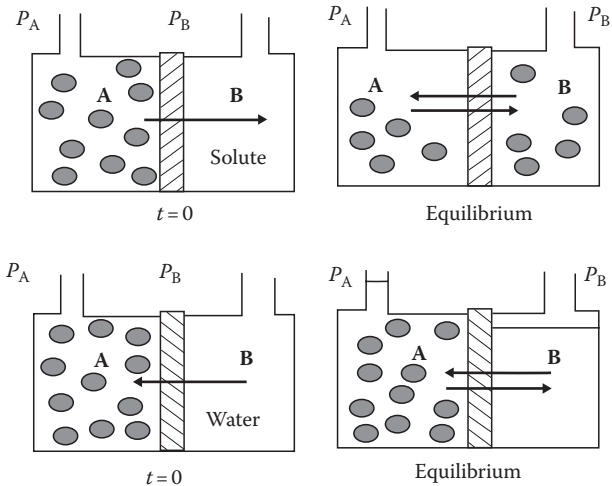

FIGURE 3.1

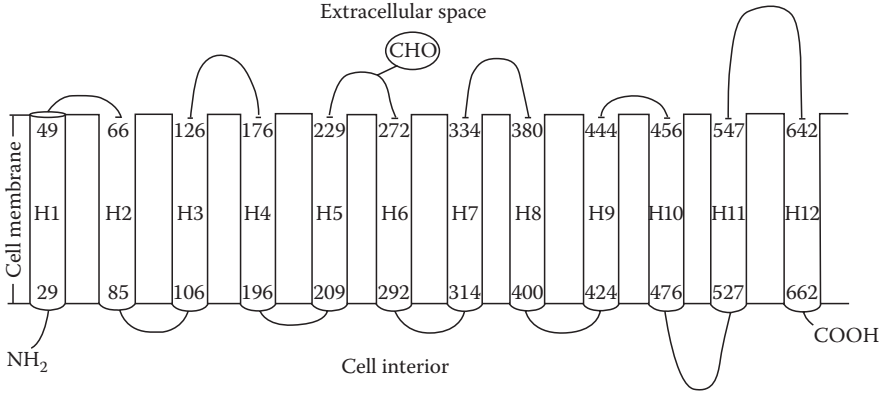

FIGURE 3.5

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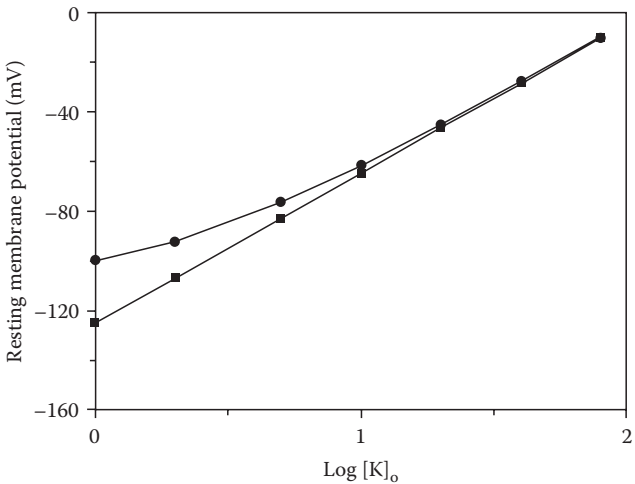

