How is this change in whole-muscle f

Work off campus? Find out about our remote access options, Yang Liu, Chuan Chang, Nicolas Diaz, Yong-Tsing Chen, Sheng Li, Ping zhou, Yinchun Chang, Trans-Kut Insinuation of the Image Area from High Density Surface Electromyography Records, Journal of Neural Engineering, 10.1088/1741-2552/ab673e, 17, 1, 1, (016070), (2020).E.G. Klaver-Krol, J.J. Rasker, M.M. Klaver, P.M. ten Klooster, M.J. Swarts, Fibromyalgia: increased muscle membrane reactivity and the role of central regulation, Clinical Neurophysiology, 10.1016/j.clinph.2018.09.030, (2018). $$-mathbf-Na 'varvec'v'$$Na v Channel excitability under mechanical stress, Biological Cybernetics, 10.1007/s00422-017-0712-3, 111, 2, (129-148), (2017). Yang Liu, Yong Ning, Sheng Li, Ping zhou, William S. Reimer, Ingchun Chang, Three-dimensional Inertia area Images from multi-channel Surface EMG Recordings, International Journal of Neural Systems, 10.1142/S0129065715500240, 25, 06, (1550024), (2015). Jack A. Roll, Jack A. Rall, Excitement-Compression Compound and The Role of Calcium in Reduction and Relaxation in the 1950s and 1960s, Muscle Reduction Mechanism, 10.1007/978-1-4939-2007-5, (105-154), (2014). Javier Rodriguez, Armando Malanda, Luis Gila, Ignacio Rodriguez, Javier Navallas, Procedure identification in the model of action potential of single fiber - Part II: Global Approach and Experimental Results, Journal of Electromyography and Kinesiology, 10.1016/j.jelekin.2009.03.003, 20, 2, (274-283), (2010). Yingchun Chang, Deng Wang, Gerald W Timm, 3D method of visualization of muscle activity to assess pelvic muscle function, Reverse problems, 10.1088/0266-5611/26/11/11/115018, 26, 11, (115018), (2010).J.R. Falce, Falce A.M. Trigueros, L.G. Weros, I.R. Carreno, D.N. Irugio, Mathematical Analysis of SFAP Convolution models, IEEE Biomedical Engineering Transactions, 10.1109/TBME.2005.845045, 52, 5, (769-783), (2005). Daniel Dumitru, Dick F. Stegeman, Machiel J. Swarts, Electrical Sources and Conductivity, Elextrodiagnostic Medicine, 10.1016/B978-1-56053-433-4.50008-0, (27-53), (2002). Elizabeth Kelly, William J Fry, Francis J Fry, Muscles Twitch Tension - Effect of Electrical Stimulating Conditions at Different Temperatures I. Results for Chloride Ringer, Physics in Medicine and Biology, 10.1088/0031-9155/9/3/309, 9, 3, (371-388), (2002). zoya C Lateva, Kevin C McGill, Assessment of motor units architectural properties by analyzing motor units of potential morphology, clinical neurophysiology, 10.1016/S1388-2457 (00)00495-8, 112, 1, (127-135), (2001). Daniel Dumitru, John C. King, Mahiel J. Determinants of the potential duration of the motor unit, Clinical Neurophysiology, 10.1016/S1388-2457 (99)00142-X, 110, 11, (1876-1882), (1999).R. Merletti, L. Lo Conte, E. Avignon,. Guglielminotti, Modeling myoelectric signals. I. Model Implementation, IEEE Biomedical Engineering Transactions, 10.1109/10.771190, 46, 7, (810-820), (1999). zoe C Lateva, Kevin C McGill, physiological origin of slow post-volina in muscle action potential, Electroencephalography and Clinical Neurophysiology /Electromyography and Motor Control, 10.1016/S0924-980X (98)00048-4, 109, 5, (462-469), (1998). Kai-Oge Henneberg, Fernan A. Roberge, Simulation of the spread of isolated skeletal muscle fiber in isotropic volume conductor, Annals of Biomedical Engineering, 10.1007/BF02738535, 25, 1, (15-28), (1997). Kai-Oge Henneberg, Fernan A. Roberge, Simulation spread into a bundle of skeletal muscle fibers: Modulation effects of passive fibers, Annals of Biomedical Engineering, 10.1007/BF02738536, 25, 1, (29-45), (1997). Fernand A. Roberge, Siring Wang, Herve Hoges, L. Joshua Leon, Spread on central fiber, surrounded by inactive fibers in a multifiber beam model, Annals of Biomedical Engineering, 10.1007/BF02684178, 24, 6, (647-661), (1996). K.A. Henneberg, Indefinite, Proceedings of the 16th Annual International Conference of IEEE Engineering in Medicine and Biology Society, 10.1109/IEMBS.1994.412000, (369), (1994).G. V. Dimitrov, S.K. Lateva, N.A. Dimitrova, Model of Slow Components of Skeletal Muscle Potentials, Medical and Biological Engineering, Calculation, 10.1007/BF02524697, 32, 4, (432-436), (1994).R. Merletti, L.R. Lo Conte, C. Sisari, U. Massazza, The effect of the ankle position on electrically caused surface myoelectric signals of the shin front Muscle, Archive of Physical Medicine and Rehabilitation, 10.1016/0003-9993 (93)90114-P, 74, 5, (501-506), (1993). Lars Arendt-Nielsen, Nikolai Gantchev, Thomas Sinkier, Muscle Length Influence on Muscle Fiber Conduction and Muscle Fatigue, Electroencephalography and Clinical Neurophysiology/Potential Calling Section, 10.1016/0168-5597 (92)90128-X, 85, 3, (166-172), (1992).W. DeMattei, V. Contegiacomo, Effect of temperature on conductivity rate in human muscle fibers, Journal of Electromyography and Kinesiology, 10.1016/1050-6411 (91)90015-W, 1, 4, (281-287), (1991).N.A. Dimitrova, Dimitrova, 1, 4, (281-287). G.V. Dimitrova, Effect of electrical stimulus parameters on the development and distribution of action potentials in short excitable fibers, Electroencephalography and Clinical Neurophysiology, 10.1016/0013-4694(88)90023-5, 70, 5, (453-459), (1988).I. Stefanova, Mathematical analysis of changes in the parameters of action potentials, membrane and ion currents of frog muscle fiber during the recovery cycle, Biological cybernetics, 10.1007/BF00364152, 57, 3, (207-211), (1987).A. Gidikov, L. Gerilovsky, N. Radicheva , N. Trayanova, The Effect of The Geometry of the End of Muscle Fiber on Extracellular Potentials, Biological Cybernetics, (1-8), (1986). Jacques Caill , Mich le Ildefonse, Oger Rougier, Sodium current existence in the tubular fiber membrane of the frog twitch muscle; Its possible role in activation reduction, Pfl gers Archiv European Journal of Physiology, 10.1007/BF00581298, 374, 2, (167-177), (1978).H. C. Hopf, R.-L. Herbort, M. Gnas, H. G nther, K. Lowitzsch, a fast and slow time reduction associated with the rapid and slow spike conductivity of skeletal muscle fibers in normal subjects and in spastic hemipares, seichschrift f r Neurologie, 10.1007/BF00316533, 206, 3, (193-202), (1974). Edward R. Chaplin, George W. Nell, Sheppard M. Walker, Excitement-reducing latencies in postpartum rat skeletal muscle fibers, Experimental Neurology, 10.1016/0014-4886 (70)90045-2, 29, 1, (142-151), (1970). C.-A. Tulin, K.-A. Karlsson, Regeneration of transectical ventricle roots, presented on monomolecular tube filter (Millipore), Journal of Neurological Sciences, 10.1016/0022-510X (69)90008-2, 8, 3, (485-505), (1969). F Buchthal, P Rosenfalck, Spontaneous Electrical Activity of the Human Muscle, Electroencephalography and Clinical Neurophysiology, 10.1016/0013-4694 (66)90001-0, 20, 4, (321-336), (1966). Hans Jarg Schatzmann, Erregun and Kontraktion glatter Vertebratenmuskeln, Ergebnisse der Physiology, Biologischen Chemie und Experimentellen Pharmakologie, 10.1007/978-3-642-49898-5, (28-130), (1964). Gilbert Glaser, Pharmacological Considerations in the Treatment of Myastenia Gravis, , 10.1016/S1054-3589 (08)60086-1, (113-160), (1963). Thomas W. Farmer, Fritz Buchthal, Poul Rosenfalck, Fire-resistant period of human muscle after passing manifested action potential, Electroencephalography and Clinical Neurophysiology, 10.1016/0013-4694 (60)90021-3, 12, 2, (455-466), (1960). Chapter 14 It is impossible to overestimate the importance of muscles in vertebrates. The whole lifestyle of everyone requires movement, impossible without muscles. In fact, a person has about 40% of the body weight of striped muscles, making it the most common tissue. Striped muscles are named so because of its characteristic cross-striped appearance. Most striped muscles are skeletal muscles involved in rotating bones around the joints and are therefore responsible for most of the movements we know about. Other striped muscles move the eyes and serve as valves to check the flow of blood or other fluids, such as bulbospongiosus helps erection of the penis or clitoris by compressing the deep dorsal vein. The heart muscle is also striped in appearance, but it is significantly different from other striped muscles both in its structure and its behavior. However, other muscles called smooth muscles do not have characteristic cross bands, but contain the same contracting proteins. Smooth muscles are essential as

