Chapter 3

Physiological actuation systems

INTRODUCTION

This chapter deals with the actuator block of the human motor control scheme (figure 3.1). In muscle, information from the central nervous system is transformed to a mechanical force output. Likewise, one can view muscle as a transformator of chemical energy into mechanical energy and heat. The focus of this chapter is on the functional characteristics of muscle, and less on the morphology and physiology. More information on these aspects is found in the courses *Human Movement Control* and *Functionality of the Locomotor Apparatus*.

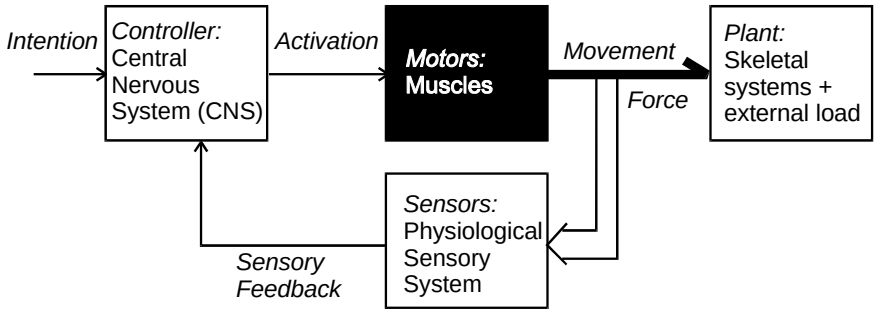

Figure 3.1 Schematic block diagram of the human motor control system. Subject of this chapter is the muscular system.

OBJECTIVES

This chapter will show:

- some morphological and physiological aspects of muscle;
- how these aspects relate to muscle function and properties;
- the common techniques to model muscle properties and function.

A Hill-type model includes a force-velocity relation, a force-length relation and an active state function. A cross-bridge model explains the contraction mechanism of a muscle.

3.1 Muscle morphology and physiology

There are three kinds of muscles: skeletal, heart and smooth muscles. Skeletal muscles make up a major part of the body; it is the prime mover of locomotion. Voluntary nerves control it. When stimulated at a sufficiently high frequency, it can generate a maximal tension, which remains (about) constant in time. In this case, the muscle is tetanized: The activity of the contracting mechanism is thought to be maximal.
Heart muscle is also striated like skeletal muscle, but is never tetanized in its normal function. Instead, it functions in single twitches. Each electrical stimulation pulse evokes one twitch. Until a certain refraction period is passed, another electric stimulation will not evoke a response.
Smooth muscles are not striated, and are not controlled by voluntary nerves. There are many kinds of smooth muscles, for example surrounding blood vessels.
We will focus on skeletal muscles. Since a resting skeletal muscle has quite ordinary

visco-elastic properties, the interesting part is the contraction. Muscles exert force when activated by stimuli from a nerve or artificially by an electrode (Functional Electrical Stimulation, FES). These stimuli start a chain reaction of chemical processes that initiate a connection between the actin filament and opposite myosin filament. Such a connection is addressed as a cross-bridge. The myo-filaments, actin and myosin, are together the smallest functional unit of a muscle, the sarcomere (figure 3.2). In a muscle fiber a large number of sarcomeres are arranged in series. The alignment of sarcomeres in series observed in parallel arranged fibers attributes to the name of striated muscle. Movement is initiated when the myo-filaments slide past one another. A large number of muscle fibers arranged in parallel form a muscle belly. Through aponeuroses (tendon-sheets) and tendons, the muscle fibers are attached to the bone structure at origin and insertion. An aponeurosis is made of tendinous tissue at which fibers are attached at an angle. At one end an aponeurosis turns into a tendon.

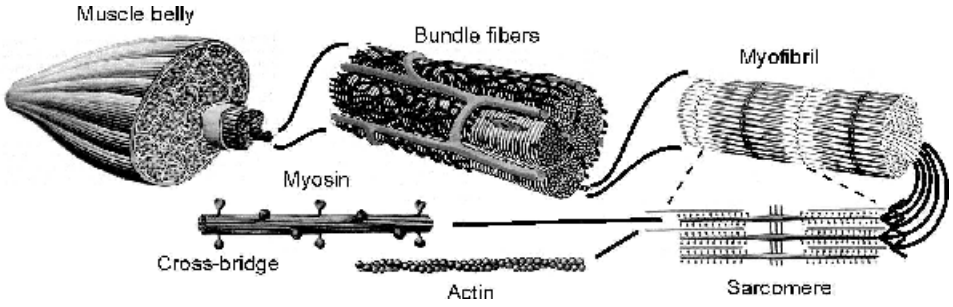

Figure 3.2 Muscle anatomy. Adapted from Gray's anatomy (Warwic and Willems, 1973).