FIGURE 3.7

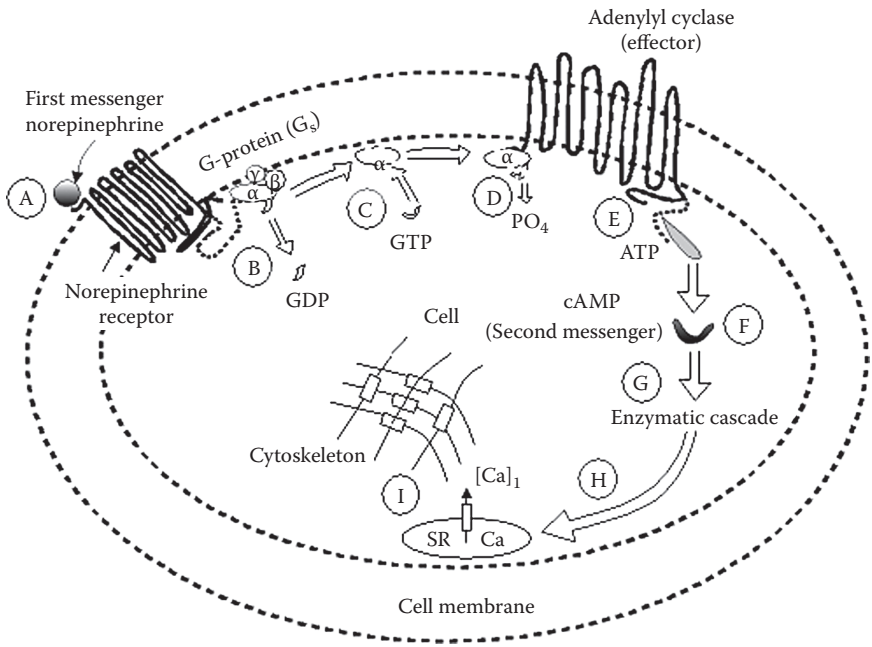

FIGURE 3.8

Ⓐ The first messenger (e.g., NE, histamine) binds to its receptor binding site on the extracellular surface of the membrane. Ⓑ This changes the 3-D configuration of the receptor and signals the G-protein to exchange GDP for *guanosine triphosphate* (GTP), which activates the G-protein. Ⓒ The G-protein dissociates freeing the α subunit, which then diffuses along the membrane. A GTP unit attaches to the vacant GDP site. Ⓓ The α subunit then interacts with an effector (e.g., adenylyl cyclase), activating the effector. After a few seconds, the α subunit converts GTP to GDP, thereby inactivating itself. The inactivated α subunit will then reassociate with a nearby β–γ complex. Ⓔ The effector adenylyl cyclase converts ATP to the second messenger, c-AMP. Ⓕ cAMP then initiates a cascade of enzymatic reactions. Ⓖ The details of these reactions have not, as yet, been worked out in cardiac muscle but may involve the second messengers IP_3 and diacylglyceride (DAG), as well as protein kinases. Ⓗ The net result of the enzymatic cascade is to open Ca channels in the SR, which releases Ca into the cytoplasm

