



TEXTBOOK:

Valentine BA, McGavin DA, Skeletal Muscle. In: [Pathologic Basis of Veterinary Disease](#), 5th Edition. Elsevier, 2012 pp 871-919.

<http://people.upei.ca/lopez/>

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SKELETAL MUSCLE MORPHOLOGY

Skeletal muscle is composed of elongated myofiber cells 1-40 mm long by 10-100 μm in diameter. Cells are multinucleated (100-300 nuclei/cell), non-branched with a parallel arrangement, and separated from each other by thin connective tissue (endomysium, perimysium, epimysium). The **sarcolemmal membrane** is formed by the endomysium and the basal lamina. The sarcoplasm of the myofibers has many myofibrils (80% of volume) arranged in sequence which gives muscle a distinct striation pattern (I/isotropic and A/anisotropic bands). "**A**" bands are larger and darker while "**I**" bands are lighter and shorter but contain a thin dark "**Z**" band in the middle. The **sarcomere** is the structural/functional unit of the muscle ("Z" to "Z" lines).

Types of muscle fibers (red and white): Based on metabolic and histochemical characteristics, muscle fibers are classified in two main types:

- **Type I fibers** are red, slow-twitch and slow-fatiguing. Their energy derives largely from oxidative metabolism. Type I fibers contain many mitochondria and a high concentration of myoglobin (cytochrome pigment). Myoglobin is largely responsible for the red color of muscles with a preponderance of type I fibers.
- **Type II fibers** white, fast-twitch and fast fatigue. Their energy derives largely from glycolysis. Type II fibers have fewer mitochondria and myoglobin than type I fibers. **Type II** fibers are also subdivided into **Type IIa** fibers (oxidative-glycolytic) which are fast contracting and fatigue resistant (more mitochondria than IIb). **Type IIb** (glycolytic) cells are also fast contracting but fatigue-susceptible due to rapid accumulation of lactic acid (less mitochondria).

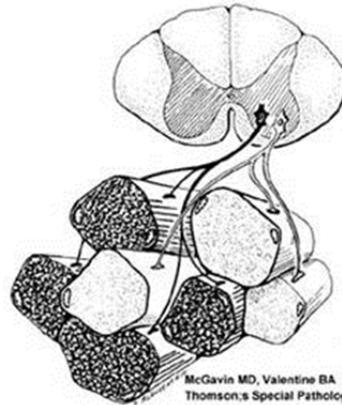
The proportion and distribution of fibers I and II

depend on animal species and functional adaptation.

The type of fiber (I or II) is determined by the type of innervating motor neurons (brainstem or ventral horn). The axon of a single motor neuron branches repeatedly and innervates individual muscle fibers.

In some animal species (i.e., avian) the predominance of specific fibers makes it possible to grossly differentiate muscle groups (i.e., breast vs. thigh).

Histochemical stains are required to differentiate types' I and II fibers.



EXAMINATION OF MUSCLES

- **Gross examination:** Check the color (pale, red or dark) and the volume (increased, normal or reduced) of the muscle group. Preferably, compare muscles with those of the opposite side or when possible with animals of the same age and breed. Be aware of possible artifacts caused by exsanguination, **algor mortis** and injection sites. Check also for changes in texture and appearance. Always cut the muscles into numerous slices.
- **Histopathological examination:** Most muscular diseases require microscopic examination of muscle. Do not forget that good fixation requires a 10:1 volume ratio (formalin to tissue). Put small (1 x 1 x 3 cm) slices of muscle (several sites) in formalin, both, from affected and normal sites. Submit tissues along with information regarding size and relative proportion (percentage) of lesions. Microscopic examination of muscle is difficult because tissue artifacts are rather common. Hyper-contraction artifacts occur when fixing muscle before **rigor mortis** has passed. Affected and non-affected muscles should be examined.

ABNORMALITIES OF GROWTH AND DEVELOPMENT

Arthrogryposis is a common congenital condition in aborted fetuses and stillborns characterized by small limbs with rigid joints(s). Rigidity of joints is caused by muscular hypoplasia which results from lack of muscle innervation during gestation. It is always associated with developmental alterations in the brain or spinal cord.

Arthrogryposis is frequently seen in:

- Dysraphism
- Spina bifida
- Syringomyelia
- Hydromyelia, etc.

These types of abnormalities in the development of the central nervous system are often associated to ingestion of a toxicant during gestation, or congenital viral infections (i.e., BVD).

