tions. The various types of tenosynovitis include idiopathic, acute, chronic, and septic (infectious). Idiopathic synovitis refers to synovial distention of tendon sheaths in young animals, in which the cause is uncertain. Acute and chronic tenosynovitis are due to trauma. Septic tenosynovitis may be associated with penetrating wounds, local extension of infection, or a hematogenous infection.

Clinical Findings and Diagnosis: There are varying degrees of synovial distention of the tendon sheath and lameness, depending on the severity. Horses are markedly lame in septic tenosynovitis. Chronic tenosynovitis is common in horses in the tarsal sheath of the hock (thoroughpin) and in the digital sheath (tendinous windpuffs). These two entities must be differentiated from bog spavin and synovial effusion of the fetlock.

Treatment: In idiopathic cases, no treatment is initially recommended. Acute cases with clinical signs may be treated symptomatically with cold packs, nonsteroidal anti-inflammatory drugs, and rest. Application of counterirritants and bandaging has been used in more chronic cases. Radiation therapy is helpful. Septic tenosynovitis requires systemic antibiotics and drainage. If adhesions develop between the tendon sheath and the tendon, persistent effusion and lameness is the rule.

CONGENITAL AND INHERITED ANOMALIES OF THE MUSCULOSKELETAL SYSTEM

Congenital and inherited anomalies can result in the birth of diseased or deformed neonates. Congenital disorders can be due to viral infections of the fetus or to ingestion of toxic plants by the dam at certain stages of gestation. The musculoskeletal system can also be affected by certain congenital neurologic disorders. *See also* CONGENITAL MYOPATHIES, p 867.

ANGULAR LIMB DEFORMITIES OF FOALS

In these congenital or acquired skeletal defects, the distal portion of a limb deviates laterally or medially early in neonatal life. In utero malposition, hypothyroidism, trauma, poor conformation, excessive joint laxity, and defective endochondral ossification of the carpal or tarsal and long bones have been implicated. One to four limbs may be affected, depending on the severity of the condition.

The carpus is affected most frequently, but the tarsus and fetlocks are occasionally involved. The deviation is obvious but varies in severity. A lateral deviation (valgus) of up to 6° of the distal portion of a limb may be regarded as normal. Most foals are asymptomatic, but lameness and soft-tissue swelling can accompany severe deviations. Outward rotation of the fetlocks invariably accompanies carpal valgus. Foals in which there is defective ossification of the carpal cuboidal bones or excessive joint laxity are frequently lame as the legs become progressively deviated. Affected limbs must be palpated carefully to detect ligament laxity and specific areas that may be painful.

Diagnosis should include a precise determination of the site and cause for the deviation. The distal radial metaphysis, physis, epiphysis, or cuboidal bones may be the site of deviation. Radiography is helpful in detecting physeal flaring, epiphyseal wedging, and deformation of carpal bones. Mildly affected foals frequently improve spontaneously without treatment.

Treatment depends on the severity of the condition and tissues affected. Excessive joint laxity, with or without cuboidal carpal bone involvement, requires tube casts or splints. The fetlock and phalangeal region should not be included in the casts, which should protect the weak joint from trauma but allow restricted exercise to maintain tendon and ligament tone. Such limb support may be required for up to 6 wk.

Physeal and epiphyseal growth disturbances are also amenable to surgical correction through hemicircumferential transection and periosteal elevation of the distal radius on the concave side of the defect or through transphyseal bridging of the physis on the convex side. These surgeries must be performed before the physeal growth plates close (as early as 2-4 mo of age), and success depends on continued growth and development of the bones. Sequential examinations and radiographs are necessary to follow spontaneous improvement or to establish a need for surgery.

Without treatment, the prognosis for severe carpal valgus is poor. The conformational anomaly leads to early degenerative joint disease. Likewise, deformity of the cuboidal carpal bones contributes to a poor prognosis. However, with early detection, careful evaluation, and proper surgical treatment, most foals respond favorably. (*See also* FLEXION DEFORMITIES, p 840.)

