Chapter 11 – Skeletal, Cardiac and Smooth Muscle Contraction

Objectives

Given the synopsis in this chapter, competence in each objective will be demonstrated by writing short essays, drawing diagrams, and responding to multiple choices or matching questions, at the level of 85% or greater proficiency for each student.

A. To explain the anatomical organization of skeletal muscle.
B. To explain the sliding filament mechanism of contraction of skeletal muscle.
C. To explain the mechanism for activation of skeletal muscle contraction.
D. To explain how different rates of activation influence contraction and tension development in different types of skeletal muscle.
E. To explain the anatomical organization of cardiac muscle.
F. To compare and contrast the contraction of cardiac muscle and the contraction of skeletal muscle.
G. To explain the mechanisms for activation and modulation of cardiac muscle contraction.
H. To explain the anatomical organization of smooth muscle.
I. To compare and contrast the contraction of smooth muscle and the contraction of cardiac muscle.
J. To explain the mechanisms for activation and modulation of smooth muscle contraction

Muscle can be divided into three types: skeletal, cardiac, and smooth. Skeletal muscle, of course, is the muscle responsible for moving the skeleton, providing expression, and producing behavior. Cardiac Muscle is the muscle found in the heart. Smooth muscle is the muscle found in the internal organs, especially blood vessels, the gastrointestinal tract, the bronchial tree, the urinary tract, and other related structures. Although these three types of muscle have many common features, there are critical differences. Skeletal muscle will be considered first and used as the foundation upon which to compare and contrast cardiac and smooth muscle.

Skeletal Muscle

General anatomical organization of skeletal muscle

Skeletal muscle cells are long, spaghetti shaped, multinucleated, and arranged in parallel. The cells are as long as the muscle. These cells are sometimes referred to as **extrafusal** muscle cells.

- The cells are connected side by side with fibrous connective tissue called the endomysium.
- Groups of skeletal muscle cells are bundled together by more fibrous connective tissue called the perimysium to form muscle fascicles.
- Muscle fascicles are bundled together by even more fibrous connective tissue called the epimysium to form a muscle.

Intermingled with the long skeletal muscle cells are short cells with single nuclei called **intrafusal** muscle cells.

- Intrafusal muscle cells are surrounded by sensory nerve receptors that are responsible for detecting the degree of skeletal muscle stretch.
- Intrafusal muscle cells are attached in parallel to groups of skeletal muscle cells by the endomysium.

Organization of individual skeletal muscle cells (muscle fibers)

Each skeletal muscle cell, whether extrafusal or intrafusal, contains a **motor end plate** controlled by motor neurons, together forming what is referred to as a neuromuscular junction. A photomicrograph of a motor end plate and its innervation by a motor neuron is shown in Figure 11.1.

Skeletal muscle cells also contain specialized contractile proteins that are arranged in a pattern that give the cells a striated or striped appearance. A photomicrograph of a skeletal muscle with this striped appearance is shown in Figure 11.2. The striped appearance is due the alternation of thin and thick contractile proteins, forming what are referred to as A-bands and I-bands.