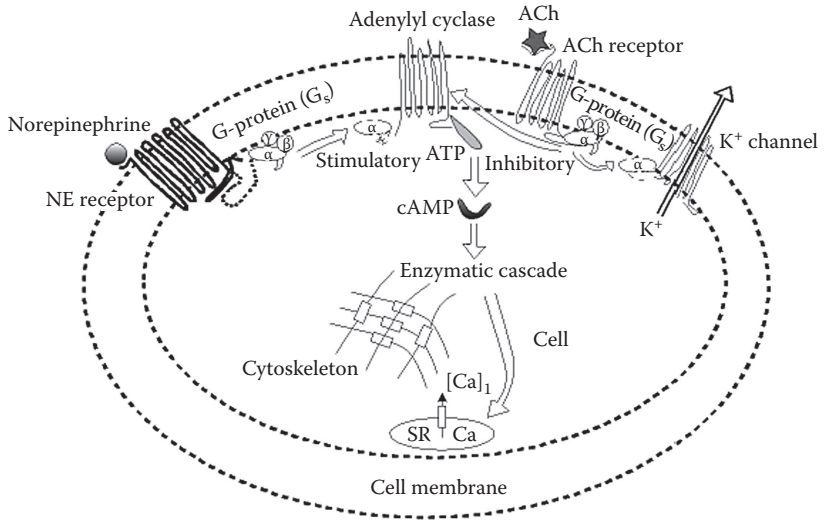

FIGURE 3.9

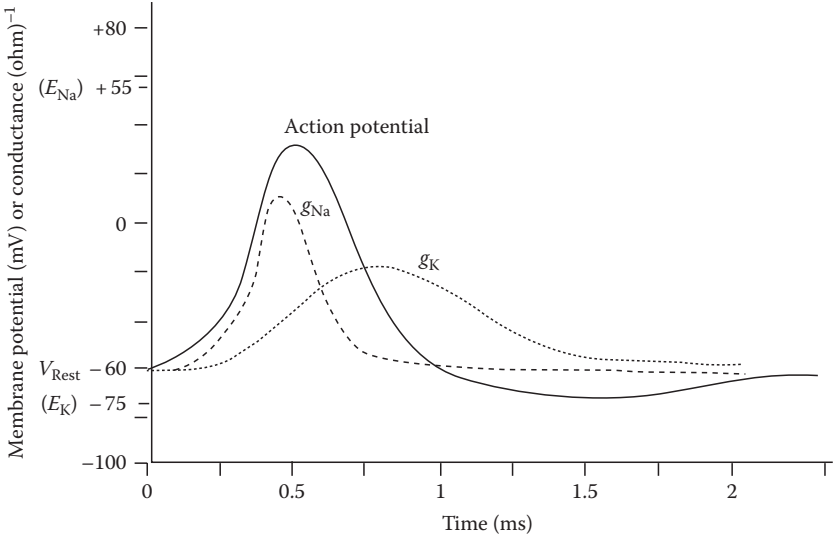

FIGURE 3.11

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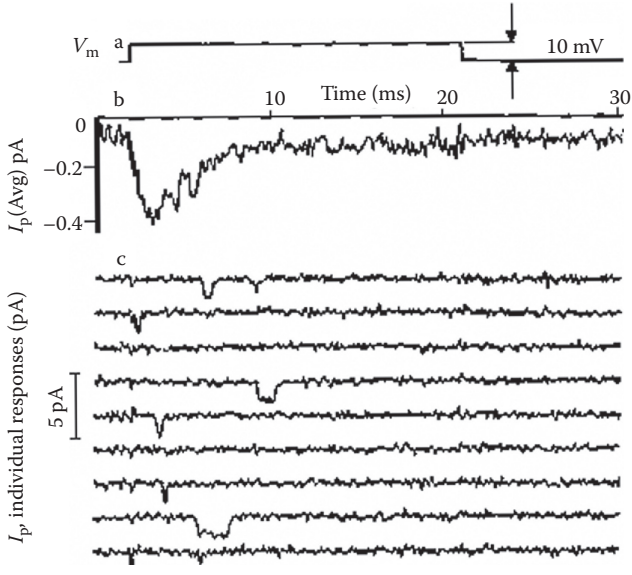

FIGURE 3.12

Typical patch clamp data. (a) Patch electrode recordings of currents recorded in rat muscle membranes in response to a 10 mV depolarization. (b) The average response to 300 individual recordings. The record-

