DOWNLOAD PDF MUSCLE WEAKNESS AND DISORDERS

Chapter 1 : Diseases and Conditions Inflammatory Myopathies

Muscle Weakness Diseases, Disorders And Treatment If you have muscle pain or unhealthy conditions related to your muscle weakness, find out what exactly your symptoms are. In case, you have muscle weakness diseases, you will know how to improve your condition and enhance your health actively.

Her early research looked at mechanisms of disease progression in mouse models of Duchenne and Congenital muscular dystrophy. More recent work relates to developing skeletal, pulmonary and cardiac outcomes in infants, boys, and men with Duchenne Muscular Dystrophy DMD. Over the last 10 years, she has been the site PI for multiple studies involving children with DMD, congenital muscular dystrophy, and spinal muscular atrophy. Community work has included service to the local Muscular dystrophy association as Camp Doctor for more than two decades. She has also collaborated in developing standard of care guidelines for treatment of children with DMD, congenital muscular dystrophy, and spinal muscular atrophy. She is the site PI for studies involving children with Duchenne muscular dystrophy and congenital muscular dystrophy. Motor neurons are the cells in the spinal cord and brainstem that control voluntary muscle movement. When these cells are damaged, the muscles become weak and get small atrophy. The muscles of the arms and legs are often involved, and even muscles of breathing and swallowing can be involved. Motor neuron diseases refers to disorders in which there is progressive weakness of muscles due to a problem with the motor neuron. The most common motor neuron disorder in children is spinal muscular atrophy SMA. Spinal muscular atrophy will not be discussed here as it is reviewed in another section of this website. However, there are other types of motor neuron disease that happen because of miscoding of the genes or because of exposures that damage the motor neurons. Description Motor neuron disease in children occurs in two ways: The symptoms common to motor neuron disorders from either cause include muscle twitching fasciculations , low tone, weakness, and low muscle bulk. These symptoms can occur at any age from neonates to adulthood. In general, the earlier the symptoms are seen, the more severe the course of weakness will be. In some people, the symptoms start when they are teens or young adults, and these patients have mild weakness and continue to be able to walk. The history and physical examination are crucial to figuring out the cause. Some questions your doctor may ask you: Testing may include an electrophysiologic tests called electromyography EMG and nerve conduction studies NCS , which tests the function of the nerve and the muscles; magnetic resonance imaging MRI ; and blood tests for genetic causes. Physical Abnormalities Weakness and loss of muscle bulk are the hallmarks of motor neuron disease. If the weakness has been long-standing and severe, then contractures of the joints can occur. In babies, if there are multiple contractures of the joints, the term arthrogryposis multiplex congenita is used to describe contractures that start from birth. In older children, the contractures may be more mild and present only in the heel cords. What other symptoms can occur with hereditary motor neuron disease? Children whose motor neuron disease results from miscoding of genes can have other neurological symptoms associated with their weakness, such as:

Chapter 2 : About Mitochondrial Disease - Mito FAQ | Mitochondrial Disease Action Committee - MitoActio

Although the metabolic muscle diseases characterized by exercise intolerance typically don't involve muscle weakness, some chronic or permanent weakness can develop in response to repeated episodes of rhabdomyolysis and to the normal loss of strength that occurs with aging.