lining the gastrointestinal tract that churn and movement of food through the tract as blood vessels that control their diameters and thus flow through them are like valves that control the passage of gases and fluids in the body, and as controllers in many other places in the body. Of the three types of muscles, skeletal and heart muscles have been studied most thoroughly. It is assumed that the reduction mechanism is the same for both types, and only the details of initiation and control of reduction vary. Not all striped muscles, however, behave in the same way. For example, skeletal vertebrate muscles all seem to initiate contractions with sodium spikes, while the striped muscles of some invertebrates initiate contractions with calcium spikes. We limit our discussion primarily to vertebrate skeletal muscles, pointing to the distinctive features of the structure and function of the heart and smooth muscles. Skeletal muscles are made up of masses of fibers, each of which is a separate cell. There are several types of muscles, each with different fiber mechanisms, but they can be divided into two main classes: those with fibers arranged parallel to those with pennath location. Figure 14-1 shows these two classes. In a parallel arrangement (A), each muscle fiber, or small group of fibers, attached to his own tendon, the tendons converge on a common point (1). Muscle fibers side by side, i.e. side by side, but the name of the class comes from the fact that muscle fibers are reduced in the direction (double arrow, f) parallel to the direction of muscle contraction (single-headed arrow, F). Pennat muscle fibers (B) are attached to the common tendon, so that the direction of shortening of individual fibers (two-headed arrow, f) differs from the direction of contraction of the entire muscle (single-headed arrow, F). As a result, the pennat muscle cannot cut as much as parallel muscles. Pennatal muscles are located in positions requiring small but powerful movements; parallel muscles are located in positions that require longer movements with less power or faster movements. Muscles, fibrillation and fila complexion To understand how muscle works, it is necessary to understand the fine structure of muscle cells, because it is the inner parts of the cells that do the work. The corresponding internal structures are myofibrills, myophilaments and sarcoplamic styculum. Muscles are made up of muscle fibers; fibers consist (partially) of myofibril; and myofibrils consist of myophilaments. Skeletal muscles have a characteristic striped appearance, because myofibrils are characteristically striped and because myofibrils are more or less in the register (the same stripes are lined up). Myofibrils are striped, because myophilation is not uniformly distributed within them, but occurs in ordinary, repetitive arrays. MyofibrilsFigure 14-2 show, on all the muscle, the bundle of muscle fibers, and their subdivisions, myofibril. Notice the striped look of all three. Each muscle fiber contains about 1000 myofibril with a diameter of 1 m and has a fiber length. Myofibrils have no membrane, just surrounded by cytoplasm. The cross bands of myofibril are consistently repetitive units called sarcomers. The sarcoma may be from 1.5-3.5 m in length, depending on the contracting state of the muscle, and it is limited at each end of the disc, called a disc or line. Each sarcoma contains anisotropic (doubly refractive, therefore dark in phase microscopy) strip, limited to two isotropic (poodino refractive, therefore, light) bands. Anisotropic group is called A band; The isotropic group is called group I. In fact, each sarcoma contains two halves of the I bands (one at each end) because one group I oscillates lines and is therefore part of two adjacent sarmers. In the center of Group A is a lighter area known as H-zone or H-band. During compression, band A does not change length (2), although the sarcoma is reduced, the distance between the q lines decreases and bands I and H narrow. Any theory of muscle contraction should take these observations into account. Myofibrils, as shown in figure 14-2, consist of protein structures called myophilaments. One thread is thick, about 11 nm in diameter and 1.5 m long, while the other is thin, 5 nm in diameter and 1 m in length. These strands are called thick strands and thin threads, respectively. Thick fila being made up of several hundred molecules of myosine, molecular-weight proteins of about 500,000 and some other small proteins whose function is unknown. The myosine molecule has a tail area, which is a birth-like area, and a head area, with two ball submarines projecting at approximately right angles with a thread. The structure was likened to two golf clubs with their shafts twisted together. The figures of the myosine molecule, and its position in the thick threads are shown in figure 14-2. The molecules of myosine thick threads are located in a sheaf with heads oriented to each end and tails to the center. Each subsequent myozin molecule is attached 14 nm further to the end of the filath, and its head rotates 60 around a filaigny from its predecessor. Thus, the thick thread is dotted with projections, except for its center, which contains only myosin tails. Note that the myosin molecules at opposite ends of the thick fila compared to the opposite direction are sort of like a bunch of unsorted golf clubs, some with heads on the right end, some with heads on the left. Thick threads match the sarcomer band. Troponin and tropomyosine are regulatory proteins that allow muscles to contract in the presence of Kaa. the thin filament contains three protein molecules: actin, troponin and troomyozine. One thin filament consists of 300-400 actin molecules and 40 to 60 troponin and troomyosin molecules. Actin is a small, almost spherical molecule that is positioned in a filalop in two spirals of fila shows, as shown in figure 14-2, about 13 actin molecules on a full spiral turn. Troponin and tropomyosine are sometimes referred to as regulatory proteins because of their central role in regulating muscle contraction. Tropomyosin is a filamentous protein that is thought to form two strands that lie in the grooves formed between the strands of actin. Troponin, a ball protein, binds to tropogiosine in only one area and is therefore thought to be sitting on the strands of tropogiosine molecules at regular intervals close to 40 nm. Figure 14-4 shows the link between the three proteins as they are now thought to exist. Thin strands attach to q disk, a flat protein structure. Thin threads can be connected at the end of the H band by thin filamentous processes. The relatively high anisotropy of band A is the result of both thin and thick strands (shown in the longitudinal section at the top and cross-section at the bottom of the fig 14-3). Group I is only slightly anisotropic because it contains only thin strands. The H strip is not as optically dense as the rest of the group because it does not contain any thin strands when the muscle is at rest. As can be seen from the cross section of the figure 14-3, the thin threads are organized into the usual hexagonal arrays in myofibrils, with a thick thread in the center of each array in band A. Three thick threads of the equinox from each thin thread, while six thin strands equinoxes from each thick thread are shown on the left panel. The cross-section through group I shows only a thin array of thread; section through band H shows only a thick array of threads (plus thin, filamentous processes associated with attaching thin threads). Myosine heads are projected from thick strands towards thin strands at intervals of about 43 nm, measured in a line parallel to the adjacent thin thread. Because they stagger around a thin thread at intervals of 60, each projects in the direction of a thin thread, and each thin thread has a projection to it of three thick threads. These predictions have been announced as either cross bridges or cross-projections, depending on whether the head is believed to contact and bind the thin strands or not. As we shall see, there are two schools of thought, in fact, two different mechanisms proposed to account for the generation of mechanical force reduction. T-tubes and sarcoplamic cells reticulumMuscle have a unique membrane structure called a transverse tube just T pipe-killing. The T-tube is the invagination of the muscle membrane, just as the insagnation is done in a balloon by pressing your finger in its direction, not piercing it, but T trumpet-murderers long and winding. The T-tube system forms a ring around each myofibril either on the line of the C, in which case there is one on the sarcomer, or at the A-I-band intersection, in which case there are two on the sarcomer. These perifibrillary rings are interconnected, forming a kind of cellular location, as shown in figure 14-5. The position of the T tubula in relation to the sarcoma is somewhat specific to the species; The skeletal muscle frog has only one tube on the sarcoma, while the human