nonlinearly on the velocity. Figure 1.4(c) is a diagram of the Hill model incorporating this internal decomposition of the *CE*.

Combining the dynamics of the *CE*, as expressed by the force-velocity relation *Eq. 1.8*, with the force-extension characteristics of the *SE* one may write a differential equation governing the basic Hill model of muscle

$$\dot{L} = \dot{P}/K(P) - V(P; P_0) \tag{1.11}$$

where $K(P)$ is the force-dependent tangent stiffness of the *SE*, L is the overall muscle length, and the superposed dots denote time differentiation.

In addition to the force-velocity relation Hill introduced another important concept - that of the *active state*. At first this idea was used in a qualita-qualitative sense to mean the internal state of a stimulated muscle which is capable of producing force and motion. But gradually the term was assigned quantitative meaning by defining it to be the force that a muscle exerted when the *CE* was neither shortening nor lengthening – that is, when the *CE* velocity is zero (Hill, 1949). Therefore, according to *Eq. 1.8*, the active state is to be identified with the internal force, P_0. This force was assumed to depend on the history of stimulation, and considerable effort was expended to measure its time course, particularly in the experimentally important cases of isometric tetanus and twitch. A discussion of these experiments may be found in Aidley (1971) and McMahon (1984) and they revealed several quantitative difficulties with the notion of active state, which suggested that this variable should best be regarded as only a qualitative measure of muscular activity. Indeed Aidley (1971) has gone so far as to state "we must conclude that the concept of the 'active state' leads more to confusion than enlightenment." Nevertheless, this concept remains an integral part of the Hill muscle model, and is generally accepted by bioengineers as providing at least an approximate measure of muscular activation [see also Chapter 5 (Winters)].

The experiments that led to the Hill model were quite restricted, involving muscle that shortened (not lengthened) at maximal (not partial) activation over a limited range of muscle lengths near the "optimal" length at which the maximal isometric force is generated. Successive investigators have attempted to generalize the model so that it is useful under circumstances more representative of those encountered in-vivo. A simple enhancement is to add a third "parallel elastic" (*PE*) element [Figure 1.4(c)] to model the fact that passive, unactivated muscle can resist stretch; it has been found, however, that this element usually generates substantial forces only at long muscle lengths, and is often ignored in simulations. It has long been known that isometric muscle under constant stimulation produces an active force that is maximal at a length L_0 close to the mean length of the muscle in the body – the optimal length mentioned above – and decreases at shorter and longer lengths (Ramsey and Street, 1940); this behavior illustrated schematically in Figure 1.6(a), was elegantly explained in terms of the cross-bridge theory by Gordon, Huxley, and Julian (1966). In order to incorporate this tension–length characteristic into the Hill model it is assumed that the active state, P_0, depends on *CE* length as well as stimulation history [Ritchie and Wilkie, 1958; see also Chapter 5 (Winters)].

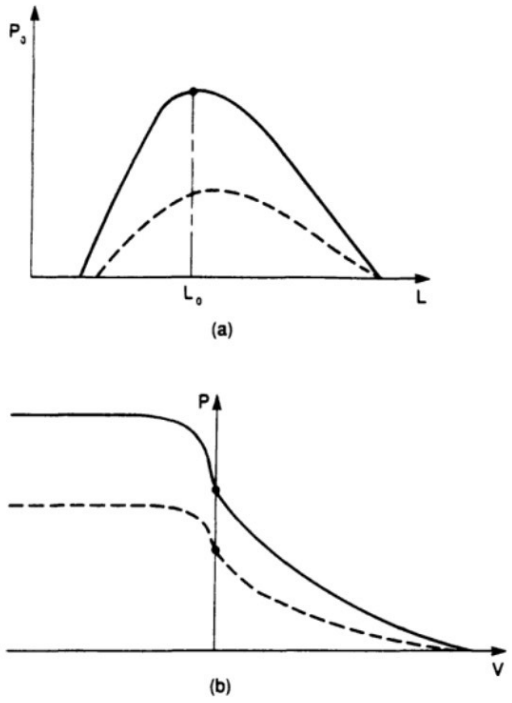

Figure 1.6: ***a)*** Solid curve: isometric force, P_0, versus muscle length, L, at maximal stimulation. The force is maximum at the "optimal" muscle length, L_0. Dashed curve: relation between P_0 and L at constant submaximal stimulation. ***b)*** Force-velocity relations of the *CE* for both positive and negative shortening velocities, V. The upper solid curve is for constant maximal stimulation, and the dashed curve is for constant submaximal stimulation.