Central muscle fatigue manifests as an overall sense of energy deprivation, while peripheral muscle fatigue manifests as a local, muscle-specific inability to do work. When a nerve experiences synaptic fatigue it becomes unable to stimulate the muscle that it innervates. Most movements require a force far below what a muscle could potentially generate, and barring pathology , neuromuscular fatigue is seldom an issue. As there is insufficient stress on the muscles and tendons, there will often be no delayed onset muscle soreness following the workout. It is this "neural training" that causes several weeks worth of rapid gains in strength, which level off once the nerve is generating maximum contractions and the muscle reaches its physiological limit. Past this point, training effects increase muscular strength through myofibrillar or sarcoplasmic hypertrophy and metabolic fatigue becomes the factor limiting contractile force. Central fatigue[edit] Central fatigue is a reduction in the neural drive or nerve-based motor command to working muscles that results in a decline in the force output. Serotonin binds to extrasynaptic receptors located on the axon initial segment of motoneurons with the result that nerve impulse initiation and thereby muscle contraction are inhibited. This is the most common case of physical fatigueâ€”affecting a national[where? This causes contractile dysfunction that manifests in the eventual reduction or lack of ability of a single muscle or local group of muscles to do work. The insufficiency of energy, i. Peripheral regulation therefore depends on the localized metabolic chemical conditions of the local muscle affected, whereas the central model of muscle fatigue is an integrated mechanism that works to preserve the integrity of the system by initiating muscle fatigue through muscle derecruitment, based on collective feedback from the periphery, before cellular or organ failure occurs. Therefore, the feedback that is read by this central regulator could include chemical and mechanical as well as cognitive cues. The significance of each of these factors will depend on the nature of the fatigue-inducing work that is being performed. Though not universally used, "metabolic fatigue" is a common alternative term for peripheral muscle weakness, because of the reduction in contractile force due to the direct or indirect effects of the reduction of substrates or accumulation of metabolites within the muscle fiber. Lactic acid hypothesis[edit] It was once believed that lactic acid build-up was the cause of muscle fatigue. The impact of lactic acid on performance is now uncertain, it may assist or hinder muscle fatigue. Produced as a by-product of fermentation , lactic acid can increase intracellular acidity of muscles. Fatigue reduced ability to generate force may occur due to the nerve, or within the muscle cells themselves. New research from scientists at Columbia University suggests that muscle fatigue is caused by calcium leaking out of the muscle cell. This causes there to be less calcium available for the muscle cell. In addition an enzyme is proposed to be activated by this released calcium which eats away at muscle fibers. They include molecules such as adenosine triphosphate ATP , glycogen and creatine phosphate. Creatine phosphate stores energy so ATP can be rapidly regenerated within the muscle cells from adenosine diphosphate ADP and inorganic phosphate ions, allowing for sustained powerful contractions that last between 5â€”7 seconds. Glycogen is the intramuscular storage form of glucose , used to generate energy quickly once intramuscular creatine stores are exhausted, producing lactic acid as a metabolic byproduct. Substrates produce metabolic fatigue by being depleted during exercise, resulting in a lack of intracellular energy sources to fuel contractions. In essence, the muscle stops contracting because it lacks the energy to do so. Grading[edit] The severity of muscle weakness can be classified into different "grades" based on the following criteria: No contraction or muscle movement. Trace of contraction, but no movement at the joint. Movement at the joint with gravity eliminated. Movement against gravity, but not against added resistance. Movement against external resistance with less strength than usual. Classification[edit] Proximal and distal[edit] Muscle weakness can also be classified as either " proximal " or " distal " based on the location of the muscles that it affects. True and perceived[edit] Muscle weakness can be classified as either "true" or "perceived" based on its cause. Perceived muscle weakness or

non-neuromuscular weakness describes a condition where a person feels more effort than normal is required to exert a given amount of force but actual muscle strength is normal, for example chronic fatigue syndrome. This is also true for some cases of chronic fatigue syndrome , where objective post-exertion muscle weakness with delayed recovery time has been measured and is a feature of some of the published definitions. Underlying factors and adaptation mechanisms". Annals of Physical and Rehabilitation Medicine. Retrieved 1 January European journal of applied physiology and occupational physiology. Archives internationales de physiologie, de biochimie et de biophysique. Int J Sport Nutr. Advances in Experimental Medicine and Biology. Acta Physiol Scand Suppl. The New York Times. Concepts and Clinical Practice 7th ed. Journal of Chronic Fatigue Syndrome.

Chapter 3 : Myasthenia Gravis Fact Sheet | National Institute of Neurological Disorders and Stroke

Muscle disorders can cause weakness, pain or even paralysis. Causes of muscle disorders include Injury or overuse, such as sprains or strains, cramps or tendinitis.