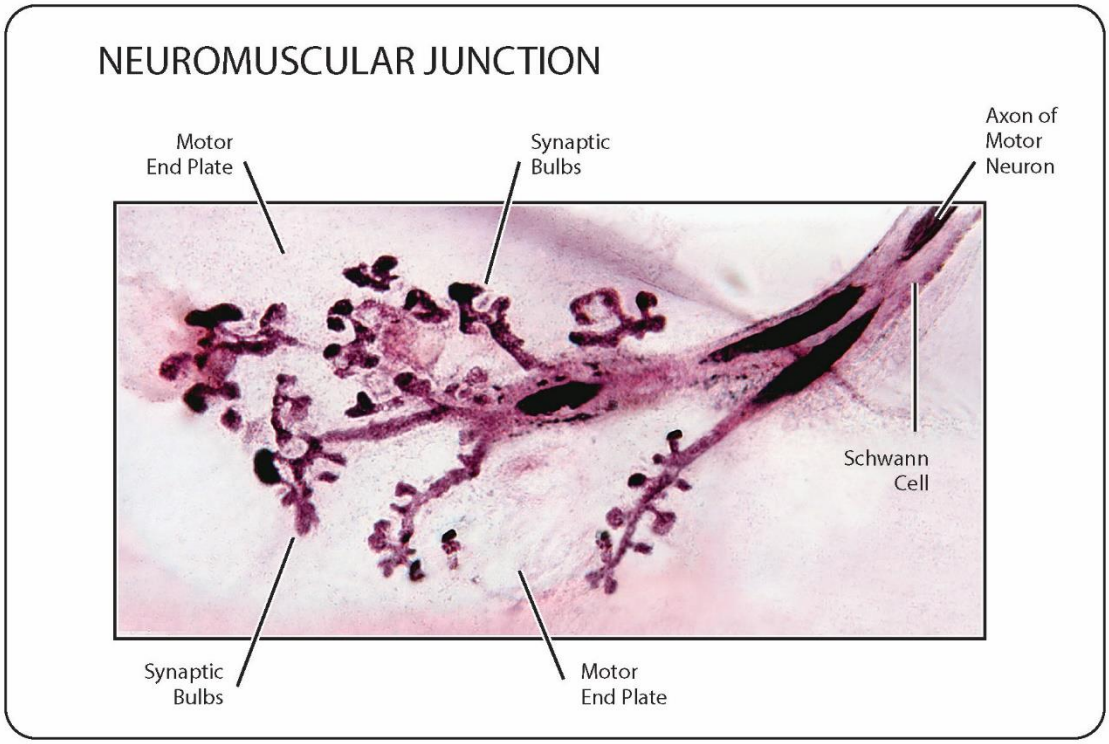


Figure 11.1 © 2018 David G. Ward, Ph.D.

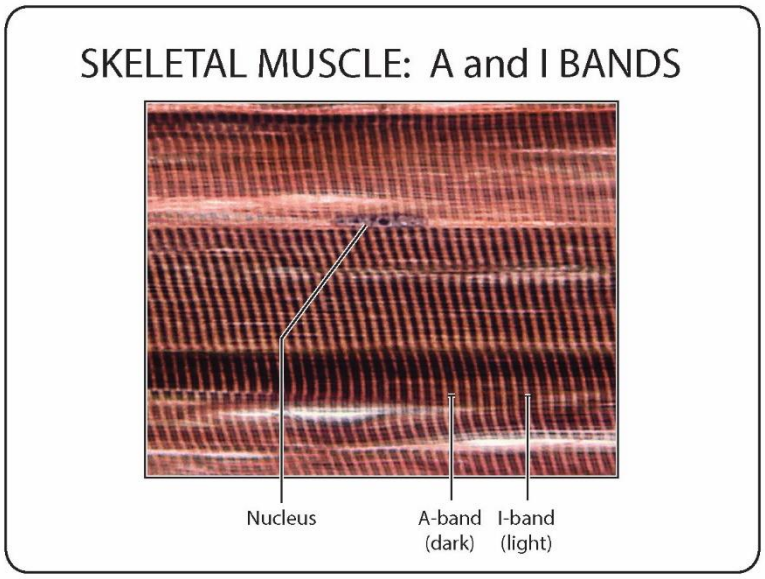


Figure 11.2 © 2018 David G. Ward, Ph.D.

- The location of the thick contractile proteins is seen as a dark stripe called the **A-band**. A major protein is **myosin** (also see Figure 11.4 later).
- The location of the thin contractile proteins is seen in part as a light stripe called the **I-band**. A major protein is **actin** (also see Figure 11.4 later).
- The protein sequence actin-myosin-myosin-actin forms a functional unit for muscle contraction called the **sarcomere**.

Long parallel groups of the thin and thick proteins are called **myofibrils**. Sarcomeres extend from one end of a myofibril to the other. Most of the volume of skeletal muscles cells is devoted to the myofibrils.

As shown in Figure 11.3, a smooth endoplasmic reticulum called the **sarcoplasmic reticulum (SR)** surrounds the myofibrils. Inward extensions of the sarcolemma (cell membrane of a muscle cell), called **transverse tubules,** come close to the sarcoplasmic reticulum. The transverse tubules conduct action potentials from the sarcolemma which play a central role in the release of calcium that is stored in the sarcoplasmic reticulum by calcium ion pumps.