skeletal muscle has two. It should be noted that in the heart muscle of a person there is only one tube on the sarcomer, as shown in figure 14-17. Inside the tubes T is continuous with extracellular space and presumably contains liquid like extracellular fluid, but since the tubular space is small and not well stirred, it is likely that the ionic movements through the tubular membrane produce significant changes in ion concentration, at least on a short-term basis. Another intracellular structure, especially important for contraction, is sarcoplamic cyticulum, a version of the muscle cell of endoplasmic cyticulum. Sarcoplamic cyticulum consists of tubes of pipes that run parallel to sarcomers from T to T-tubes (see drawings 14-5 and 14-17); Thus, there are two sets of sarcoplamic tubes of retinculum on the sarcomer in the muscles with two sets of T tubes on the sarcomer. Sarcoplamic riticulum is a bag with extended ends (tanks) adjacent to T tubes and narrow longitudinal channels connecting these extensions, one at each end. In random sections, one can find the T section tubular limited on both sides of the kind of mute bell shaped sarcoplamic stycules, as shown in Figure 14-6. T-pipes with two neighboring regions of sarcoplamic styculum are often called triads. Since T tubula and sarcoplamic stycules work together for such a long journey, most of the sarcoplamic styculum is in contact with the sarcolemma. There is a space of about 12 nm between membranes which, in electronic micrographs, appear to intersect at regular intervals of structures that have been suggested to communicate T tubes with sarcoplamic cytulum. However, large molecules such as ferritin cannot cross between two structures, and lumen sarcoplasmic reticulum contains liquid like sarcoplasm, not as extracellular liquid. In addition, electrical measurements show that sarcoplamic cyticulum does not communicate with a tubular tube through low resistance pathways. One model has a mechanical fork that closes the Ca channel, ticlum. The hypopolarization of the T tube somehow pulls the plug out of the canal hole, allowing Ca to enter the sarcoplasm. In figure 14-7, the plug is shown in red (a bucket piece) and a Ca canal in a purple (cut cylinder). Presumably, the fork is a dipole, the position of which changes by changing the polarization of the membrane. In any case, Ca efflux from sarcoplamic styculum begins to contract the muscles. Sarcoplamic cyticulum serves as a repository for Casa. In the rested muscle, Kaye is in high concentration in tanks on the triad. Lately active muscle, calcium is in the narrowed, longitudinal part from which it moves towards the triad as time passes. During the contraction, Kae is in high concentration outside the sarcoplamic stycula among myophilation. Observations that during muscle contraction, sarcoma, I and H bands become narrower, while the group does not, combined with the observation that thick and thin filams do not contract (although at very short lengths thin strands can either push through the line or fold like an accordion), suggested sliding strands of model contraction. According to the model, thick and thin threads just glide past each other. Figure 14-8 shows several positions in muscle contraction that illustrate the sliding of threads. At maximum length there are few or no overlapping strands (A), but as the muscles contract there is more and more overlap until the fibers completely overlap (D). There is general agreement that the sliding fila rate model is an accurate description of what happens during muscle contraction. According to the sliding fila1 hypothesis, thick and thin threads simply slip past each other to make a cut. Events leading to contraction Although muscle contraction can be triggered by direct electrical muscle stimulation, this is usually the result of activity in motor neurons inert muscle inertia. The potential of action initiated in alpha-motoneron is spread to motor neuron terminals and releases acetylcholin in the synaptic crevice. Acetylcholine causes the potential end of the plate in the muscles that, in normal muscle, always leads to potential action in the muscles. Muscle surge is very similar to a nervous surge, but longer in duration and with a hypopolary tail in the fall phase, which prolongs the surge by 3-4 msec. An example of muscle surge is shown in figure 14-9. The mechanism of generating a splash in mammalian striped muscles is the same as described for the nerve in Chapter 3. The long (4-5 msec) hypopolary tail muscle-building action is probably an electrotonic reflection of the potential action when it conducts in trumpeting T. At least, the tail disappears from the thorn when the muscle is treated with glycerol and then returns The Solution is Ringer, a treatment that more or less specifically breaks T trumpets, leaving the surface membrane and resting potential intact. Muscle still generates a splash, but not a contract. Carrying a splash in the T tube-murder is probably an active process, as elsewhere on the membrane, and it is the hypopolarization of T tubes that leads to a reduction. It is reasonable to ask why there is such a complex system of tubes in striped muscles. The answer may lie in synchronizing the contraction of sarmers along the length of the muscle and in its depths. Myofibril are located throughout the muscle fiber, but in mammalian muscles, usually only one neuromuscular transition to fiber. If the hypopolarization of the spike could not have been accessed into the center of the fiber, the myofibrils on the surface would have become infected earlier than those in the center. With the T-tube system, the spike conducted quickly to all parts of the cell, reaching all myofibrils for almost the same time. In the absence of such a mechanism, the contract segments of myofibrillations stretch the non-conservative segments, reducing the force transferred to the ends of the fibers and, therefore, into the joints. Hypopolarization of T tubes opens special voltage channels in opposite areas of the sarcoplamic membrane, the riticulum. According to the as-yet unexplored mechanism, this leads to the release of calcium from the tanks of the sarcoplamic sticle to the area of myophilaments. This is an important step in the reduction mechanism; muscles depleted by calcium without stopping. Calcium is scattered to thin strands and binds to troponin. Each head of the myosine molecule (the molecule has two) is ATPase, capable of hydrolyse ATP for ADP and inorganic phosphate, releasing energy; however, according to current thought, tropogiozin suppresses ATPase. The combination of troponin with Kae removes the inhibition of troomyosin, possibly causing the conformation of the thin filament change. At this point, most abbreviation theories agree. There are, however, two plausible theories about how compression force develops. First, the cross-bridge theory suggests that the actual physical binding of the head of myosin to the thin filament occurs, which causes the atF hydrolysis to rotate the head to the tail by pulling the compatible arm of the cross-bridge. This attraction leads to the relative movement of thin and thick threads and voltages and a reduction in sarcoma. In detail, ATP binds to myosine and then, in the presence of Kaa, troponin and tromyosine, the site of myosine binding is exposed to a thin fila and a physical connection is formed between actin and myosin. The activity of Meosine ATPase then turns out to be splitting the ATP phosphate link, releasing energy, and causing head in a twist. Figure 14-10 shows how the head of myosin is thought to rotate (the solid and dotted outlines of the myosin head are designed to indicate two different positions of the head as they rotate) and the relative movement of the strands that results. Since the heads of myosine molecules are oriented in opposite directions at opposite ends of the thick filament, each pulls its adjacent thin thread to the center of the sarcomer and the sarcomer shrinks. When the hypopolarizing stimulus of the T tube burst is over, calcium ceases to be released by the tanks of sarcoplamic riticulum and actively pumped into the longitudinal part of the rithula. The Ca pump, which pumps Ca from the cytosol back into the sarcoplamic lattice, is atPase, which is fospolyated and dephosphorized during the pumping process. It pumps two Ca ions for each ATF hydrolysis. In muscles, Ca ATPase accounts for almost 90% of the membrane protein and is therefore capable of pumping Ca Ions quickly. Typically, the concentration of cytosolic tsai is restored to rest level within 30 milliseconds. When calcium is removed from myofibrillations, THET replaces the ADP on the myosine complex