One symptom indicating muscular disease is weakness, usually symmetrical that is, affecting both sides of the body and mainly affecting the proximal or girdle muscles. This type of weakness may be noticed when climbing stairs, arising from a deep chair, brushing the hair, orâ€¦ Indications of muscle disease Muscular atrophy and weakness are among the most common indications of muscular disease see below Muscle weakness. Though the degree of weakness is not necessarily proportional to the amount of wasting, it usually is so if there is specific involvement of nerve or muscle. Persistent weakness exacerbated by exercise is the primary characteristic of myasthenia gravis. Pain may be present in muscle disease because of defects in blood circulation, injury, or inflammation of the muscle. Pain is rare, except as a result of abnormal posture or fatigue in muscular dystrophy â€"a hereditary disease characterized by progressive wasting of the muscles. Cramps may occur with disease of the motor or sensory neurons, with certain biochemical disorders e. Muscle enlargement muscular hypertrophy occurs naturally in athletes. Hypertrophy not associated with exercise occurs in an unusual form of muscular dystrophy known as myotonia congenita , which combines increased muscle size with strength and stiffness. Pseudohypertrophy, muscular enlargement through deposition of fat rather than muscle fibre, occurs in other forms of muscular dystrophy, particularly the Duchenne type. Tetany is the occurrence of intermittent spasms, or involuntary contractions , of muscles, particularly in the arms and legs and in the larynx, or voice box; it results from low levels of calcium in the blood and from alkalosis , an increased alkalinity of the blood and tissues. Tetanus , also called lockjaw, is a state of continued muscle spasm, particularly of the jaw muscles, caused by toxins produced by the bacillus Clostridium tetani. The twitching of muscle fibres controlled by a single motor nerve cell, called fasciculation, may occur in a healthy person, but it usually indicates that the muscular atrophy is due to disease of motor nerve cells in the spinal cord. Fasciculation is seen most clearly in muscles close to the surface of the skin. Glycogen is a storage form of carbohydrate, and its breakdown is a source of energy. Muscle weakness is found in a rare group of hereditary diseases, the glycogen-storage diseases , in which various enzyme defects prevent the release of energy by the normal breakdown of glycogen in muscles. As a result, abnormal amounts of glycogen are stored in the muscles and other organs. The best-known glycogen-storage disease affecting muscles is McArdle disease , in which the muscles are unable to degrade glycogen to lactic acid on exertion because of the absence of the enzyme phosphorylase. Abnormal accumulations of glycogen are distributed within muscle cells. Symptoms of the condition include pain, stiffness, and weakness in the muscles on exertion. McArdle disease usually begins in childhood. No specific treatment is available, and persons affected are usually required to restrict exertion to tolerable limits. The condition does not appear to become steadily worse, but serious complications may occur when the muscle protein myoglobin is excreted in the urine. Other glycogen-storage diseases result from deficiency of the enzymes phosphofructokinase or acid maltase. With acid maltase deficiency , both heart and voluntary muscles are affected, and death usually occurs within a year of birth. Muscle weakness Signs and symptoms Weakness is a failure of the muscle to develop an expected force. Weakness may affect all muscles or only a few, and the pattern of muscle weakness is an indication of the type of muscle disease. Often associated with muscle weakness is the wasting of affected muscle groups. A muscle may not be fully activated in weakness because of a less than maximal voluntary effort; a disease of the brain, spinal cord, or peripheral nerves that interferes with proper electrical stimulation of the muscle fibres; or a defect in the muscle itself. Only when all causes have been considered can weakness be attributed to failure of the contractile machinery i. The effect of weakness in a particular muscle group depends on the normal functional role of the muscle and the degree to which force fails to develop. The overall disability is not as great as weakness of more proximal closer to the body muscles controlling the pelvic or shoulder girdles, which hold large components of the total body mass against the force of gravity. If the weakness is severe, the arms cannot be raised at all. Assessment Muscle disease may be detected by assessing whether the

muscle groups can withhold or overcome the efforts of the physician to pull or push or by observing the individual carrying out isolated voluntary movements against gravity or more complex and integrated activities, such as walking. The weakness of individual muscles or groups of muscles can be quantified by using a myometer, which measures force based on a hydraulic or electronic principle. Recordings of contraction force over a period of time are valuable in determining whether the weakness is improving or worsening. The assessment of muscle weakness and wasting is directed toward discovering evidence of muscle inflammation or damage. These changes are discerned by blood tests or by measuring alterations of the electrical properties of contracting muscles. Another investigative tool is the muscle biopsy , which provides muscle specimens for pathological diagnosis and biochemical analysis. Muscle biopsies can be taken with a needle or during a surgical procedure. Classification of muscle weakness Muscle contraction results from a chain of events that begins with a nerve impulse traveling in the upper motor neuron from the cerebral cortex in the brain to the spinal cord. The nerve impulse then travels in the lower motor neuron from the spinal cord to the neuromuscular junction, where the neurotransmitter acetylcholine is released. Acetylcholine diffuses across the neuromuscular junction, stimulating acetylcholine receptors to depolarize the muscle membrane. The result is the contraction of the muscle fibre. Contraction depends on the integrity of each of these parts; disease or disorder in any part causes muscle weakness. Upper motor neuron disease Muscle weakness typical of upper motor neuron disease is seen in stroke , producing weakness of one side of the body. The arm is typically flexed, the leg is extended, and the limbs have increased tone. Some movement may be preserved, although the use of the hand is particularly limited. In comparison with muscle weakness due to disease of the lower motor neuron or muscle, in the upper motor neuron weakness the muscle bulk is usually well preserved. Other causes of upper motor neuron disorders include multiple sclerosis , tumours , and spinal cord injury. Lower motor neuron disease Degeneration of the lower neuron produces a flaccid muscle weakness. Muscle wasting is a prominent feature because the shrinkage and eventual death of neurons lead to denervation of the muscle. Diseases of the motor neurons lying in the spinal cord are called motor neuron diseases. The most common is motor neuron disease itself, also called amyotrophic lateral sclerosis and Lou Gehrig disease. Affected individuals are generally between 50 and 70 years of age and have upper and lower motor neuron weakness. Paralysis progresses rapidly, and death often results within three years. The spinal muscular atrophies are a group of disorders affecting infants, children, and young adults, often with an autosomal recessive mode of inheritance i. The infantile type of amyotrophic lateral sclerosis is fatal within one year, but the older cases tend to be less severe. No cause is yet known for any of these diseases, and no cure is available. Diseases of the peripheral nerves peripheral neuropathies, or polyneuropathies can produce symptoms similar to the motor neuron diseases. Sensory disturbance due to involvement of the nerve fibres carrying sensory impulses is usually also involved. Symptoms usually begin in the hands and feet and progress toward the body. Peripheral neuropathies can cause degeneration of the axons, the core of the nerve fibres. The axons can regenerate but only at a rate of one to two millimetres per day. Thus, after injury to a nerve at the elbow, the hand will not recover for six to nine months. Toxins and damage to blood vessels tend to cause axonal types of neuropathy. Peripheral neuropathy also can be caused by degeneration of the myelin sheaths , the insulation around the axons. These are termed demyelinating neuropathies. Symptoms are similar to neuropathies with axonal degeneration, but since the axons remain intact, the muscles rarely atrophy. Recovery from demyelinating neuropathies can be rapid. Other causes of peripheral neuropathy include diabetes mellitus , nerve trauma, inherited factors, and chronic renal failure. Neuromuscular junction disorders Diseases of the neuromuscular junction typically involve the generation of an end-plate potential that is too low to propagate an action potential in the muscle fibre. These diseases are associated with weakness and fatigability with exercise. Diseases of neuromuscular transmission may be acquired or inherited and may be the result of autoimmune disorders, such as myasthenia gravis ; congenital disorders; toxins such as those present in botulism ; and some drug-induced disorders. Primary diseases and disorders It appears that the maintenance of muscle mass and function depends on its use. For example, weight lifters and sprinters have muscle fibres with a large capacity for glycolysis and thus ATP production and sudden force generation. Striated muscles can regenerate after damage and can adapt to the loads they carry. Thus, in a muscle biopsy from an individual

