

Fig. 1.10. Hill's force-velocity curve. The shortening part of the curve was calculated from eq. (1.4) with $k = 0.25$. The asymptotes for Hill's hyperbola (broken lines) are parallel to the T/T_0 and v/v_{\max} axes. Near zero shortening velocity, the lengthening part of the curve has a negative slope approximately six times steeper than the shortening part. The externally delivered power was calculated from the product of tension and shortening velocity.

constant load, were first investigated by Fenn and Marsh (1935). They found, as Hill did later (1938), that the relation between developed force and shortening velocity is nonlinear, and therefore that the dashpot element has an acutely velocity-dependent damping.

As the mechanical circuit element suggests, engineering dashpot elements can be made by fitting a piston into a cylinder with enough clearance to allow fluid to escape past the piston as it moves. Since muscle contains a good deal of water, the dashpot model suggests that the viscosity of water ultimately determines the viscous property of active muscle. But water is a Newtonian fluid; its viscosity is not a function of shear rate, provided laminar flow is maintained. A non-Newtonian liquid would have to be postulated in order to explain the velocity-dependent damping in muscle. Furthermore, the viscosity of water varies by only a few percent as its temperature is changed by 10°C in the range near body temperature. By contrast, the damping factor B for

$$v' = (1 - T^r)/(1 + T^r/k), \quad (1.4)$$

where $v' = v/v_{\max}$, $T^r = T/T_0$ and $k = a/T_0 = b/v_{\max}$. For most vertebrate muscles, the curve described by Hill's equation has a similar shape. In fact, for most muscles, k usually lies within the range $0.15 < k < 0.25$.

It is important to note at this point that mechanical power output available from a muscle,

$$\text{Power} = Tv = \frac{v(bT_0 - av)}{v+b}, \quad (1.5)$$

has a maximum when the force and speed are between a third and a quarter their maximal values. The mechanical power output is shown also in fig. 1.10. It is apparent that the speed of shortening controls the rate at which mechanical energy leaves the muscle. The peak in the curve corresponds to about $0.1 T_0 v_{\max}$ watts. Bicycles have gears so that people can take advantage of this. By using the gears they can keep muscle shortening velocity close to the maximum-power point.

Active State

The fact that muscle develops its greatest force when the speed of shortening is zero led A. V. Hill (1922) to suggest that stimulation always brings about development of this maximal force, but that some of the force is dissipated in overcoming an inherent viscous resistance if the muscle is shortening. Thus he proposed representing the contractile element as a pure force generator in parallel with a nonlinear dashpot element (to be defined shortly), as shown in fig. 1.11a. He called the pure force generator the "active state," and proposed that it could develop a force T_0 which rose and then fell after a single electrical stimulation. In a tetanus, this active state force would rise to a constant level numerically equal to the developed isometric tension. The active state force was therefore a function of the length of the contractile element, x_1 , as was the tetanic developed tension.

Dashpot elements develop zero force when they are stationary, but resist length changes with a force $F = B\dot{x}_1$, where B may be either a constant or a function of \dot{x}_1 (fig. 1.11b). (Here and throughout, (\cdot) denotes $d(\cdot)/dt$.) Isotonic contractions, i.e., those in which the muscle is shortening against a

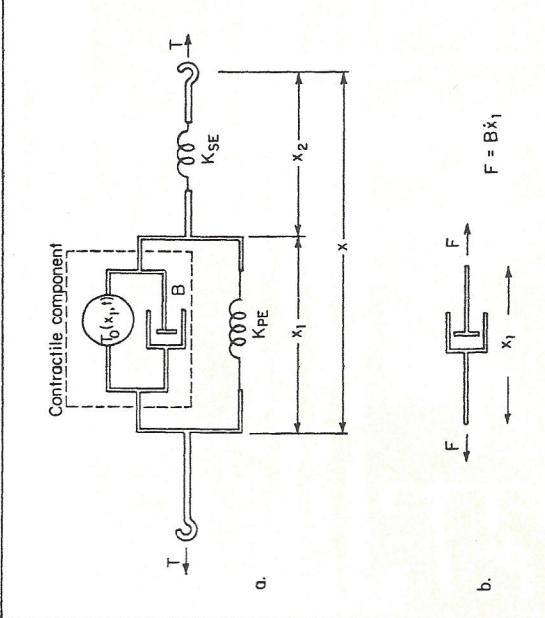


Fig. 1.11. (a) Active state muscle model. The active state $T_0(x_1, t)$ is the tension developed by the force generator in the circle. (b) The dashpot element resists with a force proportional to the velocity.

muscle was shown to be strongly dependent on shortening speed (Hill curve) and temperature (a 10°C rise in temperature increases v_{max} between two and three times). In order for muscle to suffer such a large change in internal viscosity with temperature, it would have to be filled with a viscous fluid with properties similar to castor oil (Gasser and Hill, 1924).