and myosine-actin communication is broken. Because the muscle is elastic, it will be restored to its resting length in the absence of an additional stimulus for calcium release. Reduction is an active process; lengthening is a passive process. One cycle of attachment, twist, and detachment of myosin heads will produce a linear translation of myofilaments of about 10 nm. If all the cross bridges in the myofibril cycle are once synchronous, relative movement equal to about 1% of muscle length will occur, but obviously the muscles are cut by more than 1%. The total reduction of the sarcoma during compression can exceed 1000 nm; therefore the relative movement of the thin and thick fila in the fila part will be twice that of 500 nm. To achieve this magnitude changes the total length, when each cross-bridge cycle produces a 10-nm reduction, a minimum of 50 cycles must occur. The muscles of the man's upper arm flexor can contract at a rate of 8 m/s (Wilkie DR: J Physiol (Lond) 110:249-280, 1949), during which they can be reduced by 10 cm. This reduction rate gives a reduction rate for the sarcomer 160 nm/msec. If the cross bridge is taken at a rate of 10 nm, at this speed there will be at least 16 strokes/msec. Thus, the turning point for a cross-bridge should be about 60 seconds. Calculations for the frog's sartorial muscle, which can shrink to 4 cm/sec, indicate a turning time of about 1 msec, but this reduction occurs at a lower temperature than in mammals. In any case, it is clear that washing the cross bridge should be a quick mechanical process. On the right is an animation that shows the repeated nature of Process. The cross-bridge theory says that sliding is made by physically attachmenting the heads of myosine to the actin and the arrival of the heads. The cross-bridge theory assumes that the strength generated by the muscle is proportional to the number of cross-links formed at the time, and that the probability of a cross bridge forming is proportional to the rate of reduction, i.e. the probability is high when the attachment places slowly pass by each other, small when they move fast. If voltage is only a function of cross-bridge numbers, there should be a linear relationship between length and voltage, so that the voltage increases with the reduction of length due to the greater overlap of thick and thin strands over shorter lengths. Thus, the force needed to stretch muscles at any time is also proportional to the number of cross bridges - a force needed to break the bonds of actin-myosin. Thus, (l) the tension develops by physical connections between thick and thin thread, 2) the tension depends on the degree of overlap between thick and thin thread, 3) the cross bridge arises on a thick thread and ends on a thin thread. Tension develops physical connections between thick and thin tones. The tension depends on the degree of overlap of thick and thin threads. The cross-bridge appears on a thick thread and ends on a thin thread. The arrival of motor neurone potential actionSynaptic transmission at the neuromuscular junction Action potential extends along sarcolemmaHypopolarization T tubulesCa, released into the sarcoplasm from the sarcoplamic reticulumCa, bound troponinCooperative configuration change in troponin and tropomyosinRelease inhibition of myosin-ATPA swivel of myosin headTension attachedShortening by sliding threads' removed from sarcoplasmMg-ATP-related actinomyosinCross-bridges disabledActinomyosin-ATPase inhibitedActive voltage disappearsEric elastic elements to restore resting length Properties of contract muscle When muscle is stimulated either directly or synaptically it develops tension and, if allowed, it shrinks, i.e. it shrinks (3). In this section we will discuss how muscle contracts. Isometric compared to isotonic contraction When the muscle is stimulated after its ends or tendons have been fixed, it contracts but cannot cut. This is called isometric abbreviation (iso is the same, metric - measurement or length). Muscle develops tension, but since it does not shrink, it does not do any external work (remember: work and strength x distance moved). Careful observation shows that during isometric contraction, some muscle sarcomaers contract, stretching other sarcoma chambers and, in addition, stretching the elastic elements of the muscle, increasing the tension measured on the tendon. Figure schematic diagram of the muscle, showing elastic elements both in the series and in parallel with the contracting elements (myofibrils) of the muscle. When a whole muscle rest or one resting muscle fiber is stretched, it opposes a stretch with a force that increases with an increase in stretch (such as gum). This elasticity is caused by parallel elastic elements that lie, for the most part, outside of the contracting elements in elastic tissues, including tendons and sarcolem. (When muscles contract, muscle contraction lags behind sarmer contractions due to a series of elastic elements.) Therefore, with isometric contraction, sarcomas reduce and stretch the elastic component of the series, even if the muscle as a whole does not contract, as shown in figure 14-11B. Even if there is no external work done by the muscle, there is an inner work done. If only one end of the muscle is fixed, the muscles contract and, if it shrinks with constant stress, the contraction is isotonic (iso- the same, tonic and tension). When the contracting elements are reduced, they must first stretch a number of elastic elements and develop a voltage equal to the load, until the next increments of tension leads to the lifting of the load. All the reductions that occur before the load is removed is isometric. Even if the muscle does not carry any external load, it still has to develop a tension equal to its own weight before it can cut. When contract forces exceed the load, the reduction begins; The voltage remains little more than the load throughout the contraction. The contraction stops when the active voltage drops to the point where it equals the load. At this point, the contraction becomes isometric again. Muscles lengthen (stretched) when the total tension in the muscle falls below the load. Figure 14-11C and D show changes in both series and parallel elastic elements and contract elements during isotonic contracting. When the contracting elements are shortened, stretching a number of elastic elements, but the muscles are not shortened. In D, further contraction of the contractile element leads to muscle contraction, as a number of elastic elements are already stretched. Twitch and tetanic contractionsIf a brief stimulus is applied to the muscle or one stimulus is applied to the nerve, one potential action will be caused in the muscles and, after delaying activation of about 5 msec, the muscles will contract. The time of this reduction, called twitch reduction, is shown in figure 14-12B. Muscle tension quickly rises to a maximum of about 50-80 msec after stimulus, then returns to rest tension over the next 100-200 msec or so, depending on the specific muscle. In A, the potential for muscle action is reproduced to compare with the twitch that lasts 100 times longer. The muscle contraction shown in the picture, the adductor pollicis the muscles of the thumb, is a fast muscle. As we will see, mammals also have slow muscles that require 200 msecs or more to reach their maximum twitching stress. The second stimulus, applied before the muscle is completely relaxed, causes another contraction, which adds to the first, the amount of tension is greater than that of a single twitch. This event, as you may have guessed, is called summing up. An example is 14-13. One stimulus causes twitching left in A. Repeated stimulation at a rate of 10/sec causes a summed-up tension in B. When the frequency of stimuli increases to 50/sec, the voltage rises to a more or less stable value, much more than twitch tension. This summation, as in C, is called tetanus or tetanus contraction. Since the individual contribution to tetanus twitching can still be seen as lumps in the record, this tetanus is called unemplish or incomplete. With even higher stimulation speeds, the maximum tension the muscles can withstand is received and there is no sign of individual twitches in the recording. This is called fused or full tetanus, and it is shown in figure 14-13D for 100/sec stimulation speed. Fast and slow muscles, determined by the time of twitching, also have the lowest frequency of stimuli, at which their contractions produce fused tetanus. Fast muscles cannot merge as long as the stimulus rate is equal to or exceeds 60/sec, while slow muscles can merge at a rate of just 16/sec. The potential of a single action leads to a reduction