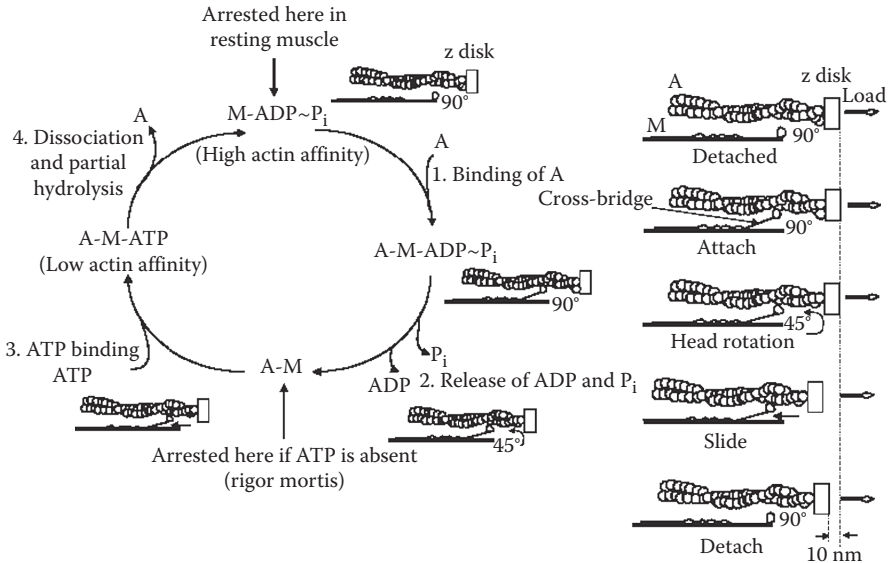

FIGURE 3.14

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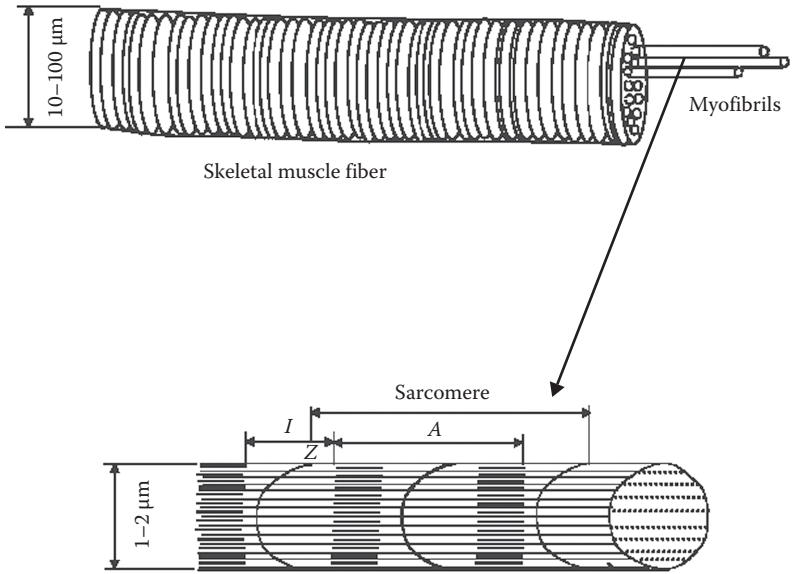


FIGURE 3.13

003x013.eps

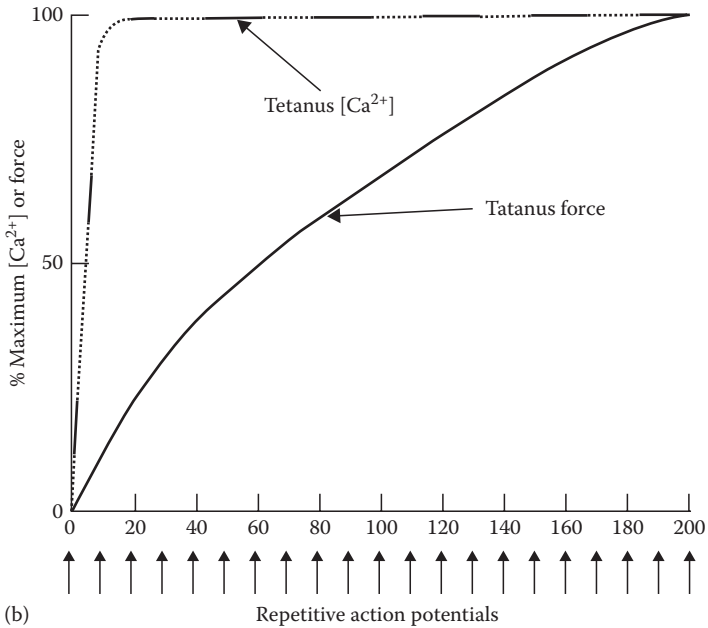


FIGURE 3.15 (continued)

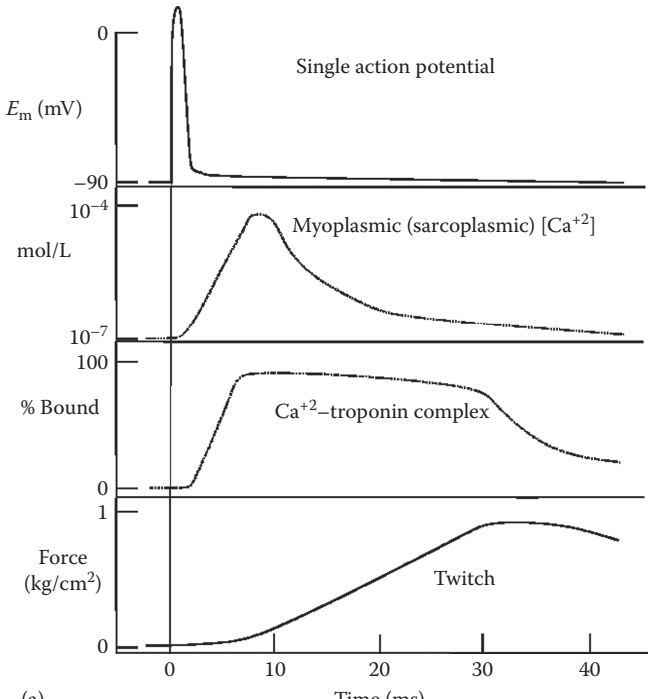


FIGURE 3.15

(a) Timing of events in the generation of a twitch force. Upper figure shows a single action potential. Middle two figures show cytosolic free calcium concentration and formation of Ca–troponin complex.

