Turkey Slaughter

Objectives

After a student completes this module, he or she will be able to accomplish the following tasks without the aid of references:

1. List two ways in which turkey slaughter differs from chicken slaughter.

2. Describe the procedure the inspector should follow when the diseases listed below are detected at the postmortem inspection station.
   a. Turkey leg edema
   b. Atrophied breast muscle
   c. Ornithosis
   d. Osteomyelitis
   e. Leukosis in the liver
   f. Granulomas of the liver

3. List two abnormalities that are often grossly visible without exploratory cuts in turkey carcasses affected with osteomyelitis.

Introduction

Turkey slaughter today is a year-round industry instead of a seasonal operation. Some factors that have contributed to this are:

- The production cycle for turkeys is shorter than that for red meat animals.
- Turkeys have a more efficient feed-conversion ration than red meat animals.
- Turkeys are cheaper for consumers than red meat animals.

Meleagris gallopavo, the domestic turkey, is now taking its place as an important source of food on a year-round basis.

This module covers several aspects of turkey slaughter, but the main emphasis is on:

- Turkey plant operations.
- Diseases of turkeys.

Turkey Plant Operations

Turkeys are hauled to the plant on truck beds or trailers in crates, fixed coops, or batteries.

When the turkeys are readied and unloaded for slaughter, the veterinarian (or a food inspector under his/her supervision) performs antemortem inspection by observing the turkeys on a lot basis.

The turkeys are hung by the shanks in shackles hooked to an overhead moving chain that conveys the live turkeys toward the stunning area prior to the neck cutting and bleeding areas.

Scalding of the bled turkeys occurs when the shackles pass through an immersion scalder filled with heated water, which is agitated by recirculation pumps.

In place of an immersion scalder, some turkey slaughter plants shower carcasses with hot water and then convey them through humidity cabinets where they are sprayed with steam. This system avoids the community bath of the immersion scalder.

Picking is done mechanically; usually there are several pickers used and each concentrates on a different area of the turkey to insure complete feather removal.

The shackled dressed turkeys sometimes are singed by a gas flame following picking, This burns the fine hair or feathers off the skin. The carcasses then pass through a wash cabinet, which is equipped with sprayers.

The hock joints are severed and the shanks are removed from the carcass prior to transfer of the carcasses to the evisceration line. The carcasses may be hung by the hocks or by the necks to make the subsequent removal of the crop and trachea (windpipe) easier.

The neck and both hocks of each carcass are placed in the shackle. This three-point suspension of the carcass facilitates the evisceration process.

Before the viscera can be removed, some cuts have to be made into the carcass. The vent area is cut free by a circular incision. Next, if a modified J-cut is used, a cut is made to the point of the keel. If a bar-cut is used, a transverse cut is made caudal to the point of the keel. Either method is approved for use provided the requirements of uniform presentation are accomplished in a sanitary manner.

Drawing, or viscera removal, is accomplished by pulling the viscera free from the body cavity and placing it consistently either to the right or left of the tail. Generally the esophagus will be the only natural body attachment remaining inside the body cavity.

The USDA food inspector inspects the eviscerated carcasses for wholesomeness. The viscera and the outside and inside of the carcass are manipulated in a manner that insures that only wholesome product is passed. Unwholesome carcasses and parts are condemned for human consumption and are positively controlled until proper disposal is completed.

Removal of the heart and liver from the viscera is part of the giblet harvest and trimming, which occurs next. The heart cap is removed from the heart, and the gall bladder is removed from the liver. Next the liver and heart are sent to an ice-and-water chiller.

The removal of the gizzard finishes the giblet harvest from the viscera.

The gizzard is removed by cutting anterior and posterior to its attachment to the gastrointestinal tract.

The gizzards are placed in a machine which splits (peels) and cleans their surfaces. The surfaces are then flushed, and the gizzards are chilled in ice and water.

After the viscera is removed, the lungs can be vacuumed from the chest cavity.

The crop and trachea are pulled free from the slit in the neck. If the oil sacs have not already been removed, they are cut off the tail.

The heads are removed and a final check of the carcasses is made to ensure all eviscerating processes have been properly completed. Then the carcasses pass through a final wash.

