13 Structure and Function of Skeletal Muscle

Muscles are biological machines that convert chemical energy, derived from the reaction between food substrate and oxygen, into mechanical work and heat. Muscle strength can be defined as the ability of skeletal muscles to develop force in order to initiate, decelerate, or prevent movement. One single factor, i. e. force alone, is not, however, sufficient to perform and to control movements, to stabilize posture, or to prevent injuries to the musculoskeletal system. Therefore, for optimum control and performance of the musculoskeletal system, muscle endurance and muscle coordination are required, in addition to force. Muscular endurance can be defined as the ability to maintain a specified force for a given period of time. However, this definition of endurance is not universally applicable, as endurance varies with the type of mechanical work performed.

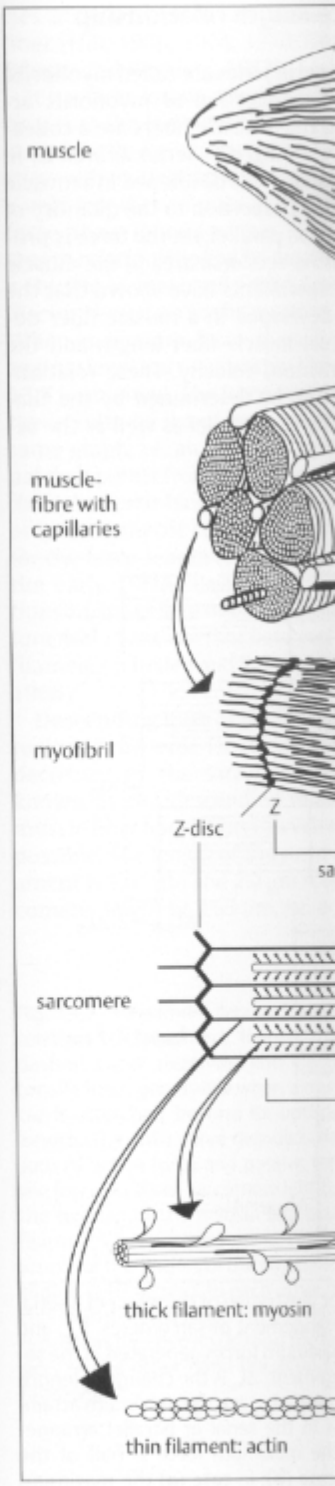
Skeletal muscle morphology

Skeletal muscle represents the largest organ of the body. It accounts for approximately 40 % of the total body weight and is organized into hundreds of separate entities, or body muscles, each of which has a specific task in enabling the great variety of movements that are essential to normal life. Each muscle is composed of a great number of subunits, muscle fibers, that are arranged in parallel and normally extend from one tendon to another (Fig. 13.**1**). Muscle fibers are cable-like structures composed of tightly packed subunits, myofibrils, that fill up most of the volume of the fibers. The myofibrils are composed of sarcomeres arranged in series. The sarcomeres, defined as the basic units of the myofibrils, are responsible for force generation and shortening of myofibrils. The basic unit is formed by two contractile proteins, or myofilaments, the myosin (thick) and actin (thin) filaments. Actin filaments are anchored at each end to the Z-disks. A sarcomere is defined as that part of a myofibril enclosed between the Z-disks. When a muscle is in a relaxed state there is some overlap between the myosin and actin filaments.

The basic units generate force by the interaction of actin and myosin filaments (Huxley, 1957, 1973, 1988; Huxley and Peachy, 1961). Cross-bridges are structures forming part of the thick (myosin) filaments of the sarcomere. They attach to the backbone of these filaments in such a way as to enable them to attach to sites on the thin (actin) filaments in their vicinity. The cross-bridges are functionally identical and act independently. The probability of attaching to an actin filament is influenced by local biochemical conditions, e. g. calcium ion concentration. At any given time during activity of a muscle, some cross-bridges are attached to the thin filaments but others are detached or moving towards new attachment sites.

When attachment of actin and myosin filaments occurs, attached cross-bridges undergo structural deformation. Force is generated and the thin (actin) filament is pulled along the thick (myosin) filament. The myofilaments slide in relation to each other; the overlap between the filaments increases and at the same time the muscle fibers, or the muscle, shorten (Fig. 13.**2**). According to the theory of contraction, the length of the myosin filaments is assumed to be constant and not to contribute to the shortening of the sarcomere. Therefore, the shortening of a sarcomere is assumed to occur only via the sliding of the filaments relative to each other. The total shortening of the myofibril is the sum of the length changes occurring in the sarcomeres arranged in series. Supposing we have two myofibrils, one consisting of 10 sarcomeres and the other of 5 sarcomeres arranged in series. It follows that the amount of shortening of the former will be twice the shortening of the latter.

It is assumed that active muscle force originates exclusively from cross-bridges. As a consequence, active force generated at any given time is dependent on the number of parallel cross-bridges attached to the thin (actin) filaments. Because of the series arrangement of sarcomeres within a myofibril, the force generated by one unit has to be maintained and transmitted to the next unit. Therefore, the force of the whole structure is equal to the force of one unit, i. e. the force of one sarcomere. If the force developed in one half of a sarcomere is to be transmitted to the other Z-disk of a sarcomere, the force in the other half of a sarcomere has to be equal in magnitude (Huijing, 1983) (Fig. 13.**3**). It follows that an equal number of active cross-bridges in both parts of the sarcomere should be attached. If the number of active cross-bridges is low, the developed force is low. If the number of active cross-bridges increases, the force developed will increase as well.



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Brinckmann P, Frobin W, Leivseth G. Musculoskeletal biomechanics. Thieme 2002.