GENERAL REACTIONS OF MUSCLE (INJURY AND HOST RESPONSE)

Rigor mortis is a postmortem contraction of skeletal muscles which results in the fixation of joints. It generally starts in the jaws and trunk and follows to the extremities. The presence and intensity of rigor mortis depend on several factors such as contents of body fat (emaciation), external and internal temperatures (fever, weather), and glycogen storage in muscle at the time of death, etc. Rigor mortis generally starts around 2-6 hours of death, peaking at 24-48 hours and disappearing by 72 hours. These times may be accelerated by body heat and exercise.

Muscle (myofiber) Atrophy: Muscular atrophy is the reduction of muscle size caused by a decrease of myofiber diameters (loss of myofilaments in the sarcomeres). Muscle atrophy is reversible providing the source of injury is removed. Histologically, there is a reduction in myofiber diameters with an unchanged amount of connective tissue (abnormal myofiber to endomysium ratios).

There are **three main types of muscle atrophy**:

i.- Denervation Atrophy (Neurogenic atrophy). There is a lack of tonic stimuli and muscle cells become atrophic. Causes of denervation atrophy include localized loss of nerve function (neuritis) or generalized loss of the entire motor unit (CNS). After denervation, muscles become rapidly atrophic and 50% of muscle mass could be lost in just few weeks. Examples of denervation atrophy are some forms of “**laryngeal hemiplegia**” (roarer horse) due to primary axonopathy or damage of the left recurrent laryngeal nerve; muscle atrophy in dogs with radial or brachial paralysis associated to trauma; atrophy of the supraspinatus muscles in horses (working horses/collar); human beings with poliomyelitis or a spinal cord injury.

ii.- Disuse Atrophy: There is intact innervation (tonic stimuli) but there is reduced movement because of pain, fracture, ankylosis, tenotomy, etc. Lesions are localized to affected groups of muscles.

iii.- Malnutrition Atrophy: A distinct type of progressive atrophy of muscle occurs in emaciation (cachexia), senility, cancer and chronic inflammatory disease (i.e., tuberculosis). In severe malnutrition, muscle ultimately becomes the source of nutrients (protein out of muscle). It may start from 24-48 hours following starvation in animals with little fat reserves. In cancer and chronic inflammation, atrophy of muscle is associated to the production of interleukin 1 (cachectin) by activated macrophages. In malnutrition atrophy, there is tonic stimulus and movement is intact. Lesions are generalized but essential muscles (i.e., diaphragm, tongue, etc.) are less affected.

Muscle Hypertrophy is the response to an increase in work demand (physiological / compensatory). There is an increase in the size but not in the number of muscle fibers. The diameter of hypertrophic muscle fibers is thickened due to formation of new myofilaments. Muscular hypertrophy can be enhanced by steroidal drugs.

Muscle Degeneration and Necrosis (myolysis): Degeneration is a common sequel to myofiber injury regardless of its cause (chemical, metabolic, traumatic, and infectious, etc.). Myofiber degeneration can be reversible, however, if injury progresses beyond the "point of no return," degeneration becomes irreversible and necrosis will follow. Muscle degeneration can only be detected grossly in severe lesions. Degenerated muscles appear pale. CAUTION: Degeneration should not to be mistaken with pale muscles of veal calves, anemia, exsanguination, fat (tongue), etc. If the calcification is extensive and severe, muscles show glistening, white, chalky foci (streaks). Red discoloration may be present when degenerated muscle coexists with hemorrhage or with sudden and extensive release of myoglobin (rhabdomyolysis) into the interstitium. Microscopic changes include vacuoles and loss of striations followed by swelling (one or more segments), hypereosinophilia, glassy or hyaline appearance (Zenker's degeneration) and finally segmental rupture of fibers and formation of retraction caps. The calcification of degenerated muscle is common in some diseases

Calcification (deposition of Ca^{++}) is a common sequel to muscle degeneration and necrosis. When the calcification is extensive and severe, there may be gross changes seen such as white, chalky foci. Microscopically (H&E), calcified fibers have granular, bluish granules resembling bacteria. Special stains such as Von Kossa are used to conclusively demonstrate calcium in affected tissues.