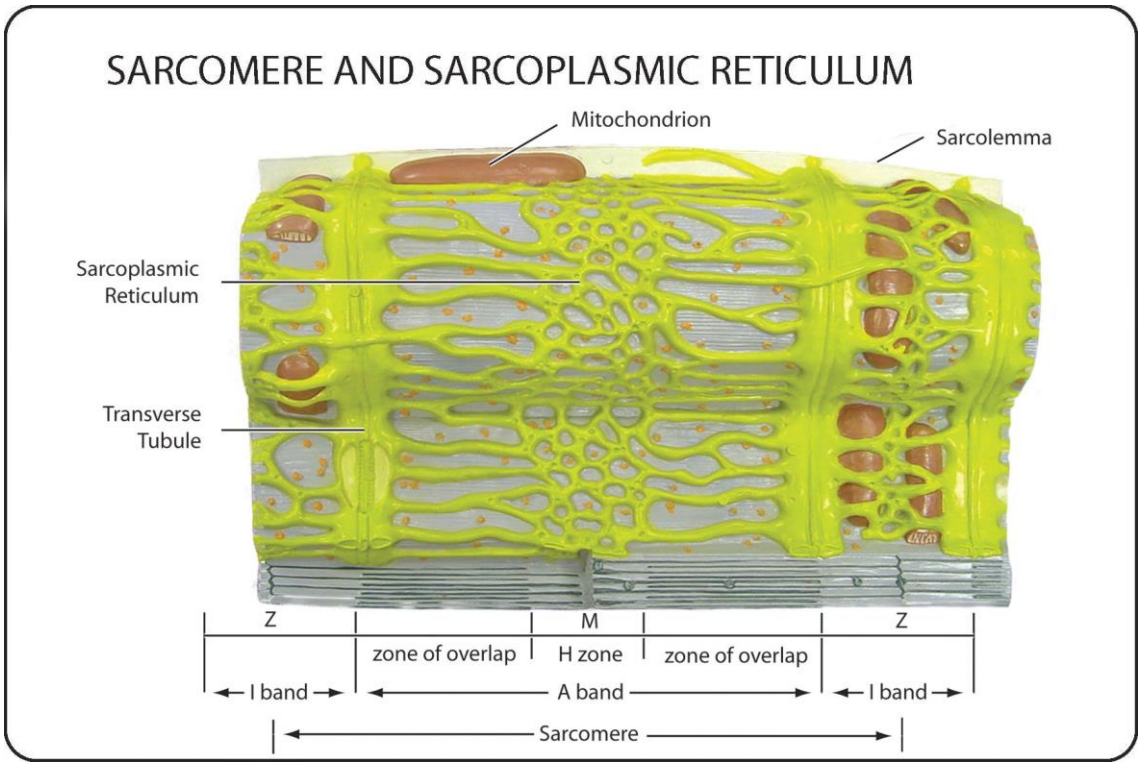


Figure 11.3 © 2011 David G. Ward, Ph.D.

Figure 11.3 also shows the relationship among the transverse tubules, the sarcoplasmic reticulum and the I-bands and A-bands. A sarcomere extends from the center of one I-band to the next.

- The center of each I-band is called the Z-line.
- A pair of transverse tubules flanks each Z-line.
- The center of each A-band is called the M-line.
- Most of the sarcoplasmic reticulum is found in an A-band.

The organization of the sarcomere and the relationship among the I-bands and A-bands and the actin proteins and myosin proteins is shown in Figure 11.4.