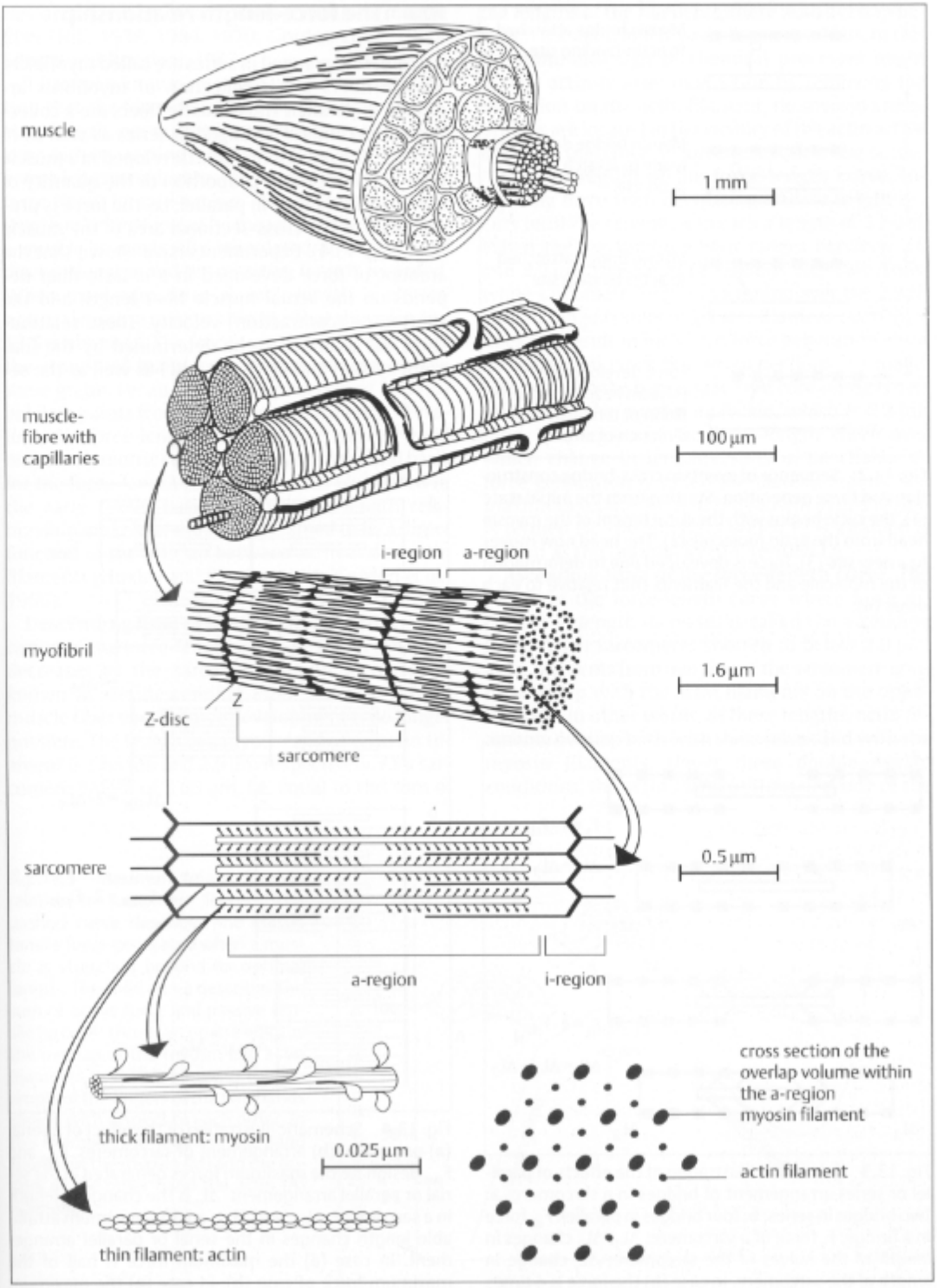

Fig. 13.1 Scheme of muscle architecture. (Adapted from Di Prampero, 1985)

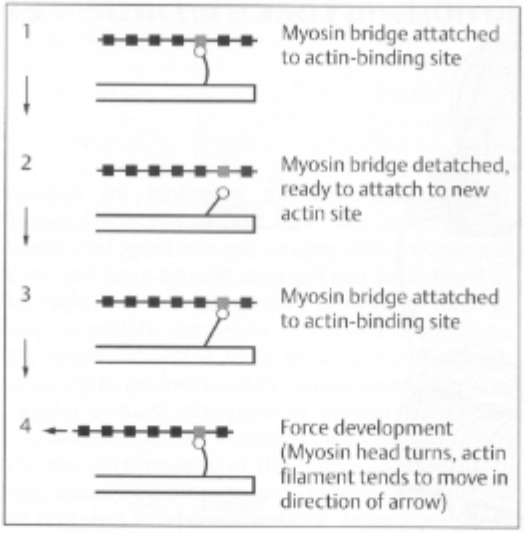

Fig. 13.2 Sequence of events in cross-bridge construction and force generation. Starting from the initial state (1), the cycle begins with the detachment of the myosin head from the actin molecule (2). The head now moves to a new site (3), force is developed due to deformation of the molecule and the filaments shift relative to each other (4).

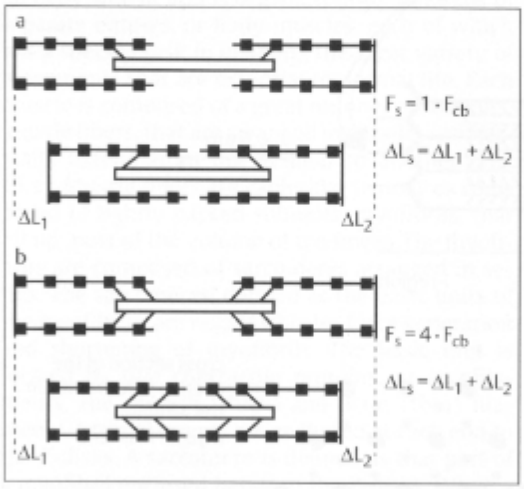

Fig. 13.3 Schematic illustration of the effects of parallel or series arrangement of bridges in a sarcomere. **a:** two bridges in series; **b:** four bridges in parallel. F_{cb} force in a bridge; F_s force of a sarcomere; ΔL_1, ΔL_2 changes in length of the halves of the sarcomere; ΔL_s change in length of the sarcomere. In case (**b**) the force is 4 times the force of case (**a**); the maximum change in length is the same in each case.