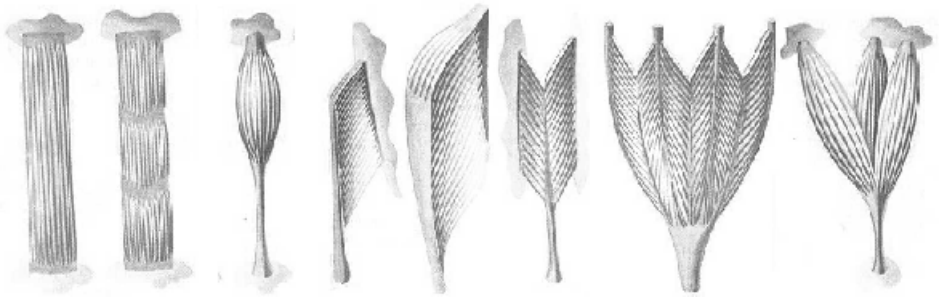

Figure 3.3 Variety of muscle architectures. Adapted from Gray's anatomy (Warwic and Willems, 1973).

The arrangement of the fibers with respect to the line of pull in muscle defines muscle architecture. A schematic representation of a classification in architectural characteristics is given in figure 3.3. The most common muscle architectures are the parallel fibered and the pennate muscles. In parallel fibered muscle is assumed that fibers are arranged along the line of pull of the muscle. In pennate muscle fibers are relatively short compared to the muscle length and have an angle of operation with respect to the muscle line of pull. That so many different muscle architectures exist suggests a relation with the function of the muscle. It can be shown that a pennate muscle, with the same fiber length and volume as a parallel fibered muscle, can exert a larger force at the cost of a smaller contraction velocity.
The active components of a muscle cannot function without the presence of passive mechanical structures. The fibers are arranged in a network of connective tissue, the endomysium (figure 3.4). The muscle is organized in bundles of fibers, each bundle containing over a hundred fibers and surrounded by the perimysium. Finally, the outer

surface of the muscle is shielded by the epimysium. Together with tendon and aponeurosis, the epimysium, perimysium and endomysium make up the passive, visco-elastic properties of the muscle. Other structures in the muscle, such as blood and lymph vessels, motor and sensor nerves, are not considered as contributing to the mechanical behavior.
A muscle fiber is a single cell, ranging in length from a few millimeters to several centimeters, and in diameter from 10 to 100 μm. Unlike other cells, it has multiple nuclei, resulting from a fusion of myoblasts in the embryonic phase.

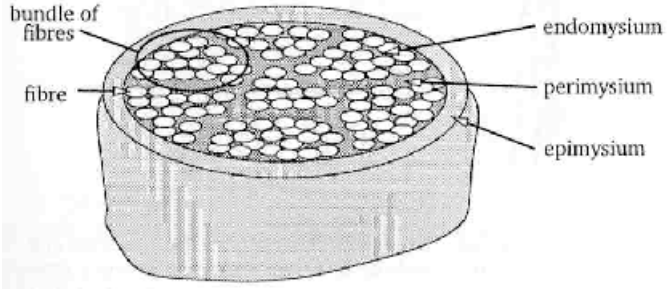

Figure 3.4 Location of the connective tissues epimysium, perimysium and endomysium (adapted from Gielen, 1998)

