Articulations are divided into synarthroses, in which the osseous components are united by fibrous tissue or cartilage, and diarthroses, in which the opposing bone ends are covered with hyaline cartilage and are separated by a joint cavity filled with synovial fluid. Synarthroses are practically immovable and are rarely associated with joint disease other than fractures. In most cases, diarthroses are movable joints, with a variable degree of mobility depending on the anatomic location of the joint. Diarthroses are frequently involved in pathologic changes involving any of their anatomic structures: the fibrous joint capsule, synovial membrane, hyaline articular cartilage, subchondral bone, and intra-articular ligaments (and also the menisci in the stifle joint). Joint disorders may be caused by trauma (acute, sharp, or blunt), chronic inflammation, developmental factors, or infections. Acute trauma frequently results in luxation, subluxation, fracture, or distortion of a joint. Direct articular trauma may also lead to septic arthritis or rupture of a collateral ligament or the joint capsule.

Developmental defects include osteochondritis dissecans, equine ataxia, and lumbar disk syndrome in certain breeds of dogs. Extension of physitis into the adjacent joint and damage due to continuous abnormal weight bearing in animals with angular limb deformities are other inciting causes of joint disease. Bacterial and fungal infections involving joints and other synovial structures, such as tendon sheaths, are usually quite clearly recognizable and require immediate and aggressive treatment.

Chronic inflammation of joints and surrounding structures is most common in articulations associated with locomotion, although other joints, such as the temporomandibular, may occasionally be affected. Normal synovial fluid lubricates the synovial tissues in a joint through boundary lubrication, including a glycoprotein expressed from the cartilage during weight bearing. The synovial fluid also nourishes the articular cartilage. Any joint injury alters the volume and composition of the synovial fluid (as a result of an increased permeability of the inflamed synovial membrane relative to blood components) and increases the intraosseous pressure in the involved bones. The increased WBC count leads to an increased concentration of proteolytic enzymes within the synovial fluid, which leads to proteoglycan washout and eventually cartilage destruction.

Diagnostic procedures to determine the nature, extent, and exact location of the joint disorder include inspection, manual palpation and manipulation, diagnostic imaging techniques, local or intra-articular anesthesia, diagnostic arthroscopy, and laboratory examination of synovial fluid or biopsy of synovial membrane.

The diagnostic and therapeutic options for management of musculoskeletal disorders have greatly expanded during the last few years and allow a return to a useful life for most animals if done early in the disease process.

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.

MULTIPLE SPECIES

Contracted Flexor Tendons

Contracted flexor tendons are probably the most prevalent abnormality of the musculoskeletal system of newborn foals and calves. An autosomal recessive gene causes this condition. 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 931.)

Dyschondroplasia

Dyschondroplasia of genetic origin is seen 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 1950s largely have been eliminated through genetic selection. Short faces, bulging foreheads, prognathism, large abdomens, and short legs are characteristic. 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 is seen 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 characteristics are an important feature of breed type. In Malamutes, the condition is accompanied by anemia.

Dystrophy-like Myopathies

Numerous examples of progressive myopathies have been described in animals; many are heritable, and many resemble various types of muscular dystrophy in humans. Affected muscles have a variety of degenerative and atrophic changes. In Meuse-Rhine-Yssel cattle of Holland, a progressive fatal myopathy of the diaphragm and intercostal muscles has been described. Another dystrophy in cattle is weaver syndrome in Brown Swiss. Hyperplasia, commonly called double muscling (p 849), is a congenital myopathy found in some European breeds of cattle. Progressive myopathies have been reported in Merino sheep in Australia (an inherited autosomal recessive), in Pietrain pigs (Pietrain creeper syndrome), and in dogs, cats, chickens, turkeys, and mink. Inherited muscular dystrophy of mice and hamsters has been studied extensively; the hamsters have severe myocardial lesions and serve as a model for studies of cardiomyopathy.

Several types of muscular dystrophy are seen in dogs. An X-linked Duchenne-like muscular dystrophy is reported in Golden Retrievers in the USA and in Irish Terriers in Europe. Affected dogs, generally males, develop progressive muscular weakness, dysphagia, stiffness of gait, and muscular atrophy. Microscopically, the distinctive alteration is lack of dystrophia, a protein concentrated in the sarcolemma and essential for normal membrane function. Some dogs die with accompanying cardiomyopathy. A similar X-linked dystrophy with a lack of dystrophia is described in cats. A second type of dystrophy involves Labrador Retrievers in North America, Europe, and Australia. Clinical signs, which include stiffness, exercise intolerance, and muscular atrophy, develop by 6 mo of age. Autosomal recessive inheritance is implicated. A further dystrophy was described in dysphagic Bouviers in Europe.