The force-length relationship

Sarcomeres arranged in series are called myofibrils. Muscle fibers are a collection of myofibrils arranged in parallel. Thus, muscle fibers are a collection of sarcomeres arranged in series as well as in parallel. The maximum force developed in a muscle fiber will increase in proportion to the quantity of myofibrils arranged in parallel, i.e. the force is proportional to the cross-sectional area of the muscle fiber (Fig. 13.**4**). Experiments have shown that the amount of force developed in a muscle fiber depends on the actual muscle fiber length and the shortening (contraction) velocity. These relationships are assumed to be determined by the filament system and by the parallel as well as the se-

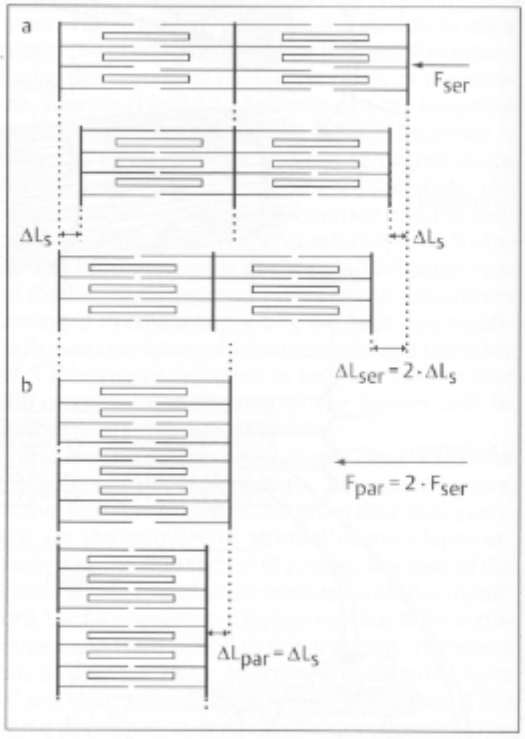

Fig. 13.4 Schematic illustration of the effect of a serial (**a**) or parallel (**b**) arrangement of sarcomeres. F_{ser} and F_{par} designate the maximum forces generated in the serial or parallel arrangement. ΔL_s is the change in length in a sarcomere; ΔL_{ser} and ΔL_{par} are the maximum attainable length changes in the serial or parallel arrangement. In case (**a**) the maximum force is half of the maximum force of case (**b**); in case (**a**) the maximum change in length is twice the maximum change in length in case (**b**).

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ries arrangement of sarcomeres within a muscle fiber (Hill, 1938, 1964, 1970; Gordon *et al.*, 1966; Edman and Reggiani, 1987).

It has been known since the late nineteenth century that the force developed by a muscle of constant length, e.g. during isometric contraction, will vary with its starting length. In a very shortened or a very lengthened state, the muscle generates less force than in mid-ranges. The isometric force-length relationship can be measured directly when a muscle is maximally stimulated at a variety of discrete lengths and the resulting force is recorded. When maximum force at each length is plotted against length, a relationship like that shown in Fig. 13.**5** is obtained. The force-length curve represents the results of many experiments plotted on the same graph, i.e. an artificial connection of individual data points from isometric experiments. Therefore, the force-length relationship is strictly valid only for isometric contractions. The structural basis for the force-length relationship was elucidated in the early 1960s. Basically, the force-length relationship in skeletal muscle is assumed to be a direct function of the overlap between actin and myosin filaments (Huxley and Peachy, 1961; Gordon *et al.*, 1966).

Descending limb of the force-length curve. The region of the force-length curve in which the force decreases as the sarcomere length increases is known as the descending limb. Above a certain muscle fiber length, force development is no longer possible. The length of a myosin and of an actin filament is 1.65 µm and 2.0 µm respectively. At a sarcomere length of 3.65 µm, i.e. equal to the sum of the lengths of the filaments, there is no overlap between the actin and myosin filaments. Thus, in this situation, although biochemical processes might permit actin-myosin interaction by removing the inhibition on the actin filament, no myosin cross-bridges are located in the vicinity of the actin active sites and therefore no force generation can occur.

Plateau region of the force-length curve. Increasing force with decreasing muscle length occurs until the sarcomeres reach a length of 2.2 µm. When the sarcomere length ranges between 2.0 and 2.2 µm, muscle force remains constant. Thus, while sarcomere length shortening over the 2.2 to 2.0 µm range results in greater filament overlap, it does not result in increased force generation since no additional cross-bridge connections are made. This is due to the bare region (not containing cross-bridges) of the myosin molecule, which is 0.2 µm long. The region of the force-length curve over which change in length results in no change in force generation is called the plateau region. The maximum tetanic force of the muscle in this region is termed F_0. The length at which F_0 is attained is known as the optimal length, termed L_0.

Ascending limb of the force-length curve. The region of the force-length curve where force increases as length increases is called the ascending limb. When sarcomeres shorten to below 2.0 µm, actin filaments from one side of the sarcomere double-overlap with the actin filaments on the opposite side. In other words, at these lengths, actin filaments overlap both with themselves and with the myosin filaments. Under these double-overlap conditions, the actin filament from one side of the

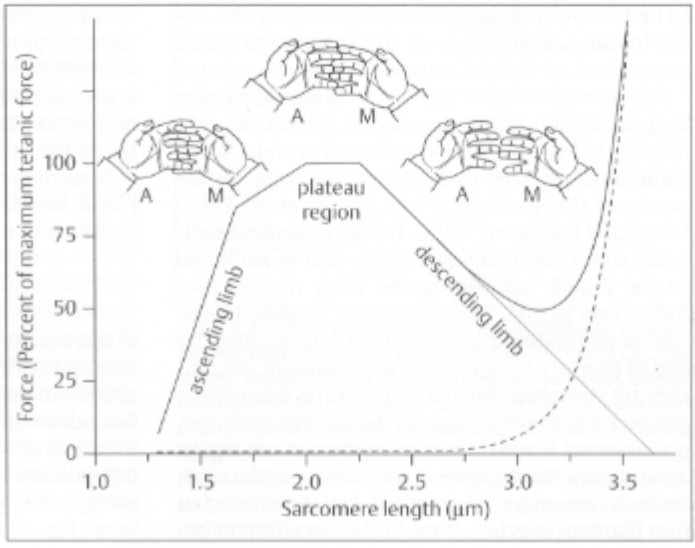

Fig. 13.5 Maximum tetanic force in relation to sarcomere length. The dashed curve describes the elastic tensile force generated when a muscle is stretched beyond its optimal length. The solid curve describes the sum of active force and passive tensile force. At three sarcomere lengths the overlap of the myosin and actin filaments is visualized using the example of the fingers of the two hands sliding with respect to each other. In this example, the fingers of one hand represent the myosin (M) and the fingers of the other hand the actin (A) filaments.

sarcomere interferes with cross-bridge formation on the other side, resulting in decreased muscle force output. The region where shortening between 2.0 and 1.87 μm occurs is known as the shallow ascending limb of the force-length curve. This region is distinguished from the next portion of the force-length curve, which is known as the steep ascending limb. At these very short lengths, the myosin filament actually begins to interfere with shortening as it abuts the sarcomere Z-disk, leading to a rapid reduction of force.