After the wash, the neck bones are cut. The necks may be placed inside the body cavity or chilled separately from the carcasses in vats of slush ice.

Next, the tails are cut, and, if they are used by the plant, hock lock wires are inserted in those carcasses that will be trussed. Tucking and trussing the legs of the carcasses is usually done prior to chilling.

Ice-and-water chillers are used to lower the product temperature. Carcasses and giblets are chilled separately.

After the initial chilling, the carcasses are hung on a drip line and drained.

Grading, if requested, is done next. Grading is a voluntary service performed at an additional expense to the plant.

Some carcasses are sent to the cut-up line. Carcass parts are packed in tray packs with plastic overlay, boxed, or bagged.

The giblets are wrapped and stuffed into the whole carcasses.

At the bagging station, the carcass is placed in a plastic bag.

The air is vacuumed out of the bagged carcass and the bag is closed with a clip. The bagged carcass then passes through a shrink tunnel, where it is sprayed with hot water. This procedure shrinks the plastic bag to conform to the shape of the carcass and results in an appealing consumer package.

The whole bagged carcasses and containers of cut-up parts are weighed to confirm, adjust, or mark the net weigh of the product. In some plants the price per pound and the total price of the product may be applied to the outside of the product package.

An immersion freezer is used by some plants to put a crust or quick chill on the product. This process helps prevent freezer burn on the carcass surfaces. Most immersion freezers contain solutions of propylene glycol or brine. As the bagged carcasses exit an immersion freezer, they must be sprayed with water in order to remove any freezing solution from the package.

The product is sorted and packed prior to entry in to the blast freezer.

Usually the air blast or plate-type freezer is used to freeze the product solid.

It is not usual for turkey plants to thaw frozen carcasses and cut-up or further process them some time after slaughter.

Once frozen, the product is ready to be shipped to food markets.

Diseases of Turkeys

Turkey diseases and conditions that maybe encountered at the slaughter plant include:

- Chlamydiosis (Ornithosis)
- Erysipelas
- Fowl cholera
- Turkey leg edema
- Breast muscle atrophy
- Turkey Osteomyelitis Complex
- Liver lesions

For most of the diseases, this section describes postmortem lesions of the disease in an organ or organs of the turkey and the differences in the characteristics of lesions caused by the different diseases.

Chlamydiosis (Ornithosis)

Chlamydiosis is known by several names. Psittacosis is the form of the disease that occurs in psittacine birds, including the zygodactyl birds (this designates the configuration of the toes-two in front and two in back). Examples are parrots, macaws, parakeets, etc. Ornithosis was named for non-psittacine birds, including turkeys, ducks, chickens, pheasants, etc. Ornithosis and psittacosis are different names of the same disease; the name indicates the type of bird affected. The name chlamydiosis, which is used by many scientists, does not refer to a specific type of bird.

The etiological agent for the disease is *Chlamydia psittaci*, which is an obligate intracellular bacterium.

The disease is endemic in parrots and other psittacine birds in South America and Australia and other tropical and subtropical areas. The smuggling of parrots, etc., is probably the biggest source of infection in this country.

The major outbreaks in the United States have involved turkeys. Since transovarian passage does *not* occur, a flock is most likely to be exposed to the disease by birds from an outside source. *Chlamydiae* are present in droppings of infected birds and these organisms remain infectious for months. The primary route of infection is *inhalation*, while a secondary route has been described via external parasites.

In natural infections, disease may spread among a large flock of birds for 2 to 8 weeks before noticeable signs appear. In experimental testing of young turkeys, the period prior to onset of signs ranged from 5 to 10 days.

The signs of chlamydiosis commonly observed in turkeys are cachexia, anorexia, hyperthermia, and, most importantly, the distinct sulfur-colored gelatinous droppings. People who work with diseased flocks have noticed a distinct stance in an affected bird, tail up and breast down on the ground when the turkeys are sitting.

Most lesions observed on postmortem inspection are related to severe damage to the lungs and heart.

- Lungs have diffuse congestion with pleural surfaces covered with fibrinous exudate.
- The heart is enlarged and covered with think fibrin plaques. The pericardial sac is thickened and coated with fibrinous exudate.
- The liver is enlarged and discolored.
- Air sacs are thickened and coated with fibrinous exudate.