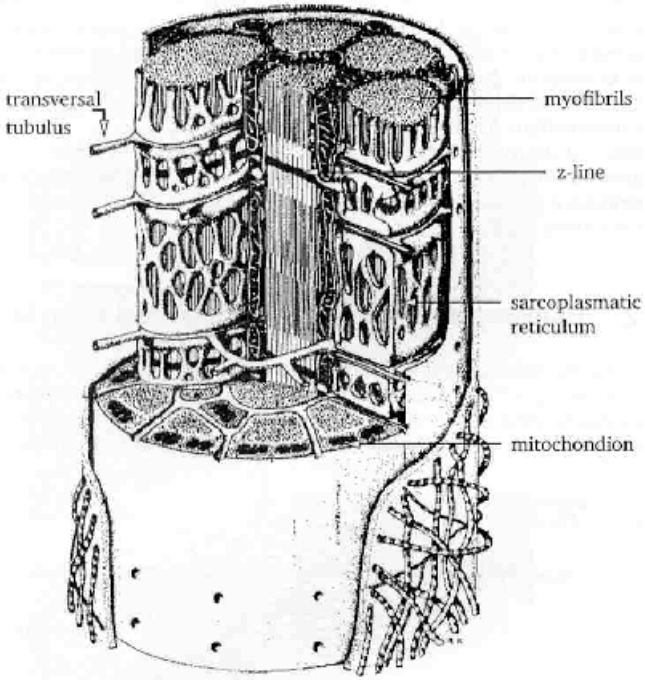

Figure 3.5 Structure of the muscle fiber or cell. Adapted from Ganong (1981).

The cytoplasm of the fiber contains myofibrils that convert chemical (metabolic) energy into mechanical energy, and a sarcotubular system needed for the release of Ca^{2+} ions into the muscle cell (figure 3.5). The sarcoplasmatic reticulum buffers the Ca^{2+} ions and the transversal tubuli (two for each sarcomere) provide the transmission of the action potential from the cell membrane to the sarcoplasmatic reticulum surrounding the myofibrils. A large number of mitochondrions provide the required energy.

The chemical reaction that performs the contraction of muscle takes place between the actin and myosin molecules of a sarcomere. The energy is provided by ATP (adenosinetriphosphate) and controlled by Ca^{2+} ions. The Ca^{2+} ions act as a catalyser: without them, the reaction would be very slow at body temperature.

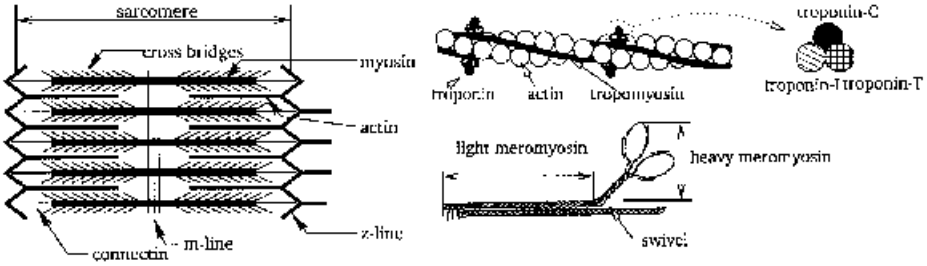

Figure 3.6 Structure of a sarcomere. The heads of the myosin (bottom right) may bind to the troponin sites on the actin filament (top right) to form cross-bridges.

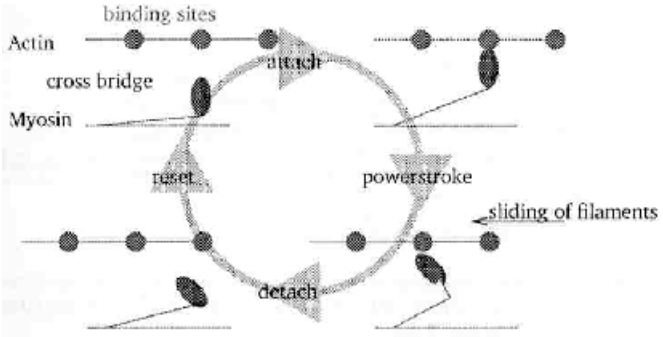

Figure 3.7 Sliding filament model: When the troponin molecules are activated by the Ca^{2+} ions, the myosin can attach to the actin. The myosinhead swivels, producing a power stroke. When detached, the myosin is reset for the next cycle.

Under influence of Ca^{2+}, the heads of the myosin attach to the troponin sites of the actin molecule to form cross-bridges (figures 3.6 and 3.7). The myosinhead then rotates about 45°: Force is generated or, in the absence of an external force, the filaments will slide along each other. With ATP the connection is detached, and the myosinhead is reset in its original position. This detachment costs energy. The myosinhead is then ready for the next cycle.

The force output of a sarcomere is regulated by the Ca^{2+}-concentration. An action potential (moving electrical impulse that locally depolarizes the cell membrane) arrives from the nervous system and moves along the fiber membrane with a speed of 1 to 5 m/s. The tubular system transports the action potential into the muscle fiber and depolarizes the sarcoplasmatic reticulum. This has the effect that the sarcoplasmatic reticulum becomes permeable for Ca^{2+}, the Ca^{2+} is released into the fibrils. The Ca^{2+} is continuously pumped back into the sarcoplasmatic reticulum. So, the action potential results in a short increase of Ca^{2+}-concentration, depending on the fiber type (figure 3.8). For fast fibers this twitch lasts shorter than for slow fibers. When the frequency of the action potentials increases, single twitches are added until no ripple is visible anymore. The constant Ca^{2+}-concentration in the so-called tetanized state results in a constant force output.