Passive force-length curve. The dashed line in Fig. 13.**5** represents the force generated if a muscle is passively stretched to various lengths. Near the optimal length L_0, passive tension is almost zero. However, as the muscle is stretched to longer lengths, passive force increases dramatically. The increase in passive force, which occurs when the muscle is stretched, may play an important role in re-establishing myofilament overlap in the absence of muscle activation. These relatively long lengths can be attained physiologically. In such situations, large passive forces directed at re-establishing muscle length will be encountered.

The structures responsible for the increase in passive resistive force are located outside and inside the myofibrils. According to Cavagna (1977) and Heerkens *et al.* (1987) the parallel elastic elements of muscle (intramuscular connective tissue) are responsible for the force exerted by a passive muscle when it is stretched beyond its optimum length. As collagen is the major protein in muscle, tensile properties of passive muscles are primarily dependent on the amount and type of collagen (Kovanen *et al.*, 1984). Although intramuscular connective tissue enables forces (actively developed by the muscle or passively imposed on the muscle) to be transmitted safely and effectively by the entire tissue, information on the connective tissue component of skeletal muscle is relatively sparse.

Recent studies have shown the origin of passive muscle tension also to be located within the myofibrils themselves. A new structural protein, 'titin', sometimes referred to as 'connectin', may be the source of this passive tension (Wang *et al.*, 1993; Labeit and Kolmerer, 1995). This large protein molecule spans each half-sarcomere and is anchored to the Z-disk, connecting the thick myosin filaments end to end. Titin is thought to play a basic role in maintaining sarcomere structural integrity and to produce passive force when muscle sarcomeres are stretched. Furthermore, titin is assumed to produce a high percentage of the passive force that is developed in most muscles in the plateau region and the descending limb of the force-length curve. Once a sarcomere is stretched beyond thick and thin filament overlap, cross-bridge attachment becomes impossible, and the forces required to re-establish myofilament overlap are thought to come primarily from the passive elastic forces of the highly stretched titin. In addition to passively supporting the sarcomere, titin stabilizes the myosin lattice, so that high muscle forces do not disrupt the orderly hexagonal array. If titin is selectively destroyed, normal muscle contraction may cause significant myofibrillar disruption.

The force-velocity relationship

Skeletal muscle has an inherent capacity to adjust its active force to precisely match the load exerted on it during shortening, or to regulate the resistive force during muscle elongation. This property distinguishes muscles from a simple elastic structure and is based on the fact that active force generation is continuously adjusted to the speed of the contractile system. The maximum force development capacity of a muscle depends, not only on muscle length, but also on the velocity of contraction. It is observed that the active force development of a skeletal muscle is high when the velocity of contraction is low and that the force decreases with increasing velocity of contraction. Thus, when the load is high, the active muscle force is increased to the required level by reducing the speed of shortening. Conversely, when the load is low, the active force can be made correspondingly small by increasing the speed of shortening.

Fig. 13.**6** shows the relationship between force and velocity of an isolated whole sartorius muscle of the frog, published by Hill (1938). Experimentally, the force-velocity relationship, like the force-length relationship, is a curve that represents the results of a number of experiments plotted on the same graph. A muscle is stimulated maximally and allowed to shorten or to lengthen against a constant load. The muscle velocity during shortening or lengthening is measured and then plotted in relation to the load.

According to Hill, the relationship between force F and shortening velocity v during a concentric contraction may be described by the equation:

$$(F + a) \cdot v = b \cdot (F_{max} - F)$$

In this equation, a and b are experimentally derived constants. The values of a and b are estimated at approximately 0.25. F_{max} is the maximum tetanic force developed at a given muscle length. The maximum force development is achieved at a shortening velocity v equal to zero. The maximum shortening velocity v_{max} is observed at force F equal to zero (Fig. 13.**6**). The Hill equation can be used to

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The force-velocity relationship is related to the cross-bridge cycling rate. The cross-bridges between actin and myosin have been found to attach and detach at certain rates (Hill, 1964; Pollack, 1983; Huxley, 1988). These are referred to as rate constants. At any point in time, the force generated by a muscle depends on the number of cross-bridges attached. When contraction velocity increases, the filaments slide past one another faster and faster; therefore, force decreases due to the lower number of cross-bridges attached. Conversely, as the relative filament velocity decreases, more cross-bridges have time to attach and generate force, so that force increases. This discussion is not aimed at giving a full description of the basis for the force-velocity relationship, but only at providing some insight into how cross-bridge rate constants can affect force generation as a function of velocity.

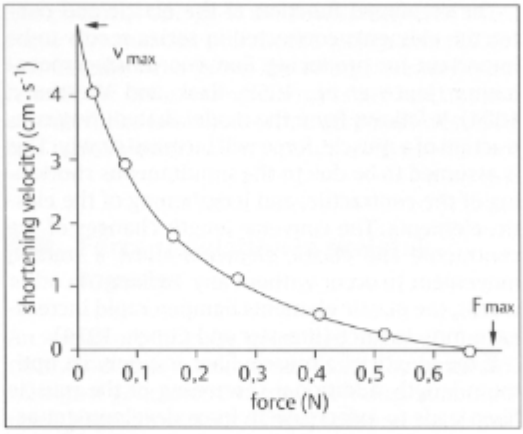

Fig. 13.**6** Empirical relation between force and contraction velocity of a muscle, documented for the sartorius muscle of the frog. The relation was described by Hill (1938) by means of a hyperbola.

Theoretical modeling of skeletal muscle behavior

The modeling of skeletal muscle serves the purpose of predicting static and dynamic behavior in different situations. The model of a muscle is constructed from single elements with known mechanical properties. Elements may be combined to represent properties of the whole muscle. Hill (1970) developed a muscle model consisting of two basic elements; a contractile element and an elastic element (Fig. 13.**7a**). The contractile element models the myofilaments responsible for active force development. The elastic element describes the passive elastic properties of the structure. The mechanical property of the elastic element equals the elastic property of a spring (see Chapter 7). Like a spring, the elastic elements store energy during elongation and release virtually all the energy as they return to their original length.

In its most simple form, the Hill model (Hill, 1958) is constructed from a contractile element connected in series with a passive elastic element. In this model, the contractile elements are considered to have force-length as well as force-velocity characteristics similar to those described for muscle fibers. As both elements are connected in series, both are subject to the identical tensile force. The change in length of the combined model is the sum of the length changes occurring in each element of the model. The velocity of shortening and lengthening of the model is the sum of the velocities of both elements of the model.