The birds usually die showing signs of acute disease.

To make a positive diagnosis of the disease, there must be a demonstration of a four-fold rise in the host'' antibody titer against chlamydial group antigens or isolation of the agent from tissues of the host. A positive diagnosis cannot be made simply on the basis of “typical” gross lesions, cellular alterations, or clinical signs.

Chlamydia psittaci is susceptible to several antibiotics, but the tetracyclines are the only ones economically feasible for large-scale treatment of flocks.

Administration of the drug via drug-coated grain or composite mash is the most common vehicle for medications.

The prevention and control of chlamydiosis depends upon good management practices since there are no effective vaccines available.

Federal regulations prohibit the movement of poultry, carcasses, or offal from any premises where this disease has been proven by isolation. Interstate movement of birds from infected flocks is prohibited. No restrictions are made on eggs from an infected flock.

Differential diagnosis for chlamydiosis would include:

- Chronic respiratory disease
- Fowl cholera-the lungs are dark fibrotic masses, not the typical lungs seen with chlamydiosis, which are hemorrhagic and covered with fibrinous exudate. A problem in diagnosis could arise is a peracute “hot” pasteurellosis was present, which would be manifested as a hemorrhagic septicemia.

Chlyamydiosis in humans was first described around 1879. Typically, if chlamydiosis were to come through a turkey plant via infected turkeys today, in 7 to 14 days approximately 10-30% of the workers would develop a severe respiratory flu. Fever and *intense* headache headache would be common.

For unknown reasons farm workers are usually not affected. But those involved in the handling, dressing, inspecting, and processing of birds are the most vulnerable to infection. There are no recorded cases of infection of homemakers handling ready-to-cook poultry.

The disease is rarely seen in children. People over fifty years of age are more vulnerable to serious infection. Immunity is not derived from an infection, which means a “cured”

person would be susceptible to subsequent infections if exposed to *Chlamydia psittaci* again.
There are two methods of diagnosing chlamydiosis in humans. A serological test demonstrating a four-fold or greater increase in serum titer, with acute and convalescent sera, is considered positive for recent infections. The second method, and probably the best, is to isolate *Chlamydia psittaci* from sputum or whole blood, which are then injected into a mouse using the I.P. route.

Treatment in humans is similar to that in poultry. Usually, tetracyclines are prescribed. Before antibiotic therapy, the human fatality rate was 20%, mostly patients over 30years of age.

To sum up, chlamydiosis is a public health hazard to veterinarians, food inspectors, and poultry plant employees. It is an infectious disease primarily in turkeys that is transmissible to humans. The disease is difficult to recognize because of its similarities to other nonzoonotic poultry diseases. Diagnosis is difficult but effective treatment is available. *Currently*, no measures are enforced to prevent or control this disease.

Erysipelas

In birds erysipelas is generally an acute, fulminating infection of *individuals* within a flock. The primary economic importance of erysipelas is its occurrence in turkeys. The etiological agent is *Erysipelothrix rhusiopathiae*, which also causes erysipelas in pigs, sheep, sea mammals, fish, and many wild animals, and erysipeloid in humans.

The disease often causes death and, in those turkeys it does not kill, generally affects the fertilizing capacity of males.

Erysipelothrix rhusiopathiae may affect humans as a local or septicemic, and occasionally fatal infection referred to as erysipeloid. It is a disease found in workers associated with handling raw fish as well as butchers, kitchen workers, veterinarians, and turkey growers.

Erysipelas is not common today since most turkeys are raised in confinement, which reduces their exposure to the organism.

Widespread artificial insemination of turkeys led to significant outbreaks of erysipelas in hens. With the use of bacterins in the early 1950’s and the availability of penicillin as a treatment in outbreaks, various programs of preventive vaccination and /or treatment have been followed. Despite this, cases of postinsemination erysipelas occur in turkey hens.

Erysipelothrix rhusiopathiae is pathogenic for turkeys at any age or of either sex following exposure by a variety of parenteral routes.

Infection can occur from ingestion of contaminated soil, water, feed, the viscera of turkeys that have died from erysipelas or contamination of breaks in the skin or mucous membranes. Stress such as inclement weather, vaccination, etc., may precede an outbreak.

