Musculoskeletal Disorders Causing Lameness in **Chickens** and **Turkeys**

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The Normal Skeleton

Many peculiarities of the avian skeleton are related to flight. Other differences are related to egg production. Many avian bones are hollow and contain air sacs. Air sacs in bones make bones lighter but are of significance because air sacculitis can result in osteomyelitis, arthritis and synovitis.

Birds have a single occipital condyle and many have a long spring-like neck. The vertebrae articulate by synovial joints. There are no intervertebral discs. The spinal column of gallinaceous birds is fused in the thoracic to sacral areas except for T4 that is articulating. The pectoral girdle is well developed with a scapula, coracoid bones and clavicle. This pectoral girdle and the large keel bone were developed to carry flight muscles. To facilitate egg laying the pelvic girdle has no pubic symphysis. Two tarsal bones are fused with the tibia giving the tibiotarsus and one with the metatarsal to form the tarsometatarsus. One tarsal bone is embedded in the gastrocnemious tendon. Four digits are present and an accessory structure, the metatarsal spur develops in males (and to a lesser extent in females). There are only 3 digits on the forelimbs.

The young chick has an egg tooth and a very immature skeleton at hatching. Cones of cartilage persist in the end of the long bones. These cones are not broken down until 7-9 days of age when a growth plate can be recognized. The growth plate in fast-growing commercial poultry is thicker and more irregular than in mammals and the epiphyses in growing birds are cartilaginous with no secondary centers of ossification. The articular cartilage develops from the growth plate. Leg tendons ossify as birds become mature.

Parathyroid glands (two on each side) are found close to the posterior poles of the thyroid. There are discrete small, calcitonin producing ultimobranchial glands, posterior to the thyroids and parathyroids.

Muscle

<u>Normal anatomy and physiology</u>

Avian muscle may be a mixture of red (dark) or white (light) fibers as it is in mammals, or groups of muscle may be predominantly one variety or the other. Red fibers have a copious blood supply, high myoglobin content, high fat content and low glycogen content while white fibers are the opposite. Red fibers metabolize fat directly and have sustained activity and slow contraction for migration. White fibers metabolize glycogen. They have rapid activity and fast contraction (fast twitch) for short, rapid flight.

Muscle mass must be evaluated in the context of the strain and species. Normal muscle mass

in a Leghorn would be classed as “reduced” in a broiler or broiler breeder. Lack of muscle mass is an important feature in evaluating the health status. Serous atrophy of fat is not as important a criterian of emaciation as it is in mammals.

Genetic and Congenital (Developmental) Anomalies

Many skeletal abnormalities are seen in embryos when unhatched eggs are broken out. Limb deformity and torticollis are seen occasionally in chicks in the hatching trays. Scoliosis and kyphosis are more frequently. These defects may be genetic or congenital and congenital defects may have a metabolic origin. Birds with torticollis have difficulty eating. Scoliosis does not usually cause a problem but may be associated with increased valgus-varus deformity.

Acquired Defects

Introduction

Many of the leg deformities and lameness problems that affect meat-type poultry are related to rapid growth and are therefore much more prominent in males. Slowing growth, particularly in the first 15-20 days of life will markedly reduce the incidence of angular bone deformity (valgus-varus), dyschondroplasia, spondylolisthesis (kinky back) and ruptured tendons, which probably make up 70-90% of the leg deformity and lameness in meat-type chickens on a high density, nutritionally adequate ration. There are genetic influences in many of the growth-related defects. Slowing growth is less effective in turkeys, although it does reduce dyschondroplasia and cardiovascular-related death. Increased exercise after 3 wks will reduce lameness in turkeys.

Leg deformity may be the result of uneven growth in the growth plate or abnormal position of the leg, however, it is more likely to be the result of muscle or tendon tension on the joints or on the bone, pulling the bones out of line or bending weak bones as they grow. Angular bone deformity at the hock is a common cause of lameness in turkeys that appears to be related to both rapid growth and increased body size and weight. It occurs at an older age in turkeys than in broiler chickens. Severe lameness in turkeys (shaky-leg lameness) is caused by tendon pain because of lack of activity related to foot-pad dermatitis and ulceration because of poor litter conditions. It usually occurs at the time tendons start to ossify.