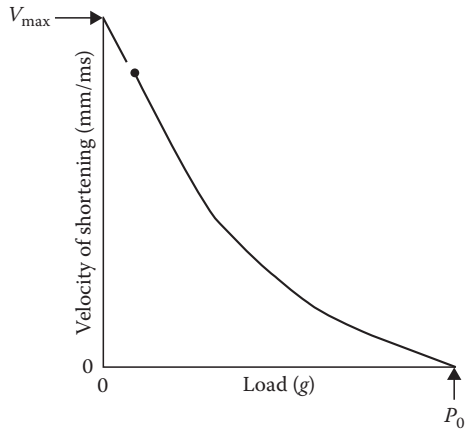


FIGURE 3.17

Relationship between load and velocity of shortening. At some load, P_0, the muscle cannot develop enough force (fiber stress) to lift the load and cannot shorten. At zero load, the velocity of shortening is maximal for a given muscle and *inotropic* state. V_{max} (the velocity of shortening at zero load) is a measure of the inherent ability of the muscle to develop force, called the *contractility* of the muscle.

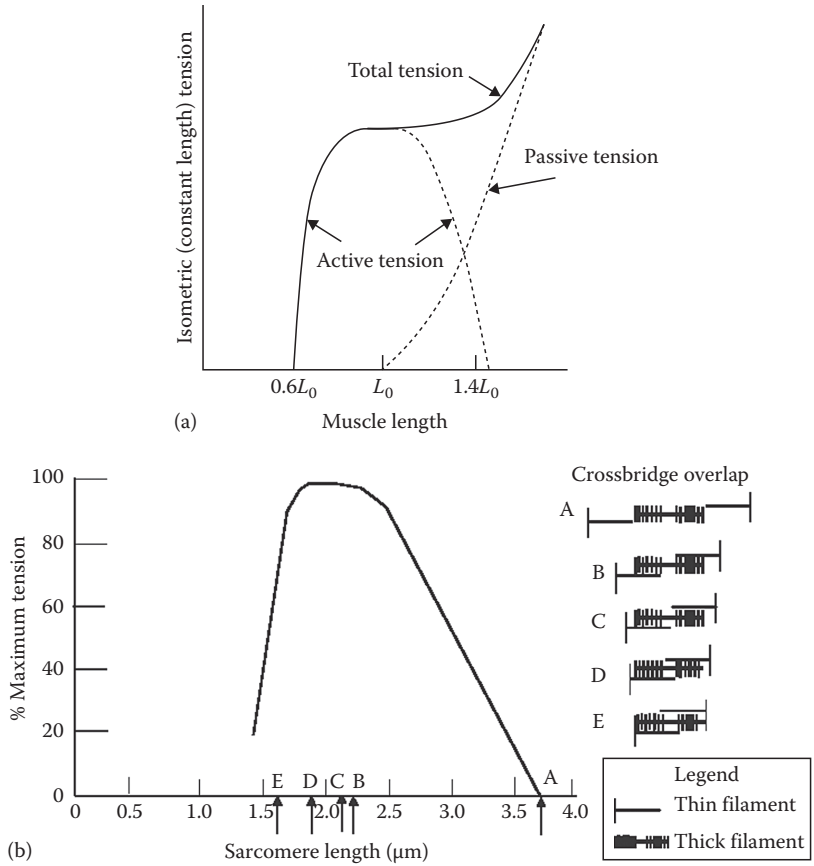


FIGURE 3.16

(a) When a muscle is stretched from some initial length, L_0, without being stimulated to contract, it follows the passive length–tension (stress) curve and acts like any distensible material (e.g., a rubber band). When the muscle is stretched and also stimulated to contract, it develops force (tension) along the active tension curve (see mechanism in Figure 3.16b). The total force developed is the sum of these two forces. (b) Force development in skeletal muscle. Within the physiological range, force development depends