with any of the muscular dystrophies, there is likely to be a mixture of the cellular changes associated with damage and those associated with regeneration and growth hypertrophy. Muscular activities in which the muscle resists an extending force eccentric contractions cause more damage to the muscle cells than contraction of the muscle at constant length isometric contraction or where shortening occurs concentric contractions. The greater damage with eccentric contraction occurs despite the fact that the metabolic rate may be one-sixth of that of an equivalent concentric or isometric contraction. Muscles that are immobilized, as by a plaster cast following fracture of a long bone , tend to waste rapidly through shrinkage of the muscle fibres. A consistent finding is that the oxidative capacity of the muscle is reduced. These changes are reversible with muscle-strengthening exercises. The muscular dystrophies The muscular dystrophies are a group of hereditary disorders characterized by progressive muscular atrophy and weakness. In most varieties the muscles of the limb girdlesâ€”the pelvic and shoulder musclesâ€”are involved. Measurement of the activity of creatine kinase in the blood, analysis of a muscle biopsy, and recordings from an electromyograph frequently establish that the muscle weakness is due to primary degeneration of the muscles. Creatine kinase is an enzyme of muscle fibres that is released into the bloodstream when the fibres degenerate, as in the muscular dystrophies. Muscle biopsies reveal the characteristic degeneration and attempted regeneration of muscle fibres. Electromyography shows differences in the electrical patterns of normal muscle, myopathy , and chronic denervation, such as in the spinal muscular atrophies. In contrast to the several varieties of muscular dystrophy that are relatively benign , the Duchenne type, which predominately affects boys , is severe. It causes difficulty in walking at about the age of four years, loss of the ability to walk at about the age of 11, and death before the age of 20, usually because of respiratory failure or pulmonary infections. There is a paradoxical increase in the size of the calf muscles, giving rise to the term pseudohypertrophic muscular dystrophy because the increase in size is the result of replacement with fat and fibrous tissue rather than growth of fibres, as in true hypertrophy. Duchenne muscular dystrophy is an X-linked condition; a defect of a gene on the X chromosome is responsible for the disease. Females do not manifest the disease but have a 50 percent probability of transmitting the gene to their sons and their daughters who themselves become carriers. Muscle degeneration is due to the lack of a protein called dystrophin, which causes a disruption of the membrane covering the muscle fibre; the results are the entry of excess amounts of calcium ions into the cell and cell degeneration. Treatment with glucocorticoid medications, specifically prednisone, may delay progression of the disease. Becker muscular dystrophy is similar to the Duchenne type except that it appears later in life and progresses more slowly. It is due to different damage to the same gene on the X chromosome that causes Duchenne muscular dystrophy; some functional dystrophin is produced. Facioscapulohumeral muscular dystrophy starts in the face, the muscles around the shoulder blades, and the upper arms.

Chapter 4 : Muscle Weakness Due to Spinal Cord Cell Disease - Child Neurology Foundation

Muscular dystrophy is a group of inherited diseases characterized by weakness and wasting away of muscle tissue, with or without the breakdown of nerve tissue. There are 9 types of muscular dystrophy, with each type involving an eventual loss of strength, increasing disability, and possible deformity.