in twitching. A few abbreviations can add up. Length-tension relationship When the resting skeletal muscle is stretched from the length of the rest, i.e. its length is at rest in the body, parallel elastic elements are stretched and the tension increases along the blue curve (A) in figure 14-15. In this figure, the length, expressed as a fraction of the length of the rest, is built on abscissa against tension (or force) on the order. If the muscle is stretched to about 180% of its resting length (we are talking about the maximum area without damage to the muscle), and the length remains constant at this value, while the reduction is taking place (the setting for this experiment is shown in the figure 14-14), it turns out the maximum isometric tension of the muscle. This voltage (full voltage) is the sum of parallel elastic tension (i.e. passive voltage) and contract voltage (i.e. active voltage). At any length of less than 180% of the resting length, the total tension developed by the muscle during isometric contraction will be smaller and will follow the red curve (B) in figure 14-15. To calculate the contract voltage, the voltage developed elements, we simply subtract the blue curve (A) from the red curve (B). The result is a solid curve, sometimes referred to as isometric isometric Curve. Obviously, the maximum isometric contract tension occurs when the muscle is at its rest length. At a shorter or longer length, the isometric voltage produced by the contracting elements is less. The exact shape of the curve for longer than resting length depends on when and how the tension is measured (for discussion see Noble MIM, Pollack GH: Molecular Constriction Mechanisms. At a length of less than 70% rest length, the muscle develops no tension at all when stimulated. It follows that during isotonic contraction, the skeletal muscle can only cut to about 70% of its resting length, and it can only develop a voltage of between 70% and 180% of the rest length. The isotonic voltage length curve is approximately superimposed on the isometric curve; hence, during isotonic contraction, the muscle is reduced to a length suitable for advanced voltage (i.e. to a length predictable from the isometric voltage length curve). The voltage length curve can be explained by the cross bridge theory. Instead of the length of the entire muscle, we could just as accurately chart the sarcomatre length on the abskisse of the drawing 14-15, with a resting length of about 2 microns. At the length of the rest, thin and thick threads are in a relative position, as shown in the figure 14-7C, with almost all thick thread overlapping with a thin thread. In this position, all myosin heads are covered with a thin thread, and therefore they are all available for the formation of cross bridges. Recall that in the theory of cross-bridges the force of compression is proportional to the number of cross-bridgeed bridges. At longer lengths, such as in A and B drawings 14-7, some of the myosin heads do not intersect the actin and are therefore not available to form cross bridges. As a result, the tension will be less. At shorter lengths such as D, actin threads from opposite ends of the sarcoma begin to interfere with each other and at a shorter length the disc can block or otherwise prevent the movement of the thick fila. Skeletal muscles produce maximum strength when they contract from the length of rest. The strength-speed relationEven although the conditions are right for isotonic contraction, i.e. the muscle is fixed only at one end and the weight is attached to the other, the reduction rate will be zero (or negative, i.e. the muscle will lengthen) when the weight is applied to the muscle more than the muscle can lift. On the other hand, when there is no weight on the muscle, it will contract at maximum speed. These are the boundary conditions of the relationship of strength and speed. Between these two extremes, the rate of reduction decreases as the load increases. Earlier measurements of the rate of reduction during isotonic contractions showed that the rate hyperbolic load lifting function (4), as shown in figure 14-16. New measurements show that this is usually not the case (Hill AV: First and last experiments in muscle mechanics. In both skeletal and heart muscle relationships, measured at the sarcoma level, are clearly not hyperbolic. The exact shape of the curve, however, is less important for this discussion than the fact that the speed decreases with increased load. Note that the hard curve does not intersect with order. This is due to the fact that it is experimentally difficult to make the load zero in practice. (In most cases, muscles should lift at least their own weight.) We can extrapolate the curve back in order to see what the zero-load speed will be (dashed line extending the curve in figure 14-16). It's top speed or Vmax. In fact, because the strength exerted by the muscle is also related to its length, in figure 14-16 there will be a family of curves. The curve shown was gained when the muscle began contracting for its rest length. The force generated at a larger or smaller initial length will be less than at the length of the rest. Thus, all length curves, except the length of rest, will be roughly parallel, but lower than shown in the figure of 14-16. They will be roughly parallel, with the exception of almost zero load, where all curves will converge on the same Vmax value (Figure 14-17). The lower the load, the faster the reduction. The cross bridge theory is able to take into account the speed curve, suggesting that the speed constants for the fastening and separation of cross bridges depend on the instant position of the cross bridge relative to the place of attachment on a thin thread. The attachment speed is zero after the cross bridge passes through the anchorage site, but the speed of separation is high. When approaching the place of attachment, the cross-bridge cannot be attached if it is at a certain distance; beyond this distance, the attachment rate is zero, and the distance rate is not. The squad requires a certain minimum time. At low speeds, there is enough time to detpc, but at high speeds may not be. Thus, the cross bridge can remain attached longer than it should, and actually resist the movement of the thin thread in the direction in which it has just moved it. This will lead to a force opposite to what is caused by sarcomar reduction and, therefore, reduce effective contract force. The greater the speed, the greater the effect of the squad. Mechanisms of classification of the force of compression We have already discussed one mechanism of classification of muscle strength, that is, by classifying the frequency of discharge in motor neurons and, therefore, temporal twitching cuts. The endpoints of this continuum are of course the one twitching into a weak element (minimum) and fused thetanic contraction into the strongest (maximum one block twitch). Each muscle fiber is contacted by only one motoneuron, but motoneuron can contact many muscle fibers. When the motor neuron discharges, it activates all the muscle fibers with which it makes synaptic contact. The motor neuron and muscle fibers with which it comes into contact are called the motor block. The motor unit for the muscles of the lower leg can contain up to 1,700 muscle fibers, while the motor block for the outer muscles of the eye can contain only 7 fibers. The fibers that make up the motor unit are not grouped into one bundle in the muscle, but are scattered in small bundles of several fibers. Thus, the forces produced by the contraction of one motor unit are distributed through the muscles. Because all muscle fibers of the motor unit contract simultaneously, it is more difficult to achieve a subtle gradation by virtue of contraction when motor units are large. Suppose one motor unit contains 20 fibers and the other only 5, and let's assume that each fiber can produce 1 gram of force. The minimum forces produced by the motor units will be 20 grams and 5 grams in twitching, and the temporal summation will produce forces graded in 20- and 5-gram increments. When small gradations are needed, as they are in eye movements, motor units are usually small. Muscle contraction strength can also be varied by changing the number of active motor units, i.e. spatial summation. Because the muscle fibers are connected in parallel and exert force on the same tendon, the forces produced in all fibers will add up to the tendon. Thus, the greater the number of active motor units, the greater the strength on the tendon. Not all motor units in muscles of the same size. As the reduction in contract population increases, larger and larger motor units are