The articular cartilage of rapidly growing poultry is thick and is not produced from an ossification center in the epiphysis. Focal avascular or ischemic necrosis (osteochondrosis) of this cartilage frequently results in joint lesions. Avulsion fractures and ligament damage at the intertarsal joint or femorotibial joints are frequently missed during a necropsy examination for lameness in turkeys. Spontaneous fracture of the femur occurs sporadically in heavy males, occasionally as a flock or farm problem.

Nutritional deficiency may be the cause of lameness. Vitamin deficiency may be the result of insufficient nutrient for the growth rate of the birds, which is why it is seen first in the males. The problem may be poor quality vitamin, failure to add the vitamin premix, inadequate mixing of feed, or it may be secondary to viral or bacterial damage to the intestinal epithelium affecting digestion and absorption of nutrients.

Muscle lesions are rare in poultry, however, nutritional (selenium deficiency), exertional (deep pectoral myopathy, turkey leg edema) and toxic (ionophore) myopathies do occur. Rupture of

the peroneus muscle, at the place where tendons ossify, causes downgrading at processing in turkeys but does not cause lameness. Spontaneous rupture of the gastrocnemius tendon above the hock is frequently seen in broilers, but is rare in turkeys.

A) Leg problems associated with egg storage and incubation.

The longer eggs are held before setting, the more cull chicks and leg deformities will be seen. This may be associated with abnormal position of the yolk after long storage. Low humidity (increased fluid loss) will increase the problem.

Incubation temperature & leg deformity. Incubation temperature influences metabolic rate and leg deformity in chicks and poults, because metabolic rate affects the developing embryo. Cool temperature from day 2 to 5 may increase crooked toes, angular bone deformity, spraddled, slipped tendon and rotated tibia. Overheating after day 10 can have a more serious effect on these deformities particularly in turkey poults. Egg size influences internal egg temperature and larger eggs are more apt to overheat as embryos mature because of the heat generated by the developing embryo.

B) Growth and environment related leg deformity.

1) **Crooked toes, curled toes:** Curled toe is a common developmental anomaly in both young growing turkeys and chickens, affecting a few birds in most flocks. Toes are bent either laterally or medially in a horizontal plane. Examination reveals twisting of the phalanges. Infrared brooding and wire floors appear to increase the incidence. Use of roosts decreases the incidence. This condition must be differentiated from curled toes due to riboflavin deficiency in young birds, in which the toes are curled ventrally and in which the primary lesion is in the peripheral nerves.

2) **Slipped tendon:** The gastrocnemious tendon has slipped off the condyles of the distal tibia and the bird has lost control of the lower leg. Primary slipped tendon can occur without bone or cartilage abnormality and is not necessarily caused by manganese or other deficiency. Bone strength is normal. Slipped tendon causes only 1-3% of the lameness in chickens but is rare in poults.

3) **Spondylolisthesis (kinky-back)** is ventral dislocation of the anterior end of the articulating 4th thoracic vertebrae, which tips up the posterior end to cause pinching of the spinal cord. Damage to the spinal cord causes partial posterior paralysis. The affected broilers sit on their tail with their feet extended or fall to one side. The lesion must be differentiated from scoliosis (which usually causes no clinical signs) and osteomyelitis or osteochondrosis/dyschondroplasia of the same vertebrae, causing compression of the spinal cord, which cause similar clinical signs usually in males. Dislocation may also occur between other cervical and thoracic vertebrae. Spondylolisthesis is a frequent lesion, more common in female than male broilers. It is rare in turkeys. Affected birds cannot reach food or water and should be removed and killed. Slowing growth particularly in the first 14 to 21 days will reduce the incidence.