Myositis, or general muscle inflammation, may be caused by: The inflammatory myopathies are rare and can affect both adults and children. Dermatomyositis is the most common chronic form in children. Polymyositis and dermatomyositis are more common in females while inclusion body myositis affects more men. Inclusion body myositis usually affects individuals over age General symptoms of chronic inflammatory myopathy include slow but progressive muscle weakness. Inflammation damages the muscle fibers, which causes weakness, and may affect the arteries and blood vessels that pass through muscle. Other symptoms include fatigue after walking or standing, frequent episodes of tripping or falling, and difficulty swallowing or breathing. Some individuals may have muscle pain or muscles that are tender to touch. Polymyositis affects skeletal muscles the type involved in body movement on both sides of the body. It is rarely seen in persons younger than age Generally, the onset occurs between age 30 and Signs and symptoms of polymyositis vary considerably from person to person, which can make it difficult to diagnose. Untreated progressive muscle weakness may lead to difficulty swallowing, speaking, rising from a sitting position, climbing stairs, lifting objects, or reaching overhead. Some people with polymyositis may also develop arthritis, shortness of breath, heart arrhythmias irregular heartbeats , or congestive heart failure when the heart is no longer able to pump out enough oxygen-rich blood. Dermatomyositis is characterized by a skin rash that precedes or accompanies progressive muscle weakness. The rash appears patchy, with purple or red discolorations, and characteristically develops on the eyelids and on muscles used to extend or straighten joints, including knuckles, elbows, knees, and toes. Red rashes may also occur on the face, neck, shoulders, upper chest, back, and other locations. There may be swelling in the affected areas. The rash sometimes occurs without obvious muscle involvement and often becomes more evident with sun exposure. Adults with dermatomyositis may experience weight loss or a low-grade fever, have inflamed lungs, and be sensitive to light. Adult dermatomyositis, unlike polymyositis, may accompany tumors of the breast, lung, female genitalia, or bowel. Children and adults with dermatomyositis may develop calcium deposits, which appear as hard bumps under the skin or in the muscle called calcinosis. Calcinosis most often occurs one to three years after disease onset but may occur many years later. These deposits are seen more often in childhood dermatomyositis than in dermatomyositis that begins in adulthood. In some cases of polymyositis and dermatomyositis, distal muscles, which are the muscles away from the center of the body, such as those in the forearms and around the ankles and wrists , may be affected as the disease progresses. Polymyositis and dermatomyositis may be associated with collagen-vascular or autoimmune diseases such as lupus. Inclusion body myositis IBM is the most common form of inflammatory myopathy in people age 50 years and older and is characterized by slow, progressive muscle weakness and wasting over the course of months or years. IBM affects both proximal and distal muscles, typically in the thighs and forearms, and is often occurs on both sides of the body, although muscle weakness may affect only one side of the body. Falling and tripping are usually the first noticeable symptoms. The disorder often begins with weakness in the wrists and fingers that causes difficulty with pinching, buttoning, and gripping objects. People may experience weakness in their wrist and finger muscles and atrophy thinning or loss of muscle bulk in their forearm muscles and quadriceps muscles in the thighs. Difficulty swallowing occurs in approximately half of IBM cases due to involvement of the throat muscles. Symptoms of the disease usually begin after the age of 50, although the disease can occur earlier. Unlike polymyositis and dermatomyositis, IBM occurs more frequently in men than in women. Necrotizing autoimmune myopathy NAM is a rare and relatively newly recognized subgroup of inflammatory myopathies. NAM can occur at any age but usually affects adults. Its symptoms are similar to polymyositis and dermatomyositis, with weakness in both the upper and lower body, difficulty rising from low chairs, climbing stairs, or lifting objects. However, the onset of these symptoms can be more severe and sudden, reaching their

peak over a period of days or weeks. Other symptoms include fatigue, weight loss, and muscle pain. NAM occurs alone or after viral infections, in association with cancer, in people with connective-tissue disorders such as scleroderma, or, rarely, in people taking cholesterol lowering medications statins. Muscle weakness and pain may continue to worsen even after individuals stop taking the drugs. Childhood inflammatory myopathies have some similarities to adult dermatomyositis and polymyositis. They typically affect children ages 2 to 15 years. Symptoms include proximal muscle weakness and inflammation, edema an abnormal collection of fluids within body tissues that causes swelling , muscle pain, fatigue, skin rashes, abdominal pain, fever and contractures. Contractures result from shortening of muscles or tendons around joints, are caused by inflammation in the muscle tendons, and prevent the joints from moving freely. Children with inflammatory myopathies may have difficulty swallowing and breathing. The heart may also be affected. Between 20 to 40 percent of children with juvenile dermatomyositis develop calcinosis, which can cause significant muscle weakness and pain, joint contracture, skin ulcers, and decreased muscle bulk. Diagnosis is based on medical history, results of a physical examination that includes tests of muscle strength, and blood samples that show elevated levels of various muscle enzymes and autoantibodies. A biopsy sample of muscle tissue should be examined for signs of chronic inflammation, muscle fiber death, vascular deformities, or other changes specific to the diagnosis of a particular type of inflammatory myopathy. A skin biopsy can show changes in the skin associated with dermatomyositis. Chronic inflammatory myopathies cannot be cured in most adults but many of the symptoms can be treated.