The muscle fibers are organized in motor units with about 100 fibers not clustered but distributed over the muscle volume. Each motor unit is excited by a single motor

neuron. When a low force output is required, the small motor units, containing a relatively small number of slow fibers, are excitated first. At larger force levels, larger motor units are excitated as well, until finally the largest motor units containing the fastest fibers are used. This mechanism is called the size principle and reduces fatigue in natural contractions. Fatigue is also minimized by motor unit rotation: After a certain stimulation time, an activated motor unit is allowed some rest and replaced by another motor unit. So, muscle force output can be increased by increasing the activation frequency and by increasing the number of active motor units.

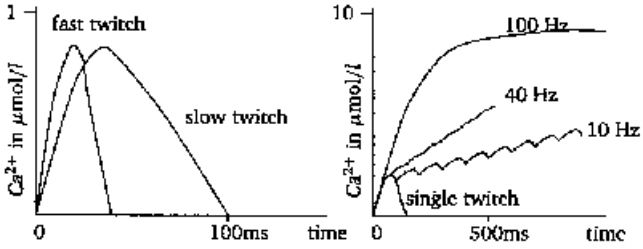

Figure 3.8 *Ca^{2+}-concentration for a single action potential (left) for a pulse train at different frequencies (right).*

3.2 Functional characteristics of a sarcomere

3.2.1 FORCE-VELOCITY RELATION

The functional characteristics of muscle are based on experiments performed by A.V. Hill (1938). He related the force output F of a fully tetanized muscle to the contraction velocity v:

$$(v+b)(F+a) = b(F_0 + a) \tag{3.1}$$

Where a, b and F_0 are constants. Roughly, if we ignore the constants a and b on the left-hand side, equation 4.1 states that the rate of work done, or the rate of energy conversion from chemical reaction, is a constant. This seems reasonable for in the tetanized condition.
Usually, the force-velocity equation is put in a dimensionless form $g(v)$:

$$\begin{aligned} F &= F_0 \frac{1-(v/v_0)}{1+c(v/v_0)} = F_0 \cdot g(v) \\ c &= F_0 / a \\ v_0 &= b \cdot c \end{aligned} \tag{3.2}$$

Where c is a shape factor and v_0 the maximal contraction velocity of the sarcomere when not loaded. It is often assumed that c and v_0 are constant for a certain muscle, only depending on the fiber type and rest-length l_r. For fast fibers, c=0.1 and $v_0 = 8l_r$ per second, for slow fibers c=1 and $v_0 = 2l_r$ per second. However, in equation 3.2 is shown that c and v_0 both depend on the tetanic force F_0.

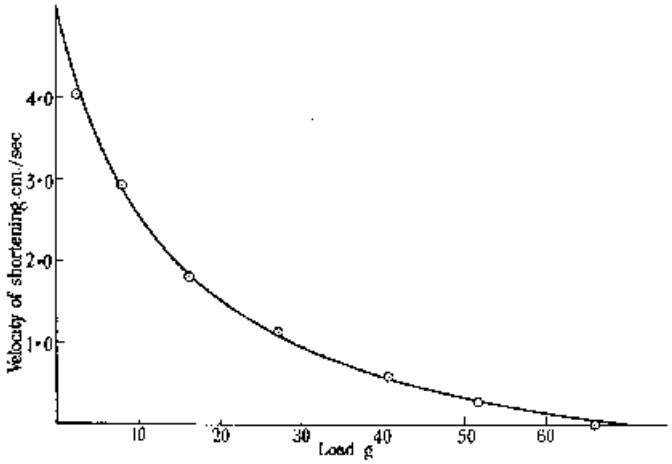

Figure 3.9 Force-velocity curve: Experimental data from Hill (1938) with the fitted equation 3.1.

