hungry in 4-5 days and seek a host, although they may live for several months without feeding. They feed on blood for 4-5 days and then leave the host for a hiding place nearby and molt (shed skins) in 3-9 days to the first nymphal stage.

Nymphs are active and feed only at night, but can do without food for as long as 15 months. The first-stage nymphs feed in 10-45 minutes, leave the host and hide for 5-8 days, and molt to a second nymphal stage that is ready to feed in 5-15 days. Similarly, these second-stage nymphs feed and hide; adult ticks emerge from the nymphal skins ready to engorge with blood and mate about 1 week later. Oviposition commences 3-5 days after mating. This complete life cycle takes about 7-8 weeks during warm weather and longer during cold seasons. Fowl ticks remain inactive in cracks and crevices during cold weather, and adults may live without a blood meal for more than 4 years.

Birds suffer chiefly from attacks of these ticks during the warm dry season. Loss of blood may reach proportions of a fatal anemia; at least there may be emaciation, weakness, slow growth, and lowered production. Ruffled feathers, poor appetite, and diarrhea are signs suggesting tick infestation. The fowl tick is the most important poultry ectoparasite in many tropical countries, being a limiting factor in successful rearing of standard breeds of poultry. Turkeys usually suffer even more than chickens; recently hatched poult's and chicks show the highest mortality. These ticks cause skin blemishes on turkeys, reducing market price.
The fowl tick is capable of transmitting the highly pathogenic spirochete *Borrelia anserina* in many parts of the world. Tick-borne avian spirochetosis has been reported in chickens and turkeys in the United States; epizootics of avian spirochetosis in Arizona are associated with infestations by the fowl tick. Fowl ticks have been reported to transmit *Aegyptianella pullorum* and fowl cholera (*Pasteurella multocida*) in some regions of the world. All postembryonal stages of the common fowl tick have been found infected with *A. pullorum* in some areas. However, in other areas transmission of fowl cholera was not shown even though fowl ticks harbored *P. multocida* for 25 days. *Aegyptianellosis* has not been reported from the Americas.

Tick paralysis in chickens, a flaccid, a febrile motor paralysis, may result from attacks by *A. persicus* as well as by *A. walkerae* in Africa. Etiology of this sporadic disease is not understood, but most probably a specific paralytic toxin is contained and transmitted in the tick salivary secretions. Clinical signs may be confused with botulism, neural signs of Marek's disease, transient paralysis, Newcastle disease, and possibly conditions caused by other bacterial or chemical toxins.

*Control.* Control requires treatment of premises because adult and nymphal ticks are on their hosts only a short time and then hide in the surroundings. The litter, walls, floors, and ceilings must be sprayed thoroughly, forcing spray into cracks and behind nest boxes. Outdoor runs and feed troughs, woodpiles, and tree trunks may be treated using approved insecticides. Other methods for fowl tick control include use of metal construction, elimination of tree roosting, using roosts suspended from ceilings, and converting to cage operation. Frequent inspection is necessary to combat ticks before their number has increased to a harmful extent. Fowl ticks are rare in modern, large commercial cage-layer operations.

*Rodents*

Rodents are common external pests in and around poultry facilities and can parasitize poultry. Therefore, they are included in this chapter. However, other such books have chosen to list them as a separate chapter. Rodents' burrowing and gnawing activity can undermine foundations and destroy curtains and insulation. Rodents can eat or contaminate feed, which increases feed costs and affects feed conversions. Additional problems can be produced by the presence of these pests, since they may carry a variety of diseases and ectoparasites.

*Rats*

Figures 18.4 and 18.5. Rat control includes clean up (left) and exclusion (right)
The Norway rat (*Rattus norvegicus*) is the most common rat around poultry houses. Rats have three basic requirements: food, water, and harborage. If one of these is missing there will not be a rat problem. Unfortunately, all of these are usually present in and around poultry production facilities. Rats eat almost any type of food, including eggs and poultry feed; however, rats prefer fresh food. When fresh food is available, rodents will totally ignore spoiled food. An adult rat will eat and drink approximately 0.5-2 oz of food and water each day, with 200 adult rats consuming 25 lbs of feed daily.

Rat harborage around a poultry house is seen in the form of burrows in the ground and under the foundation, litter under the slats, and in wood-piles, old nests, and other debris near the poultry houses. This type of harborage must be removed for any control program to be successful. Most rat activity, including feeding, occurs at night. Rats observed outside their harborage during the day indicate a large population. Rats are very territorial; when crowded they become increasingly aggressive. The stronger and more aggressive rats drive the weaker rats away from the food, forcing them to feed during the day.

Rats have high reproductive rates, which can lead to large numbers of rodents in a fairly short period of time. A single pair of rats and their offspring could produce as many as 1500 rats in 1 year if all the offspring survived. Rats will breed at 3-5 months of age and give birth approximately 3 weeks after mating. Four to seven litters are produced in 1 year with each litter having 6-12 young; the female will breed again 1-2 days after giving birth. Although breeding occurs all year, increased breeding frequently occurs during the spring and fall. Generally, rodent populations decrease during the winter; however, in a poultry house, the opposite is frequently true, because of immigration of rats into the building when the weather cools, not because of increased reproduction. The best time for search for rodents and their excrement is at night in the dark with a flash light.

**Mice**

The house mouse (*Mus musculus*) is the most common mouse found in and around poultry facilities. Mice require food, water, and harborage, and will eat almost any kind of food with about 0.1oz of food consumed by an adult mouse each day. Mice are active throughout the day, often feeding every hour. However, peak activity occurs at dusk and dawn. Mice require much less water than rats and are capable of utilizing water from the food they eat. Mice will burrow in the ground and in insulation or live in rolled-up curtains. Mice are able to reproduce 6-8 weeks of age. They will give birth to five or six young approximately 3 weeks after mating; the female can breed again 2-4 days after giving birth. Generally, five to eight litters are produced in a year. Mice breed regularly throughout the year with no seasonal peak.

**Control.** There are three aspects to rodent control: rodent-proofing, sanitation, and rodent killing. Rodent-proofing can be an effective long-term control measure. However, it is impossible to rodent-proof a poultry facility with curtains, wooden side-walls, and/or dirt floors. Access to the building can be restricted by patching or screening holes in the foundation, thus forcing the rodents to burrow into the house, which makes them easier to detect (figures 18.4 and 18.5).

Sanitation involves cleaning around the facility. Rodents are secretive creatures. They do not like to move in open areas. Therefore, mowing the grass and weeds on a regular basis creates a less
favorable habitat. Removing piles of old wood, nests, or any other debris helps to make the area less attractive to rodents and aids in making early detection possible. (figure 18.6). When debris or tall grass is present, rodents can burrow into a facility and go unnoticed. Rolling the house curtains up and down a couple of times a week during summer months will disturb any rodents that are in the curtains and discourage them from living and/or nesting in them.

Figure 18.6. Rodent harborage

After the cultural control measures have been completed, rodent killing can begin using baiting, fumigating, trapping, or even shooting. A properly conducted baiting program is easiest and most effective.

There are many products that will kill rodents. The first safe and commonly used baits are the multiple-dose anticoagulants (Table 18.2). Products that contain warfarin, fumaric, chlorophacinone, or diphacinone as an active ingredient are examples of this type. Multiple-dose anticoagulants must be consumed for several days to be lethal. The effects are cumulative: if a rodent feeds on this type of bait for a day or so, then feeds on something else before returning to the bait for another few feedings, the rodent will not be controlled. Therefore, it is imperative that enough bait be available for the rodents to eat for several days. The specific number of days that a rat or mouse must feed on a multiple-dose anticoagulant poison depends on the bait being used and the amount consumed. These chemicals are safe for people and for non-target animals, because a single dose will not cause death.

Table 18.2.

<table>
<thead>
<tr>
<th>Active Ingredient</th>
<th>Type of Bait</th>
<th>Trade Names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warfarin</td>
<td>Multiple-dose anticoagulant</td>
<td>Bar Bait®, Contrax®</td>
</tr>
<tr>
<td>Pival</td>
<td>Multiple-dose anticoagulant</td>
<td>Rivalyn®, Contrax®</td>
</tr>
<tr>
<td>Diphacinone</td>
<td>Multiple-dose anticoagulant</td>
<td>Promar®, Ramik®</td>
</tr>
<tr>
<td>Chlorophacinone</td>
<td>Multiple-dose anticoagulant</td>
<td>Rozal®</td>
</tr>
<tr>
<td>Zinc Phosphidea</td>
<td>Acute single dose, rapid death</td>
<td>Ridal®, Rodent Pellets®</td>
</tr>
<tr>
<td>Bromethalin</td>
<td>Single dose, affects central nervous system</td>
<td>Assault®, Vengeance®</td>
</tr>
<tr>
<td>Brodifacoum</td>
<td>Single-dose anticoagulant</td>
<td>Ropax®, Talon®</td>
</tr>
<tr>
<td>Bromodiolon</td>
<td>Single-dose anticoagulant</td>
<td>Boothill®, Hawk®</td>
</tr>
<tr>
<td>Cholecalciferol</td>
<td>Single-dose/multiple dose, affects blood calcium levels</td>
<td>Rampage®</td>
</tr>
</tbody>
</table>

*aRestricted to use by licensed pest-control operators.
The second type includes the single-dose anticoagulants. Products that contain brodifacoum are examples of this category of baits. A single feeding is sufficient to kill a rodent. They are safe; however, care should be taken to keep them from pets, livestock, and children. They are potentially lethal if a large dose is consumed.

Currently, there are two other types of single-dose baits that are not anticoagulants. The first contains the active ingredient bromethalin, which affects the central nervous system. Baits containing bromethalin are effective, but they are also fairly toxic so extra care should be taken when using this compound. The second type of bait contains cholecalciferol, which is vitamin D₃. Cholecalciferol causes a calcium imbalance in the blood. Since other mammals can tolerate much larger changes in their blood calcium level, this compound is very safe for people and for non-target animals.