added. This, of course, means that compression power will be a non-linear function of the number of motor units. With strong stimulation at a rate above 5/sec, the compression force will increase for the most part twitching caused by a weaker, lower stimulation frequency. This is the result of spatial and temporal summation. Maximum muscle tension can develop, obviously, occurs when all motor units produce fused tetal contractions (absolute maximum). This occurs at stimulation frequencies for approximately 50/sec, and within the speed limits of the discharge speed of motor neurons. This forms the limits of muscle voltage control: the minimum voltage produced by a single twitch in the smallest motor unit, and the maximum voltage of the fused reductions in all motor units. Almost any force between them can be produced by some some reductions of different motor units and different frequencies of motor neuron discharge, i.e. different quantities of spatial and temporal summation. The minimum voltage is produced by one twitchin smallest propulsion unit and the maximum simultaneous fused contraction in all motor units. Tension develops smoothly during normal muscle contractions, not like the subthetanic contractions we've seen. However, motor neurons have a stable rate of firing in a wide range of developed tensions, and they often shoot at speeds below those needed to produce theta synthetic synthesis. At these low frequencies, motor units must behave. This means that the smoothness of voltage development is due to asynchrony when the various motor units are narrowed. There are two different types of skeletal muscles in vertebrates, red and white muscles. Red muscles are red because they contain myoglobin protein, which, like hemoglobin, contains an iron-rich group of gems. It is a group of hem that gives both hemoglobin and myoglobin red color and the ability to bind oxygen. White muscles are white because they contain little myoglobin. The red muscles are more slowly contracted by twitching muscles, slow muscles, while white muscles more quickly contract the fast muscles. As noted, slow muscles also require a lower minimum stimulation rate for thetaan synthesis. The concentration of myosine is about the same in both red and white muscle, but the concentration of myosin-zinpas is much higher in white muscle fibers. The red muscle contains a lot of mitochondria and gets most of its ATP from oxidative phosphorylation. This source delivers ATP quickly, and thus the red muscles are able to maintain contractions for longer without tedious. In addition, the red muscle is highly vascularized, receiving and using more oxygen than white muscles. It can also be a function of high concentration of myoglobin. The white muscle, on the other hand, contains several mitochondria and gets most of its ATP from glycolysis, a breakdown of glycogen (which occurs in high concentrations in the white muscle) into lactic acid. This ATP source is not as effective as oxidative phosphorylation, and therefore white muscles get tired faster than red muscles. The white muscle is also more poorly vascular. These deficiencies may not be a big problem because white muscles are usually only active for short periods in normal behavior. Because of these differences, white muscles have been called twitching now, paying later muscles, while red muscles have been called pay as you twitch your muscles. Different muscles contain different types of muscle fibers. Some fibers quickly contract, glycolytic and fatigue quickly. They are known as FG fibers, for a quick, Other fibers are slowly contracted, oxidative, and slowly tiring. They are known as SO fibers, for Oxidative. However, other fibers contract quickly and are both oxidative and glycolic and are therefore known as FOG fibers. Although the motor unit consists of only one type of muscle fiber, most muscles are mixtures of FG, SO and FOG fibers. The sole of the muscle is a red muscle, and it contains almost exclusively SO fibers (87-100%, depending on the type), while gastrocnemius, the white muscle, has a mixture of FG, FOG and SO fibers (41-66%, 14-38%, 5-45%, depending on the species). Although the motor unit consists of only one type of muscle fiber, most muscles are a mixture of FG, SO and FOG fibers. Fibers in red and white muscles also receive different inertia. Fibers in red muscles are inert with small-diameter motor neurons, which reduces the speed of conductivity, which discharges almost continuously at low frequency. Fibers in white muscles receive inertia from large motor neurons, which have longer periods of silence between discharges, but discharge at high frequencies. Twitch Compression Time, msec Fast, 50-80 Slow, 100-200 Minimum Entgeneric Frequency 60/sec 16/sec Content of myoglobin Low High Primary Source OCH Glycolysis Oxidative Glycogen High Activity myosine-ATE High Low Capillary Blood Flow Low Fiber High Fiber Level Easy Difficult Nervous Dimension Large Nervous Fiber High-frequency continuous, low-frequency voltage is produced more than the properties of both red and white muscles are summed up in the table 14-2. The properties of slow muscle fibers make them most suitable for long periods of contraction when minimal strength is required, for example, when maintaining posture. Fast muscle fibers are better suited for short periods of rapid contraction at higher strength, such as in sprints. In fact, during a workout there may be a differential effect on two types of muscles. Strength training leads to hypertrophy in mostly white muscles with the conversion of FOG into FG fibers. The number of fibers does not increase, but the size of the fibers and the amount of myofibril do increase. This increases both strength and rate of contraction. Endurance training appears to affect red muscle fibers, causing an increase in the concentration of oxidative phosphorylation enzymes, increased muscle vascularization and conversion of FG into FOG fibers, but no changes in the ratio of fast and slow fibers and no changes in muscle size. When muscles are contracted, they generate action potential. The potential for action is the result of transmembrane currents in muscle fibers that can be recorded extracellularly. This can be done in non-anggetized people by using small metal electrodes on the skin above the muscle or by using the subcutaneous needle-electrodes inserted into the muscle. muscle contraction, obtained in this way, is an electromyogram, abbreviated to EMG. When a needle needle used, it is often possible to detect discharges in single muscle fibers near the electrode. Discharges in different fibers can sometimes be distinguished by the amplitude of their spikes. Figure 14-18 shows EMG from human biceps (upper footprint) and triceps muscles (lower footprint) during alternative flexion and elbow expansion. Notice the spikes of different amplitude and the overall increase in the density of spikes with each reduction. Structurally, the heart muscle is similar to the skeletal muscle in that it is striped, having both thick and thin strands. It has a well-developed T tubular system, although the sarcoplamic stickula is not as large or extensive as in the skeletal muscle. Figure 14-19 shows the basic structure of the heart muscle for comparison with the figure of 14-5. Unlike skeletal muscles, the triads of a person's heart muscle are located on the line of the z, giving only one on the sarcomer. The mechanism of arousal-reduction of the compound is the same as for skeletal muscles: the membrane potential of action leads to an increase in ca' around myophilaments, which activates myosin-ATPase and leads to the slip of thin and thick filaments. The source of calcium is different in the heart muscle. Since sarcoplamic cyticulum is poorly developed, it cannot sequester the large amount of calcium that skeletal muscles can. Thus, most of the calcium to contract must come from extracellular sources; it comes in during the potential of the action. There are a large number of different types of cells in the heart muscle. These include cells of the synoathral node, the athrioventricular node, the atrium, his beam, and the ventricle, each with a different form of action potential. The details of these differences go beyond this treatment. For our purposes, it is convenient to distinguish between two types of heart muscle cells: a pacemaker cell, like Purkinje fiber, and a contracting cell. Examples of Purkinje (A) fiber action potential and contract cell action potential (B) are shown in Figure 14-20. Both actions have potentials much longer in duration than spikes in nerve cells and skeletal muscle cells, 0.5 sec compared to 0.5 to 5.0 msec. The hypopolar phase of purkingye