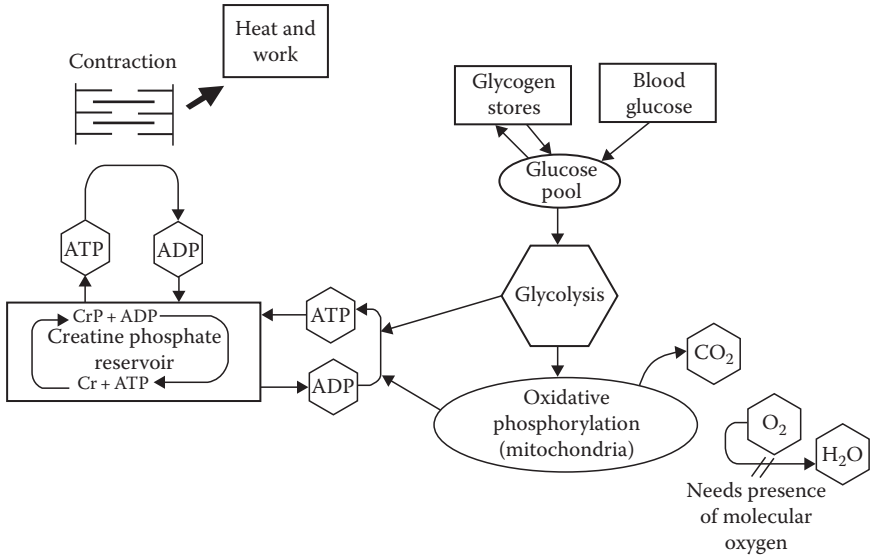
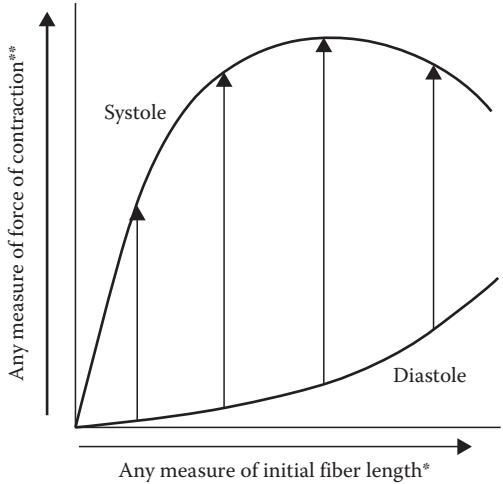


FIGURE 3.18



*End-diastolic volume, end-diastolic pressure
**Stroke volume, cardiac output

FIGURE 3.19

Relationship between force of contraction and stretch of the ventricle prior to contraction. When the ventricle is passively stretched (e.g., by an increase in venous return due to a transfusion or exercising skeletal muscle) over the physiological range, it responds with a larger force of contraction up to a certain point. This mechanism, called the *Frank–Starling* mechanism or *heterometric autoregulation*, is independent of neural control. It allows the heart to handle increases in venous return without invoking neural reflexes and is an important adaptive mechanism in the failing heart. The heart actually operates on one of a family of Frank–Starling curves, depending on its *inotropic* state (see Chapter 5).

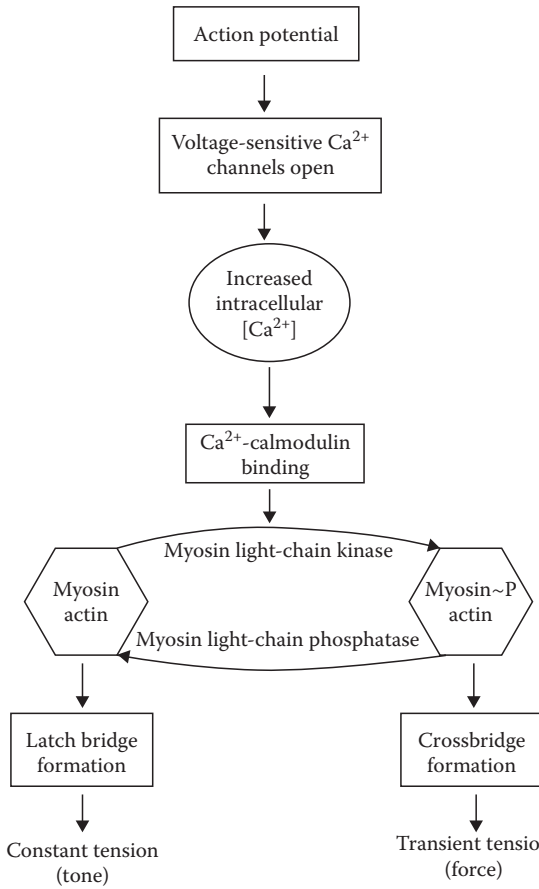


FIGURE 3.20

Actin–myosin crossbridge formation in smooth muscle, showing latch-bridge formation and formation of transient force. All larger blood vessels (arteries, arterioles, veins, venules) have smooth muscle cells, either embedded in their walls or nearby in the interstitial space. Latch-bridge formation allows these muscle cells to provide structural support for the blood vessel by providing a constant basal level of vasoconstriction, called *tone*. The myosin content is much lower and the actin content higher than in