The last category of baits includes acute single-dose rodenticides such as zinc phosphide. These chemicals are very effective and useful for a quick knockdown of a large population. However, these chemicals are highly toxic, and most are restricted to use by licensed pest-control operators. Except under extreme circumstances, the other types of bait are equally effective and much safer.

Bait rotation is practiced to initially knock down the rodent population to a manageable population with an acute rodenticide. The next step is to create a rodent-free environment by rotation between anticoagulant and non-anticoagulant baits.

For any baiting program to be effective, rodents must consume a lethal amount of bait. In order to accomplish this, care must be taken in placement of bait. Random placement of bait around a poultry facility is rarely very effective. Always remember that rodents will not go out of their way to eat poisoned bait if they have food readily available. Baits should be systematically placed at equal distance intervals entirely around all poultry facilities, so that rodents entering a facility will have a high probability of encountering bait before entering a house (figures 18.7, 18.8, and 18.9).
Figure 18.9. Rat control
19. Internal parasites

Introduction

Helminth or worms are very common internal parasites. Nematodes constitute the most common helminth parasites of poultry.

General morphology

Nematodes, or roundworms, are spindle shaped with the anterior and posterior ends attenuated (thinner). The body covering, or cuticle, is often marked by transverse grooves. Cuticular fins, or alae, may be present at the anterior (cervical alae) or posterior (caudal alae) part of the body. The latter are found on the tail of the male worm, and in the case of certain groups, are modified to form bursa. Cuticular ornamentations are occasionally found on the anterior extremities and may take the form of spines, cordons, or shields.

The mouth opening, located at the anterior end of the body, is usually surrounded by lips bearing sensory organs. In more generalized types of nematodes the mouth leads directly into a cavity immediately anterior to the esophagus. The mouth cavity may be reduced or absent in more specialized groups of nematodes. The esophagus may be simple (consisting of one undivided part) or more complex (consisting of a short anterior muscular part and a long posterior glandular part). A bulb may or may not be present at the posterior end. The intestine follows the esophagus and is connected with the anal or cloacal opening in the posterior end of the body by a short rectum.

The nematodes are, with very few exceptions, sexually distinct. Sexual dimorphism (differences) is remarkably demonstrated by some species of nematodes such as Tetrameres americana in which the elongate male worm is much smaller than the globular-shaped female. The male can be distinguished from the female by the presence of two (rarely one) chitinous structures known as spicules, located in the posterior end of the body. The spicules have been considered as intermittent organs for use during copulation, keeping the vulva and vagina open and, to some extent, guiding the sperm into the female. Eggs or larvae are discharged through the vulva, the position of which varies considerably in different groups of nematodes.

Development

Nematodes of poultry have either a direct or an indirect type of development; about one-half require no invertebrate intermediate hosts, whereas the others depend on such intermediate hosts as insects, snails, and slugs for the early stage of development.

Nematodes normally go through four developmental stages before reaching the fifth or final stage. Successive stages are preceded by shedding of the skin (molting). In some nematodes the loosened skin or cuticle is retained for a short time as a protective covering; in others it is shed at once.

Eggs deposited in the location in which the female worms are found ultimately reach the outside in the droppings. Excorporeal (out of the host’s body) existence is necessary for eggs to become
infective for avian or arthropod hosts. The conditions existing within the definitive host are important to the development of the eggs. Outside the host in the required optimum moisture and temperature, these undergo development. Eggs of some nematodes require only a few days to complete embryonation; others require several weeks. For nematodes with direct life cycles, the final host becomes infected by eating embryonated eggs or free larvae. For those internal parasites with indirect life cycles, the intermediate host ingests embryonated eggs or free larvae and retains the larvae within the body tissues. The final host becomes infected either by eating the infected intermediate host or by injection of the larvae by a blood-feeding arthropod.

_Asccridia galli_

**Location**

This worm exists in the lumen of the intestine, occasionally in the esophagus, crop, gizzard, oviduct, and body cavity.

**Morphology**

Worms have a large, thick, yellowish white head with 3 large lips. The male is 50-76 mm long, 490-1.21 mm wide (figure 19.1). It has a pre-anal sucker oval or circular, with strong chitinous wall with a bacilliform interruption on its posterior rim; tail with narrow caudal alae or membranes and 10 pairs of papillae. The first pair of ventral caudal papillae is anterior to the pre-anal sucker, the fourth pair is widely separated (compare with _A. dissimilis_); spicules nearly equal and narrow, and end blunt with a slight indentation. The female is 60-116 mm long, 900-1.8 mm wide; the vulva is in anterior part of body, eggs are elliptical, thick-shelled, and not embryonated at time of deposition (figure 19.0).

*Figure 19.0. Worm egg*
Life cycle

The life history is simple and direct. Infective eggs hatch in either the proventriculus or the duodenum of the susceptible host. The young larvae, after hatching, live free in the lumen of the posterior portion of the duodenum for the first 9 days. They then penetrate the mucosa and cause hemorrhages. The young worms enter the lumen of the duodenum by 17 or 18 days and remain there until maturity, at approximately 28-30 days after ingestion of embryonated eggs. Larvae may enter the tissues as early as the 1st day and remain there as long as 26 days after infection. The majority spend from 8-17 days in the intestinal mucosa. A few of the larvae penetrate deep into the tissue, while the majority undergoes only a brief and shallow association with the intestinal mucosa during the "tissue phase". *A. galli* eggs ingested by grasshoppers or earthworms hatch and are infective to chickens, although no development of the larvae occurs.

Under optimum temperature and moisture conditions, eggs in the droppings become infective in 10-12 days; under less favorable conditions a longer time is necessary. Eggs are quite resistant to lower temperatures.

Pathogenicity

*A. galli* infection causes weight depression in the host, which correlates with increasing worm burden. In severe infections, intestinal blockage can occur. The nutritional state of the host is also important, since weight depression is greater with high dietary levels of protein (15%) than with low levels (12.5%). Chickens infected with a large number of ascarides suffer from loss of blood, reduced blood sugar content, increased urates, shrunken thymus glands, retarded growth, and greatly increased mortality. However, no effects of infection on blood protein level, packed-cell volume, or hemoglobin levels were found. *A. galli* can also have detrimental effects through interaction (synergism) with other disease conditions such as coccidiosis and infectious bronchitis. *A. galli* has also been shown to contain and transmit avian reoviruses.

One of the most striking effects of infection, at least from an aesthetic standpoint, is the occasional finding of this parasite in commercial egg. Presumably the worms migrate up the oviduct via the cloaca, with subsequent inclusion in the egg. Infected eggs can be detected by candling, thus eliminating a potential consumer complaint.
Immunity

The age of the host and severity of exposure play a role in *A. galli* infections. Chickens 3 months or older manifest considerable resistance to infection with *A. galli*. In older fowl, larvae are recovered that have undergone little or no development since emerging from the egg. Larval development is arrested in the third stage at high dose rates as a result of resistance rather than a density-dependent phenomenon. Heavier broiler breeds are more resistant to ascarid infections than are the lighter White Leghorns.

*Capillaria* (crop worm)

Location

This worm is found in the mucosa of mouth, esophagus and crop.

Morphology

The body is thread-like, attenuated anteriorly and posteriorly; head with (*C. annulata*) or without (*C. contorta*) a cuticular swelling (figure 19.2). The male is 8-17 mm long, 60-70 mm wide, has two terminal laterodorsal prominences on tail end a spicule very slender and transparent, about 800mm long and a spicule sheath covered with fine hair-like processing. The female is 15-60 mm long, 120-150 mm wide; the vulva is prominent, circular, and 140-150 mm posterior to beginning of intestine.

![Figure 19.2. Capillaria worm](image)

Life cycle

The cycle is direct for *C. contorta* and indirect for *C. annulata*, which requires the earthworm as an intermediate host. Eggs pass out the rectum and embryonate on the ground or in the
earthworm in about 1 month. Embryos or earthworms, containing the embryos, are consumed by the bird. Worms mature in the host in about 1 month.

**Pathogenicity**

When present in large numbers, these worms are extremely pathogenic and can result in death especially in turkeys, partridges, pheasants, guinea and quail. They are less common and less pathogenic in chickens. Signs are principally malnutrition and emaciation, associated with severe anemia. Usually there is inflammation (thickening and roughening) on the crop and esophageal walls.

*Heterakis gallinarium* (*cecal worm*)

**Hosts**

Chickens, turkeys, ducks, geese, grouse, guinea fowl, partridges, pheasants, quail can be infected.

**Location**

The worms are found in the lumen of the cecum.

**Morphology**

The worms are small and white, with the following characteristics: the head end bent dorsally, mouth surrounded by 3 small equal-sized lips; 2 narrow lateral membranes extend almost entire length of body; and esophagus ending in a well-developed bulb containing a valvular apparatus. The male is 7-13 mm long, with a straight tail ending in a subulate point. It has two large lateral bursal wings, a well-developed pre-anal sucker, with strongly chitinized walls and a small semicircular incision in posterior margin of the sucker wall. It has 12 pairs of caudal papillae, the 2 most posterior parts stout and superimposed; and dissimilar spicules, the right one 0.85-2.8 (generally 2) mm long, the left one 0.37-1.1 mm long with a curved tip. The female is 10-15 mm long, with the following characteristics; long, narrow, pointed tail; non-prominent vulva, slightly posterior to the middle of the body; and thick-shelled eyes, ellipsoidal, unsegmented when deposited, similar in appearance to those of *A. galli*, 63-75 mm x 36-50 mm.