Glycogen Storage Disease
(Glycogenosis)

Progressive muscular weakness and inability to rise properly may be seen in animals with glycogen storage diseases. To date, 5 of the 8 types of glycogen storage diseases characterized in humans have been identified in animals (types I, II, III, VII, and VIII). Affected species include cattle, sheep, dogs, cats, horses, Japanese quail, rats, and mice. Type II glycogenosis in Shorthorn and Brahman cattle has been well documented and is inherited as an autosomal recessive disorder. Affected cattle develop muscular weakness and die at 9-16 mo of age, often with accompanying cardiomegaly and congestive heart failure. Morphologic and biochemical study reveals extensive intralysosomal and cytoplasmic glycogen deposits. Corriedale sheep and Lapland dogs also develop type II glycogenosis.

Myophosphorylase deficiency (type V glycogenosis) is an autosomal recessive disorder in Charolais cattle. Affected cattle show exercise intolerance and may have increased serum activities of skeletal muscle-origin enzymes.

Muscular Steatosis

In muscular steatosis, which has been seen occasionally in cattle, sheep, and pigs at slaughter, fat replaces muscle fibers. No clinical disease results, and the cause is unknown. The gross lesions are symmetric, pale areas in affected muscles, especially of the back, neck, and upper limbs. Microscopically, many muscle fibers are replaced by fat cells.

Myopathy Associated with Congenital Articular Rigidity
(Arthrogryposis)

This syndrome, one of the more common congenital defects of calves, is characterized by rigid fixation of the limbs in abnormal postures; it often produces dystocia. Affected animals may have other anomalies, including hydrocephalus, palatoschisis, and spinal dysraphism. The condition may be lethal, but some mildly affected animals recover completely. The muscle lesions may be primary in some types of the disease, but the neural lesions generally are primary, and the muscular alterations represent denervation atrophy. Congenital articular rigidity is seen in cattle, sheep, horses, and pigs. Numerous etiologic factors have been recognized. In cattle, these include viral (Akabane virus [p 501], bluetongue virus [p 590]) and plant (*Lupinus* sp [p 2420]) teratogens, and a heritable recessive trait in Charolais (*see* ARTHROGRYPOSIS, p 849). In sheep, plant (locoweed) and viral (Akabane, Wesselsbron [p 624] teratogens, Rift Valley fever [p 617]), parbendazole exposure, and inherited autosomal recessive primary myopathies of Merino and Welsh Mountain lambs may cause congenital articular rigidity. In pigs, the condition may be inherited as an autosomal recessive, or result from deficiency of vitamin A or manganese or from exposure of pregnant sows to plant toxins (eg, tobacco, thornapple, hemlock, and black cherry).

Osteochondrosis

Osteochondrosis is a disturbance in endochondral ossification that is sometimes classified as dyschondroplasia. The immature articular cartilage may separate from the underlying epiphyseal bone, which sometimes dissects completely free and floats loose in the synovial cavity, resulting in accompanying synovitis or the retention of pyramidal cores of physeal cartilage projecting into the metaphysis. Often, these two lesions are seen simultaneously in the same bone. The disease develops during maximal growth when the biomechanical stresses are greatest in the immature skeleton (4-8 mo in dogs, 80-120 lb [36-54 kg] in pigs). It is most common in large and giant breeds of dogs and in rapidly growing pigs, horses (p 963), turkeys, and chickens.

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. It 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. Foramina 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.

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 1 or all 4 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 dewclaw is missing. The toes may be fused to give rise to polysyndactyly. Rarely 1 or all 4 limbs have the condition. Polydactyly in cattle appears to be polygenic with a dominant gene at one locus and a homozygous recessive at another.

CATTLE

Arthrogryposis

Arthrogryposis is ankylosis of the limbs, usually combined with a cleft palate and other growth deformities. It is seen 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. In the spinal cord, necrosis of neurons and lesions of the white matter may be seen. Arthrogryposis has more than one etiology and pathologic entity. The arthrogryposis syndrome in Charolais is 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 501) or bluetongue (p 590) virus can also cause arthrogryposis.

Brown Atrophy

(Xanthosis, Lipofuscinosis)

In dairy cattle with brown atrophy, the skeletal muscles and myocardium are yellow-brown to bronze. The masseter muscles and the diaphragm are affected most frequently. No clinical disease results. Microscopically, brown lipofuscin pigment granules accumulate under the sarcolemma or centrally in the muscle fibers. A genetic cause is presumed because certain breeds (eg, Ayrshire) are more predisposed than others.