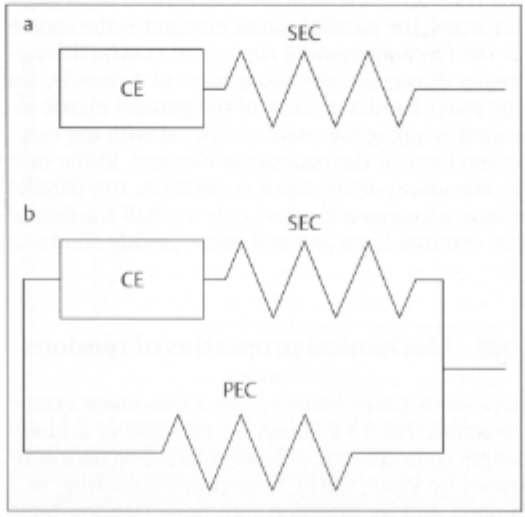

Fig. 13.**7** Hill's muscle model. The contractile properties are represented by the contractile element CE. The elastic properties are represented by the series elastic component SEC (in series to the contractile element) and the parallel elastic component PEC (parallel to the contractile element). (**a**) basic model for description of the active properties of a muscle. (**b**) basic model for description of the active and passive properties of a muscle.

The combined function of the elastic and contractile elements connected in series seems to be important for producing fine, coordinated movements (Joyce *et al.*, 1969; Rack and Westbury, 1984). It follows from the model that, during contraction of a muscle, force will increase slowly. This is assumed to be due to the simultaneous shortening of the contractile, and lengthening of the elastic, elements. The converse length changes of the contractile and elastic elements allow a smooth movement to occur without any 'rocking'. In other words, the elastic elements dampen rapid increases in muscle force (Bressler and Clinch, 1974).

If the length of a muscle fiber is below its optimum length, additional shortening of the muscle fiber leads to a decrease in force development accompanied by a simultaneous shortening of the series elastic elements. When the velocity of shortening of a muscle fiber increases, the force is reduced (because of the force velocity relationship) and the series elastic element is again allowed to shorten. As a consequence, the total length change in the combined model is greater than the isolated length change in the contractile element.

To explain the mechanical behavior of passive muscles, a third element is introduced: the parallel elastic element (Fig. 13.7**b**). The parallel elastic element is arranged in parallel to the contractile element. As the stiffness and the tensile strength of the contractile element are assumed to be negligibly small, the parallel elastic element is the source of the force preventing rupture of contractile elements during passive elongation of a muscle. On the other hand, the force of the parallel elastic element is negligibly small compared with the maximum force of the contractile element. In the case of maximum active force generation, the parallel elastic element will resist only a small fraction of the external force and will consequently shorten.

Mechanical properties of tendons

Tendinous tissue behaves like a non-linear elastic structure. Fig. 13.**8** shows an example of a force-length characteristic of tendon, based on data published by Woo (1981). A specimen consisting of a tendon and its insertion into bone (tendon-bone complex) was investigated. Starting from its resting length, the bone-tendon complex was elongated up to rupture or failure at the site of insertion, and the opposing (resistive) force was measured. The stiffness of the complex at each point of the curve is defined as the ratio of force increase and length increase, i.e. $\Delta F/\Delta L$ [N/m]. The figure shows that the stiffness of the tendon is a function of elongation. With minor elongation of the tendon, stiffness is low, e. g. small changes in force have relatively large effects on tendon length. As elongation increases, stiffness increases as well. In the linear portion of the curve, stiffness is virtually constant. The stress σ_{max} (force divided by cross-sectional area) of a tendon at the point of rupture amounts to approximately 100 MPa; the strain ε_{max} at rupture is in the region of 10 % of the starting length.

The series elastic elements located in tendinous tissue are important for an optimum function of active muscles. When a muscle develops force, the length of the tendon increases and the muscle is allowed to shorten. Because of the change in tendon length, a muscle-tendon unit will have an increased operating range relative to the range of muscle fibers alone. If the muscle-tendon unit is strained by small forces during a movement, the length change in the unit will occur at the tendon because of its lower stiffness. The result is that a fraction of the length change that would have to be accommodated by muscle fibers is actually taken up by tendons. Thus, by attaching muscles to bones via compliant (elastically elongating) tendons, the length changes of the muscle fibers are reduced. Length changes of muscles and their tendons may also have opposite signs. When the force of a muscle-tendon unit with constant length is reduced, the tendon is allowed to

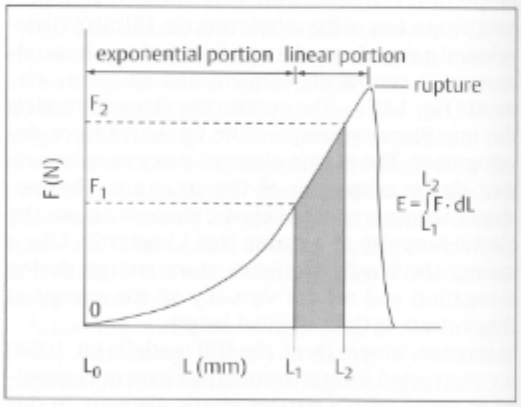

Fig. 13.8 Force-length characteristic of a tendon, using the example of the flexor digitalis tendon of the pig (Woo, 1981). The graph shows the force of the tendon in relation to its length, starting from its length L_0 in the unloaded state. Force as function of length shows first an approximately exponential behavior pattern followed by an approximately linear portion. Upon further increase of the force, rupture occurs at the tendon-bone interface. The shaded area below the curve is a measure of the energy E stored when the force changes from F_1 to F_2 and the tendon length changes from L_1 to L_2.

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Plastic deformation of tendons is negligible, i.e. after release a stretched tendon will return to its original length. The energy expended on stretching the tendon is stored as potential energy and released almost completely as the force exerted on the tendon decreases to zero. The loss of energy is less than 10 %. The force-length characteristics of tendons are dependent only to a minor extent upon the velocity of stretching. Therefore, the deformation energy stored in a tendon during stretching is given by the area below the force-length curve (Butler *et al.*, 1978; Fung, 1981; Woo, 1981)

$$E = \int F \cdot dL \ [Nm]$$

In this formula E denotes the energy, F the resistive force, and dL the change in length. The integral extends from the resting length of the tendon (with zero force F) up to the length reached under the force applied. The amount of stored energy increases as stretching increases (see Chapter 7). At higher forces the tendon is rather stiff and the change in length relative to the change in force will be small. Therefore, in this situation the increase in stored energy will be small as well. A greater amount of energy could be stored if the length change in the tendon were increased. In real life these increased ranges of elongation are obtained during stretch-shortening cycles occurring in a variety of movements. During a stretch-shortening cycle the initial stretching of the muscle-tendon unit is followed by active shortening of the complex. During the stretching phase energy is stored in the unit.