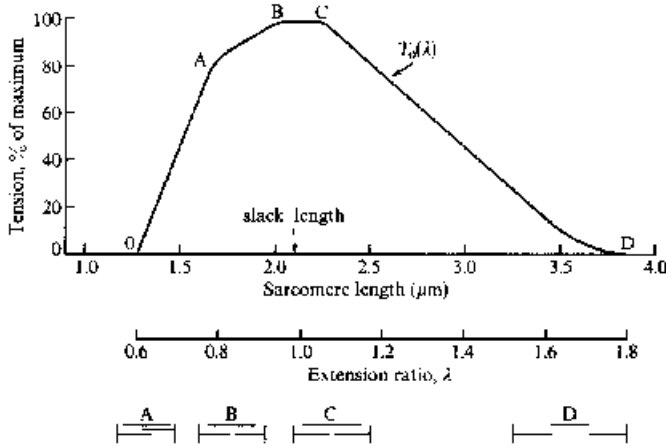

Figure 3.10 The (isometric) force-length curve for frog skeletal muscle fibers. The relative positions of the actin and myosin filaments for A to D are shown at the bottom. From Gordon et al. (1966).

3.2.2 FORCE-LENGTH RELATION

The number of cross-bridges that can be formed depend on the amount of overlap of the actin and myosin filaments (figure 3.10). Therefor, the force F_0 in equation 4.6 depends on the length of the sarcomere. The maximum overlap occurs between B and C in figure 3.10. The part between O and B is called the ascending limb and between C and D the descending limb of the force-length curve. The force-length relation is usually expressed as a dimensionless function *f(l)*:

$$F_0 = F_{\max} \cdot f(l) \tag{3.3}$$

Where $F_{\max}$ is the maximal isometric force at optimum sarcomere length and l is the actual sarcomere length. $F_{\max}$ is considered proportional to the fiber cross-area with a constant fiber stress (estimated by different authors between 10 and 100 N/cm^2).
The total force output of the sarcomere is then:

$$F = F_{max} \cdot f(l) \cdot g(v) \tag{3.4}$$

Note that this equation is valid for a tetanized fiber only. It is assumed that the length and contraction velocity of the sarcomere are independent, this is not necessarily true.

3.3 Muscle models

Numerous models are developed to describe aspects of muscle functioning, with the purpose to describe or predict how muscle behaves under certain conditions. As the knowledge of muscle and the numerical capacities increase, these models tend to increase in complexity as well. However, it is often more fruitful to apply as simple as possible models for each specific problem to develop a clear insight in the underlying mechanisms. For example, to study the effect of the pennation angle on muscle force, planimetric models seem appropriate (Huijing and Woittiez, 1984). To describe the relation between neural activation and the resulting joint rotation, the model should at least describe the force-length and force-velocity characteristics (Winters and Stark, 1985). When the shape of the muscle or the interaction between fibers is considered, finite element-like models are unavoidable (van der Linden, 1998).
We will focus on the second type of model, which seems most appropriate to be applied in human motion control. There are basically two different models developed: a Hill-type model that describes muscle function on a macroscopic level, based on empirical relations; and a cross-bridge model that explains muscle behavior on a microscopic level.

3.3.1 General concepts

When expanding the block diagram of figure 3.1, one should note that several muscles might apply a torque at the same joint, each with a specific moment arm. Also passive structures such as ligaments apply a torque at the joint. It is assumed that the segments model (chapter 2) is extended with a muscle attachment model that defines the anatomical origins, insertions and functional moment arms of muscles and other structures (e.g. Brand et al., 1982).
The muscle model should describe the properties of the sarcomere, i.e. the force-length and force-velocity characteristics (contraction dynamics), and a model to relate the neural input to the muscle activation (activation dynamics). It is assumed these are independent. The muscle and skeletal system block in figure 3.1 then expands to the structure of figure 3.11.

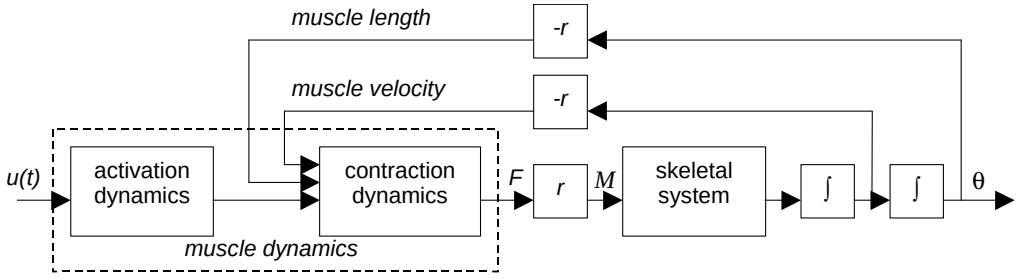

Figure 3.11 Expansion from figure 3.1 of the muscle and skeletal system part of the human motor control system. Input is the neural activation u(t), output a movement represented by θ. Muscle contraction dynamics depends on muscle length and velocity, the muscle moment arm r is used to determine the joint moment of force M from the muscle force F.