It may have been these thoughts which led Fenn to doubt that anything as simple as a mechanical dashpot was responsible for the force-velocity behavior of muscle. He proposed, correctly, that a biochemical reaction controlled the rate of energy release and therefore the mechanical properties.

Nevertheless, the model of fig. 1.11a has proven enormously useful in calculating the purely mechanical features of skeletal muscle working against a load. If T_0 is specified as a function of time, and if $B(\dot{x}_1)$, $K_{PE}(x_1)$, and $K_{SE}(x_2)$ are given as empirical relations, then the overall length x and tension T of the muscle may be calculated as it works against an arbitrary mechanical system opposing it. In the worked problems at the end of the chapter, this model is used to calculate the two-twitch behavior illustrated in fig. 1.3.

Muscles Active While Lengthening

In ordinary exercises such as running, muscle functions to stop the motion of the body as often as it does to start it. When a load larger than the isometric tetanus tension T_0 is applied to a muscle in a tetanic state of activation, the muscle lengthens at a constant speed. The surprise turns out to be that the

steady speed of lengthening is much smaller than would be expected from an extrapolation of the Hill equation to the negative velocity region. In fact, Katz (1939) found that $-dT/dv$, the negative slope of the force-velocity curve, is about six times greater for slow lengthening than for slow shortening.

Another anomaly is that the muscle "gives," or increases length rapidly, when the load is raised above a certain threshold, as shown in fig. 1.10. This "give" becomes a very large effect, almost as if the muscle had lost its ability to resist stretching, when the load is about $1.8 T_0$ (Katz, 1939).

Time Course of Active State in a Twitch

In 1924, Gasser and Hill proposed applying a sudden stretch to a muscle in the early phase of rising tension following stimulation. This quick change of length by a controlled amount would instantaneously lengthen the series elastic element, thus allowing its force to match the force in the contractile component, hastening the plateau of tetanic tension.

A variation of this idea was applied by Ritchie (1954), who released a muscle to a new, shorter isometric length at different times after a single stimulus. The experiments were carried out in the plateau region of the length-tension curve. The series of curves shown in fig. 1.12 shows how the tension was redeveloped after the muscle reached its new isometric length (the final length was the same for each curve). Each of the curves has a maximum, where the rate of change of tension is zero. If $\dot{T} = 0$, then $\dot{x}_2 = -\dot{x}_1 = 0$ in fig. 1.11. Because $\dot{x}_1 = 0$ the dashpot element contributes no force and $T = T_0$. (Since the muscle has been released to a length below its rest length, the parallel elastic element contributes no force.) Ritchie therefore connected up all the maxima of the tension curves and concluded that this locus of points (broken line in fig. 1.12) corresponds to the falling phase in the time course of the active state following a single stimulation. The duration of the active state was found to depend on the initial length of the muscle, with a shorter initial length corresponding to a more rapid decay.

It was already known that the onset of activity begins very soon after stimulus. Frog muscle at 0°C begins isotonic shortening in less than 20 msec (Hill, 1951). Since it was known that isotonic shortening begins at its full maximum speed, it was concluded that the active state had already reached its full intensity by this time.

To explore more carefully the onset of the active state, Edman (1970) modified Ritchie's method. A single muscle fiber was given a train of stimulations which produced an incompletely fused tetanus. At various times after a particular stimulus, the fiber was released to a new, shorter isometric length, as in Ritchie's experiment. But unlike Ritchie's experiment, the fiber was now stimulated again by the next impulse in the train. The force measured was a series of peaks and troughs (fig. 1.13). The tension recorded at the