Chapter 5 : Neuromuscular Disorders | MedlinePlus

Muscle weakness happens when your full effort doesn't produce a normal muscle contraction or movement. It's sometimes called reduced muscle strength, muscular weakness, or weak muscles.

Myasthenia gravis affects both men and women and occurs across all racial and ethnic groups. It most commonly impacts young adult women under 40 and older men over 60 , but it can occur at any age, including childhood. Myasthenia gravis is not inherited nor is it contagious. Occasionally, the disease may occur in more than one member of the same family. Although myasthenia gravis is rarely seen in infants, the fetus may acquire antibodies from a mother affected with myasthenia gravisâ€”a condition called neonatal myasthenia. Rarely, children of a healthy mother may develop congenital myasthenia. This is not an autoimmune disorder it is caused by defective genes that produce abnormal proteins in the neuromuscular junction and can cause similar symptoms to myasthenia gravis. How is myasthenia gravis diagnosed? A doctor may perform or order several tests to confirm the diagnosis, including: A physical and neurological examination. In a neurological examination, the physician will check muscle strength and tone, coordination, sense of touch, and look for impairment of eye movements. This test uses injections of edrophonium chloride to briefly relieve weakness in people with myasthenia gravis. The drug blocks the breakdown of acetylcholine and temporarily increases the levels of acetylcholine at the neuromuscular junction. It is usually used to test ocular muscle weakness. Most individuals with myasthenia gravis have abnormally elevated levels of acetylcholine receptor antibodies. A second antibodyâ€”called the anti-MuSK antibodyâ€”has been found in about half of individuals with myasthenia gravis who do not have acetylcholine receptor antibodies. A blood test can also detect this antibody. However, in some individuals with myasthenia gravis, neither of these antibodies is present. These individuals are said to have seronegative negative antibody myasthenia. Muscle fibers in myasthenia gravis, as well as other neuromuscular disorders, do not respond as well to repeated electrical stimulation compared to muscles from normal individuals. Single fiber electromyography EMG , considered the most sensitive test for myasthenia gravis, detects impaired nerve-to-muscle transmission. EMG can be very helpful in diagnosing mild cases of myasthenia gravis when other tests fail to demonstrate abnormalities. Diagnostic imaging of the chest using computed tomography CT or magnetic resonance imaging MRI may identify the presence of a thymoma. Measuring breathing strength can help predict if respiration may fail and lead to a myasthenic crisis. Because weakness is a common symptom of many other disorders, the diagnosis of myasthenia gravis is often missed or delayed sometimes up to two years in people who experience mild weakness or in those individuals whose weakness is restricted to only a few muscles. A myasthenic crisis is a medical emergency that occurs when the muscles that control breathing weaken to the point where individuals require a ventilator to help them breathe. Approximately 15 to 20 percent of people with myasthenia gravis experience at least one myasthenic crisis. This condition usually requires immediate medical attention and may be triggered by infection, stress, surgery, or an adverse reaction to medication. However, up to one-half of people may have no obvious cause for their myasthenic crisis. Certain medications have been shown to cause myasthenia gravis. However, sometimes these medications may still be used if it is more important to treat an underlying condition. Today, myasthenia gravis can generally be controlled. There are several therapies available to help reduce and improve muscle weakness. This operation to remove the thymus gland which often is abnormal in individuals with myasthenia gravis can reduce symptoms and may cure some people, possibly by rebalancing the immune system. A recent NINDS-funded study found that thymectomy is beneficial both for people with thymoma and those with no evidence of the tumors. The clinical trial followed people with myasthenia gravis and no visible thymoma and found that the surgery reduced muscle weakness and the need for immunosuppressive drugs. Medications to treat the disorder include anticholinesterase agents such as mestinon or pyridostigmine, which slow the breakdown of acetylcholine at the neuromuscular junction and thereby improve neuromuscular transmission and increase muscle strength. These drugs improve muscle strength by suppressing the production of abnormal antibodies. They include prednisone, azathioprine, mycophenolate mofetil, tacrolimus, and rituximab. The drugs can cause significant side effects and must be