![Figure 19.3. Adult Heterakis worms in the cecum.](image-url)
**Life cycle**

The greatest production of eggs for each egg ingested is with the ring-necked pheasant, followed by the guinea fowl and chicken. Eggs pass out in the feces in an unsegmented state. In approximately 2 weeks or less, under favorable conditions of temperature and moisture, eggs reach the infective stage. When they are swallowed by a susceptible host, the embryos hatch in the upper part of the intestine. By the end of 24 hours most of the young worms have reached the ceca. The larvae are closely associated with or occasionally embedded in the cecal tissue until 12 days post-exposure, with peak association at 3 days. Tissue association increases with age of birds; nevertheless, a true tissue phase rarely occurs with *H. gallinarum*. At necropsy most of the adult worms are found in the tips or blind ends of the ceca (figure 19.3). Earthworms may also ingest the eggs of the cecal worms and may be the means of causing infection in poultry.

**Pathogenicity**

The ceca of experimentally infected birds show marked inflammation and thickening of the walls. In heavy infections, nodules form in the mucosa and submucosa.

The chief economic importance of the cecal worm lies in its role as a carrier of the blackhead organism *Histomonas meleagridis*. Blackhead may be produced in susceptible birds by feeding embryonated eggs of *H. gallinarum* taken from blackhead-infected birds. The protozoan parasite is found incorporated in the worm egg and its presence identified in the gut wall and in the reproductive systems of the male and female and in the developing eggs of this cecal worm. Direct transmission of *Histomonas meleagridis* can be accomplished using larvae and male worms.

**Syngamus trachea (gape, red or forked worm)**

**Hosts**

Chickens, turkeys, geese, guinea fowl, pheasants, peafowl, quail can be infected.

**Location**

The organism is found in the trachea, bronchi, and bronchiolus.

**Morphology**

It is sometimes designated as "redworm" because of its color or "forked worm," because the male and female are in permanent copulation so that they appear like the letter "Y". The mouth is orbicular, with the hemispheric chitinous capsule, usually with 8 sharp teeth at the base, surrounded by a chitinous plate, the outer margin of which is incised to form 6 festoons opposite each other. The male is 2-6 cm long, 200mm wide, with the following characteristics; obliquely truncated bursa, provided with rays, sometimes with strikingly asymmetrical dorsal rays; and equal, slender, short (57-64 mm) spicules (figure 19.4). The female is 5-20 mm long (longer in the turkey), 350 mm wide has a conical tail end, bearing a pointed process; prominent vulva,
about 1/4 of body length from anterior end, position varying with age; and 90 mm x 49 mm, ellipsoidal, operculated eggs.

Figure 19.4. Adult *Syngamus* worm in the trachea.

**Life cycle**

The life history of this gapeworm is peculiar in that transmission from bird to bird may be successfully accomplished either directly (by the feeding of embryonated eggs or infective larvae) or indirectly (by ingestion of earthworms containing free or encysted gapeworm larvae they had obtained by feeding on contaminated soil). The female gapeworm deposits eggs through the vulvar opening underneath the bursa of the attached male onto the lumen of the trachea. The eggs reach the mouth, are swallowed, and pass to the outside in the droppings. Following incubation of approximately 8-14 days under optimum moisture and temperature, eggs embryonate, and soon after some may hatch, with the larvae living free in the soil. The earthworms become infected with gapeworm larvae. Within the earthworm the larvae penetrate the intestinal wall, enter the body cavity, and finally invade the body musculature in which they may encyst for an indefinite period.

Gapeworm larvae in the earthworm remain infective to young chickens for as long as 4-1/3 years. Slugs and snails may also serve as transfer or auxiliary hosts of larvae, and live larvae have been recovered from snails over a year after infection. Snails are not true intermediate hosts in the strict sense, since they are not necessary for the transfer of gapeworms to their bird hosts. *S. trachea* taken from various wild and domestic birds was more readily transferred to young chickens with a greater degree of success if the earthworm was employed as an intermediary.
Some infective larvae penetrate the wall of the crop and esophagus and then penetrate the lungs directly. However, the majority penetrates the duodenum and is carried to the lungs by the portal bloodstream via the liver and heart. Larvae are found in the liver as early as 2 hours post-inoculation and in the lungs as early as 4 hours. Larvae probably break out of the capillaries in the lung into the interlobular connective tissue and migrate into the parabronchia and atria via air capillaries. Molting and development to the adult stage can occur as early as 4 days post-infection, with copulation by 5 days in pheasants.

Copulation of the worms in chickens is seen 1 day later. Larvae can be recovered in the lungs up to 7 days. Worms are also found in the parabronchi and secondary bronchi up to 9 days. Adults enter the trachea as early as 7 days, and males are firmly attached to the tracheal wall by 11 days post-infection. Approximately 2 weeks are required for the infective larvae to reach sexual maturity and for eggs to appear in the droppings. Although the role played by wild birds in the spread of gapeworm disease is still undecided, wild birds probably do not spread the disease in this country.

**Pathogenicity**

In the United States, *S. trachea* is the causative agent of "gapes" (labored breathing due to parasites) in chickens, turkeys, peacocks, and pheasants.

In artificial rearing of pheasants, gapes is a serious menace in the United States. Confinement rearing of young birds has reduced the problem in chickens compared to a few years ago. However, this parasite continues to present an occasional problem with turkeys raised on range.

Young birds are most seriously affected with gapeworms. The rapidly growing worms soon obstruct the lumen of the trachea and cause suffocation. Turkey poults, baby chicks, and pheasant chicks are most susceptible to infection. Turkey poults usually develop gapeworm signs earlier and begin to die sooner after infection than young chickens. Full-grown birds rarely show characteristic signs unless heavily infected.

Birds infected with gapeworms show signs of weakness and emaciation and usually spend much of their time with eyes closed and head drawn back against the body. From time to time they throw their heads forward and upward and open the mouth wide to draw in air. An infected bird may give its head a convulsive shake in an attempt to remove the obstruction from the trachea so that normal breathing may be resumed. Little or no food is eaten by birds in the advanced stages of infection, and death usually ensues.

Examination of the trachea of infected birds shows that the mucous membrane is extensively irritated and inflamed. Coughing is apparently the result of this irritation to the mucous lining. Lesions are usually found in the trachea of turkeys and pheasants but seldom if ever in the trachea of young chickens and guinea fowl. These lesions or nodules are produced as a result of an inflammatory reaction at the site of attachment of the male worm, which remains permanently attached to the tracheal wall throughout the duration of its life. The female worms apparently detach and reattach from time to time in order to obtain a more abundant supply of food.
Prevention and control

Modern poultry practices, especially confinement rearing of broilers and pullets and caging of laying hens, have significantly influenced the quantity and variety of nematode infections in poultry. Many that caused extensive problems in "backyard" or "farmyard" flocks are seldom seen in commercial operations. Others such as Ascaridia are still found in commercial birds. In addition, increased pen rearing of game birds has led to increasing nematode problems in these species.

For most nematodes, control measures consist of sanitation and breaking the life cycle rather than chemotherapy. Confinement rearing on litter largely prevents infections with nematodes using intermediate hosts such as earthworms or grasshoppers, which are not normally found in poultry houses. Conversely, nematodes with direct life cycles or those that utilize intermediate hosts such as beetles, which are common in poultry houses, may prosper. Treatment of the soil or litter to kill intermediate hosts may be beneficial. Insecticides suitable for litter treatment include carbaryl, tetrachlorvinphos (stirofos), or Ronnel®, Safecide® from Schering-Plough or Tempo* from Bayer. However, treatment is usually done only between grow-outs. Extreme care should be taken to ensure that feed and water are not contaminated. Treatment of range soil to kill ova is only partially successful. Changing litter can reduce infections, but treating floors with oil is not very effective. After the old litter has been removed, spraying with permethrin or Ravap® (a mixture of Rabon and Vapona) has proven effective for beetle control.

Raising different species or different ages of birds together or in close proximity is a dangerous procedure as regards parasitism. Adult turkeys, which are carriers or gapeworms, can transmit the disease to young chicks or pheasants, although older chickens are almost resistant to infection.

Ascaridia and Heterakis - approved compounds

Only piperazine is currently approved for use in chickens. Piperazine compounds have been widely adopted as a method of treatment for Ascaridia, since they are practically nontoxic. Piperazine may be administered to chickens in the feed (0.2-0.4%) or water (0.1-0.2%), or as a single treatment (50-100 mg/bird). The rate for feed or water medication in turkeys is the same; however, the single-treatment dose is 100 mg/bird (under 12 weeks) and 100-400 mg/bird (over 12 weeks). A high concentration of piperazine in contact with worms at a given time is very important for maximum elimination. Therefore, to be most effective, piperazine should be consumed by birds in a period of a few hours. Piperazine in drinking water is the most practical method of application for commercial flocks. Since piperazines are available as a wide variety of salts, the level should be calculated on the basis of milligrams of active piperazine. Piperazine compounds exert a narcotizing effect, thus enabling worms to be removed and expelled alive by means of natural peristalsis. The adult worms quickly die when exposed to the outside environment. Fenbendazole (SafeGuard) from Intervet, Inc. of Millsboro Del., is approved for use in controlling ascarids in turkeys. It is administered in the feed at 16 ppm.
Capillaria

Tramisol (levamisole hydrochloride) from Schering-Plough has been effective when give at 16mg/kg body weight by drinking water for 3 to 4 hours. It requires a Veterinary prescription.

Heterakis

Piperazine, levamisole, and benzamidzole type wormers such as tetramisol and allbendazole in a form which is highly soluable in water and can be distributed with a medicator.

Syngamus

Thiabendazole is currently approved for use only in pheasants at a level of 0.05% for 2 weeks. Thiabendazole is effective when administered in the feed. Mash containing 0.5% thiabendazole fed to 4-week-old turkey poults for 9-20 days removed 98% of the gapeworms from 117 birds. The drug appeared effective whether treatment was initiated on post-infection day 30 or started on the day of infection. Continuous medication of pen-reared birds at levels of 0.1-4% has been recommended, but is not economical.

Several other compounds have been shown effective against Syngamus. Mebendazole® (methyl 5-benzoyl-2-benzimidazole*, or Carbamate®) was 100% efficacious when fed prophylactically at 0.0064% and curatively at 0.0125% to turkey poults. A level of 0.044% for 14 days has also been effective.