3.3.2 HILL-TYPE MODELS

The original model developed by Hill is shown in figure 3.12. The muscle is considered as a large sarcomere (contractile element CE) with some additional passive visco-elastic properties to contribute for tendon and aponeuroses (series element SE) or the connective tissues epimysium, perimysium and endomysium (parallel element PE). It should be no surprise that the equations describing this muscle are based on the equations in section 2:

$$\begin{aligned} F_{mus} &= F_{SE} + F_{PE} \\ F_{SE} &= \frac{F_{CE}}{\cos\alpha} \\ F_{CE} &= F_{\max} \cdot q(t) \cdot f(l) \cdot g(v) \end{aligned} \tag{3.5}$$

Where the pennation angle α is included to account for the pennation effects and *q(t)* is the so-called active state function (see below).

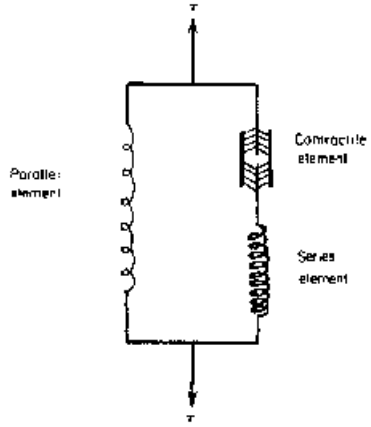

Figure 3.12 Hill muscle model with active properties in the contractile element and passive properties in the series and parallel element.

3.3.2.1 Force-velocity relation

The force-velocity relation *g(v)* is extended to account for all muscle loading situations: The original relation derived by Hill is valid for concentric contractions (i.e. shortening muscle, the muscle generates mechanical energy) only. For eccentric contractions (i.e. lengthening muscle, the muscle dissipates energy) is found that the muscle force can be 1.2 to 1.8 times larger than the isometric force (figure 3.13). The actual shape of the force-velocity relation is defined by a number of muscle-dependent parameters.
It is obvious that the mechanical power output has an optimal value between zero and maximal velocity. At zero velocity (isometric contraction) the mechanical power output is zero. This does not mean that no chemical energy is required, as can easily be verified by carrying a weight.

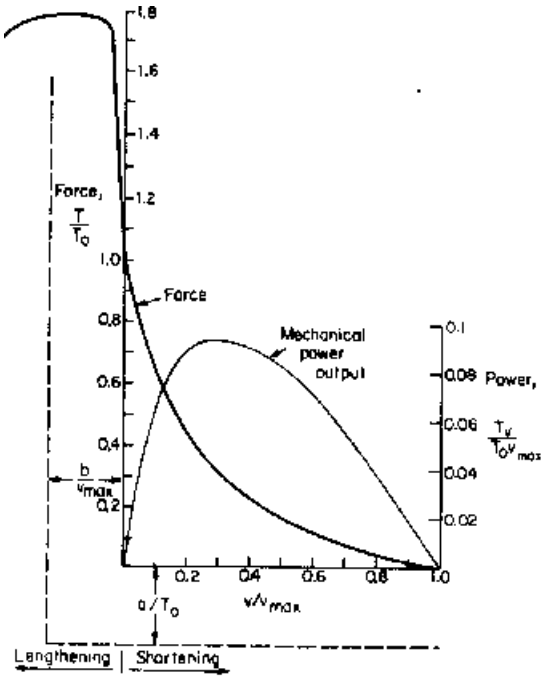

Figure 3.13 The muscle force velocity relation and mechanical power output. From McMahon (1984)

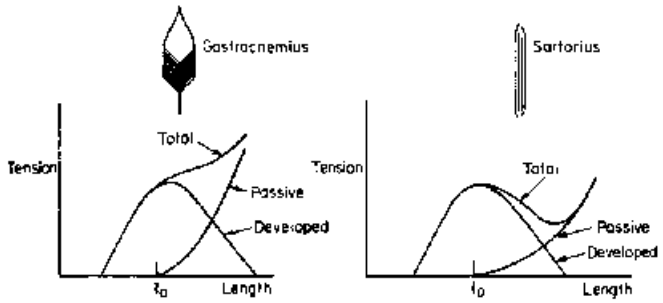

Figure 3.14 Force-length relation for two muscles under isometric conditions. From McMahon (1984).