carefully monitored by a physician. Plasmapheresis and intravenous immunoglobulin. These therapies may be options in severe cases of myasthenia gravis. Individuals can have antibodies in their plasma a liquid component in blood that attack the neuromuscular junction. These treatments remove the destructive antibodies, although their effectiveness usually only lasts for a few weeks to months. Plasmapheresis is a procedure using a machine to remove harmful antibodies in plasma and replace them with good plasma or a plasma substitute. Intravenous immunoglobulin is a highly concentrated injection of antibodies pooled from many healthy donors that temporarily changes the way the immune system operates. It works by binding to the antibodies that cause myasthenia gravis and removing them from circulation. With treatment, most individuals with myasthenia can significantly improve their muscle weakness and lead normal or nearly normal lives. Sometimes the severe weakness of myasthenia gravis may cause respiratory failure, which requires immediate emergency medical care. Some cases of myasthenia gravis may go into remissionâ€”either temporarily or permanentlyâ€”and muscle weakness may disappear completely so that medications can be discontinued. Stable, long-lasting complete remissions are the goal of thymectomy and may occur in about 50 percent of individuals who undergo this procedure. The mission of the National Institute of Neurological Disorders and Stroke NINDS is to seek fundamental knowledge about the brain and nervous system and to use that knowledge to reduce the burden of neurological disease. Although there is no cure for myasthenia gravis, management of the disorder has improved over the past 30 years. There is a greater understanding about the structure and function of the neuromuscular junction, the fundamental aspects of the thymus gland and of autoimmunity, and the disorder itself. Technological advances have led to more timely and accurate diagnosis of myasthenia gravis and new and enhanced therapies have improved treatment options. Researchers are working to develop better medications, identify new ways to diagnose and treat individuals, and improve treatment options. Medication Some people with myasthenia gravis do not respond favorably to available treatment options, which usually include long-term suppression of the immune system. New drugs are being tested, either alone or in combination with existing drug therapies, to see if they are effective in treating the disease. Studies are investigating the use of therapy targeting the B cells that make antibodies rituximab or the process by which acetylcholine antibodies injure the neuromuscular junction eculizumab. The drugs have shown promise in initial clinical trials. Diagnostics and biomarkers In addition to developing new medications, researchers are trying to find better ways to diagnose and treat this disorder. For example, NINDS-funded researchers are exploring the assembly and function of connections between nerves and muscle fibers to understand the fundamental processes in neuromuscular development. This research could reveal new therapies for neuromuscular diseases like myasthenia gravis. New treatment options Findings from a recent NINDS-supported study yielded conclusive evidence about the benefits of surgery for individuals without thymoma, a subject that had been debated for decades. Researchers hope that this trial will become a model for rigorously testing other treatment options, and that other studies will continue to examine different therapies to see if they are superior to standard care options.

Chapter 6 : Muscle weakness - Wikipedia

Muscle Pain/Weakness in Autoimmune Disease People with autoimmune conditions such as scleroderma, vasculitis and myositis, can experience muscle pain many times because of the symptoms linked with the condition.

Learning disabilities Becker muscular dystrophy Signs and symptoms are similar to those of Duchenne muscular dystrophy, but tend to be milder and progress more slowly. Symptoms generally begin in the teens but may not occur until the mids or even later. Other types of muscular dystrophy Some types of muscular dystrophy are defined by a specific feature or by where in the body symptoms first begin. Myotonic muscular dystrophy is the most common form of adult-onset muscular dystrophy. Facial and neck muscles are usually the first to be affected. Muscle weakness typically begins in the face and shoulders. The shoulder blades might stick out like wings when a person with FSHD raises his or her arms. Onset usually occurs in the teenage years but may begin in childhood or as late as age This type affects boys and girls and is apparent at birth or before age 2. Some forms progress slowly and cause only mild disability, while others progress rapidly and cause severe impairment. Hip and shoulder muscles are usually the first affected. People with this type of muscular dystrophy may have difficulty lifting the front part of the foot and as a result may trip frequently. Onset usually begins in childhood or the teenage years. When to see a doctor Seek medical advice if you notice signs of muscle weakness â€” such as increased clumsiness and falling â€” in yourself or your child. Request an Appointment at Mayo Clinic Causes Certain genes are involved in making proteins that protect muscle fibers from damage. Muscular dystrophy occurs when one of these genes is defective. Each form of muscular dystrophy is caused by a genetic mutation particular to that type of the disease. Many of these mutations are inherited. Risk factors Muscular dystrophy occurs in both sexes and in all ages and races. However, the most common variety, Duchenne, usually occurs in young boys. People with a family history of muscular dystrophy are at higher risk of developing the disease or passing it on to their children. Complications The complications of progressive muscle weakness include: Some people with muscular dystrophy eventually need to use a wheelchair. Shortening of muscles or tendons around joints contractures. Contractures can further limit mobility. Progressive weakness can affect the muscles associated with breathing. People with muscular dystrophy may eventually need to use a breathing assistance device ventilator , initially at night but possibly also during the day. Weakened muscles may be unable to hold the spine straight. Muscular dystrophy can reduce the efficiency of the heart muscle. If the muscles involved with swallowing are affected, nutritional problems and aspiration pneumonia may develop. Feeding tubes may be an option.

Chapter 7 : Muscle Weakness and Fatigue | Causes and Treatment | Patient

Learn about Muscle Weakness on theinnatdunvilla.com, including information on symptoms, causes and treatments.