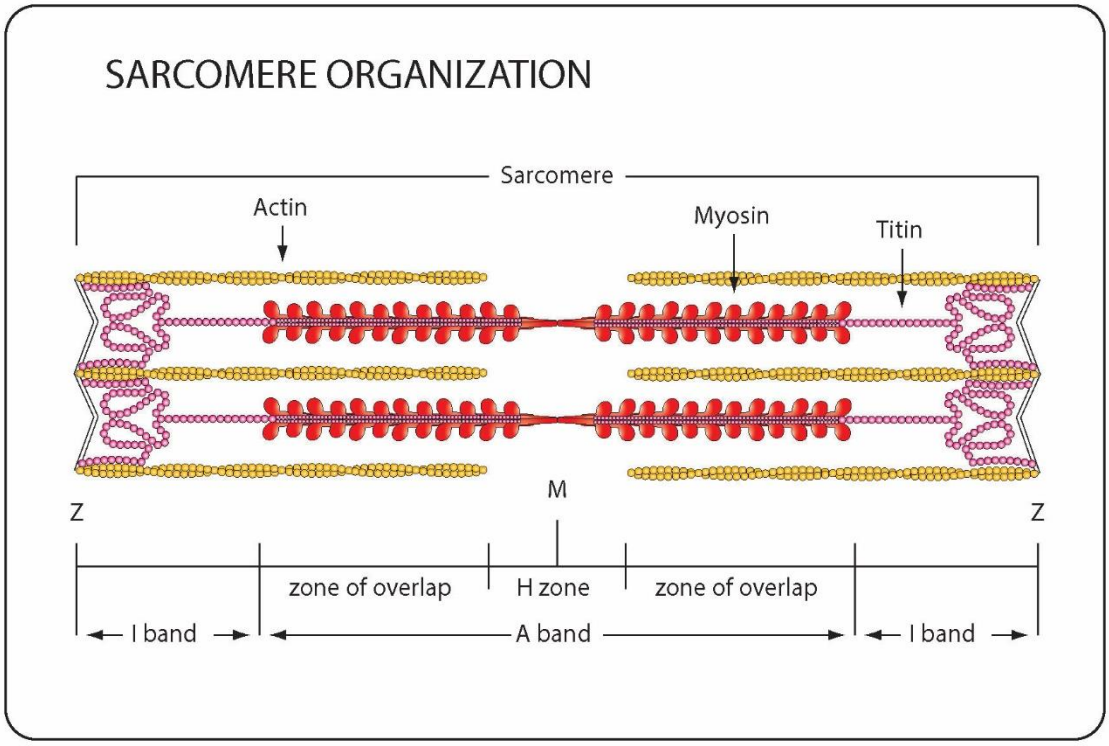


Figure 11.4 © 2018 David G. Ward, Ph.D.

- A sarcomere extends longitudinally from one Z-line to the next.
- Adjacent actin proteins connect at the Z-line in the center of each I-band.
- **Titin** proteins attach at the Z-lines in order to anchor the myosin proteins.
- Adjacent myosin proteins connect at the M-line in the center of each A-band.
- The actin and myosin proteins overlap in a region called the zone of overlap.
- The center of the A-band where myosin does not overlap with actin is called the H zone.

Figures 11.4 and 11.6 show the general structure of the actin and myosin chains.

- Actin is composed of chains of actin molecules wound around each other.
    - Each actin molecule contains binding sites for myosin.
    - Tropomyosin, held in place by troponin, runs parallel to the actin chains and covers the binding sites (not shown in Figure 11.4).
- Myosin is composed of a helical array of myosin molecules, each with an enlarged head often called a cross bridge.
    - The cross bridges are cocked and held close to the array by ATP.

Sliding filament mechanism of contraction

An overview of the mechanism responsible for muscle contraction is illustrated in Figures 11.5 and 11.6 and summarized below.

1) When actin is covered by tropomyosin, myosin cross bridges cannot bind to the actin molecules, and the muscle is relaxed.
2) However, when tropomyosin is moved and binding sites on actin are exposed, myosin cross bridges can and will bind to the actin molecules.
3) When the myosin cross bridges bind to the actin molecules, they bend, and pull the actin toward the center of the sarcomere (M-line), making the sarcomere shorter; the muscle is contracted.

The process just described involves energizing the myosin cross bridges and moving the tropomyosin. ATP and calcium is necessary for this process, as shown in Figure 11.6.