Regeneration: Skeletal muscle has a remarkable ability to regenerate, providing the lesion is segmental and the sarcolemmal tube and myosatellite cells remain intact. Macrophages and to lesser extent neutrophils clean cell debris within 12 hours of necrosis. The sarcolemmal tubes (endomysium + basal lamina) serve as scaffold for the myoblasts and as a barrier to prevent fibroblasts from getting into the sarcoplasm. Myoblasts (Myosatellite cells) undergo mitosis and repair takes place by the formation of new sarcomeres at the edges of ruptured myofibers. The edges then become bridged by newly formed sarcomeres. If sarcolemmal tubes are disrupted (trauma, infarction, infection), partial regeneration can occur but is generally complicated with fibrosis (scar).

Other abnormal colors are occasionally found in muscles. Black discoloration is seen in melanosis (rare); brown discoloration (tan) occurs in the so-called xanthomatosis (lipofuscinosis/wear and tear pigment). Both types of discoloration are considered incidental findings.

MYOPATHIES (NUTRITIONAL, METABOLIC, EXERTIONAL, TRAUMATIC)

NOTE: Muscular dystrophy is a group of rather important hereditary and idiopathic diseases often fatal in humans characterized by progressive weakness and muscular degeneration because the absence or presence of abnormal muscular proteins. Hereditary muscular dystrophies are rare and of little importance in animals (cattle, sheep, dogs). "**Muscular dystrophy**" is often misused as a synonym of "**myopathy**" in the veterinary jargon.

NUTRITIONAL MYOPATHY

White Muscle Disease (Nutritional Myopathy; Nutritional Myodegeneration):

It is a very common and serious condition in farm animals. It has variable morbidity and mortality affecting up to 50% of animals. WMD generally affects rapidly growing, young, well thrift sheep, cattle, pigs, and is less common in foals and goats. It is also seen in captive minks (WMD / steatitis). WMD is associated to vitamin/selenium deficiency but it is exacerbated by other factors such as exercise, environment (climatologic conditions may be involved), nutrition and some toxicants. The occurrence of WMD is unpredictable and the theory of a geographic predisposition has been recently challenged. WMD is occasionally found in neonates. WMD in pigs may be independent or coexist with other vitamin E or selenium deficiency syndromes (mulberry heart, hepatitis dietetica).

The pathogenesis of WMD is related to the oxidation of cell membrane lipids (lipoperoxidation) by free radicals due to lack of oxygen radical-scavengers such as tocopherol (Vitamin E) and selenium containing enzymes glutathione peroxidase /reductase). Membrane peroxidation induces a positive influx of Ca^{++} into sarcoplasm and mitochondria. A considerable amount of energy is required to remove Ca^{++} out of the cell. Once cell energy is exhausted, myofibers degenerate and Ca^{++} accumulates up to 50 times the normal amount. Intracellular enzymes such as CPK leak out from the cell into serum. Finally, degenerated myofibers undergo necrosis. Since muscle activity relates to the production of free radicals, muscles with higher activity such as diaphragms, intercostals, tongue and heart are more severely affected (type I fibers).

Gross Pathology: It is difficult to see in mild cases. In severe cases (fatal), affected muscles appear pale with calcifications. **CAUTION:** WMD should not be mistaken with normally pale muscles of veal calves or animals with anemia. Do not forget to inspect the heart (LV in calves; RV in sheep). For confirmation take samples of Type I muscles (diaphragm, intercostal, etc.) and fix them in formalin.

Histopathology: Segmental degeneration (*Zenker's*, flocculant degenerations), hypercontraction with classic loss of striation, hypereosinophilia, fibre fragmentation (retraction caps), calcification (severe, see pathogenesis), proliferation of myosatellite cells (attempts to repair), and macrophages cleaning cell debris.

METABOLIC MYOPATHIES

Porcine Stress Syndrome (PSS), also known as **Porcine Malignant Hyperthermia** and **Pale Soft Exudative Pork** is a hereditary (» Landrace, Pietran, » Hampshire, Yorkshire, rare in other breeds), life-threatening, hypermetabolic syndrome. PSS is characterized by respiratory and metabolic acidosis, myoglobinemia, hyperkalemia, high blood lactate, hyperthermia, cardiovascular collapse and death. Any type of stress such as fighting, exercise or heat can trigger PSS. The pathogenesis is related to an inherited defect in the intracellular uptake, storage and release of Ca^{++} ions. Excessive Ca^{++} in the cell (consumption of ATP) progresses to degeneration and necrosis of fibers. Denaturation of sarcoplasmic proteins leads to movement of intracellular water into the interstitium (see gross lesions). In the past, exposure to halothane was used to detect susceptible pigs (unclear mechanism). Currently, there is a blood test for PSS. **Gross Pathology:** muscles with higher proportions of type II fibres (back: longissimum, psoas; semitendinosus) are pale and wet (an interstitial edema). To facilitate postmortem diagnosis, make several longitudinal cuts with a knife in affected muscles to promote interstitial fluid to escape (water oozes and drips). Extra-muscular lesions include a severe pulmonary edema (froth in the trachea) and accumulation of a clear fluid in abdominal and thoracic cavities. **Histopathology:** Early myofiber degeneration, necrosis, and interstitial edema.