Outbreaks usually start suddenly, with losses of one or several birds; one may suspect that the deaths are due to poisoning, stampede injuries, or predators. A few droopy birds (especially toms) may be noticed, but are usually easily aroused. Some may have cutaneous lesions or swollen, purplish, turgid snoods. Gradual emaciation, weakness, and signs of anemia occur in some cases where endocarditis is the cause of death.

Sudden losses of hens with peritonitis, perineal congestion, and skin discoloration 4-5 days after artificial insemination have been reported.

In immunized flocks, some of the affected birds will recover.

The gross lesions represent a septicemic disease with many of the septicemia and toxemia indicators present. The most characteristic lesions seen in a field study involving turkeys were:

- Congestion of viscera and intramuscular and subpleural ecchymotic hemorrhages.
- Tubular leader or snood turgid with an irregular reddish-purple color in toms. When present this lesion is *probably pathognomonic.*
- Liver and spleen swollen with hemorrhages.
- Ecchymotic and suffusion hemorrhages under the gizzard serosa.
- Intense catarrhal or sanguino-catarrhal enteritis.
- Skin usually diffusely red and muscles a dirty brick-red color.

The immunity induced by proper use of a good bacterin prevents disease under field and experimental conditions. The use of a bacterin in conjunction with a rapid-acting form of penicillin at the beginning of an outbreak will usually control losses.

Fowl Cholera

The etiological agent for fowl cholera is *Pasteurella multocida.* It is a contagious disease affecting domesticated and wild birds. This disease usually manifests itself as a septicemia associated with high morbidity and mortality, but chronic conditions do occur. This particular disease has historical importance because it was one of the diseases Veterinary Services of USDA was created to investigate.

Chickens become more susceptible to fowl cholera as they reach maturity. Waterfowl seem particularly susceptible. The disease is rarely diagnosed in chickens less than 12 weeks of age. Ranged turkey flocks are more likely to be exposed to infected wild birds and mammals since the disease is found universally.

Most of the time, the mode of introduction into a flock is difficult to pinpoint. Adding newly purchased stock to a breeding flock could explain the occurrence of the disease in some cases. Free-flying birds having contact with poultry may be a reservoir for the fowl cholera organism. The organism is seldom transmitted through the egg. There is no limit to the duration of the chronic carrier state other than the life of the bird. Generally the “healthy” nasal carriers of the organism are considered to be the reservoir of infection.

Most dissemination of *Pasteurella multocida* within a flock is primarily by excretions from the mouth, nose, and conjunctiva of diseased birds that contaminate their environment, such as feed and water.

Feces are not considered a source of infection. There have been no experiments that have shown viable *Pasteurella multocida* organisms present in affected birds' feces.

The signs of infection in the acute form of fowl cholera usually exist only a few hours before death.

Unless infected birds are observed prior to death, signs of acute fowl cholera will be missed. Therefore death may be the first indication of the disease. Signs of acute fowl cholera are as follows:

- Fever, anorexia, ruffled feathers, mucous discharge from the mouth, diarrhea, and increased respiratory rate.
- Cyanosis often is observed, around the unfeathered areas of the head, such as the wattles and comb.
- Fecal material is initially watery and whitish in color but later becomes greenish with the presence of mucus being observed.

The chronic form of fowl cholera may follow an acute stage of the disease *or result* from infection with organisms of low virulence. The signs of chronic fowl cholera are usually manifested in the following manner:

- Localized lesions found in areas such as wattles, sinuses, leg or wing joints, foot pads, and sternal bursae often are swollen.
- Exudative conjunctival and pharyngeal lesions may be observed.
- Torticollis sometimes occurs as a result of meningeal infection.

In summarizing the signs of the disease in a flock, individual birds could show:

- Acute stages of the disease.
- Partial recovery with relapse followed by death.
- Chronic infection.
- Complete recovery
- No signs of infection.

The lesions of fowl cholera vary in type and degree of severity. The signs of infection and the lesions *present* are difficult to categorize as either totally acute of chronic in nature.

In acute fowl cholera most of the postmortem lesions are related to vascular disturbances.