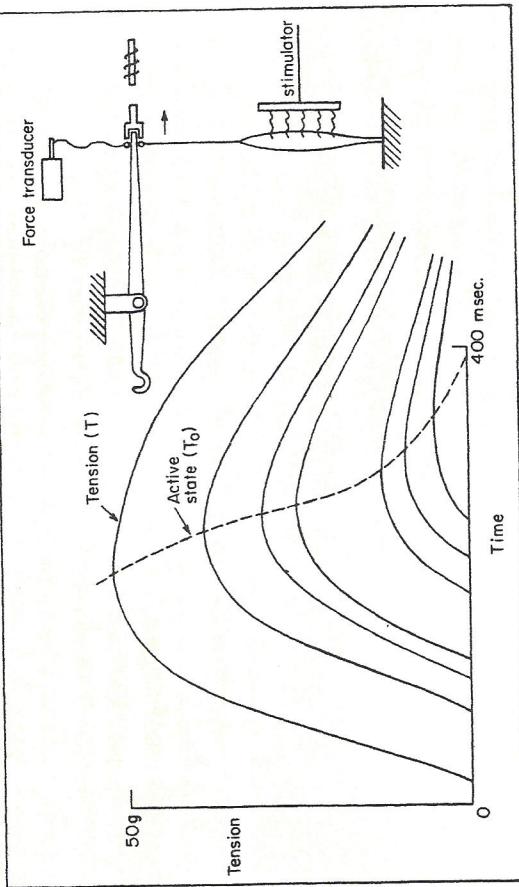


Fig. 1.12. Ritchie's experiment for determining the falling phase of the active state tension during a twitch. A frog sartorius muscle at 0°C is given a single stimulation. The top curve shows the isometric tension as a function of time, when the catch was withdrawn before stimulation. In each of the lower curves, the muscle was released from a particular length an increasing time after stimulation and allowed to shorten against no load until the redundant link to the force transducer became taut. Thereafter, the muscle developed tension isometrically. The maxima of the tension curves define the active state tension, T_0 , as a function of time. Adapted from Ritchie (1954).

bottom of the troughs as well as at the top of the peaks must be understood as the tension produced by the active state element at that moment, for the reasons discussed above. Edman found that after a latency period of about 12 msec from the time of stimulation, the active state rose very rapidly, requiring only 3–4 msec to increase from 25 to 65% of its maximum.

Returning to the model of fig. 1.11, if the parameters K_{SE} , K_{PE} , and B are known, it is possible to calculate the time course of the active state from a single measurement of the isometric twitch tension. Using a parameter estimation method based on the response of a tetanized muscle to a step change in length, Inbar and Adam (1976) calculated the active state curve shown in fig. 1.14, assuming B , K_{SE} , and K_{PE} were all constants. The shape of the curve was not changed importantly when nonlinear parameters were substituted for the linear ones.

Difficulties with the Active State Concept

In a famous review written in 1954 entitled "Facts and Theories About Muscle," Douglas Wilkie begins:

Facts and theories are natural enemies. A theory may succeed for a time in domesticating some facts, but sooner or later inevitably the facts revert to

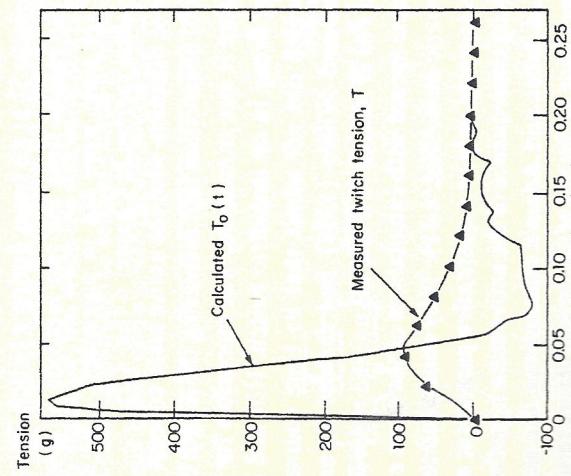


Fig. 1.13. Edman's experiment for determining both the rising and falling portions of the active-state curve. The top curve shows the tension in a series of twitches at constant length for a single fiber of frog semitendinosus muscle at 1°C. Lower curves show the tension when the muscle was released to the same length at various times during the next-to-last contraction cycle. The maxima and minima active-state curves (broken lines) are extrapolated from lines connecting the maxima and minima of the tension curves. These extrapolations intersect near the level of maximum tetanic tension for this fiber. From Edman (1970).

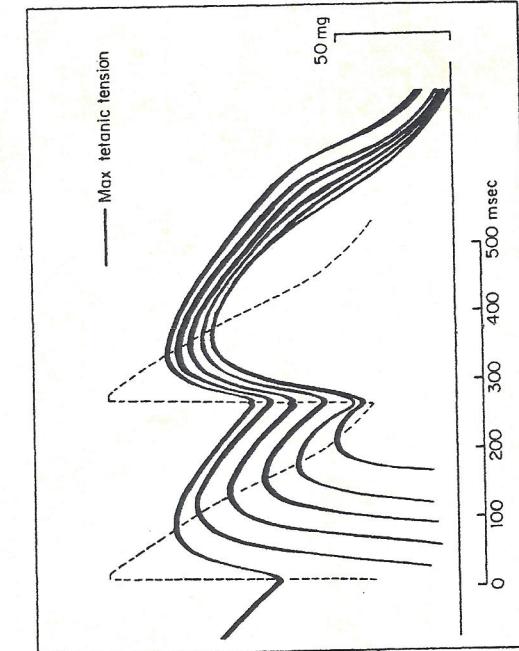


Fig. 1.14. Measured twitch tension and calculated active state $T_0(t)$ for frog gastrocnemius muscle at 24°C. The calculations assumed the model of fig. 1.11 with linear elements determined from measurements of the muscle's response to a change in its length while tetanized. A significant feature of the calculations is the negative T_0 at the end of the falling phase of twitch tension. From Inbar and Adam (1976).