Cambendazole®[5-isopropoxycarbonylamino-2-(4-thizoly)-benzimidazole] was found to be more efficacious than thiabendazole or disophenol (2, 6-diodo-4-nitrophenol). The level of control with three treatments of cambendazole* on days 3-4, 6-7, and 16-17 post-infection was 94.9% in chickens (2mg/kg x 50mg/kg) and 99.1% in turkeys (2mg/kg x 20mg/kg).

Levamisole*, fed at a level of 0.04% for 2 days or 2 g/gal drinking water for 1 day each month, has proven effective in game birds. Fenbendazole at 20 mg/kg for 3-4 days is also effective.
*Not approved for use in commercial poultry.

Cestodes

Introduction

Fifty percent of the intestinal tracts of chickens or turkeys may contain tapeworms if they are reared on range or in backyard flocks. These parasites are found more frequently late in the summer when intermediate hosts are abundant. In contrast, birds confined within poultry houses seldom become infected. Tapeworm infestations are now considered rare in intensive poultry-rearing regions. Beetles and houseflies inhabiting poultry houses still act as intermediate hosts for Raillietina cesticillus.
Some larger tapeworms may appear to completely block the intestine of infected birds, producing serious problems to poultry. Different species vary considerably in pathogenicity so identification as to species is desired. Unfortunately, diagnosticians have often been satisfied with a diagnosis of "cestodiasis".

Tapeworms or cestodes are flat, ribbon-shaped, segmented worms. The term proglottid is used to describe these individual "segments", since the latter term is reserved by classical zoologists as a characteristic demonstrated in other phyla. One to several gravid proglottides is shed daily from the posterior end of the worm. Millions of eggs may be required to complete the complicated two-host or three-host life cycle. Each proglottid contains one or more sets of reproduction organs, which may be crowded with a mass of eggs as the maturing proglottid becomes gravid.

Tapeworms are characterized by complete absence of a digestive tract and obtain their nourishment by absorption from the gut contents of the host. Although the duodenum, jejunum, or ileum is the usual site for attachment of the scolex ("head"), one species (*Hymenolepis megalops*) from ducks is found attached to the cloaca or bursa of Fabricius. Birds become infected by eating an intermediate host, which transmits a larval stage of the tapeworm to the intestine of the definitive host. This larval tapeworm is known as a cysticercoid. The intermediate host may be an insect, crustacean, earthworm, slug, snail, or leech depending upon the species of tapeworm.

Most cestodes are usually host specific for a single or a few closely related birds. Identification of the genus and species may provide a clue to the probably intermediate host. The diagnostician may then be able to suggest practical control measures. Completion of a two-host life cycle depends upon a unique set of ecologic conditions. Thus minor changes in flock management may cause a break in the life cycle and affect a useful control measure.

**Classification**

Over 1400 species of tapeworms have been described from wild and domestic birds. Since most of them have no common name, they are best recognized by their genus and species names. Descriptions of species together with keys and more complete classification into higher taxa will be found in separate works.

The 3 families (*Davainidae, Dilepididae, Hymenolepidae*) and 10 genera (*Amoebotaenia, Choanotaenia, Davainea, Diorchis, Drepanidotaenia, Imparmargo, Metroliasthes, Raillietina, Hymenolepis, Fimbriaria*) recognized here may appear in birds brought to diagnostic laboratories in the United States.

**Morphology and life cycles of adults**

The anatomic features needed to identify poultry tapeworms are illustrated by *Davainea proglottina*. This species differs from most other tapeworms in possessing only one or two each of immature, mature, and gravid proglottides compared to dozens or hundreds in other species. The entire connected chain of proglottides constitutes the strobila. Besides the strobila, the scolex and the neck are recognized. Anchorage is accomplished by the scolex with the assistance of four pairs of suckers or acetabula, which may possess one or two rows of acetabular hooks. If
hooks are present, the species is described as armed; if absent, it is unarmed. A plunger-shaped organ known as the rostellum is frequently present at the anterior end. The rostellum may assist in anchorage by means of one or two rows of rostellar hooks and by the suction created by partial withdrawal of the rostellum into the scolex. The host tissue thus becomes firmly embedded in the rim of the scolex. The neck is an elongated undifferentiated area between the scolex and the strobila from which new proglottides proliferate. The neck area in _D. proglottina_ is so short that it is sometimes listed as missing.

A set of both male and female reproductive organs is found in each proglottid. Morphological differences in size and location of these organs are used in taxonomic descriptions of different species. Older gravid proglottides, containing numerous eggs, are shed individually or in short chains late in the day after the worm has absorbed and stored glycogen from the gut contents of the host. _D. proglottina_ generally sheds 1 gravid proglottid per day while _Raillietina cesticillus_ may produce as many as 10-12 (figure 19.5).

**Figure 19.5. Tapeworm life cycle**

[Image of Tapeworm life cycle]

**Figure 19.6. Adult Raillietina worms found in the intestine**

[Image of Adult Raillietina worms]
**Onchosphere**

Within the uterus the fertilized egg develops into a multicellular embryo. Six hooks are a prominent feature of this stage. The onchosphere (6-hooked or hexacanth embryo) represents a multicellular larva that contains penetration glands and has numerous muscular attachments to activate the hooks. Each proglottid may contain several hundred of these multicellular embryos or "eggs", which may be surrounded by distinctive membranes, which may be useful in identifying the species of chicken tapeworm (figure 19.6).

**Cysticercoid**

Intermediate hosts such as beetles, houseflies, slugs, or snails become infected by swallowing individual eggs from the feces or they devour the entire proglottid after being attracted by odor or movement. The 6-hooked embryo hatches from the egg in the gut of the intermediate host by action of the hooks and penetrates the gut wall. After radical reorganization and a change in polarity, the larva transforms into a cysticercoid. This development requires a minimum of 2 weeks depending upon outside temperatures. The cysticercoid remains within the body cavity of the intermediate host until the latter is eaten by the bird. The cysticercoid is activated by the bile in the definitive host, and the scolex becomes attached to the intestine. The first gravid proglottides appear in the feces 2-3 weeks after the cysticercoid is swallowed by the definitive host.

**Raillietina cesticillus**

*Diagnostic characteristics*

The scolex of this large robust tapeworm (15 cm long) embeds deeply in the mucosa of the duodenum or jejunum. It has a distinctive, wide, flat, rostellum, which bears a double row of 300-500 hammer-shaped hooks. Flattened rostellum acts as a retractable piston drawing into an outer sleeve of the scolex, thus providing a firm grip on the mucosa. It has four unarmed weak suckers; genital pores alternate irregularly, 20-30 testes posterior in proglottid; and single eggs are encapsulated in uterine membranes. Mature eggs occur with two distinctive funnel-shaped filaments between the middle and inner membranes.

*Life history*

Over 100 species of beetles belonging to 10 families are proven to have natural or experimental intermediate hosts. A minute hysteroid beetle (*Carcinops pumilio*) is the natural intermediate host in broiler houses. Darkling beetles (*Alphitobius diaperinus*), houseflies, grasshoppers, ants, and lepidopterous larvae have proved negative as experimental hosts. As many as 930 cysticercoids have been found in a single ground beetle.

*Pathogenicity*

This parasite may cause emaciation, degeneration and inflammation of villi, reduction of blood sugar and hemoglobin, and reduced growth rate.
**Diagnosis and identification**

Distinctive characteristics of different species of chicken tapeworms may best be demonstrated by examining the scolex or individual proglottides of recently shed, live specimens. Preservation in alcohol or formalin, although required before staining, often obscures useful characteristics needed for rapid identification. The intestine is best opened with scissors under water thus permitting the strobila to float free revealing the area to which the scolex is attached. Recovery of the scolex is worth considerable effort as its characteristics alone may indicate the species. Wet-mount preparations of the scolex examined under a cover glass with 100 times, or higher, magnification may reveal sufficient characteristics to make a species identification. Hook characteristics may require measurement with an ocular micrometer under higher magnification. Semipermanent cleared preparations of scolices may be made by using a drop of Hoyer's solution.

**Prevention and control**

Prevent the birds from having contact with the intermediate host with the use of broad-spectrum insecticides. For layers in cages, collect feces over a water flushable pit, connected to a lagoon to control flys. No drugs are available for treating this worm.
20. Miscellaneous conditions

The following conditions are included here because the etiology is unknown or because the etiology is not due to a single entity.

*Spondylolisthesis (kinky back)*

Species of bird--Broilers.

Action--Chronic.

Age of bird--Young fast growing.

Etiology--It is a developmental disorder influenced by conformation and growth rate, resulting in a deformity of the 6th thoracic vertebra, which causes spinal cord compression and posterior paralysis.

Mode of transmission

1. It is a noninfectious metabolic disease of broilers, which is aggravated by fast body development.

2. It does not occur in breeders, where growth rate is slowed by restricted feeding. It can be increased by genetic selection.

Clinical signs

1. The incidence of affected birds may reach 2% of the flock.

2. Peak incidence occurs at 3-6 weeks of age.

3. Severely affected birds often become laterally recumbent (lay on their sides) and may die from dehydration, if not culled.

4. Lordosis (curving forward) and subclinical spondylolisthesis are common in broilers and develop soon after hatching (figure 20.0).
Figure 20.0. Broiler with kinky back.

Postmortem lesions

1. The posterior paralysis results from rotation of the body of the 6th vertebra along the axis of the spine with the posterior part of the body moving dorsal (backward) and anterior (forward), relative to the anterior part.

2. The rotation causes a kyphotic (humpback) angulation of the floor of the spinal canal between the 6th and 7th thoracic vertebrae, and spinal cord compression. The deformation of the spinal column can be readily recognized by palpating the ventral surface of the spinal column during necropsy (figure 20.1).