Double Muscling

Double muscling is an overdevelopment of the musculature in the neonate. The condition is seen in various beef breeds including Charolais, Santa Gertrudis, South Devon, Angus, Belgian Blue, Belgian White, and Piedmontese. The muscles of the shoulder, back, rump, and hindquarters are separated by deep creases, particularly between the semitendinosus 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. Succinic dehydrogenase activity is significantly decreased in affected calves.

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 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.

HORSES

Angular Limb Deformities

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 4 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 with 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.

Defects of the Spine

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 (roach-back) 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 (T5-10) in lordosis and in the cranial lumbar region (L1-3) in kyphosis.

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. (*See* HYPERKALEMIC PERIODIC PARALYSIS, p 954.)

Polysaccharide Storage Myopathy

See CHRONIC EXERTIONAL RHABDOMYOLYSIS, p 952.

Glycogen Branching Enzyme Deficiency

Glycogen branching enzyme (GBE) deficiency may be a common cause of neonatal mortality in Quarter Horses that is obscured by the variety of clinical signs that resemble other equine neonatal diseases. Clinical signs of GBE deficiency may include transient flexural limb deformities, stillbirth, seizures, respiratory or cardiac failure, and persistent recumbency. Leukopenia, high serum CK, AST, and γ-glutamyl transferase are present in most affected foals. Gross postmortem lesions are inconclusive. Muscle, heart, or liver samples contain abnormal periodic acid-Schiff-positive globular or crystalline intracellular inclusions in amounts proportional to the foal's age at death. Accumulation of an unbranched polysaccharide in tissues is suggested by a shift in the iodine absorption spectra of polysaccharide isolated from the liver and muscle of affected foals. Skeletal muscle total polysaccharide concentrations are reduced, but liver and cardiac muscle glycogen concentrations are normal. Several glycolytic enzyme activities are normal, whereas GBE activity is virtually absent in cardiac and skeletal muscle, as well as in liver and peripheral blood cells. GBE activities in peripheral blood cells of dams of affected foals and several of their half-siblings or full siblings are ~50% of controls. GBE protein in liver is markedly reduced to absent in affected foals. Pedigree analysis supports an autosomal recessive mode of inheritance.

SHEEP

Spider Lamb Syndrome

Hereditary chondrodysplasia, or spider lamb syndrome, is an inherited, semilethal, musculoskeletal disease affecting lambs primarily of 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.

PIGS

Splayleg

(Spraddleleg, Myofibrillar hypoplasia)

In this condition of neonatal pigs, the hindlegs are spread apart or extended forward due to weakness of the adductor muscles relative to the abductors. Affected pigs are susceptible to overlaying, starvation, and chilling because of poor mobility. Mortality may reach 50%. Genetic influence has been demonstrated. There are significant differences in the incidence among litters of different sires and breeds. It is seen more frequently in males than females and in pigs of lower birthweight. The syndrome also may be produced if glucocorticoids are administered during pregnancy, and it appears possible that stress-sensitivity of the heavily muscled parent(s) may be a contributing factor. However, any cause of stretching of the adductor muscles increases the incidence. Stretching can result from slippery or sloping floors, struggling while legs are caught in cracks in the floor, or as the result of damage to nerve pathways from intrauterine viral infections. Mycotoxins have been suggested to play a role in some cases. The general nutrition of the sow (choline, methionine, and vitamin E levels) may influence the incidence, but benefits from feeding supplements to sows is questionable.

The clinical signs are distinctive. In utero infections with hemagglutinating encephalitis virus, enteroviruses, other viruses, and postpartum bacterial meningeal infection and trauma should be considered. The affected muscles are generally hypoplastic, and the small muscle fibers contain few myofibrils, as would be found in muscles of normal fetuses nearing parturition. Frequently affected muscles include the semitendinosus, longissimus dorsi, and triceps.

Dry, nonslippery floors should be provided, with no cracks in which the legs can become trapped, especially for the first 2 days. Pigs should be protected from injury by the sow, and adequate suckling should be ensured. In affected piglets, the hindlegs should be secured together above the hocks with a loose "figure 8" of adhesive tape for 2-4 days. Appropriately treated pigs usually recover within a week, although few recover if the front legs are also affected. Glucocorticoids should not be administered late in gestation. Highly susceptible blood lines should be eliminated.

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 3 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 vitamin D or its activity is decreased, calcium and phosphorus absorption are 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.