3.3.2.2 Force-length relation

In the force-length relation, two contributions can be identified (figure 3.14): The passive behavior from the parallel element and the active behavior from the contractile element. The total muscle force is the summation of these two. The passive muscle force is measured by stretching a not-activated muscle. Above a certain length, usually the rest length of the muscle, a force is developed. Below the rest length the muscle does not resist compression. The passive force may depend on the stretching velocity (i.e. visco-elastic instead of elastic), although this is not always included in the model. The total muscle force is measured for a fully tetanized muscle under isometric conditions (otherwise the force-velocity relation would also contribute). From the total and the passive curve the active force-length relation is determined. In principle, this is not a correct procedure, since the passive properties may depend on the activation of the muscle: The muscle geometry in both states is different. The active force is

smoother than for a single sarcomere (see figure 3.10), because the local effects of numerous sarcomeres are averaged for the entire muscle. The active force-length relation is often described by a set of polynomials or an exponential function. It should be noted that the relative contribution of the passive force to the total force might depend on the geometry of the muscle (figure 3.14).

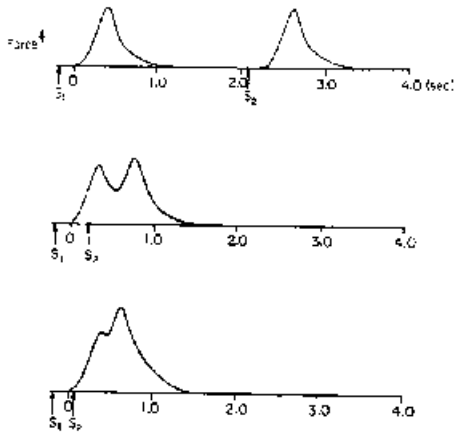

Figure 3.14 Superposition of two twitches with decreasing time-intervals. Note the time delays for excitation and the Ca^{2+}-concentration.

3.3.2.3 Active state function

One needs the active state function to describe other than fully tetanized muscle. It relates the neural input *u(t)* to the muscle activation *q(t)* and is normalized between zero and one. The most simple relation would be a linear one: *q(t)=u(t)*. A force output of half the maximal force would then require a neural input of 0.5. This implicitly assumes that at each force level the muscle is tetanized, which is of course not correct. A single twitch could not be modeled with it (see also figure 3.15).
Winters and Stark (1985) proposed to model the active state with two first-order differential equations:

$$\begin{aligned} &\tau_1 \frac{dN_a}{dt} + N_a = u(t)\,; \\ &\tau_{a/d} \frac{d\psi}{dt} + \psi = N_a\,; \\ &q(t) = h(\psi) \end{aligned} \qquad (3.6)$$

The first equation describes the excitation dynamics: The relation from the neural input to the excitation N_a of the sarcoplasmatic reticulum. The from this process resulting time delay τ_1 is about 30 ms and is shown in figure 3.15 as the time difference between S_1 and the start of the twitch. The second equation describes the Ca^{2+}-concentration in the sarcomere ψ as a function of the excitation. As it takes more time to pump the Ca^{2+} back into the sarcoplasmatic reticulum than to insert it into the sarcomere, the time constant for activation τ_a is shorter than for deactivation τ_d, with magnitudes of about 10 and 50 ms respectively (see figure 3.15). The last equation describes how the Ca^{2+}-concentration relates to the force output of the fiber. It is often assumed that this is a linear relation.

3.3.2.4 Passive properties

The parallel element is already discussed with the force-length relation. The series element shows the same type of behavior, but with other parameters. Usually an exponential function is assumed to describe this behavior.

3.3.2.5 Discussion

Hill-type models are very useful to make a link between the neural system and a resulting force production on and movement of a body segment. However, it is difficult to assess the validity of these models. The model is the result of single, well-defined experiments; the overall performance may deviate from the processes occurring in nature. Some points require attention:

- It is implicitly assume that the time, muscle length and muscle velocity are independent in the formulation of the contractile force (equation 3.5). Most likely these are not, as is discussed before. It is known that the force output is contraction history dependent (Meijer, 1998) and effects like fatigue are not considered.
- The equations for the different elements of the muscle are dependent on a large number of parameters, such as maximal force, maximal contraction velocity, and optimal muscle length. These parameters are not well known for human muscle, and most of the time based on animal experiments. Also, an isolated muscle may behave different from a muscle *in vivo*.
- The parameters are not really constant, otherwise exercise and training would not help. Muscle is a living tissue that adapts to the required performance.
- Hill-type models are not linked to the microscopic mechanisms within a muscle; they are the result of experimental curve fitting.
- The descending limb of the force-length relation can show a negative slope. In a numerical model this relates to a negative stiffness, which may become numerically unstable. In real life this negative stiffness is unlikely to occur. One should keep in mind that the force-length relation is the result of a large number of isometric experiments. For the purpose of modeling these separate points are fitted with a curve.