ARTHROGRYPOSIS

Arthrogryposis is ankylosis of the limbs, usually combined with a cleft palate and other growth deformities. It occurs in all breeds of cattle, particularly Charolais. At birth, affected calves exhibit joints fixed in abnormal positions and frequently have scoliosis and kyphosis. They are usually unable to stand or nurse. Muscle changes, notably atrophy, have also been seen. Changes may also occur in the spinal cord with necrosis of neurons and lesions of the white matter. Athrogryposis has more than one etiology and pathologic entity. Analysis of pedigrees and matings that produced affected calves revealed that the arthrogryposis syndrome in the Charolais is genetic in origin and caused by an autosomal recessive gene with complete penetrance in the homozygous state. Teratogens identified as causing arthrogryposis include plants such as lupines (anagyrine as the toxic agent) that are ingested by pregnant cows between day 40 and 70 of gestation. Prenatal viral infections with the Akabane (p 451) or bluetongue virus (p 520) can also cause arthrogryposis.

CONTRACTED FLEXOR TENDONS

Contracted flexor tendons are probably the most prevalent abnormality of the musculoskeletal system of newborn foals and calves. This condition is caused by an autosomal recessive gene. In utero positioning may also affect the degree of disability.

At birth, the pastern and fetlocks of the forelegs and sometimes the carpal joints are flexed to varying degrees due to shortening of the deep and superficial digital flexors and associated muscles. A cleft palate may accompany this condition in some breeds. Slightly affected animals bear weight on the soles of the feet and walk on their toes. More severely affected animals walk on the dorsal surface of the pastern and fetlock joint. If not treated, the dorsal surfaces of these joints become damaged, and suppurative arthritis develops. Rupture of the common digital extensor can occur as a sequela. This condition should be differentiated from arthrogryposis.

Mildly affected animals recover without treatment. In moderate cases, a splint can be applied to force the animal to bear weight on its toes. The pressure from the splint must not compromise the circulation, or the foot may undergo ischemic necrosis. Frequent manual extension of the joints, attempting to stretch the ligaments, tendons, and muscles, aids in treating these intermediate cases. Severe cases require tenotomy of one or both flexor tendons. A plaster-of-Paris cast may also be indicated in some cases. Extreme cases may not respond to any treatment. (*See also* FLEXION DEFORMITIES, p 840.)

DEFECTS OF THE SPINE OF FOALS

Defects of the spine include scoliosis, synostosis, and lordosis. Although all of these conditions are uncommon in foals, congenital scoliosis is encountered most frequently. On clinical examination, it is often difficult to assess the severity. A better appreciation of the condition can be obtained by radiographic examination. In mild cases, improvement is spontaneous and may be complete. Even in the more severe cases, there is rarely

any obvious abnormality in gait or maneuverability. However, these foals frequently are not raised because they appear unlikely to be able to withstand being ridden or worked.

Another occasional congenital deformity is that of synostosis (fusion of vertebrae), which may be associated with secondary scoliosis. Radiography is necessary for confirmation.

Congenital lordosis (swayback) is associated with hypoplasia of the intervertebral articular processes. In adult horses, degrees of acquired lordosis and kyphosis (roachback) are occasionally seen, which contribute to back weakness. Diagnosis is based on the clinical appearance and can be confirmed by radiography, which reveals an undue curvature of the vertebral column usually in the cranial thoracic region ($T_{5\text{-}10}$) in lordosis and in the cranial lumbar region ($L_{1\text{-}3}$) in kyphosis.

DOUBLE MUSCLING

Double muscling is an overdevelopment of the musculature in the neonate. The muscles of the shoulder, back, rump, and hindquarters are separated by deep creases, particularly between the semitendinosis and biceps femoris, and between the longissimus dorsi muscles of either side. Necks of double muscled cattle are shorter and thicker, and their heads appear smaller. Associated disorders include hypoplastic reproductive tracts, delayed reproductive age of maturity, and lengthened gestation and increased birth weights combined with dystocia. Double muscling is caused by a pair of incompletely recessive genes that result in various degrees of the condition in the animal. Succinic dehydrogenase activity is significantly decreased in affected calves.

DYSCHONDROPLASIA

Dyschondroplasia of genetic origin occurs in most breeds of cattle. The forms range from the so-called Dexter "bulldog" lethal, in which the calf is invariably stillborn, to those animals that are mildly affected.