- General hyperemia of visceral veins is common.
- Large numbers of the organisms can be observed microscopically from the blood of the engorged veins.
- Pneumonia is more severe in turkeys than in chickens.

- Large amounts of viscid mucus in pharynx, crop, and intestines are seen.
- Ovaries of layers are affected by hyperemia.

Lesions of chronic fowl cholera generally are characterized by infections of a localized nature. The lesions become suppurative and are widely dispersed throughout the carcass.

- Pneumonia is common in turkeys.
- Middle ear and cranial bone involvement is common in turkeys with torticollis manifested in the live bird.

Pasteur did some work with a vaccine for fowl cholera but was not very successful. Since Pasteur, there have been several attempts to produce efficient vaccines for fowl cholera. Substantial but not absolute immunity can be induced in fowl using killed *Pasteurella multocida* vaccines under controlled conditions. The vaccination process is performed by a subcutaneous injection. Under field conditions there are losses from fowl cholera even in vaccinated flocks. The probable cause of death is the presence of other disease, environmental stress, or an improperly prepared or administered vaccine.

A positive diagnosis of fowl cholera should be based on three findings:

- Clinical observation.
- Necropsy findings.
- Isolation of *Pasteurella multocida.*

Several drugs have been used to treat fowl cholera cases with varying degrees of success depending to a large extent on the promptness of treatment and the drug used.

Prevention of fowl cholera is done best by trying to eliminate the reservoirs of *Pasteurella multocida* organisms and preventing poultry flocks from contacting reservoirs of infection.

This bacterial disease is *not* a disease of the poultry hatchery and infection occurs after the birds are in the possession of the producer.

Therefore, good management sanitary practices must be directed toward elimination of the sources of infection.

Turkey Leg Edema

This condition has been recognized for many years. Turkey leg edema has occurred since 1967 in the U.S. The condition occurs primarily in heavy tom turkeys, 25 weeks of age or older, although it is sometimes reported in heavier hen turkeys. The syndrome is more prevalent from August through October but occurs to some extent throughout the year.

The specific cause of this disease is unknown.

The condition can be identified on antemortem in high-incidence flocks by feeling crepitation of gas under the skin of the leg area.

On postmortem inspection, from 2 to 20% of carcasses may be affected. An occasional flock may have as high as 70% involvement.

Approximately 2% of all turkey flocks slaughtered show some evidence of the problem.

Gross pathological findings as postmortem inspection include a blanched appearance of the skin over the thigh, a slick-feeling sensation on palpation, and an accumulation of amber or red-colored gelatinous fluid in the inguinal space and in the subcutaneous tissues of the leg. The condition is often *unilateral*. Numerous gas bubbles are present in the edema fluid and can be detected by palpation of crepitation prior to opening the inguinal space. The amount of gas bubbles is variable but they are present to some extent in all affected carcasses. The more chronically involved carcasses show a greenish discoloration of the edema fluid or the presence of yellow-colored inflammatory exudate. There is no evidence of injury to the skin or to the knee of hock joints. The adductor muscles of the thigh appear swollen and contain hemorrhages.

Inspectors should observe and palpate the skin of the leg and inguinal space for evidence of crepitation to detect this condition.

Turkey leg edema is a localized inflammatory condition and the affected tissue must be trimmed.

If there is evidence of systemic disturbance in the carcass, the whole carcass is required to be condemned. Indicators of septicemia and/or toxemia justify condemnation of the carcass. Turkey leg edema is *not* a justification for carcass condemnation.

Breast Muscle Atrophy

This disease has several names, including green atrophy, green breast, and green muscle degeneration. The disease is often found in a slaughtered flock of broad-breasted hen turkeys five months of age or older.

This condition is characterized by greenish discoloration of all or a portion of the deep pectoral muscle on either or both sides of the keel bone in breeder hen turkeys. The greenish discolored muscle has a hardened, woody texture, is shrunken, and is surrounded by a zone of inflammatory tissue. External examination of the skin-covered breast reveals a change in contour. The change varies from a slight depression to a marked wasting away of the breast.

There is a definite genetic relationship involving certain blood lines of broad-breasted bronze turkeys. The condition is not usually found in turkeys less than 5 months of age. The disease sometimes occurs in slaughtered chickens that are 12 weeks old or older. The incidence in affected flocks ranges from 1 to 12 percent.