Loss of the sensation IV. In general, treatment for muscle weakness involves a multifaceted protocol in order to address cause, aid to build up strength as well as reduce the risk of the increased complications, like muscle atrophy, and assists the patient to live an active life. Common treatment includes appropriate rest, nutrition, regular medical care, good hydration, rehabilitation, occupational therapy, physical therapy and regular exercises. Generally, the treatment for muscle weakness caused by most bacterial infections including antibiotic medications. To treat muscle weakness caused by anemia,they generally include the treatment of underlying conditions and blood transfusions. To treat muscle weakness caused by dehydration and electrolyte imbalance, they generally include treatment for underlying conditions of electrolyte correction and rehydration with intravenous fluids or oral and electrolytes. Below are more details of treatments of muscle weakness as well as muscle weakness diseases and disorders. By supplying your body with these supplements from different sources, you can increase your strength and get rid of muscle weakness fast. Especially, you can add more foods rich in these nutrients, vitamins and minerals to your diet for improving your muscle weakness diseases and disorders. Typically, you can make use of collagens in order to improve muscle weakness in individuals with myasthenia gravis. On the other hand, you can take advantage of those in order to cure your muscle weakness. So, to improve your condition, you should stop this root cause. Try to eliminate all stressful and anxious factors. Keep your mind relaxed and free. As a result, you will have good moods, balanced mind and fight off pain and improve your overall health. Walk Walking is a physical activity that can help you improve your blood flow and keep your muscle active. By taking appropriate walk everyday, you will be able to get rid of muscle weakness while preventing diseases related to your heart, mental health, and memory and so on. Control Your Breath Exactly I mean the breathing techniques. By controlling your breath, you will be able to reduce effects of hyperventilation. Make sure that you will have concentrated and slow breath that are not too deeply or quickly. Hold each of your breath about a few seconds for regaining carbon dioxide you have spent through the hyperventilation. Then breathe out slowly. You can practice distracting yourself through phone calls, mental exercises, bath, music or anything you want to get rid of discomfort of your muscle weakness. Actually, this is a way to cheat your feelings due to adrenaline coursing in your vain, and some other ones are because of genuine tiredness that muscles feel after you got tension for a long time. Rest and water are effective ways to get rid of the feelings of muscle weakness. For instance, you can take a cold or warm bath if you feel tired and exhausted. Water will help you refresh your mind and feel more comfortable. As a result, you can get rid of pain and stress. You will feel more energy running in your muscles. On the other hand, you can try music therapy in order to distract yourself against muscle weakness. Just listen to music and make your brainwave work. In other words, listening to music is a way to cheat your mind for relaxation. Drink Carrot Juice As I mentioned above, water can help refresh your state. Drink cold water to boost your mind and awareness. In order to get rid of your muscle weakness diseases and disorder, do the same but add carrot juice. This juice is proven to improve the blood circulation through your brain and body. Physically, your stiffness and weakness can be caused by the lack of proper blood circulation. By drinking about just 12 oz. Eat Healthy Proteins Next to diet tips to improve muscle weakness diseases and disorders, take healthy proteins to strengthen your health and get rid of fatigue fast. There are different sources of healthy protein you can add to your daily diet such as nuts, yogurt and eggs. Some particular minerals and vitamins can strengthen your nervous system for muscle functions including amino acids, vitamin B complex, magnesium and calcium. Muscle weakness irritability and fatigue are among symptoms of the lysine deficiency. Besides, Coenzyme Q10 can aid in absorbing essential particular calcium, minerals and vitamins. So, in order to improve your muscle weakness diseases and condition, supply your body with coenzyme Q10 and Lysine. You can buy these supplements on drugs stores. Drink Horsetail Tea Besides the diet plan and some supplements for muscle weakness diseases and disorders, you can try horsetail tea. This herbal tea is considered a natural remedy to increase your muscle strength, as it is high in silica. This agent helps improve

your mood and helps your muscles contract. Simply, make a cup of hot horsetail tea and drink it daily for at least 2 months. Then, you will notice your improved muscle strength. Stay Motivated Exercising is a way to stay motivated. You can walk and run to increase the blood circulation for getting rid of muscle weakness diseases and disorders. Besides, you can focus on your decreased muscle weakness in the body such as legs or hands or backs. Next, you will practice specific exercises and workout to strengthen the affected areas. For instance, beside walking or running, you can do exercises such as Yoga, Pilates, Tai Chi or swimming.

Chapter 8 : Evaluation of the Patient with Muscle Weakness - - American Family Physician

Muscle disease, or myopathy, may occur as a result of having an underactive thyroid (hypothyroidism) or an overactive thyroid (hyperthyroidism). As a rule, muscle problems related to these concerns generally are mild and eased with prompt treatment of the thyroid disorder.

Chapter 9 : Muscle Disorders | MedlinePlus

Muscle weakness is commonly due to lack of exercise, ageing, muscle injury or pregnancy. It can also occur with long-term conditions such as diabetes or heart disease. There are many other possible causes, which include stroke, multiple sclerosis, depression, fibromyalgia and chronic fatigue syndrome (ME).