Figure 20.1. Decalified “pinched” spinal cords with kinky back

Diagnosis

1. Spondylolisthesis is best confirmed by removing, decalcifying and splitting the spinal column along a midline longitudinal plant to allow visualization of the spinal cord compression.
2. It simulates other developmental skeletal disorders such as valgus (twisted outward) and various deformations of the intertarsal joint, tibial dyschondroplasia, osteochondrosis, degenerative joint disease and osteoperosis.

3. A twisted spinal column resulting in a definitive diagnosis can be seen without decalcifying in extreme cases.

**Prevention**

1. Slowing growth rate as is done in breeders (by restriction of light or feed), and genetic selection of birds that are less susceptible is helpful.

2. Permitting a dark period allows rest, melatonin synthesis and reduces stress. Melatonin, produced by the pineal gland, controls circadian pacemaker and promotes sleep.

3. Common light programs start with 24 hours of 60 lux (intensity) light. That is slowly reduced to 12 hours of 5 lux light at 2 weeks. The lights are slowly increased to 23 hours of 5 lux light at 5 weeks of age.

**Special Note**

It is one of many skeletal problems caused by rapid growth rate in broilers.

**Deep pectoral myopathy (Green Muscle Disease)**

**Species of bird**--Turkeys and broilers.

**Action**--Chronic.

**Age of bird**--Growing.

**Etiology**--Ischemia (inadequate circulation of blood) causes the swelling in a tight fascia (band) of vigorously exercised muscle. There is some evidence of hereditary predisposition, rapid growth rate, and increased handling in turkey breeder hens during artificial insemination.

**Mode of transmission**

1. The cause is a lack of oxygen due to improper blood supply or necrosis around tissues or blood vessels.

2. It is non-contagious and no specific nutritional factors may influence the condition.

**Clinical signs**
1. This is a processing plant problem. No problem is noted in the field.

**Postmortem lesions**

1. Unilateral or bilateral lesions, which do not affect the health, of the bird may occur followed by more chronic lesions resulting in dimpling or flattening of the breast muscle which can be palpated.

2. The whole deep pectoral muscle is swollen, pale and edematous with necrosis in the middle 1/3-3/5 of the muscle, which is only evident after processing or necropsy (figures 20.2 and 20.3).

3. In other lesions, the edema disappears and the necrotic muscle becomes more prominent and drier with greenish areas.

4. Necrotic muscle shrinks and may be enclosed in a fibrous capsule. The sternum adjacent to the necrotic muscle becomes roughened and irregular.

Figure 20.2. Green muscle disease

Figure 20.3. Green muscle disease (lower side)
**Diagnosis**

1. Gross (greenish muscles) and microscopic lesions are characteristic.

2. Microscopically the fibers are swollen and eosinophilic with discoid necrosis. Nuclei are absent or faint. Surrounding the necrotic tissue are inflammatory reactions. Vascular lesions consist of thromboses, intima proliferation, and aneurysm formation.

3. It simulates bacterial (gangrenous), fungal (favus) or nutritional (exudative diathesis) dermatitis.

**Prevention**

1. Slowing growth rate as is done in breeders (by restriction of light or feed), and genetic selection of birds that are less susceptible is helpful.

**Special Note**

It is one of many musculo-skeletal problems caused by rapid growth rate in broilers.

*Acute death syndrome (sudden death syndrome, heart attack and flip-over)*

**Species of bird**—Broilers and turkeys.

**Action**—Peracute.

**Age of bird**—It occurs in 1-8 weeks old bird.

**Etiology**—Unknown cause, but probably a metabolic disease due to genetic, nutritional and environmental factors affecting the incidence and severity.

**Clinical signs**

1. The greatest losses in birds occur from 3-6 weeks of age.

2. Birds squawk during a sudden attack characterized by loss of balance, convulsions, and violent flapping.

3. Most birds die on their backs with one or both legs extended or raised.

**Postmortem lesions**

1. Birds are in good health and flesh.

2. Livers are enlarged, pale and friable and gall bladder is empty (figure 20.4).
3. Kidneys may be pale and lungs are often congested and edematous.

4. The ventricles of the heart are generally contracted and the thyroid, thymus and spleen congested. There may be hemorrhages in the kidney.

5. There can be blood in the abdominal cavity.

![Figure 20.4. Enlarged edematous lung with sudden death syndrome](image)

**Diagnosis**

1. Clinical signs, gross and microscopic pathology are characteristic.

2. Microscopically there is congestion, edema and lymphoid edema in the lungs. Mild degeneration and infiltration of lymphoid cells in the heart is seen.

3. It simulates aortic rupture and ascites.

**Prevention**

Reduce bird density and slow growth and move slowly and quietly through the house to reduce the disease. Acclimate flock to sounds using a radio placed in the house.

*Aortic rupture (dissecting aneurysm) - turkey - heart attack*

**Species of bird**--Fast growing, healthy, heaviest turkeys or broiler (14-20 weeks).

**Action**--Peracute.
Age of bird: It occurs at 7-24 weeks of age and has a higher incidence in males.

Etiology—Intimal plagues in abdominal aorta. Higher blood pressure plays a role.

Mode of transmission

1. Unknown mode of transmission, but genetics and/or nutrition may play a role.
2. It is not contagious.

Clinical signs

1. Heavy fast growing birds die suddenly due to internal hemorrhage and flop on their backs.
2. Mortality rarely reaches 2% of the flock. It is most common between 12 and 16 weeks.

Postmortem lesions

1. A dissecting aneurysm close to kidneys or testes is seen with blood in the abdominal cavity (figure 20.5).
2. The head, skin, and musculature are anemic. Occasionally, blood will run out of the mouth or the oral cavity will be blood-stained.

Figure 20.5. Aortic Rupture

Diagnosis

1. There is a longitudinal slit in the aorta between the external iliac and sciatic arteries.
2. A marked intimal thickening or a large fibrous intimal plaque often occurs in the region or rupture. These gross and microscopic lesions with clinical signs are characteristic.
3. It simulates flip-over syndrome and ascites.
**Prevention**

Slowing growth rate with reduced energy level in the feed, or feed restriction using a lighting program, or use of mash feed. These management factors will reduce the disease, but at a cost of reduce growth.

**Treatment**

Reserpine in the feed can control the condition.

**Special note**

Genetic strains differ in blood pressure level and incidence of the disease. Reduction in incidence due to breeding has been seen.

**Ascities (water belly)—right ventricular failure**

**Species of bird**—Broilers, ducks.

**Action**—Acute to chronic.

**Age of bird.** It is prominent in young fast growing broilers.

**Etiology**—Hypoxia (lack of oxygen) is caused by many agents and leads to constriction of arteries, inducing increased pulmonary arterial pressure, which results in right ventricle hypertrophy. The right ventricular valve becomes weak (flabby), which causes back flow of blood and fluid (ascities) in tissues.

**Mode of transmission**

1. It is noncontagious and influenced by genetics, breeding, nutrition, growth rate, ambient temperature and amount of oxygen in the air.

**Clinical signs**

1. Water belly, abdominal distension, reluctance to move, and dyspnic and cyanotic lesions maybe seen.

2. Affected birds are smaller than normal and listless with ruffled feathers.

3. Mortality ranges from 0.5-20% of the flock.

**Postmortem lesions**
1. Water belly (straw-colored fluid in abdominal cavity) (figure 20.6), blood clots in lungs, enlargement of right side of the heart, liver scarred, congested or mottled with a grayish capsule and irregular surface and shrunken and the lungs are congested, edematous and hemorrhagic (figures 20.8 and 20.9). The normal heart seen in figure 20.7.

**Figure 20.6. Water belly**

![Figure 20.6. Water belly](image)

**Figure 20.7. Normal Heart**

![Figure 20.7. Normal Heart](image)

**Diagnosis**

1. Clinical signs and postmortem lesions (water belly with enlarged right heart valve) are diagnostic.

![Figure 20.8. Enlarged heart on far right](image)  **Figure 20.8. Enlarged heart on far right**

![Figure 20.9. Enlarged heart valve on far right](image)  **Figure 20.9. Enlarged heart valve on far right**

2. Microscopically, cartilagous and osseous nodules occur in the lung, congestion of kidney, edema, congestion and hemorrhages in the heart.

3. It simulates salt, crotalaria and furazolidone toxicity.
**Prevention**

1. Change the environment with less altitude, or change the ration. Use less dense (less energy) nutrients or mash feed instead of pellets or restrictive lighting to reduce growth rate during the first 2 weeks of age.

2. More aeration of house during cooler months when gas brooders are in longer use will also help.

3. Adequate temperature control of house, good air, and litter management.

**Treatment**

Reduced sodium and furazolidone content of diet, and water, feed and/or light restriction will reduce the mortality.

**Special note**

It has a genetic susceptibility. It occurs more in males. Hypoxia caused by altitude or gas fumes, carbon monoxide from brooders, worse in winter (tight house and lower temperatures), stress of mycotoxins, toxic fat, increased salt in the diet and/or coal tar disinfectants causes ascities. It is an important cause of broiler mortality at 5-7 weeks of age, especially in the northern United States and Canada in the winter, and year around in areas around the world with high altitude. New lighting programs (reduced photoperiod) to reduce growth rate during the first 2 weeks of age can reduce mortality due to ascities, flip over syndrome, and leg problems. Such programs might include: 24 hrs of 60 lux green light for the first week; 12 hours of 5 lux green light for the next 2 week; and then 23 hours of 5 lux blue light for the remainder.

**Fatty liver hemorrhagic syndrome (FLS)**

**Species of bird**--Chickens - commercial layers and broiler breeders.

**Action**--Chronic.

**Age of bird**: It occurs in laying hens (usually early in production).

**Etiology**--Excessive consumption of high energy diets, where exercise is restricted in cages or overcrowded breeder houses. It may be compounded in hot weather.