The condition appears to result from a lack of blood supply to the deep pectoral muscle. The subsequent degeneration and absorption of the muscle and the breakdown of myoglobin to cholemyoglobin explains the greenish discoloration of the muscle.

Outlined below is the procedure to follow when the inspector-in-charge determines the incidence is sufficiently high to indicate a flock problem.

- The lot(s) run from the affected flock are retained.
- Trimming related to “atrophied turkey breast” is not conducted at the postmortem inspection station.
- Control of the retained lot(s) must be maintained until the carcasses are “raw deboned” *or* each turkey breast is slashed bilaterally and any necessary trimming completed.

Turkey Osteomyelitis Complex

FSIS recognized that the Turkey Osteomyelitis Complex (TOC) had become a significant problem in young turkeys during the 1980’s. In 1988 the Agency implemented a policy that requires additional inspection procedures for any lot of turkeys in which TOC is identified. These additional procedures are conducted by plant employees and represent a form of salvage.

While any bone may be affected by TOC, most commonly the epiphyseal growth plates of long bones are involved. The proximal end of the tibia is the bone most often affected. The inflammation may remain confined within the bone and cartilaginous growth plate or it may affect adjacent soft tissues and joints as the reaction progresses. Many organisms can be found in TOC lesions. *Staphylococcus* and *Escherichia coli* are isolated frequently. The lesions can take a variety of forms, ranging from mild inflammation to severe, purulent reactions.

TOC-affected carcasses can seldom be identified positively during routine postmortem inspection. However, carcasses suspicious for the condition are easily identified. Two external signs are frequently seen in TOC-affected carcasses—joint swelling and green discoloration of the liver. The latter sign is the most consistent indicator that TOC may be present. However, it is not pathognomonic. Although, most carcasses affected by TOC exhibit a green liver, most carcasses exhibiting a green liver do not have TOC.

In order to distinguish lots of turkeys affected by TOC from those showing external sings compatible with TOC but caused by other conditions, the Agency requires VMO’s to conduct special diagnostic examinations on suspicious lots. If the presence of TOC is confirmed during the diagnostic exam, the VMO requires the plant to conduct additional examination procedures on all carcasses identified as suspects at the postmortem inspection stations.

When osteomyelitis is detected during the plant examination, all tissues to the next normal joint must be removed and condemned. Product that is salvaged must be held for reexamination by FSIS personnel before it is allowed to enter normal production flow. All aspects of the TOC procedure must be consistently performed in accordance with FSIS policy and in a manner acceptable to the IIC or approval can be rescinded and the procedure discontinued. If the procedure is discontinued, FSIS postmortem condemnations could increase dramatically in some lots.

Liver Conditions

Granulomas: Granulomas are among the most common liver lesions seen in slaughtered turkeys. Occasionally whitish foci or spots are embedded in an otherwise normal-appearing liver. The inspector may have difficulty in recognizing these lesions as granulomas until some correlation is provided. The poultry inspection team *must* differentiate granuloma-type lesions from malignant tumors, including leukosis lesions. The appearance of “atypical” liver lesions grossly resembling leukosis has resulted in turkey carcasses being condemned in the past. Today, with the field supervisor’s expertise and through laboratory investigation, inspectors should accurately identify such lesions.

There have been discussions in the past about lymphoid tissue that is not leukosis that appears in the liver. Supposedly, according to one source, these are islets of misplaced “bursa cells” that are “homesteading” the liver.

Leukosis: Turkey liver lesions that are leukosis are considered to be uncommon although there have been incidences in which atypical granulomatous lesions have been incriminated as leukosis.

Blackhead: The liver lesions of blackhead in turkeys are well described in the literature. The liver contains irregularly round, depressed lesions that vary in color. The lesions are yellow to gray and sometimes are green or red. The diameter of the lesions vary but are often 1 to 2 cm and may coalesce to produce larger lesions.

This disease occurs when unmedicated turkeys under 12 weeks of age are exposed to *Histomonas meleagridis* complicated by secondary bacteria. The common cecal worm of turkeys, *Heterakis gallinarum*, as well as earthworms, play a role in dissemination of this disease.