EXERTIONAL MYOPATHIES

Exertional Myopathies comprise a group of diseases which result in severe muscle degeneration following strenuous exercise. Lesions are similar to WMD but affect mainly major muscle masses with predominantly glycolytic type II fibers. Postulated pathogenesis includes rapid utilization of glycogen, accumulation of lactic acid, alteration in protein structure, loss of water, an interstitial edema, compressive circulatory disturbances, ischemia, degeneration, necrosis, myoglobinemia and myoglobinuria.

The most notable forms of exertional myopathies are:

- **Azoturia (paralytic myoglobinuria, Monday morning disease, sacral paralysis).** In the old days it was thought that Azoturia was an equine disease associated to strenuous exercise after a long resting period along with a diet rich in carbohydrates predisposing to lactic acidosis. The carbohydrate-rich theory is no longer accepted and current literature suggests that the basic underlying problem is an intrinsic muscle “polysaccharide storage disease” known as **Equine Polysaccharide Storage Myopathy (EPSSM)**. Azoturia or EPSSM is clinically characterized by muscle weakness, diaphoresis (sudoresis), reluctance to move, myoglobinuria, and in severe cases, recumbence (prelude to death), renal failure, acidosis and death. **Gross Pathology:** Muscles of the extremities (gluteal/lumbar) are swollen, edematous, dark (myoglobin stain), and the kidneys appear dark-black. In horses that survive, affected muscles become pale and atrophic. **Histopathology:** In acute cases, there is segmental degeneration and necrosis with little or no calcification and in chronic cases there is fibrosis and atrophy. In severe cases of azoturia, there is also toxic injury to the kidneys (myoglobinuric nephrosis) which may result in death from renal failure.
- **Tying Up (Setfast, Acute Rhabdomyolysis, Transient Exertional Rhabdomyolysis):** It is considered by some to be a mild, rapidly regressive form of azoturia. There are no gross lesions (euthanized horses), but microscopic examinations of tissues reveal a mild to severe rhabdomyolysis. It rarely produces visible myoglobinuria. A similar syndrome has been reported in racing greyhound dogs (lumbar muscles).
- **Capture Myopathy** is an acute and often fatal myopathy of wild mammals and birds. The disease is generally preceded by a chase, struggle or transport. The pathogenesis is similar to Azoturia. Lesions: muscle degeneration, hemorrhage with occasional rupture of tendons.
- **Compartment Syndrome** is characterized by degeneration and necrosis of muscles that are surrounded with a heavy aponeurosis (connective tissue). It occurs in poultry (deep pectoral myopathy) and well-conditioned athletes (pre-tibial muscles). The pathogenesis is based on muscular expansion in a non-expandable compartment. This results in vascular compression, ischemia and infarction of the muscle fibers. **Deep pectoral myopathy** is more frequently seen in poultry, affecting the deep pectoral musculature (degeneration and necrosis of supracoracoid muscles). This disease of some breeds of turkeys and chickens is preceded by a brief but vigorous flapping of wings. The lesions appear a few hours later. **Pathology:** There is localized necrosis of the supracoracoid muscles.

TRAUMATIC MYOPATHIES

- **Downer Syndrome:** It is an ischemic necrosis of ventral and limb muscles following prolonged recumbence (disease/anesthesia). Mature animals (cows > sheep, pigs, horses) in good bodily condition are most susceptible. Lesions appear as early as 6-12 hours. There is a vicious circle

(prostration ► muscle injury ► edema ► compression ► venous occlusion ► ischemia ► injury ► prostration...). **Gross Pathology:** Combination of ischemia / degeneration/fibrosis (pale) and congestion/hemorrhage /myoglobin (red).