**Mode of transmission**
1. There is an over consumption of feed, positive energy balance, and excessive fat deposition.

2. It usually occurs because of too low calcium in layer diet when birds are just coming into production. Birds in cages overeat to achieve increased calcium requirement.

3. Elevated serum calcium and cholesterol in affected chickens may be due to a hormone imbalance.

4. Diets high in rapeseed meal may aggravate the condition.

**Clinical signs**

1. Signs include straining during oviposition, sudden drop in egg production, thin-shelled eggs, cage layer fatigue, weak, rubbery legs, and depressed sternum and rib cages.

2. Hens are over weight with large pale combs and wattles covered with dandruff.

**Postmortem lesions**

1. Lesions include internal hemorrhage, and a fatty, friable, hemorrhagic liver (figure 20.10).

2. Hematomas (blood tumors) are dark red to brown within the parenchyma (specific cells within the organ).

3. Large amounts of fat in the abdominal cavity and around the viscera can be seen.

![Figure 20.10. Organs (liver and head) seen with FLH](image)

**Diagnosis**

1. Postmortem lesions (enlarged, yellow, hemorrhagic liver) are characteristic.

2. The fat content of livers exceeds 40% dry weight. Hepatocytes are distended with fat vacuoles, varying sized hemorrhages and organizing hematomas.
3. It simulates aflatoxicosis.

**Prevention**

1. Lower calorie intake, increase Ca during the onset of lay and lower rapeseed in the feed.

**Tibial dyschondroplasia (TD)**

**Species of bird**--Chickens (broilers), turkeys, ducks.

**Action**--Chronic.

**Age of bird**--Young.

**Etiology**--1) Failure of the chondrocytes to hypertrophy (increase in size) results in abnormal cartilage, which cannot be invaded by blood vessels; 2) vascular invasion of the cartilage from the metaphysis is not adequate; and/or 3) defective chondrolysis occurs.

**Mode of transmission**

1. This disease is not contagious, and genetic and/or dietary (cation - anion ratio) of the ration or high phosphorus relative to calcium) factors may be involved.

2. Grain high in *Fusarium roseum* or fungicide (tetra methylthiuram sulfate) can cause the disease.

**Clinical signs**

1. Lameness in as many of 30% of the flock (reluctance to move, a stilted gait, and bilateral swelling of the femoral-tibial joints) can be seen.

2. Bowing of the bones is evident and is more severe in roaster birds or males kept over 8 weeks for de-boning with body weight over 5 lbs or 2.2 kilograms.

**Postmortem lesions**

1. Proximal tibiotarsal bone is enlarged and contains an abnormal mass of cartilage (failure of cartilage in growth plate to become calcified) (figures 20.11 and 20.12).

2. Fractures below the abnormal cartilage may occur.
Figure 20.11. Bent tibia (right leg) due to TD

Figure 20.12. Tibial dyschondroplasia (abnormal mass of cartilage)

**Diagnosis**

1. History, clinical signs and postmortem lesions (abnormal mass of cartilage in tibial head) are characteristic.

2. Microscopically, dyschondroplasia is characterized by persistence and accumulation of pre-hypertrophic cartilage and begins as early as the first week of age. Chondrocytes in abnormal cartilage are smaller and shrunken.

3. It simulates perosis, rickets, and osteochondrosis (necrosis of growth plate primarily in the vertebrae and femoral head).
4. Lithoscope can detect TD in birds as young as 1 week of age. This machine is used by basic breeder companies to select out TD in their genetic lines (figure 20.12).

**Prevention**

Slow the growth rate and select strains with a lower incidence of TD.

**Treatment**

1. Diet changes will reduce the disease such as reducing phosphorus relative to the level of calcium.

2. Feed a diet free of *Fusarium* and tetramethylthiurams sulfate.

**Special note**

It is common in broilers, resulting in bowing of tibia tarsus (drum stick). TD, perosis, rickets, viral tenosynovitis, bacterial or mycoplasma synovitis, pale bird syndrome (femoral head necrosis), and Marek's disease may all cause condemnation of parts (wings, thighs, legs) in broilers. Trimming of carcass parts is the fourth leading cause of condemnation in broilers in the US. It is the one of the most common cause of leg weakness in broilers. Genetic selection of grandparent strains using the lithoscope has greatly reduced the incidence and severity of this disease.

![Figure 20.12. Radiographic view of abnormal tibia on the left](image-url)
21. Environmental diseases and Vices

Environmental diseases are normally due to housing large amounts of poultry in a rather restricted, enclosed area. Vices are undesirable behavior patterns resulting in injury to the birds and financial losses.

**Heat prostration**

Older birds (over 5 weeks) and adults (especially heavy breeds) in production are more susceptible to high temperatures accompanied by high humidity. Lacking sweat glands, birds' only method of cooling is by rapid respiration with mouths, a form of evaporative cooling, open and wings relaxed and hanging loosely at their sides. When outside temperatures approach 100°F and relative humidity reaches 90%, body temperatures rise, the birds become week and feed consumption is decreased. If this continues for several days or weeks, the birds may die due to respiratory, circulatory, or electrolyte imbalances. Birds in production will show a dramatic decrease in egg production and shell quality will deteriorate. Every attempt should be made to improve air circulation with additional fans. Insulation of the building and use of white or minum paint on the outside of the building will reflect heat. Water sprinklers can be installed on the roof or over side curtains to cool the house. Installation of high-pressure foggers in the ceiling or evaporation coolers on the side-walls will also significantly reduce inside temperatures when the relative humidity falls below 75% (figures 21.0 and 21.1.)

A system of tunnel ventilation (exhaust fans at one end of the house and air inlets at the other end) will provide significantly more efficient cooling and heating throughout the year (figure 21.2). Reducing bird concentration and providing bird’s cool water with vitamin C, electrolytes and/or aspirin will also reduce heat stress. Increasing the nutrient density of the feed (increased energy and protein) and/or feeding early in the morning or late in the evening are also important during hot weather when the birds' feed intake is less. This is a common practice in the Southeastern US during the summer months.

![Figure 21.0. Evaporative cooled house](image1)

![Figure 21.1. Side wall fans to increase air circulation](image2)
Smothering

Smothering is caused by crowding or piling in a corner. It may occur when birds are moved to new quarters, when they are frightened by a loud noise or intruder in the house, or when birds are chilled. It is often more common at night. Smothering of chicks occurs in boxes due to overcrowding or when boxes are staked too high, or when there are insufficient air ventilation holes. A postmortem exam of birds reveals congestion of the trachea and lungs.

The condition can be reduced by encircling chicks around the brooding area with a guard. Guards should be made of a netting material, which provides no blockage of air or light and can be easily disinfected. The house should be prewarmed 24 hours before receiving the chicks. When the birds are moved to new quarters, lights should be dimmed and birds frequently checked for piling. Care should be made to prevent frightening the birds by loud intruders. Condition birds to humans and outside noises (trucks, machines, airplanes) with an inside radio.

Dehydration

It is caused by failure of the chicks to find water or newly hatched chicks being kept in machines and hatchery over 24 hours before reaching the farm. Chicks can survive several days without water, but will generally become weak or die after 3-4 days with no water. Dehydration can be determined by the inability of the chick to "peep", insufficient weight for the size and age, and dehydrated (dark and wrinkled) skin around the shanks. Blood vessels on the skin will be very prominent. Other changes include blue discoloration of the beak, dry and dark breast musculature, dark kidneys, accumulation of urates in the ureters, and darkening of blood.

To prevent this condition adequate watering space should be available in the brooding area inside a circular brooding guard. Mini-chick drinkers should be placed in the brooding area prior to the reception of the chicks. Care should be taken to show the birds how to drink from the automatic mini drinkers. Larger automated drinker systems including troughs, nipple drinkers, and bell-shaped drinkers, if used, should be in place no later than 7 days of age. The two watering systems should overlap by at least 3 days to ensure a smooth transitional phase between the two systems. If nipple drinkers are used, rubber bands can be placed over a few or drip catching devices used to aid chicks in learning to drink. Timing of setting, hatching and chick pulling
needs to be coordinated so that chicks spend no more than 12 hours in the machines after hatching and are processed (vaccinated, debeaked and sexed) in less than 8 hours and reach the house in less than 24 hours after hatching. Chicks should be pulled from the hatcheries when 75% have a small humid ring around the head.

**Vices**

**Cannibalism**

**Vent-picking**

Picking of the vent or region of the abdomen several inches below the vent is the most severe form of cannibalism (figures 21.3 and 21.4). This is generally more commonly in high-production or overweight pullet flocks. Predisposing factors are prolapse or tearing of the tissues by passage of an abnormally large egg. Vent picking can result in anemia.

![Figures 21.3 and 21.4. Vent picking](image)

**Feather-pulling**

Frequently seen in flocks kept in close confinement resulting in lack of sufficient exercise (figure 21.5). Nutritional deficiencies may contribute to the problem.

**Toe-picking**

Most commonly seen in young birds. Inadequate feeder space or inability of the chick to find the feed (starting chicks over paper) will lead to toe-picking.
**Head-picking**

Follows injuries to the comb or wattles. Area around eyes and ears may be black and blue with hemorrhage.

**Egg-eating**

Follows conditions that favor egg breaking. Need to reduce light and have frequent egg collection and removal of floor eggs.

**Etiology**

Predisposing factors include insufficient feed or feeder space, high-density rearing, excessive light, too much heat, nutritional deficiencies or irritation from external parasites.