4) **Tibial dyschondroplasia (TD), osteochondrosis:** Proliferation with lack of removal of avascular prehypertrophying cartilage is called dyschondroplasia. If the lesion is small, bony change is minimal. If the lesion is large, the end of the affected bone enlarges, becomes weakened and may bend backward. The cortex becomes thin and the bone may fracture spontaneously or at processing. When the end of the bone enlarges and becomes weaker, the bone compresses when heavy birds stand. These birds are in pain when they walk and quickly sit again. As bone growth slows with approaching maturity the lesion may be removed or occasionally necrosis develops around the

cartilage. This results in long bone necrosis or fracture and severe lameness. Thirty to 50% of male meat-type poultry have dyschondroplasia, but lameness only occurs if there is bone weakness, necrosis, deformity or fracture.

The cause is multifactorial, but rapid growth, particularly without a long daily rest period, and electrolyte imbalance (anion/cation imbalance) are primary. Anions should be increased and cations reduced. Some mycotoxins and chemicals also increase the incidence. Dyschondroplasia causes 5-25% of the lameness in meat-type chickens, turkeys and ducks with occasional higher levels in turkeys. It caused up to 50% of the lameness in some flocks in countries where animal protein is used in the ration.

Alkalizing the ration by removing 1 kg of NaCl and replacing it with 2 kg of $NaHCO_3$ per tonne will reduce the incidence of dyschondroplasia. The addition of 5 to 10 μg 25$(OH)_2$cholecalciferol/kg of feed may prevent TD.

5) **Valgus-varus deformity (VVD); angular bone deformity; twisted legs:** VVD is lateral or medial deviation of the distal tibiotarsus with a corresponding deviation of the metatarsus and secondary displacement and occasionally complete slippage of the gastrocnemious tendon. Affected birds are "bow legged" or "knock-kneed" until they go "off their legs". This deformity occurs in rapidly growing birds from a few days of age to processing. Growth plates are normal in the distal tibiotarsus but the proximal metatarsus may be enlarged. Interstarsal ligaments become stretched and the joint is slack. Bone strength is normal by the time birds are seen lame but spontaneous fracture may occur through the growth plate between the tibia and attached tarsal bones. The etiology is not known although the defect is related to over-nutrition and rapid growth.

This deformity occurs at an older age in turkeys than in broiler chickens, usually occurring after six weeks of age. VVD also occurs in ratites and some zoo birds (storks or egrets) if they are grown rapidly on high protein turkey feed, indicating that VVD is not genetic. The etiology may have to do with uneven growth of the two tarsal bones, the growth plate at the end of the distal tibia, or asymmetrical tendon tension of fast growing or weakened bones. It may also be caused by some B vitamin deficiencies It is a frequent cause of lameness in turkeys causing 15 to 30% of leg deformity. It is the most frequent cause of lameness in broiler chickens causing up to 60% of the skeletal disease.

Slowing growth rate in the first 10 to 14 days will reduce the incidence of this deformity in meat-type chickens. Reducing the protein in the feed is also effective, but reduces feed conversion. Increased exercise is more effective in turkeys, perhaps because it increases bone strength.

C) Physical and mechanical causes of lameness and musculoskeletal disorders

1) **Epiphyseal separation:** When the legs of normal, young, rapidly growing broiler chickens are disarticulated at necropsy, the articular cartilage often pulls off the femoral head and trochanters by the joint capsule, leaving the smooth, shiny growth plate. Occasionally part of the growth plate also pulls away, leaving rough, irregular, necrotic-looking, subchondral bone. Some people describe this separation at post-mortem as "femoral head necrosis" but it is normal in broilers.

Broiler chickens that are caught and carried by one leg to be crated for trucking to the processing plant may struggle causing epiphyseal separation at the proximal femur. These broilers bleed from the end of the femur and may die of hypovolemic shock or be condemned.

2) **Fracture.** Free-range and perchery hens frequently suffer from trauma-induced fracture of leg or wing bones. If the hens have osteoporosis, fractures are more frequent and may also occur in caged hens as spontaneous fractures of the spine or legs, or from injury. Many fractures occur when fowl

MUSCULOSKELETAL SYSTEM

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Two sets of legs.

Chicken with crooked neck.

Crooked neck with skin removed, X2, (1 severe, 1 mild) and vertebrae from one of affected birds showing scoliosis.

Reduced muscle mass in a Leghorn. This hen died from emaciation.