The **brachycephalic dwarfs** that were common in Hereford cattle in the 1950's largely have been eliminated through genetic selection. They are characterized by short faces, bulging foreheads, prognathism, large abdomens, and short legs. They are approximately half normal size. The **dolichocephalic dwarf**, most commonly seen in Angus cattle, is of the same general body conformation as the brachycephalic dwarf, except that it has a long head and does not have either a bulging forehead or prognathism. The short-faced calves are frequently referred to as "snorter" dwarfs because of their labored and audible breathing. Both types are of low viability and susceptible to bloat. Their carcasses are undesirable, and they are rarely kept except for research purposes.

Dyschondroplasia of the appendicular and axial skeletons also occurs in dogs. The former is reported in Poodles and Scottish Terriers, the latter in Alaskan Malamutes, Basset Hounds, Dachshunds, Poodles, and Scottish Terriers. In some breeds (Bassets, Dachshunds, Pekingese), the appendicular dyschondroplastic characters are an important feature of breed type. In Malamutes, the condition is accompanied by anemia.

HYPERKALEMIC PERIODIC PARALYSIS

Hyperkalemic periodic paralysis (HPP) is a hereditary condition of Quarter horses that is the result of a genetic mutation in the skeletal muscle sodium channel gene. It is inherited as an autosomal dominant trait. Most affected horses are heterozygotes. The classic signs are muscle fasciculation, spasm, and weakness associated with hyperkalemia. However, these signs are only rarely observed in affected horses. Potential sequela of attacks are abrasions and involuntary recumbency. These problems are not specific for HPP but occur more frequently in affected horses. It is also likely that HPP results in greater muscle mass. There are suggestions that homozygotes may be more severely affected and show signs of upper respiratory obstruction as foals.

The test of choice is the gene probe for the HPP type sodium channel DNA. This is the most sensitive test for detecting the mutation in horses susceptible to HPP. Diagnosis can also be based on the appearance of clinical signs, increased plasma potassium lev-

els, and associated increased plasma potassium concentrations in response to oral potassium loading. All affected horses are descendants of the American Quarter horse sire Impressive.

LIMBER LEG

Limber leg is a hereditary condition of Jersey cattle, apparently controlled by a simple recessive gene. Some affected calves are born dead. Living calves appear normal at birth but are unable to stand. They may struggle to stand but cannot because of incompletely formed muscles, ligaments, tendons, and joints. The shoulder and hip joints can be rotated in any direction without apparent discomfort. Diagnosis is based on signs, necropsy findings, and identification of carrier animals.

OSTEOGENESIS IMPERFECTA

Osteogenesis imperfecta is a generalized, inherited bone defect in cattle, dogs, and cats, characterized by extreme fragility of bones and joint laxity. The long bones are slender and have thin cortices. Calluses and recent fractures may be present. The sclera of the eyes may be bluish. The inheritance is most likely polygenic.

OSTEOPETROSIS

Osteopetrosis is a rare disease that appears to be inherited as a simple autosomal recessive trait in Angus, Simmental, and Hereford cattle, and that is also seen in dogs and foals. It is characterized by premature stillbirth 10 days to 1 mo before term, brachygnathia inferior, impacted molar teeth, and easily fractured long bones. Bone marrow cavities are absent and replaced by primary spongiosa. The fetal-like abnormal intramedullary bone consists of chondro-osseous tissue. Foraminas of the skull and long bones are hypoplastic or aplastic. The cranium is thickened and compresses the brain. Extensive mineralization is present in vessel walls and neurons of the brain. Diagnosis is confirmed by a longitudinal bisection of long bones revealing the diaphyses filled with a plug of bone instead of marrow.

SPIDER LAMB SYNDROME OF SHEEP

Hereditary chondrodysplasia, or spider lamb syndrome, is an inherited, semilethal, musculoskeletal disease affecting lambs primarily of the Suffolk or Hampshire breeds. Lambs have pronounced medial deviation of the carpus and hock and are unable to stand without distress. Pathologic changes in the skull reveal a rounding of the dorsal silhouette, producing a "Roman nose" appearance and a narrowed elongation of the occipital condyles. The thoracic and lumbar vertebrae are moderately kyphotic, which causes a dorsal rounding of the backline. The sternebrae are dorsally deviated, leading to a flattening of the sternum. The forelimbs have a medial deviation of the carpal joints with a bowed radius and ulna and irregular thickening of the growth plate cartilage. The hindlimbs have medially deviated hocks and bowed tibiae, which also have thickened, irregular growth plates. Muscle atrophy is also predominant. The regulation of liver insulin-like growth factor (IGF) and the IGF-binding proteins may be involved in the physical manifestations of this disorder. It is suggested that the condition is inherited in a simple autosomal recessive pattern.