![Figure 21.5. Back Picking](image)

**Prevention**

Provide adequate feed and feeding space, reduced bird density, reduced light, beak trimming, toe and comb trimming in breeders and wattle trimming in cage birds. Chick feeder lids or paper should be filled with feed and be in placed prior to the reception of the chicks. Automatic feeders (pan-shaped or troughs) should be available no later than 7 days of age. Feed lids and automatic feeders should overlap by 3 days to ensure smooth transition between the two feeding systems.
Table of Equivalents

Weight of Water

1 cubic foot of water weighs 62.4 lb
1 U.S. gallon of water weighs 6.33 lb
1 Imperial gallon of water weighs 10.00 lb
1 Imperial gallon of water weighs 4.54 kilos

Liquid Measure

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Linear Measure Equivalents

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Weights Equivalents

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## Liquid Volume Equivalents

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<th>Quart (IMP)</th>
<th>Gallon (U.S.)</th>
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Management Surveys

Broiler farm visit
Breeder Farm visit
Hatchery visit

BROILER FARM VISIT

By: __________________________ (Person)
Date: _________________________

1. Farm name _______________________
   A. Source of Chickens
      Hatch due ____________________
      1. Hatchery: _____________
      2. Breeder Flock: ___________
         Breed: ___________ Name: ______________

   B. Number of chicks: _____________
      Number of houses per farm: _____________

   C. Entrance to farm
      1. fence: ______________
      2. drive-through disinfection: ____________
      3. vegetation: ____________
      4. trash: ____________
      5. antiroom: ____________
      6. record room: ____________
      7. shower room: ____________
      8. change of coveralls: ____________
      9. other: ____________

   D. House Number
      Inside of house: ______________
      Enclosed: ______________
      1. lighting: ______________
      2. litter quality: ______________
      3. ventilation: ______________
      4. air quality: ______________
      5. temperature °C: ______________
      6. % relative humidity: ______________
      7. feed system: ______________
         a. type: ______________
      8. water system: ______________
         a. type: ______________
         b. condition: ______________
         c. disinfection: ______________
d. TDS: _______________

9. curtains: _____________

10. insulation: ___________

11. sanitation and hygiene: ___________
   a. cleanliness: ___________
   b. exterior parasites: ___________
   c. internal parasites: ___________
   d. foot bath: ______________
   e. anteroom: ______________
   f. record room: ___________

12. miscellaneous: ___________

13. heating system: ___________

14. cooling system: ___________

E. Condition of birds
1. comfort: ___________

2. quality: ___________
   a. debeaking: ___________
   b. sexing: ___________

3. morbidity: ___________

4. % mortality: ___________
   1 wk __ 2 wks __ 3 wks __
   4 wks __ 5 wks __ 6 wks __

5. vaccination program
   a. MDV: ___________
   b. NDV: ___________
   c. IBV: ___________
   d. IBDV: ___________
   e. Fowl pox: ___________
   f. reovirus: ___________

6. medication program: ___________
   a. feed
      1) starter: ___________
      2) grower: ___________
      3) finisher: ___________
   b. water: ___________

F. Overall management of house: ___________

G. Overall management of farm: ___________

H. Specific deficiencies
1. ___________

2. ___________

3. ___________

4. ___________

I. Farm manager: ___________
BREEDER FARM VISIT

By: ___________________
Date: ___________________

I. Farm Name: ___________
Hatch date: ______________

A. Source of chickens: ___________
   1. hatchery: ___________
   2. grandparent flock: ___________
      breed ___________
      name ___________

B. Number of males placed: ___________
Females placed: ___________
Number of house per farm: ___________

C. Entrance to Farm
   1. fence: ___________
   2. drive-through disinfection: ___________
   3. vegetation: ___________
   4. trash: ___________

D. House number
   Inside of house: enclosed: ___________
   Curtain: ___________
   1. lighting
      a. type: ___________
      b. program: ___________
   2. litter quality: ___________
   3. ventilation: ___________
   4. air quality: ___________
   5. temperature °C: ___________
   6. relative humidity: ___________
   7. feed system: ___________
      a. type: ___________
      b. condition: ___________
      c. program: ___________
   8. water system: ___________
      a. type: ___________
      b. condition: ___________
      c. disinfection: ___________
      d. TDS: ___________
   9. curtains: ___________
   10. insulation: ___________
11. sanitation and hygiene:
      a. cleanliness: ___________
      b. external parasites: ___________
      c. internal parasites: ___________
      d. foot bath: ___________
      e. antiroom: ___________
f. record room: __________
12. heating system: __________
13. cooling system: __________
14. nest boxes: ______________
   a. amount: ______________
   b. size: ______________
   c. positioning: __________
   d. condition: ___________
15. nest litter: ______________
16. slates: ________________

E. Condition of birds:
1. comfort: ______________
2. quality: ______________
3. morbidity: ______________
4. % mortality:
   a. growing: __________
   b. laying: __________
5. body weight
   a. males: __________
   b. females: __________
6. body weight uniformity
   a. males: __________
   b. females: __________
7. vaccination program
   a. MDV: __________
   b. NDV: __________
   c. IBV: __________
   d. IBDV: __________
   e. Fowl pox: __________
   f. Reovirus: __________
   g. Cholera: __________
   h. AE: __________
   i. ILT: __________
   j. Coccidiosis: __________
   k. Other: __________
8. medication program
   a. feed: __________
   b. water: __________
9. debeaking: __________
10. sexing: ____________

F. Hen house production
1. egg collection
   a. trolley: __________
   b. manual: __________
2. egg sanitation
a. cleaning: __________
b. spraying: __________
c. culling: __________

3. egg production
   a. hen day production: __________
   b. hen house production: __________
   c. fertility: __________
   d. hatchability: __________
   e. shell quality: __________
   f. egg size: __________

4. egg storage
   a. cleanliness: __________
   b. temperature: __________
   c. % relative humidity: __________
   d. fumigation: __________
   e. egg shell quality: __________

G. Overall management of house: __________
H. Overall management of farm: __________
I. Specific deficiencies: __________
   1.: __________
   2.: __________
   3.: __________
   4.: __________
J. Farm manager: __________
HATCHERY VISIT

By: __________________
Date: ________________

I. Hatchery Name: _________________
   A. Number of eggs set per week: ______________
   B. Actual capacity of hatchery: ______________

II. Outside of Hatchery
   A. Fence: ______________
   B. Drive-through disinfection: ______________
   C. Vegetation: ______________
   D. Trash: ______________
   E. Chick waste disposal: ______________
   F. Ventilation system: ______________
   G. Road to farm: ______________

III. Inside of Hatchery
   A. Shower room: ______________
   B. Records room: ______________
   C. Fumigation system: ______________

IV. Egg Storage Room
   A. Temperature: ______________
   B. % Relative humidity: ______________
   C. Organization: ______________
   D. Cleanliness: ______________
   E. Egg size: ______________
   F. Egg shell quality: ______________

V. Egg Setter Room
   A. Temperature °C: ______________
   B. % Relative humidity: ______________
   C. Machines: ______________
      1. type ______________
      2. condition: ______________
      3. number: ______________
   D. Cleanliness: ______________
   E. Odor: ______________
   F. Exploders: ______________
   G. Sanitation system: ______________

VI. Egg Hatcher
   A. Temperature: ______________
   B. % Relative humidity: ______________
   C. Machines: ______________
      1. type ______________
      2. condition: ______________
      3. number: ______________
   D. Uniformity of hatch: ______________
   E. Sanitation system: ______________
VII. Chick Pool Room
A. Organization: ___________
B. Handling of chicks: ___________
C. Cleaning of hatching trays: __________

VIII. Chick Processing Room
A. Temperature: _____________
B. Organization: _____________
C. Vaccination crew: ___________
   1. MDV: ___________
      a. automatic: ___________
      b. manual: ___________
      c. efficiency: ___________
   2. NDV-IBV: _____________
      a. spray: ___________
         1) automatic: ___________
         2) manual: ___________
      b. eye drop: ___________
      c. efficiency: ___________
D. Debeaking: ___________
E. Sexing: _____________

IX. Vaccination Room
A. Separate: ___________
B. Enclosed: ___________
C. Sterilization system: ___________
D. Liquid nitrogen: ___________
E. Diluents: ___________
F. Antibiotics: ___________
G. Cleanliness: ___________
H. Organization: ___________
I. Cooling System: ___________
J. Thawing of vaccines: ___________
K. MDV type ___ source ___
   NDV type ___ source ___
   IBV type ___ source ___
   Reovirus type ___ source ___
   IBDV type ___ source ___

X. Chick Bus System
A. Separate room: ___________
B. Cleanliness: ___________
C. Temperature: ___________
D. Condition of bus: ___________
   1. heating system: ___________
   2. cooling system: ___________
   3. ventilation: ___________

XI. Records Room Breeds: ___________
A. Breeder flock performance: ___________
   1. % hen house production: __________
2. % hen day production: ____________
3. % fertility: ____________
4. % hatchability: ____________
5. specific gravity: ____________

B. Hatchery performance: ____________
   1. sanitation check: ____________
      a. plating-microbiology: ____________
      b. fungus bacterial: ____________
      c. storage: ____________
      d. setters: ____________
      e. hatchers: ____________
      f. vaccination room: ____________
      g. chick processing room: ____________

2. egg breakout: % mortality
   early: ____________
   mid: ____________
   late: ____________
   true fertility: ____________

XII. Hallways
A. Air flow: ____________
B. Cleanliness: ____________
C. Foot bath: ____________
D. Air sanitation: ____________

XIII. Overall Management of Hatchery: ____________

XIV. Specific Deficiencies
A.: ____________
B.: ____________
C.: ____________

XV. Hatchery manager: ____________
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**Other Articles**

Glossary

abscess: A pocket of pus (chronic inflammation).

active immunity: Immunity produced either by natural exposure or by vaccination.

acute disease: A disease of short and severe duration.

agglutination test: A test for the presence of antibodies performed by mixing serum and an antigen.

agglutinin: A substance that causes bacteria or blood corpuscles to coalesce or clump together.

airsacculitis: Inflammation of the air sacs.

anamnestic response: The improved immune response exhibited by an individual with former exposure to the specific antigen.

anemic: A shortage of hemoglobin or red blood cells.

anion: A negatively charged ion.

anionic (detergent): Any class of synthetic compounds whose anions are alkali salts, a soap, or whose ions are ammonium salts.

anthelmintic: Capable of expelling or destroying parasitic worms especially from the intestines.

antibiotic: A substance produced by microorganisms that has the power to kill or inhibit the growth of other organisms.

antibody: A substance formed in the body as a result of infection or administration of suitable antigens.

antigen: A protein or carbohydrate that produces antibodies when injected into the bird.

antiseptic: A substance applied to animals that reduce microorganisms to a harmless state, either by killing them or preventing their growth.

antitoxin: A specific antibody capable of neutralizing a specific toxin.

artery: A blood vessel which carries blood away from the heart.

arthritis: An inflammation of a joint.

ascites: A collection of fluid in the abdominal cavity (water belly).

aseptic: Free from pathogenic organisms.
ataxia: Uncoordinated muscular movements (as in avian encephalomyelitis).

atrophy: Shrunken tissue or organ.

attenuated: A disease organism that has been weakened to reduce its virulence.

autogenous vaccine: A vaccine prepared from cultures derived from infected birds and used to immunize the host against that same strain of organism.

avirulent: An organism that is not virulent or pathogenic.

avitaminosis: A disease or malfunction caused by a vitamin deficiency.