3.3.3 CROSS-BRIDGE MODELS

Huxley (1957) developed the first cross-bridge model to explain on a molecular level how a muscle produces force. The cross-bridge model is in fact a replacement for the contractile element in figure 3.12 or for the contraction dynamics block in figure 3.11. It assumes that the cross-bridge can be in one of two different states (figure 3.16): Attached or detached[1]. A function *f(x)* describes the rate with which detached cross-bridges can attach, a function *g(x)* describes the rate of the reverse process. As this is a cyclic process, these rates are equivalent to the probabilities that cross-bridges attach or detach.
The functions *f* and *g* depend on a distance *x*, which is the distance that a myosin molecule is stretched. The myosinhead may attach to the troponin sites on the actin filament (see also figure 3.6). It is assumed that a certain range of stretch lengths is available ($0<x<h$) for attachment at unoccupied troponin sites, and with increasing *x* the probability of an attachment *f* increases. Outside this range the cross-bridges can only detach. For a negative *x*, i.e. a compressed myosin filament, the probability of detachment *g* is so large that almost immediately detachment occurs. The force a single cross-bridge can deliver is assumed proportional with stretch length *x*.
Suppose *N* is the total number of available cross-bridges, and *n* is the number of

[1] Later models included more, intermediate states to explain more aspects of muscle behavior.

attached cross-bridges with stretching length *x* and at time *t*. Alternatively, *n(x,t)* can be viewed as the distribution (over *x*) of attached cross-bridges at time *t*. The rate of change of *n(x,t)* in time is proportional to the attachment rate times the number of detached cross-bridges minus the detachment rate times the number of attached cross-bridges:

$$\frac{dn(x,t)}{dt} = \frac{\partial n}{\partial t} + v\frac{\partial n}{\partial x} = f(x)\{N - n(x,t)\} - g(x)n(x,t) \tag{3.7}$$

Where *v* equals the contraction velocity of the sarcomere. All cross-bridges are acting in parallel, so the total sarcomere force equals the sum of all cross-bridge forces:

$$F_{CE} = k\int_{-\infty}^{\infty} x \cdot n(x,t) \cdot dx \tag{3.8}$$

Where *k* is the stiffness modulus of a single cross-bridge. With known sarcomere dimensions and initial conditions the partial differential equation (3.7) can be integrated, although this requires considerable numerical effort. The cross-bridge model predicts the force-velocity curve well: For eccentric contractions, a relatively large number of cross-bridges is at a large stretch length *x*, thus increasing the force. For concentric contractions *x* is relatively small, resulting in a lower force.

Although the cross-bridge models explain how muscle force is developed, they are rather complex for application in large-scale muscle models. Simulation times would be very large. It should also be noted that the shape of the functions *f* and *g* is rather arbitrary, other functions could be found that behave equally well. The model explains the force-velocity relation; the force-length relation has to be added more or less artificially. Also, the activation dynamics that has to be added is more complex. On the other hand, the method can be extended in a natural way to include the chemical (metabolic) energy release.

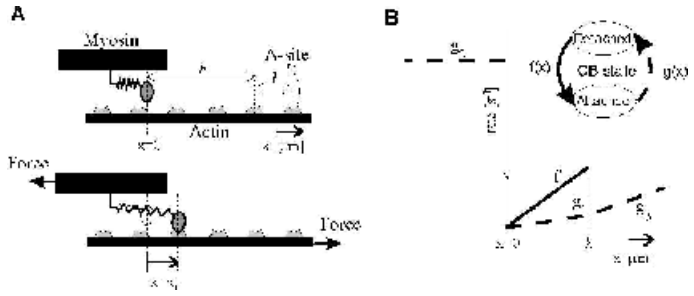

Figure 3.16 Cross-bridge model with the molecular mechanism (a) and cross-bridge states (b).

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