The potential energy stored in a muscle-tendon unit is released by decreasing the force exerted on the structure. If this is done slowly, the energy will become available slowly; if it is done at a rapid rate, the energy will be released rapidly as well. The amount of energy released per unit of time, the power P, is given by

$$P = F \cdot v$$

where F is the force and v the shortening velocity of the muscle-tendon unit.

It is of practical importance that tendons can store energy during a stretch-shortening cycle at a relatively low rate. Upon lengthening, the tendon then acts as an energy reservoir that can be emptied at a high rate to effect movements with high speed and power (Huijing, 1983). This situation may be compared to shooting an arrow with a bow. The potential energy of the bow is increased when the active muscles pull on the string, thereby deforming the bow. When the string is suddenly released, the deformation energy is set free and the arrow will be launched with high power, resulting in a high velocity.

Force regulation in skeletal muscles

An excitatory impulse generated naturally in the central nervous system or artificially by an electrical signal generator creates a so-called action potential (specific electrical field) at the relevant muscle fibers. The tensile force developed by a single fiber in response to a single action potential invading the motor endplates is called a twitch. As the frequency of the stimulating impulses increases, twitches begin to overlap. At frequencies above a certain limit, single twitches can no longer be discriminated and tetanic contraction develops. The frequency limit, where tetanic contraction or tetanic force generation occurs, varies among different fibers and individual motor units. This limit is normally observed in the range between 10 and 100 Hz (Fig. 13.**9**). The higher the frequency of stimula-

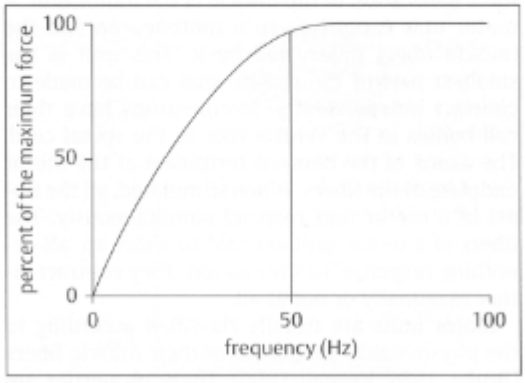

Fig. 13.**9** Dependence of the force of a muscle fiber on the stimulation frequency, the force-frequency curve. At stimulation frequencies higher than about 10 Hz, single twitches merge to form a constant output force. In the lower region of the force-frequency curve, small changes in stimulation frequency effect large variations in force output. In contrast, at high frequencies a change in frequency effects only a small change in force, if any. Changing the frequency from 50 Hz to 100 Hz effects, for example, only a negligibly small change in force. Stimulation frequency of motor units is usually in the order of 10 to 60 Hz. In this region, force depends strongly on frequency.

Table 13.1 Relation between motor units and muscle fiber types

Designation of motor unit	Muscle fiber type
fast fatiguable unit (FF)	Type IIb, fast glycolytic fiber
fast fatigue resistant unit (FR)	Type IIa, fast oxidative glycolytic fibers
slow unit (S)	Type I, slow oxidative fiber

tion of the muscle fibers, the greater is the force produced in the muscle as a whole.

Skeletal muscle fibers possess a wide spectrum of morphologic, contractile, and metabolic properties. Muscle fibers are classified as fast or slow, oxidative or non-oxidative, and glycolytic or non-glycolytic (Brooke and Kaiser, 1970; Bruke, 1981). Often, only three different types are described; for details see Table 13.**1**. The three types differ characteristically with respect to speed of shortening, force generation, and endurance. Fast-contracting fiber types (Type IIb) shorten approximately two to three times faster than slow-contracting fibers (Type I). The specific tension (force divided by cross-sectional area) of fast muscle fiber types is higher than that of slow muscle fibers. In general, slow muscle fibers (Type I) have the greatest endurance, followed by Type IIa and Type IIb fibers.

The functional unit of force generation in a muscle is the sarcomere (in fact the half-sarcomere due to sarcomere symmetry). The functional unit of force generation of the muscle is the motor unit. A motor unit comprises an α-motoneuron and the muscle fibers innervated by it. This unit is the smallest part of the muscle that can be made to contract independently. Motoneurons have their cell bodies in the ventral root of the spinal cord. The axons of the neurons terminate at the motor endplate of the fibers. When stimulated, all the fibers of a motor unit respond simultaneously. The fibers of a motor unit are said to show an 'all-or-nothing response' to stimulation: they contract either maximally or not at all.

Motor units are usually classified according to the physiological properties of their muscle fibers (Bruke, 1981, Kernell 1996). These properties are the motor unit twitch tension, the tetanic tension recorded at an intermediate stimulation frequency, and the fatiguability of the unit in response to a specific stimulation protocol (Fig. 13.**10**). In general, motor units belong to three different groups. Those that have a fast contraction time and a low fatigue index are known as fast fatiguable (FF) units. Those that have a fast contraction time and a high fatigue index are designated fast fatigue resistant (FR) units. Those that have slow contraction times, and which are most resistant to fatigue, are classified as slow (S) units.

A whole muscle is subdivided into many motor units, each of which comprises a single motoneuron and its related muscle fibers. The muscle fibers belonging to one motoneuron have the same physiological, biochemical, and ultrastructural properties. The number of muscle fibers belonging to a motor unit, and the number of motor units within a whole muscle, vary widely. This is closely related to the degree of control required of the muscle. The muscle fibers within a motor unit are interspersed among fibers of other motor units. The functional consequence of this dispersion is that the forces generated by a unit will spread over a larger tissue area. This may minimize mechanical stress in focal regions within the muscle.

The nervous system can vary muscle force output by two mechanisms. By varying the stimulation frequency, the force will be changed, i.e. when the frequency of stimulation is increased, the force output will increase as well. Thus the force output is positively related to the discharge rate of a motor unit. This phenomenon is termed temporal summation. Alternatively, muscle force can be varied by changing the number of motor units that are active at a given time. For relatively low-force contractions, few motor units are activated, while for higher force generation, more units are activated (Bodine *et al.*, 1987). The process by which motor units are added as muscle force increases is termed recruitment.