1) When not bound to actin, the myosin head has a low activity of ATPase.
   The ATPase splits ATP, causing ADP and Pi to bind to the myosin, putting the myosin head in a high energy conformation, ready to bind to exposed actin. However, when tropomyosin covers actin it prevents the binding of the myosin heads to the actin.
2) When calcium binds to troponin the tropomyosin moves and exposes the binding sites on actin, allowing the myosin heads to bind to actin. In the process Pi is released.
3) The myosin head bends and pulls actin toward the center of the sarcomere. In the process ADP is released.
   When bound to actin, the myosin head has a high activity of ATPase.
4) ATP binds to the myosin and releases the head from the actin. The ATPase splits ATP, causing ADP and Pi to bind to the myosin, putting the myosin head in a high energy conformation, ready to bind to actin when exposed.

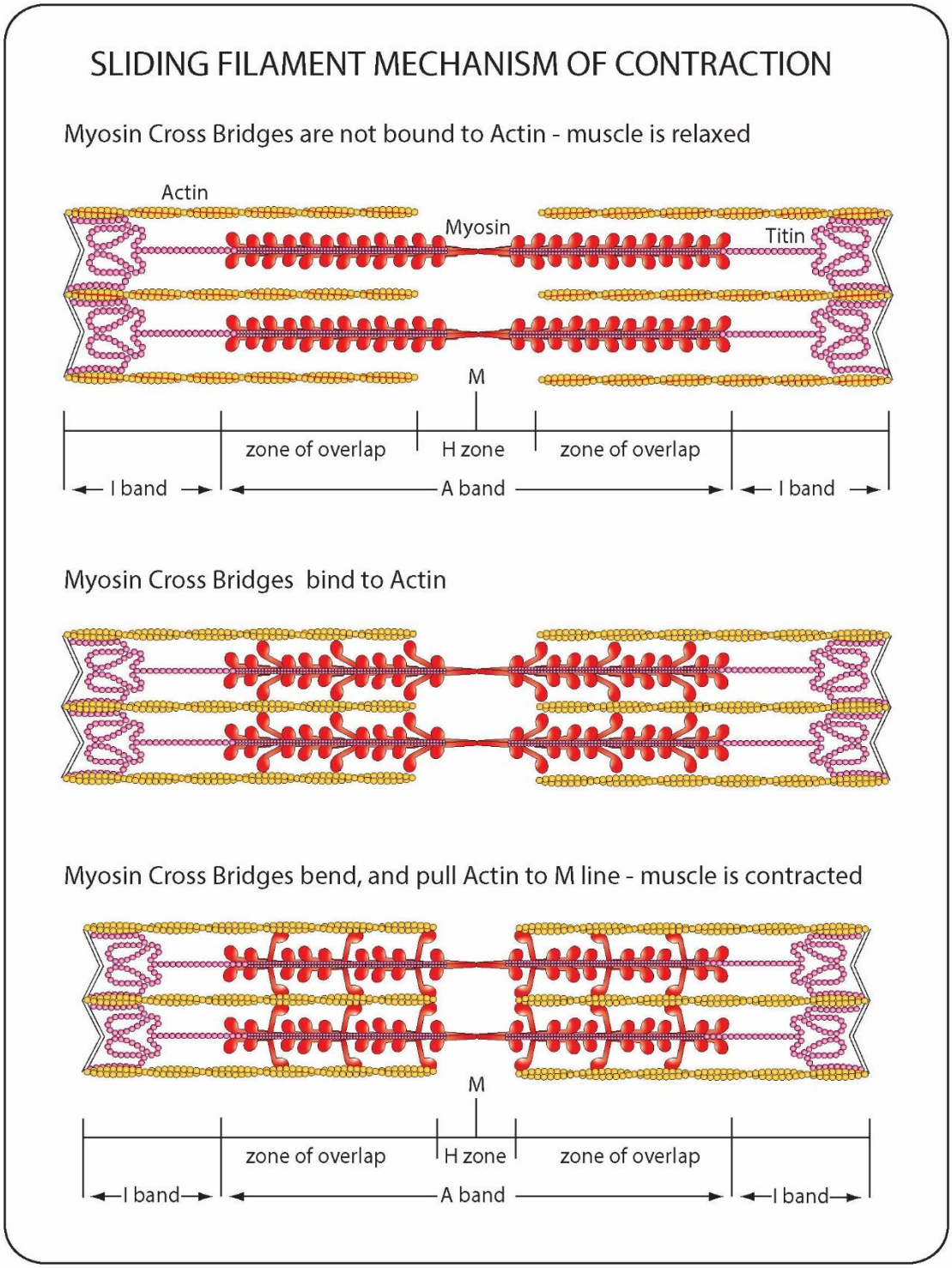


Figure 11.5 © 2018 David G. Ward, Ph.D.