fiber action is no different from the phase in the skeletal muscle, and appears to have the same ion mechanism, i.e. a sharp increase in sodium conductivity. The growing phase of the contracting cell's potential is shown in an extended sweep in figure 14-20C (right) along with The Purkinje fiber for comparison (5). The potential of the contracting cell has two phases of growth, a rapidly growing phase, both in the Purkinje fiber (6), and a slower rising phase. The fast phase has the same mechanism as the upward phase of the Purkingier fiber potential, but the slower phase is the result slow inner current, carried mostly ca. Calcium current is activated at a more hypopolarized level of membrane potential than sodium activation, and calcium current inactivation is less rapid by about two orders of magnitude. The long plateau of action potential in the heart muscle serves two functions: It provides a longer contraction without resorting to tetanus, and it provides a longer fire-resistant period to prevent the heart from contracting prematurely. This plateau is produced by a number of factors, the most important of which is a decrease in potassium conduction with hypopolarization, followed by slow-growing growth, which brings potassium conduction to a finship value slightly more than the resting levels in The Purkinje fibers and resting levels in the contractile cell at about 300 msec. Changing membrane conduction with changes in the membrane potential of biophysics is called a correction. This change in potassium conduction is called an abnormal correction. Changes in sodium and potassium conductivity are shown in figure 14-21B in the same timeline as the potential action of Purkigner fiber in A. During plateau potential action, membrane stability is high. Thus, after peaking the potential of action, GNAS begins to decrease, albeit more slowly than in skeletal muscles and nerves, and in the contractile cell a slow internal current is maintained. In addition, there may be a small external current due to chloride ions (7). These currents would have actually been reversed by a large external current of the CK if potassium conduction had remained normal, but since GCC is depressed due to abnormal correction, the amount of external iK and external iCl is just a little more than the inside of the iNa' iCa amount, and the membrane repolarizes only very slowly. However, as the GCC increases during the burst (because the membrane is slowly repolarized), iK' increases, while gNa and gCa' decrease, and the membrane begins to repolar faster and faster (impact action potential) until the potential resting membrane, Vr, is reached. In pacemaker cells, the membrane potential almost changes; so there is no true potential for rest. In Purkinje fibers the membrane does not really have Vr, because the membrane potential is constantly changing. It is a property of pace-stimulated cells, cells that have their own internal rhythms of activity. Once one potential action is complete, the membrane immediately begins to generate another, even in the absence of any neural connections. This behavior never happens in skeletal muscles; Muscle is not contracted in the absence of inertia. When the membrane of the fiber Purkinje repolarizes, it returns to the most negative diastolic potential of the membrane, Vd, where the current membrane is zero, im'0, but it stays there only for before the membrane starts to hypopolarize again. In Vd the current membrane is zero because the outer current of potassium is precisely balanced by the internal currents of sodium, calcium and chloride. The membrane begins to hypopolarize, because the conduction of potassium, after its initial decline and slow rise to a level just above the level of rest, begins to decline to the level of rest. As it does so, potassium current decreases and the point is reached where iK' no longer balances the internal currents, and the pure internal current hypopolarizes the membrane. When the critical level of shooting is reached, the potential of action is initiated. The rhythm of the pacemaker that develops is myogenic (muscle origin) rather than neurogenic (neural origin), but it may be influenced by neurotransmitters; they reduce or increase the rate of action potential formation by reducing or increasing the rate of hypopolarization after a potential action. With the increase in speed, the subsequent action potential begins earlier; with a decrease in speed, it starts later. Not all muscle cells are pacemaker cells, but the whole heart must behave like a unit in order to be an effective pump. The contractions are partially synchronized with the electrotonic spread of action potentials from one cell to another. The heart muscle is a network of branching muscle fibers connected to each other by ruptured compounds that are strung together in a structure called an intercalated disk. The intercaled disk is shown in figure 14-19. It is believed that the ruptured intersection is low, much like electrotonic synapses. It seems likely that the transmission of the action potential from cell to cell in the heart muscle is the same as transmission from cell to cell in electrotonic synapse. The heart muscle behaves in the same way as skeletal muscles, but it exerts passive tension when stretched to much shorter lengths. In fact, when the muscle is stretched from the length even shorter than the rest length, there is resistance to the stretch. In other words, the heart muscle experiences elastic tension even at the length of rest (skeletal muscles are not present). In addition, the most developed tension in the heart muscle occurs not at the length of rest, but at its stretch beyond the length of rest (figure 14-22). The result is that when more blood returns to the heart from the veins, the muscle fibers of the heart will stretch more and the blood will be automatically pumped out more strongly than when the heart is just usually filled. This is the basis of Frank-Starling's mechanism in the heart. The strength speed curve for the heart muscle is the same shape as for skeletal muscles, and similar parallel curves are generated for different initial lengths, with permanent Vmax. The increase in the amount of blood in the heart thus does not increase ability or muscle capacity. On the other hand, The actions of some agents such as norradineline or circulating hormones include an increase in Vmax or contracting of the heart muscle. The heart muscle differs from the skeletal muscle in its structure, calcium source and the fact that it can develop tension longer than the length of rest. There is a significant variety among smooth muscles, but they all share the lack of cross bands characteristic of the heart and skeletal muscles and the inertia of the autonomic nervous system as the heart muscle. It's not like a skeletal muscle that's innericated by the fibers of the somatic system. Smooth muscle fibers are smaller than skeletal muscle fibers and are filled with strands oriented roughly along the long axis of fiber. There are both thick, myosin-containing threads and thin, actin-containing threads, but they are not intercigitated, as in striped muscles. Thin filament appears to be attached to a plasma membrane or to some structure in the cytoplasm. In general, the smooth muscle has twice as much actin, but only a third more myosin than in striped muscles. Smooth muscles develop tension that varies depending on muscle length in the same way as in skeletal muscles, but over a much wider muscle length range, almost twice as much skeletal muscle. This property is suitable for their functions as a lining of hollow organs; even when the organ is stretched, smooth muscles can still exert significant tension. Many people assume that the presence of a voltage length curve means that the reduction occurs through a sliding filath mechanism, but how this happens is not known. It has been shown that substantial voltage can be developed in myofibrils to which myosin heads were added in the presence of Ca and ATP, but in which thick fila shows are absent (Oplatka A, Gadasi H, Borejdo J: Biochemphys Res Comm 58:905-912, 1974). This may be due to the contraction mechanism in the smooth muscle. Stretching the denervated smooth muscle causes him to actively contract, a phenomenon never seen in skeletal muscles. Presumably, stretching, like the appearance of a potential action, causes an increase in cytoplasmic ca, which comes either from the sarcoplamic cytocal or directly through the cell membrane. Smooth muscle contraction is much slower than in skeletal muscles. This may be due to the slow spread of Kaa from outside the cell or the slow rate of ATF hydrolysis or both. Smooth muscle, like the heart muscle, is subjected to spontaneous, rhythmic contraction, driven by activity in certain pacemakers, which behave like pacemaker cells in the heart muscle. This pacemaker activity is carried out through gaping connections with adjacent smooth muscle fibers that do not generate pacemaker activity. This is typical of the type of smooth muscle one unit of smooth muscle. Contract activities One unit of smooth muscle can be altered by neural activity or hormones. This kind of smooth muscle also contracts in response to a quick stretch. A one-time smooth muscle is found in the gastrointestinal tract, uterus and blood vessels. Multifunctional smooth muscles are found in the lungs, arteries and erectile tissue of the hair follicles. These smooth muscles contain multiple ruptured compounds, and so contractions do not extend from cell to cell, as in one unit of smooth muscle. The multifunctional smooth muscles are richly innately inductive, with each cell receiving inertia from more than one nerve fiber and each fiber innervating multiple