In their classical study, Henneman *et al.* (1965) showed that, at very low forces, low amplitude voltage signals of short duration ('spikes') were observed at the nerve. As muscle force increased, the size of the spikes also increased in a very orderly fashion, i.e. as force continued to increase, the units recruited exhibited larger and larger spikes. The entire process was reversed as force decreased. From these observations it was concluded that, at low muscle force levels, motor units with the smallest axons and the lowest threshold and depolarization frequency were first recruited. As force increased, larger and larger axons with higher activation thresholds and higher excitation amplitudes were recruited. This is known as the 'size principle' and provides an anatomic basis for the orderly recruitment of motor units to produce a smooth contraction. In later studies, e.g. Binder and

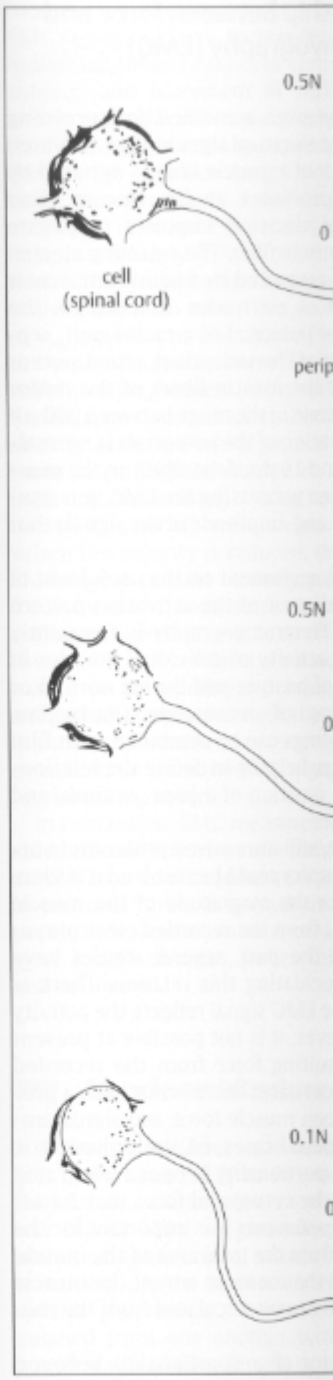

Fig. 13.10 Schematic illustration o
physiologic properties of three diffe
units. Type IIb (upper diagram) has
vating a large number of muscle fib
duce large forces but are subject to
IIa (middle diagram) has medium siz

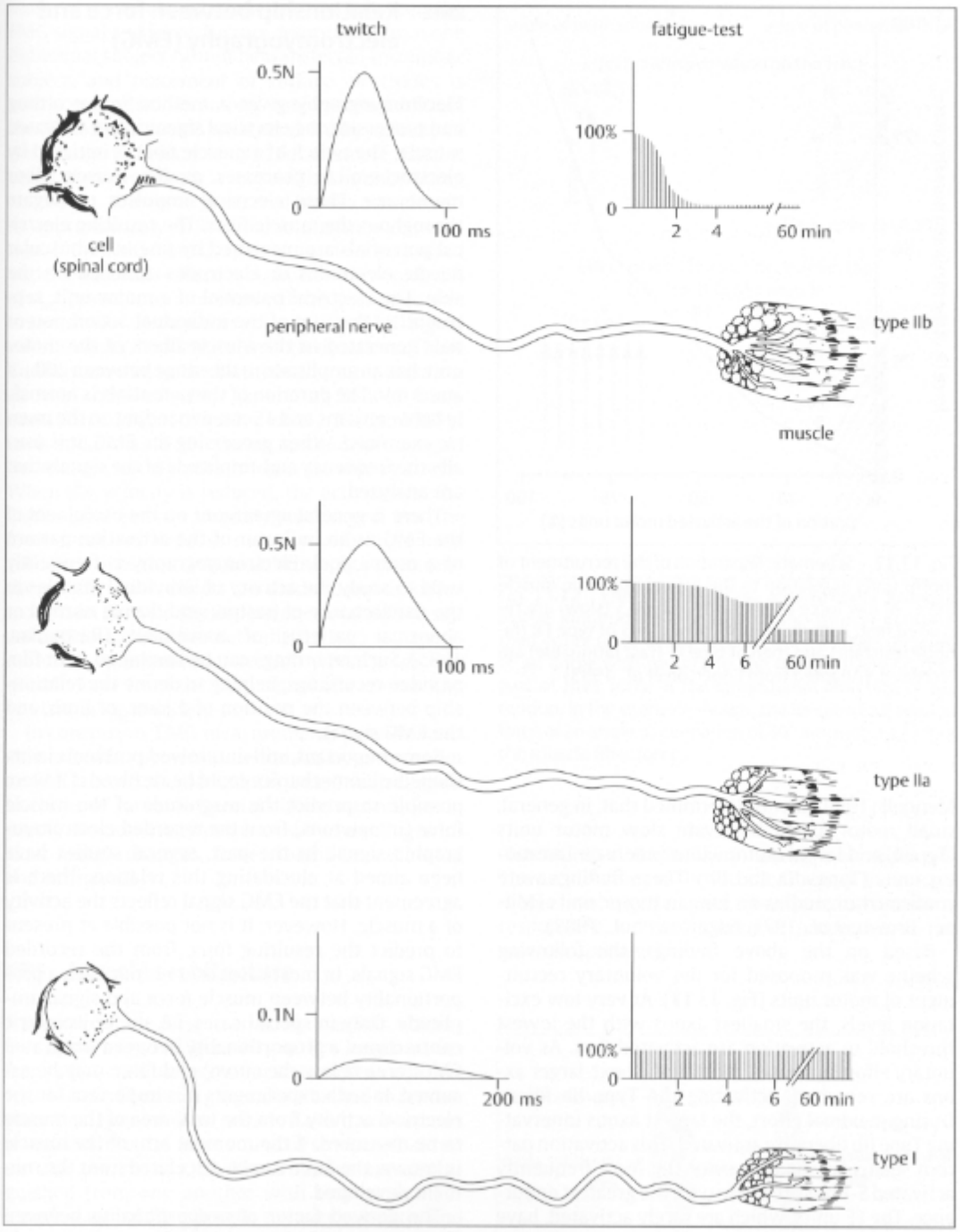

Fig. 13.**10** Schematic illustration of the anatomic and physiologic properties of three different types of motor units. Type IIb (upper diagram) has large axons innervating a large number of muscle fibers. Such units produce large forces but are subject to rapid fatigue. Type IIa (middle diagram) has medium size axons innervating a large number of muscle fibers. Such units produce lower forces than Type IIb but are less subject to fatigue. Type I (lower diagram) has small axons innervating only few fibers. Such a unit develops only a low force but is able to maintain this force over a longer period of time. (Adapted from Edington and Edgerton, 1976)