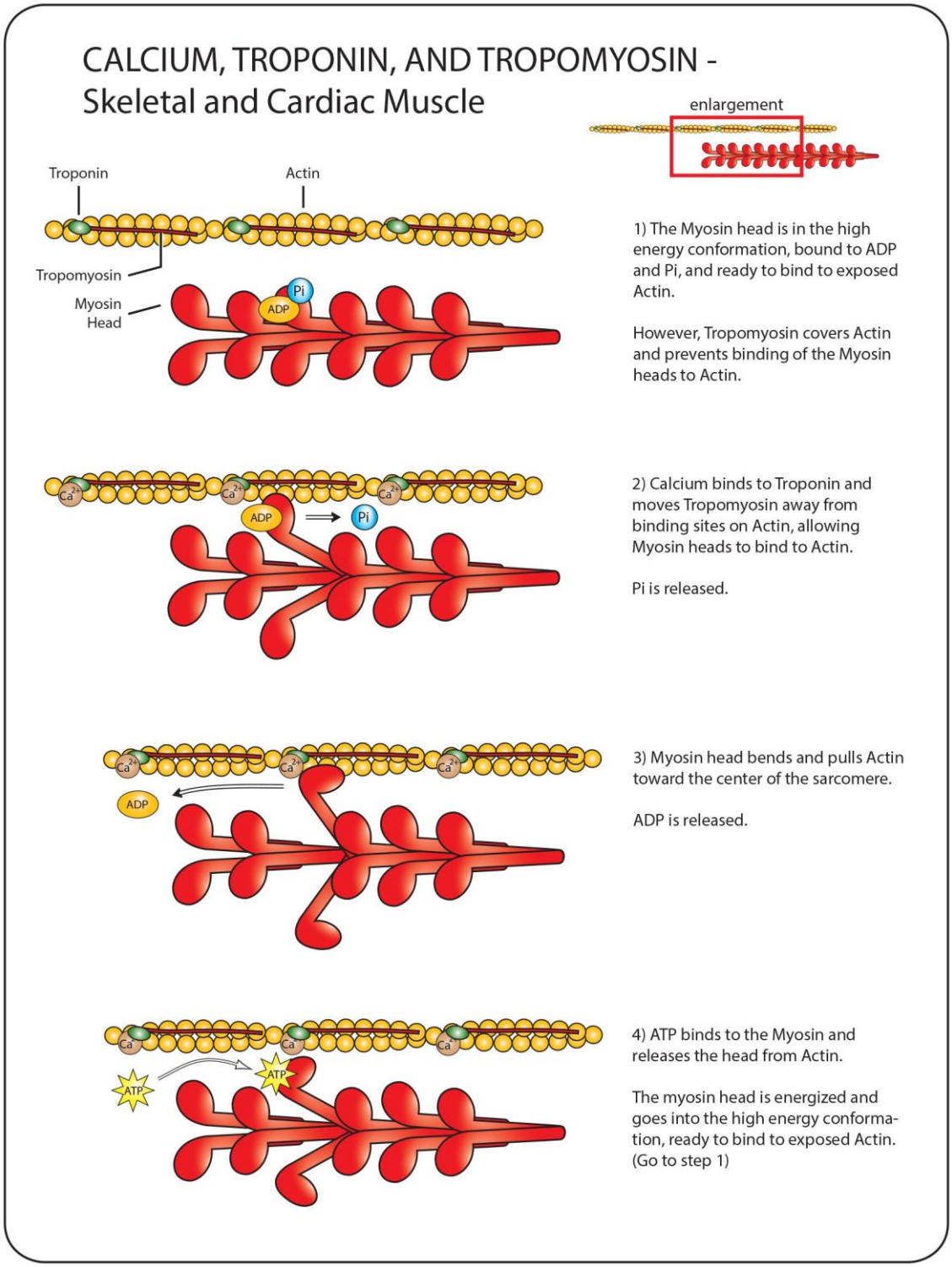


Figure 11.6 © 2019 David G. Ward, Ph.D.