- **Equine Post-anesthetic Myopathy:** It occurs in 3-6% of anesthetic cases. Severity varies from muscle swelling with lameness to paresis with renal failure and shock. Sequels include permanent loss of muscle function.
- **Crush Syndrome** is an acute degenerative myopathy caused by severe trauma to a muscle group. *Pathogenesis and lesions* are similar to Downer's syndrome.

MYOSITIS (Bacterial, parasitic, immune mediated, idiopathic)

Blackleg (**Black quarter** , **symptomatic anthrax** , **emphysematous gangrene**) is an acute fatal emphysematous myositis caused by *Clostridium chauvoei* . The pathogenesis is complex:

- **First step.-** Soil-containing spores ► ingestion ► spores to GIT/liver ► spores in muscle (latent).
- **Second step.-** Muscle injury/hemorrhage ► change in local environment (O₂ tension, pH, etc.) ► germination of spores in the muscle ► exotoxins ► edema, myonecrosis ► emphysema ► generalized toxemia ► death (□24 hours).
- **Gross Pathology:** It generally affects large muscle masses such as those of the pectoral, pelvic, crural and scapular regions. It occasionally involves the musculature of the tongue, heart or diaphragm. Affected muscles are black, with gas (emphysema) and crepitate on palpation. A rancid (butter) odor is reported in affected tissues. Peripherally to the lesion, there is a mild interstitial and subcutaneous edema (gelatinous fluid). Fibrin tags are typically found on the epicardial and pleural surfaces. Other lesions include diffuse serosal hemorrhages, an early bloat and severe pulmonary edema. For laboratory diagnosis, submit formalin-fixed tissues for histopathology and fresh (non-fixed) tissues for a FA test (a confirmatory test).
- **Histopathology:** Microscopically, lesions are somehow disappointing as compared to the magnitude of gross lesions. There are segmental degeneration, necrosis, discrete edema, occasional neutrophils and emphysema in affected muscle. Bacteria are rarely seen in the tissues.

Gas Gangrene (Malignant Edema) is an acute fatal infection caused by *Clostridium septicum*, *Cl. perfringens* , *Cl. novyi*, *Cl. sordelli* and *Cl. chauvoei* alone or in combination. It generally affects ruminants, horses and swine, and carnivores are only sporadically affected. The route of entry of these anaerobic, highly toxigenic, opportunistic organisms is always through a wound. Spores of these clostridia are commonly present in soil and feces. Not all wounds contaminated with these organisms result in gas gangrene since optimal conditions are required, i.e., deep wounds with low oxygen tension. Affected areas appear cold on palpation (live animals), there is crepitating, generalized signs of toxemia, and finally prostration death.

- **Gross Pathology:** Lesions are locally extensive involving mostly connective tissue and

characterized by severe edema (gelatinous fluid), minimal emphysema, hemorrhage, discoloration of overlying skin.

- **Histopathology:** Edema, cellulitis, minimal myositis. Final diagnosis also requires FA tests.

Overall (subjective) differences between black leg and malignant edema				
Disease	Myositis	Cellulitis	Edema	Emphysema
Black Leg	+++	+	++	++++
Malignant Edema	+	++++	++++	+++

Viral Myositides are rare in North America. The most common viral diseases causing myositis is Foot and Mouth Disease and Bluetongue

Parasitic Myositis: Several important parasites cause myositis in domestic animals.