B-cells: Cells of the immune system, which are capable of being transformed to plasma cells, which produce antibody.

bacteria: Microscopic organisms that are composed of a single cell.

bactericide: A substance that kills bacteria, but not necessarily their spores.

bacteriostat: A substance that inhibits the growth of bacteria without killing them.

benign: Localized tumor.

blood test  See agglutination test.

broad-spectrum antibiotic: An antibiotic that inhibits the growth of many kinds of microorganisms (the more numerous the kinds, the wider the spectrum).

bursa of Fabricius: A small lymphoid gland adjacent to the upper part of the cloaca involved in the processing and maturation of B-cells of the humoral or antibody mediated immune system.

carrier: A host that shows no evidence of a disease yet harbors the organism and is capable of transmitting it to others.

caseous: Cheesy exudates (chronic inflammation).

catarrhal: An inflammation of the mucous membranes.

cation: Positively charged atom.

cellulitis: Inflammation of cellular tissue. Also called infectious process (IP).

chronic disease: One that has a long duration, usually evidenced by morbidity rather
than mortality.

clot: Disturbance of circulation.

coccidiostat: A chemical compound added to the feed or drinking water to combat coccidiosis, by inhibiting the life cycle of the parasite.

COFAL-free: (Complement fixation avian leukosis). A test used on fertile eggs to determine that they are free of avian leukosis virus.

congestion: An over accumulation of blood in the blood vessels causing excessive blood in the tissues.

contagious disease: An infectious disease that is readily transmitted to other birds.

coryza: Head cold (chronic inflammation).

culture (noun): A group of microorganisms grown on artificial media in a laboratory.

culture (verb): A procedure used to remove organisms from the bird and to isolate them.

cyanosis: Bluish in color as a result of lack of oxygen.

cyst: Any sack containing a liquid.

cytoplasm: All the contents of the cell, excluding the nucleus.

degeneration: breaking down of an organelle (reversible or irreversible).

detergent: Usually a soap-less, synthetic, water-soluble agent that reduces surface tension and thus emulsifies oils and has cleansing properties.

diphtheria: Cankerous (false membrane) growth in the mouth.

disease: An impairment of the normal function of any body organ or part of the bird.

disinfectant: A substance that kills pathogenic organisms, but not necessarily spores and is usually applied to inanimate objects.

edema: An excess of fluid in the tissues of the bird.

emaciated: Thin, loss of body weight.

endemic: A disease confined to a small locality.

endocardium: Tissue lining the heart.
enteritis: An intestinal inflammation.

erthrocyte: A red cell or corpuscle of the blood used for transporting oxygen.

estrogens: Certain hormones secreted by the ovary that are capable of governing some of the secretions of the oviduct.

etiology: Cause of a disease.

fomites: Inanimate objects such as clothing, feed bags, etc., that harbor disease-producing organisms.

gangrene: Death of a large mass of tissue (gaseous and green in color).

germinicide: Any agent that kills bacteria, especially those bacteria that are disease-producing.

gram-negative bacteria: Those that retain a violet color even in the presence of alcohol or acetone when using a gram stain. The test is based on the chemical structure of the cell wall.

gram-positive bacteria: Those that lose their color in the presence of alcohol or acetone when using a gram stain.

hematoma: Blood tumor.

hemorrhage: A condition occurring when blood escapes from the circulatory system.

hepatitis: An inflammation of the liver.

histology: Study of the body tissues.

hormone: A substance produced by specialized body cells, which when transported by the blood system, has the power of effecting a change in other body cells.

host: An animal that supports a parasite or a pathogenic organism.

host cell: A cell invaded by a foreign infection.

host-specific: An organism confined to a single host species.

hydrogen-ion concentration: A value that indicates the acidity or alkalinity of a solution, running from 1 to 13 (exponentially). Seven is neutral; above seven is alkaline; below seven is acid. Also called pH.

hyperplasia: Increase in size of an organ due to increased in number of cells.

hypertrophy: Increase in size of an organ due to increase in the size of the cells.
immune: A bird is said to be immune when it has some degree of resistance to a particular disease causing organism.

immunity: The state of being resistant or immune.

inclusion bodies: Bodies, virus particles, found in the cell contents when the bird is infected with certain viral diseases.

induced immunity: Immunity resulting from vaccination.

infection: The invasion of a pathogen into susceptible tissue resulting in disease.

infectious disease: A disease produced by the invasion of living microscopic organisms.

infectious organism: An organism that has the capability of producing disease.

isolation: Keeping poultry in areas separate from other poultry and other vectors.

lentogenic: Low virulence.

lesion: A variation in the normal appearance of tissue as the result of a pathogen or injury.

leukocyte: White blood cell.

ligament: Connects bone to bone.

lymph: Circulating body fluid mainly concerned with transporting the components of the immune system.

lymphocyte: White blood cells produced by the lymphatic system.

lyophilized: Freeze-dried.

macrophage: Phagocytic cells which can engulf and destroy foreign substances.

macroscopic: Observable without magnification.
malignant: Tumor which invades and destroys tissues.

memory cells: Cells that "remember" previous immune responses and accelerate repeated responses.

mesogenic: Medium virulence.

microscopic: Visible with a microscope.

morbidity: A sickness in a bird or flock caused by disease.

mortality: Death of birds in the flock.

mycosis: Any disease caused by a fungus.

necrosis: Death of a cell or tissue in a living host.

nematodes: Round worms.

neoplasm: Tissue that develops abnormally and usually has no physiological function, such as a tumor.

nephritis: Inflammation of the kidneys.

parasite: An organism that lives in or on another organism, from which it derives its nourishment.

passive immunity: Usually that parental immunity passed from mother to offspring through the egg (by antibodies), or artificially by the administration of an antiserum.

pathogen: An organism capable of causing disease.

pathogenicity: The capability of an organism to produce a disease; a quantitative term.

Peracute: Very acute (a few hours) - no clinical signs.

pericarditis: Inflammation of the sac surrounding the heart.

peritoneum: Lining of abdominal cavity.

peritonitis: Inflammation of the peritoneum, the thin membrane found in the abdomen.

persosis: A deformity of the leg bones, usually due to a nutritional deficiency.

pH: See hydrogen-ion concentration.

plasma: The clear solution remaining after the corpuscles have been removed from the blood.
polyvalent: A vaccine containing several antigens, which are derived from different strains of an organism or organisms.

prolapse: Protruding of oviduct through the vent.

protozoa: Minute protoplasmic acellular or unicellular animals with varied morphology and physiology.

renal: Relating to the kidneys.

resistant: Cannot be infected or have disease.

sanitizer: A preparation capable of reducing the number of bacteria present, sometimes combined with a detergent.

septicemia: Invasion of the bloodstream by pathogenic microorganisms.

serological test: A test performed on blood serum to determine the presence or absence of specific antibodies.

serotype: A particular strain of a microorganism as determined by a serologic assay.

serum: The clear fluid remaining after the corpuscles and clotting properties have been removed from the blood.

sinus: A cavity, a hollow space.

sterilizer: Any chemical or agent (steam, heat, etc.) that destroys all forms of life (bacteria, mold, viruses, etc.)

stress: Anything that affects the bird's well-being and lowers its resistance to disease. Border line condition between health and disease.

subacute: Between acute and chronic.

subtype: A strain of microorganism that is closely related to another as determined by a serologic test. It is within the same serotype.

surfactant: Chemicals that lower the surface tension of the solvents in which they are dissolved, such as detergents.

syndrome: A group of symptoms (clinical signs) common to a specific disease.

T-cells: Cells of the immune system, which are transformed and matured in the thymus.

tendon: Attaches muscle to bone.
thymus: Lymphoid glands located in the neck responsible for the T-cell dependent immune system development in the young chick.

titer: A value placed on the potency of a biological substance (antigen or antibody); when applied to the agglutination test, it is the highest dilution at which clumping of the antigen occurs.

toxin: A poison produced by the metabolic processes of microorganisms.

tranquilizer: A drug that slows the metabolic rate of the bird as exemplified by reduced heartbeat, lowered blood pressure, reduced mental awareness, etc.

trauma: A wound or injury.

tumor: New tissue which grows independently of surrounding structure.

vaccine: preparation of microorganisms (killed, living attenuated, temperature sensitive mutant, recombinant or living totally virulent) that when placed in the body of the bird produces or increases immunity to a certain disease.

variant: In microorganisms, one that exemplifies a variation from the original form, resulting in change of its genetic makeup.

vector: A living or inactive substance that carries and transmits parasites to poultry, such as an earthworm, wild bird or man.

vein: Carries blood to the heart.

velogenic: A virulent virus capable of causing high morbidity and mortality.

viability: Ability to live.

virulence: The relative ability of a microorganism to produce disease, usually a quantitative term.

viscera: Organ contained within body cavity.

wounds: Torn or broken tissues.