cells. Like skeletal muscles, multifunctional smooth muscles have motor units; Unlike skeletal muscles, nerve inputs into these smooth muscles can be either excitatory or inhibitory. The reaction of the entire muscle depends on the number of active motor units, the frequency of discharge in the fibers and the relative amount of excitatory and inhibitory input. Multifunctional smooth muscle activity can be triggered by hormones, but it is not strongly affected by rapid muscle stretching. Under normal circumstances, the contraction of the skeletal muscle is triggered by the potential of action in motor neurons, which arrive at the neuromuscular junction and cause the release of acetylcholine from their terminals. Acetylcholine produces in muscle, hypopolarization post-synaptic potential, the potential end of the plate, which always initiates the potential of action in normal muscle fiber with normal inertia. The potential action of the muscles sweeps down the muscle membrane into the T tubes and somehow causes the release of calcium from the tanks of the sarcoplasm to the styculum. Calcium binds to troponin, and there is a release of inhibition of myosine ATPase, hydrolysis ATP, and relative translation (sliding) of thick and thin strands, causing the sarcomer to cut and strain the muscles to increase and possibly cause the muscles to contract. It is believed that actual connections are formed between thick and thin tones (cross bridges), and bridges rotate. The contractions are stopped by removing calcium from the sarcoplasm into thin longitudinal tube-murder sarcoplasm. The cross-bridge theory states that relaxation occurs when the cross bridges are disconnected, a number of elastic elements then restore the muscles to the length of rest. Muscle contraction can be isometric or isotonic in an experimental situation. Isometric contractions are those where tension develops in the muscles, but that does not change the length. Isotonic contractions are those in which the muscle experiences constant tension but can contract. During movement, muscle contraction is probably a mixture of contractions that are isotonic, isometric, and none like with the length and tension of the different. The potential of one action in a motor neuron short, short, contraction in the muscle that he innervates. If multiple action potentials come to a muscle close together in time, twitches can add up. Summation can lead to a steady reduction, which is known as tetanus. Striped muscles develop their maximum isometric stresses at their rest lengths and develop only smaller voltages at a length greater or less than the length of rest. The rate of muscle contraction depends on its load, the more the load, the lower the speed. Expressed in another way, the greater the rate of contraction, the less stress that can be raised by the muscles. Muscle contraction strength can be assessed by changing the discharge frequency in active motor units and by changing the number of active motor units, resulting in the force graded between the twitch voltage in the smallest motor unit to the voltage of the fused tetanus in all motor units of the muscle. Fast muscle differs from slow muscle in its faster twitching contractions, higher maximum thetonic frequencies, reduced myoglobin content, reduced blood flow, more tedious, and inertia of larger axons that discharge periodically at a higher frequency. The motor unit contains either fast or slow muscle fibers, but the muscles are usually a mixture of both fast and slow fibers. EMG is a recording of the action potentials of groups of muscle fibers that lie next to the record electrodes. If needle electrodes are used to record EMG, discharges of a single fiber can often be distinguished. The heart muscle differs from the skeletal muscle by the form of its action potential. All the potentials of action in the heart muscle develop more slowly (the phase of hypopolarization is longer) and longer duration. These differences are due, at least in part, to the slow internal flow of calcium and possibly various kinetic channel activation and abnormal correction, respectively. The heart muscle also shows rhythmic activity, which is myogenic, never seen in skeletal muscles. In addition, unlike skeletal muscles, the heart muscle experiences elastic tension even at the length of rest and is able to develop tension at a length shorter than the length of rest. The smooth muscle behaves either as a whole, i.e. there are many ruptured compounds that cause the whole muscle to contract more or less at once, or as several units, i.e. there are several ruptured compounds, but a rich inertia, each cell is capable of independent contraction. In one unit of smooth muscle rhythmic contractions are myogenic; in multifunctional smooth muscles they are neurogenic. One unit of smooth muscle contracts in response to rapid stretching; there are no multifunctional smooth muscles. Ariano MA, Armstrong RB, Edgerton VR: Hindlimb muscle fibers populations of five mammals. J Cytochem 21:51-55, 1973. Bendall JR: Muscles, molecules and movements. New York, American Elsevier, 1970. Carlson FD, Wilkie DR: Muscle Muscles Englewood Cliffs, New Jersey, Prentice Hall, 1974. Ebashi S, Endo M, Ohtsuki I: Muscle Contraction Control, Rev Biophys 2:351-384, 1969. Gordon AM, Huxley AF, Julian FJ: Tension development in heavily stretched vertebrate muscle fibers. J Physiol (London) 184:143-169, 1966. Gordon AM, Huxley AF, Julian FJ: Variation of isometric tension with sarcomer-length vertebrate muscle fibers. J Physiol (Lond) 184:170-192, 1966. Hill AV: First and last experiments in muscle mechanics. London, Cambridge Univ. Press, 1970. Huxley AF, Simmons RM: Proposed mechanism of power generation in striped muscles. Nature 233:533-538, 1971. Huxley AF: Muscle reduction. J Physiol (Lond) 243:1-43, 1974. Julian FJ, Moss RL, Solens MR: Mechanism for vertebrate striped muscle contractions. Circ Res 42:2-14, 1978. Noble MIM, Pollack GH: Molecular Reduction Mechanisms. Circus Res 40:333-342, 1977. Oplatka A, Gadasi H, Borejdo J: Reducing the spectre of myofibrili and glysterini corrugated muscle fibers irrigated by heavy meomezin subfrage-1. Biochemical Biophysics Res Comm 58: 905-912, 1974. Wilkie DR: The link between strength and speed in the human muscle. J Physiol (London) 110: 249-280, 1949. Tzurler KL: Mechanism of muscle contraction and its energetic energy. In: Mountcastle VB :: Medical Physiology. 13th o.p. Volume 1, St. Louis, Mosby 1974. Footnotes: 1. In rice. 14-1, muscle fibers rectangles or parallelograms, tendon lines emitting from muscle fibers or vertical lines extending from the two sides of muscle fibers, arrows marked F indicate the direction of force rendered by the fibers, and arrows marked F indicate the direction of force rendered throughout the muscle. 2. In fact, it is generally accepted that in Limulus, horseshoe crabs, bands change length when contractions occur at a length less than the length of the rest. They're probably not in the muscles of mammals. 3. Active muscle always develops tension, but not always shrinks. 4. Another word of strength is the load. 5. Differences in this figure are exaggerated for didactic purposes. 6. Do not confuse the fibers of Purkinier (muscles) of the heart with the cells of Purkinier (nerve) of the cerebellum. 7. The current of chloride outwards, because the driving force on the plateau is outward (membrane potential positive from the outside); chloride ions actually get into the cell. (TOC) (Chapter 15) (Glossary) (Abbreviations) (Abbreviations) how is this change in whole-muscle force achieved in vivo. how is this change in whole muscle force achieved