- **Trichinosis** (*Trichinella spiralis*) is an important zoonotic disease (rare in Canada/USA except in the Arctic) acquired through consumption of incomplete cooked meat from pork, bears, or aquatic mammals. The adult stage of the parasite lives in the small intestine. Larvae migrate from the intestine to tissues, with some predilection for muscles of tongue, masseter, diaphragm intercostals and eye. Larvae become encysted in the muscle. The larvae induce only minimal host response and therefore it is difficult to see them grossly without the help of a trichinoscope. Larvae penetrate myofiber (ratio 1:1), induce a transient (localized) lymphoplasmacytic response that become "encapsulated." The larvae remain viable for years, or die and become calcified (small visible white nodules).
- **Cysticercosis** is another important zoonotic disease prevalent in many developing countries and only occasionally seen in Canada and the USA. The cysticercus is the larval stage of a taenia (Platyhelminth) with indirect life-cycle; adult stages (tapeworm) reside in the intestine of definitive hosts (carnivores); the larval stage (cysticercus) resides in the muscle of an intermediate host (i.e., pigs and cattle). Cysticercosis in humans implies fecal/oral contamination involving the ingestion of taenia eggs (not from ingestion of cysticerci). In some hosts, including human beings, the cysticercus may reside in the brain in the so-called neurocysticercosis. There are various species of cysticerci and taenias. **Gross Pathology:** Cysticerci form large visible cysts (1-2 cm) which generally contain a clear fluid and larvae. **Histopathology:** Encysted cysticerci in muscle fibres with minimal inflammatory response (few lymphocytes and/or eosinophils). Dead larvae generally become intensively calcified.
- **Sarcocystosis (Sarcosporidiosis)** is an important protozoal disease affecting primarily herbivores and pigs. The parasite has an indirect life-cycle where carnivores (dogs, cats, human beings) are definitive hosts and herbivores, pigs and birds are the intermediate hosts. **Gross pathology:** Most infections do not produce gross lesions except some rare cases in which a large cyst contains trillions of protozoal parasites (sheep). Sarcocystis are so common that most herbivorous have parasites in their musculature, however, these protozoa cause no inflammatory response. A few animals develop nonspecific focal myositis when the microscopic cyst surrounding the parasite is broken into the host cell. **Histopathology:** Parasites are frequently present in the muscle fibers causing little if any injury or host response. Because of this, many pathologists considered *Sarcocystis* in muscles just an incidental finding. Although muscle lesions are not important, sarcocystosis is considered by some a serious clinical disease and important cause of abortion.

Recent studies associate *Sarcocystis* with Eosinophilic Myositis, a condition seen sporadically in cattle and sheep (meat inspection). Eosinophilic myositis appears grossly as focal or locally extensive, well-demarcated green discoloration in the muscle.

Note: Other parasites such as Toxoplasma, Trypanosoma (rare in Canada/USA), will be covered in other systems (cardiovascular, nervous, etc.).

IDIOPATHIC AND IMMUNE MEDIATED MYOSITIS

Masticatory Muscle Myositis (MMM) is a rare and acute (relapsing) **eosinophilic myositis** in dogs characterized by swollen painful jaws, and blood eosinophilia. Another progressive condition called **atrophic myositis** has also been described in dogs (long-nosed breeds). Some investigators suggest that MMM is the same condition as atrophic myositis, but at different stages of development (Acute vs. Chronic). The pathogenesis is not completely understood by auto-antibodies against a particular protein present in fibers IIM (unique fiber in masticatory muscles) has been suggested. This specific type of myosin shares antigenic determinants with some bacteria and this may explain why antibodies are formed. **Gross Pathology:** Lesions are bilateral. In acute cases there are edema and extensive infiltration of eosinophils in the masseter, temporal, pterygoid muscles. In the chronic form, the cellular infiltrates change to lymphocytes and plasma cells and the fibers become atrophic.

Canine Polymyositis: It is a rare condition affecting dogs of presumed autoimmune etiology. It involves most muscles and is characterized by degeneration, necrosis with infiltration of lymphoplasmacytic cells and occasional eosinophil.

Dermatomyositis is a familial disease of Collies and Shelties, possibly immune-mediated clinically characterized by **dermatitis** (face, tip of tail, bony prominence) and **myositis** (masticatory muscles). Most dogs recover spontaneously.

Myasthenia gravis, a rare but important disease of human beings, is seen sporadically in dogs and cats. It is a disease of the neuromuscular junction (post-synaptic membrane -motor end plate), which causes weakness and severe muscular fatigue from the mildest exercise. Two types of myasthenia gravis are described:

- **Hereditary** in which animals are born with reduced number of acetylcholine (ACh) receptors
- **Acquired** in which animals develop antibodies (IgG) against ACh receptors later in the postnatal life.

There is dramatic improvement in the clinical signs following administration of anti-cholinesterase drugs (neostigmine). **Histopathology:** There is muscle atrophy (disuse type). In human beings, 70% of patients with myasthenia also have thymus abnormalities (thymitis, follicular hyperplasia, thymoma). Megaesophagus, dysphagia, and aspiration pneumonia are common complications in dogs.

NEOPLASTIC DISEASES OF MUSCLE

Spontaneous tumors of striated muscles are rare in veterinary medicine but common in experimental pathology (toxicology). Neoplasia arising in muscle may include tumors from striated muscle, adipose cells (lipoma/sarcoma), fibrous connective tissue (fibroma/sarcoma), nerves (neurofibroma) or vascular cells (hemangioma/sarcoma). Tumors of striated muscle likely originate from embryonic remnants of myofibers. Striated muscle neoplasia occurs most frequently in the heart, muscles and sporadically from non-muscular sites such as kidney, urinary bladder and lung.