their predatory ways. Theories deserve our sympathy, for they are indispensable in the development of science. They systematize, exposing relationship between facts that seemed unrelated; they establish a scale of values among facts, showing one to be more important than another; they enable us to extrapolate from the known to the unknown, to predict the results of experiments not yet performed; and they suggest which new experiments may be worth attempting. However, theories are dangerous too, for they often function as blinkers instead of spectacles. Misplaced confidence in a theory can effectively prevent us from seeing facts as they really are.

The facts as they really are fell to bickering with the active state theory about ten years after Hill invented it. By this time, Hill (1951) had extended the definition of the active state to include the capacity to shorten, measured as the speed of shortening under a very light load. Jewell and Wilkie (1960) investigated the time course of active state during a twitch, using two procedures which should have given equivalent results.

First, they repeated the Ritchie quick-release method to obtain a measure of muscle T_0 as a function of time following a single stimulus. Then they released the same muscle to shorten under a very light load at various times during the twitch. Employing Hill's extended definition of the active state, the initial velocity of the lightly loaded muscle should have been proportional to the active state tension T_0 at all times during a twitch, but this was found not to be the case. The results from the two methods of determining the time course of the active state were not in agreement. The speed of unloaded shortening is discussed further in Chapter 4 (fig. 4.2).

A second point of difficulty with the active state concept arose from Jewell and Wilkie's (1960) observation that increasing the length of a muscle, even if this was done before it was stimulated, was responsible for an increase in the half-time of force decay in an isometric twitch. Hill had defined the active state in a way which did not admit that its time course might be length dependent. Jewell and Wilkie (1958) had showed in another paper that neither the initial rise of tension in a tetanus nor the redevelopment of tension after a quick release agreed with calculations of the same events based on the active state schematic model (fig. 1.11). They concluded that the active state idea should be interpreted only as a qualitative parameter of muscle activity.

Another alarming consequence of the active state hypothesis is shown in fig. 1.14. According to Inbar and Adam's calculations, the active state must go through a negative phase at the end of the twitch before returning to zero. This result was found to be independent of the details of the model parameters employed. It is hard to imagine what a negative force generator would mean physically in this context.

Summary and Conclusions

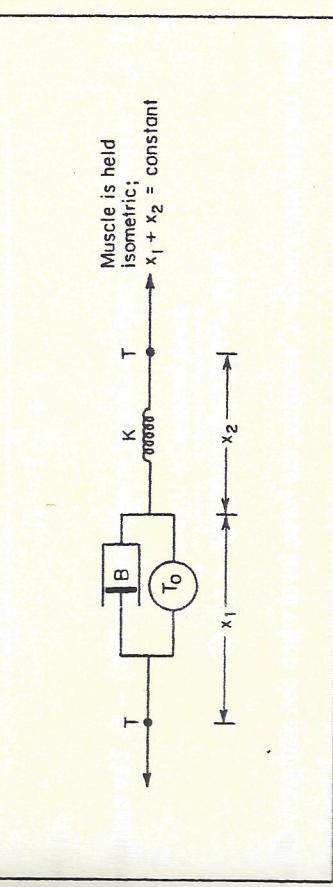
The main purpose of this chapter has been to introduce the schematic diagram of fig. 1.11, whose parameters can be obtained empirically from mechanical experiments. The force-length characteristics of the parallel elastic spring K_{PE} and active state force generator T_0 can be found from passive and tetanic force-length experiments, respectively. The nature of the series elastic element K_{SE} and the dashpot element $B(\dot{x}_1)$ is determined from the initial (instantaneous) length change and early slope of the length record in the quick-release experiments. Experiments involving a quick length change can be used to establish the time course of the active state tension, T_0 , during a twitch.

I should emphasize that no one has believed in fig. 1.11 as a comprehensive representation of the way muscle actually works since about 1924, for some of the reasons touched on above, and for a variety of reasons still to be mentioned. Most notable among the failures of this "viscoelastic" model is its inability to account for the Fenn effect, a major subject of the next chapter.

Solved Problems

Problem 1

In real muscle, the peak tension developed by two twitches sufficiently far apart is practically the same, but when the second twitch is given immediately after the first, the peak tension of the second twitch is higher. Take the linear three-element model below and answer the following questions:



- (a) Assume the initial tension is zero. Then the pure force generator develops tension according to the schedule shown below. Derive the differential equation describing the system and solve it to obtain the force T as a function of time for $0 \leq t \leq 2C + A$.