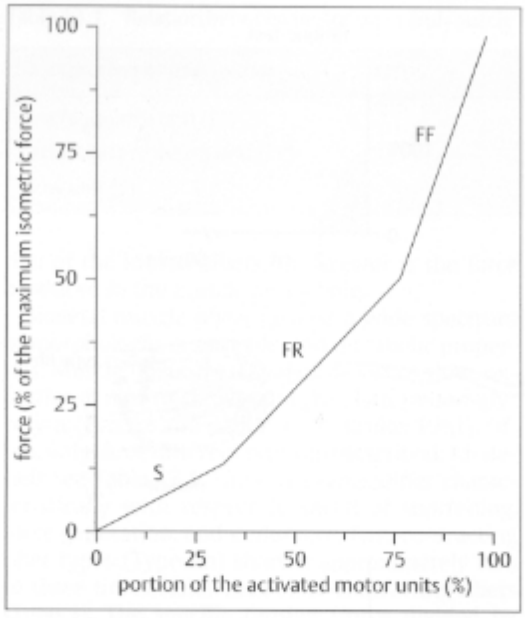

Fig. 13.**11** Schematic illustration of the recruitment of motor units in relation to the active, isometric muscle force. At low force levels, units of type S (slow) are recruited first; with increasing force, units of type FR (fatigue resistant) and then of type FF (fast fatiguable) are recruited. (Adapted from Edgerton *et al.*, 1983)

Mendell (1990), it was determined that, in general, small motor axons innervate slow motor units (Type I) and larger motor axons innervate fast motor units (Types IIa and IIb). These findings were confirmed in studies on human motor units (Milner-Brown *et al.*, 1973, Edgerton *et al.*, 1983).

Based on the above findings, the following scheme was proposed for the voluntary recruitment of motor units (Fig. 13.**11**). At very low excitation levels, the smallest axons with the lowest threshold to activation are activated first. As voluntary effort increases, most of the next-larger axons are recruited, activating the Type IIa fibers. During maximal effort, the largest axons innervating Type IIb fibers are activated. This activation pattern seems reasonable, since the most frequently activated S-units are those with the greatest endurance. The FF units, which are rarely activated, have the lowest endurance. In addition, the S-units (Type I), which are activated first, develop the lowest tension, so that low tension is generated as contraction begins. This provides a mechanism for smoothly increasing tension as first S-units, then FR-units, and finally FF-units are recruited.

Relationship between force and electromyography (EMG)

Electromyography gives a method for recording and processing the electrical signals of an activated muscle. The twitch of a muscle fiber is initiated by electrochemical processes at the muscle fiber membrane. These electrical impulses propagate throughout the muscle fiber. The resulting electrical potentials are measured by fine intramuscular needle electrodes or electrodes attached on the skin. The electrical potential of a motor unit, representing the sum of the individual action potentials generated in the muscle fibers of the motor unit, has an amplitude in the range between 200 µV and 3 mV. The duration of the potentials is normally between 2 ms and 15 ms, depending on the muscle examined. When processing the EMG, it is usually the frequency and amplitude of the signals that are analyzed.

There is general agreement on the usefulness of the EMG as an indicator of the activation pattern of a motor pool. Electromyography is frequently used to study the activity of individual muscles in the maintenance of posture and during normal or abnormal patterns of movement (Basmajian, 1985). Such recordings can be combined with film or video recordings, helping to define the relationship between the position of a joint, or limb, and the EMG signal.

Some important, still unresolved problems in orthopedic biomechanics could be resolved if it were possible to predict the magnitude of the muscle force (in newtons) from the recorded electromyographic signal. In the past, several studies have been aimed at elucidating this relation. There is agreement that the EMG signal reflects the activity of a muscle. However, it is not possible at present to predict the resulting force from the recorded EMG signals. In most cases there is not even a proportionality between muscle force and signal amplitude. Only in special cases, i.e. under isometric contractions, a proportionality between integrated EMG (area below the curve) and force may be assumed. In such experiments it is important for the electrical activity from the total area of the muscle to be measured. If the moment arm of the muscle is known, the force can be calculated from the moment developed.

The derived factor of proportionality between EMG signal and force can be used to predict muscle force from the EMG signal only. However, this method has its limitations. The factor of proportionality between EMG signal and muscle force determined for one muscle may not be valid for another muscle. Other muscles may have different moment arms, changing the rel
EMG signal and force. Factors de
individual subject cannot be tra
subject, and placement of su
poorly reproducible among su
the EMG signal seems to depen
the change in muscle length. Fo
being measured from a muscle
at 5 % v_{max}. The force generated
be approximately 75 % of maxi
sion. If the EMG from the same
under conditions where it is sh
of v_{max}, much more tension wi
to the slower contraction veloc
will, however, be identical in bo
findings are due to the fact that
already activated when genera
mum force (Bigland-Ritchie et
Loeb and Gans, 1986). Owing t
the muscle, not all cross-bridge
When the velocity is reduced, t
EMG signal remain virtually u
force increases because more
sufficient time to attach. In an a
these difficulties, efforts are ma
mation on the velocity depende
nal and to apply a theoretically
mentally determined correctio
muscle force from the EMG sig
or elongating muscle.

In conclusion, EMG measure
able information on muscle acti
an EMG record should be inter
in terms of force. In special ci
purely isometric situations or
slow length changes, forces
and estimated.

Muscle architectur

A typical muscle fiber in an ad
eter in the range of 50 to 70 µ
from a few millimeters to mor
ters. Muscle fibers are generally
plates or aponeuroses, which
transition into the more round
outside the muscle. Skeletal
guished from one another w
length, ratio of fiber length to
the orientation of the fibers rel
of the muscle (Woittiez *et al.*,
Vree, 1987; Otten, 1988, Huijin
et al., 1990). While muscle fib
consistent fiber diameter betw
ferent sizes, the geometric arra