SYNDACTYLY AND POLYDACTYLY

Syndactyly or mule foot is the partial or complete fusion of the digits of one or more feet. Reported in numerous cattle breeds, it is most prevalent in Holsteins and is inherited as a simple autosomal recessive condition. The forefeet are affected most often, but one or all four feet may be affected. Animals affected with syndactyly walk slowly, usually have a high-stepping gait, and may be more prone to hyperthermia.

Polydactyly is a genetic defect of cattle, sheep, pigs, and occasionally horses. In its

most common form, the second digit is developed but the medial declaw is missing. The toes may be fused to give rise to polysyndactyly. Rarely one or all four limbs will have the condition. Polydactyly in cattle appears to be polygenic with a dominant gene at one locus and a homozygous recessive at another.

DYSTROPHIES ASSOCIATED WITH CALCIUM, PHOSPHORUS, AND VITAMIN D

The principal causes of osteodystrophies are deficiencies or imbalances of dietary calcium, phosphorus, and vitamin D. Their interrelationships are not easily defined, and their interrelationship with the parathyroid gland must also be considered. Deficiencies of any of the three may be absolute or relative and must be assessed in relation to availability and growth rate.

The primary source of calcium and phosphorus is the diet. These elements are absorbed in amounts depending on the source of the minerals, intestinal pH, and dietary levels of vitamin D, calcium, phosphorus, iron, and fat. If there is a decrease in vitamin D or its activity, calcium and phosphorus absorption will be reduced. Vitamin D is obtained either through the diet or by production when the skin is exposed to sunlight (ultraviolet radiation). Before vitamin D can be used, it must be processed into its metabolically active form by the liver and kidney. Vitamin D_3 (cholecalciferol) acts primarily on the GI tract to increase absorption but also affects the bone, thereby increasing availability of elemental calcium.

Parathyroid hormone (PTH) is secreted in response to a low circulating calcium ion concentration. In general, it plays a role in increasing available calcium. The three target organs of PTH are the kidney, the bone, and the intestine. In the kidney, PTH promotes renal tubular absorption of calcium while enhancing renal excretion of phosphorus, thereby maintaining an appropriate calcium to phosphorus ratio. In the intestine, PTH promotes absorption of calcium. PTH also facilitates mobilization of calcium from bone by allowing utilization of calcium from the osteoid matrix.

Specific bony lesions are associated with abnormalities in absolute or relative amounts of vitamin D, calcium, phosphorus, and PTH. Often, in addition to the deficiency or excess in one element, this also causes a secondary pathology due to feedback mechanisms, altered ratios, or concomitant metabolic deficiencies. Specific disease syndromes can be classified as nutritional or metabolic in nature.

NUTRITIONAL OSTEODYSTROPHIES

RICKETS

Rickets is a disease of young, growing animals. The most common causes are dietary insufficiencies of phosphorus or vitamin D. Calcium deficiencies can also cause rickets, and while this rarely occurs naturally, poorly balanced diets that are deficient in calcium have been said to cause the disease. As in most diets causing osteodystrophies, the abnormal calcium to phosphorus ratio is most likely the cause.

Clinical Findings and Lesions: The characteristic lesions of rickets are failure of both vascular invasion and mineralization in the area of provisional calcification of the physis. This pathology is most obvious in the metaphyses of the long bones. There may be a wide variety of clinical signs, including bone pain, stiff gait, swelling in the area of the metaphyses, difficulty in rising, bowed limbs, and pathologic fractures. On radiographic examination, the width of the physes is increased, and the nonmineralized physeal area is distorted. In advanced cases, angular limb deformity can be seen due to asynchronous bone growth.

Animals fed all-meat diets are commonly affected. Kittens that are fed beef heart ex-