- **Rhabdomyoma** is a benign and generally congenital tumor of skeletal muscle. It is most frequently found in the cow, sheep and pig. As many as 66% of rhabdomyomas originate in the heart (see cardiovascular system). **Gross Pathology:** These tumors appear as a large pedunculated mass embedded in the heart. **Histopathology:** Neoplastic cells have characteristic cross-striation resembling skeletal muscle.

- **Rhabdomyosarcoma** is the malignant counterpart of the rhabdomyoma and it frequently metastasizes to surrounding tissues and distal organs. Rhabdomyosarcoma is most commonly found in the cow, sheep, dog and horse but have never been reported in pigs. **Gross pathology:** The tumors are poorly encapsulated spherical nodules formed by pink/grey tissue. Metastasis to lung, spleen, lymph nodes and kidneys are common. **Metaplastic Rhabdomyosarcomas** arising from sites with no striated muscle such as kidney and urinary bladder are sporadically reported. **Histopathology:** Extremely variable, with or without striation, or giant cells. In the most anaplastic tumors it is necessary to do immunohistochemistry to demonstrate myosin in neoplastic cells.

GLOSSARY

A/anisotropic, "A" bands Acetyl-choline receptors Acute Rhabdomyolysis Algor mortis Ankylosis Aponeurosis Aspiration pneumonia Atrophy Azoturia Basal lamina Black quarter Blackleg Blue tongue Brachial paralysis Budding Cachexia Calcification Canine Polymyositis Capture Myopathy Cellulitis	Gangrene Gas Gangrene Giant cell Glutathione peroxidase Glycolysis H&E Histochemical stains Hypercontraction artifacts Hypereosinophilia Hyperkalemia I/isotropic, "I" bands Idiopathic Infarction Injection site Intermediate host Ischemic necrosis Lactic acid Laryngeal hemiplegia Lipofuscinosis	Perimysium Poliomyelitis Porcine Stress Syndrome Post-synaptic membrane PSS Pulmonary edema Radical-scavengers Regeneration Retraction cap Rhabdomyolysis Rhabdomyoma Rhabdomyosarcoma Rigor mortis Sarcocystosis Sarcolemma Sarcolemmal tube Sarcomeres Sarcoplasm Sarcoplasmic reticulum Sarcosporidiosis Selenium
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Chronic atrophic myositis <i>Clostridium chauvoei</i> <i>Clostridium novyi</i> <i>Clostridium perfringens</i> <i>Clostridium sordelli</i> Compartment Syndrome CPK Crush Syndrome <i>Cysticercosis</i> <i>Cysticercus</i> <i>Cysticercus bovis</i> <i>Cysticercus cellulosae</i> Definitive host Denervation Diaphoresis Disuse Downer Syndrome Dysphagia Emphysematous gangrene End-stage muscle Endomysium Eosinophilic myositis Epimysium Exertional Myopathies Exertional Rhabdomyolysis FA test Fecal/oral contamination Fast-twitch Fibroblasts Foot and Mouth Disease Free radicals	Lipoperoxidation Malignant Edema Malignant Hyperthermia Masticatory Muscle Myositis Megaesophagus Melanosis Mitochondria Monday morning disease Motor end plate Motor neurons Muscle hypertrophy Muscular dystrophy Myasthenia gravis Myoblasts Myofibers Myofibrils Myoglobin Myoglobinemia Myoglobinuria Myolysis Myonecrosis Myopathy Myosatellite cells Myositis Myotubules Neurogenic atrophy Neuromuscular disease Neuromuscular junction Nutritional myopathy Pale Soft Exudative Pork Paralytic myoglobinuria	Skeletal muscle Slow-twitch Suprascapular muscle Symptomatic anthrax <i>Taenia saginata</i> <i>Taenia solium</i> Thymitis Thymoma Tocopherol Tonic stimuli Toxaemia Toxoplasmosis <i>Trichinella spiralis</i> Trichinosis Trypanosome Tying Up Type IIa fibres Type IIb fibres Vasculitis Vitamin E Von Kossa stain Wear and tear pigment White Muscle Disease WMD Xanthomatosis Zenker's degeneration Zoonotic diseases
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THE END

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