Skeletal muscle activation

Skeletal muscle cells are almost exclusively activated by motor neurons releasing acetylcholine that acts on nicotinic-m receptors, as shown in Figure 11.7.

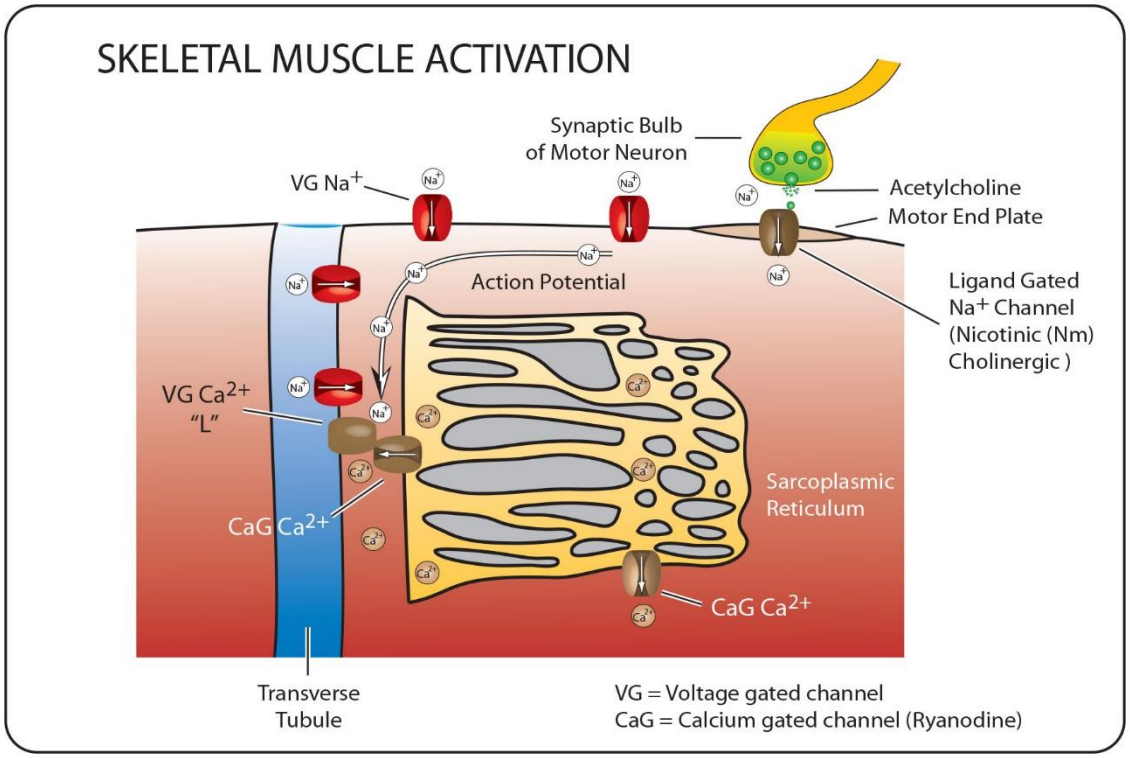


Figure 11.7 © 2014 David G. Ward, Ph.D.

- Acetylcholine is released by synaptic bulbs of a motor neuron.
- Ligand gated sodium channels in the motor end-plate with Nicotinic-m receptors bind to the acetylcholine and open, allowing sodium ions to diffuse in causing a graded depolarization.
- The influx of sodium causes the opening of voltage gated sodium channels.
- The sequential opening and closing of voltage gated sodium channels and potassium channels along the membrane produces an action potential like that seen in axons (refer to Chapter 8).
- The action potential is conducted across the sarcolemma and down each of the transverse tubules.
- The action potential activates voltage gated L-type calcium channels in the transverse tubules that are mechanically coupled to calcium sensitive ryanodine receptors (calcium gated calcium channels) in the sarcoplasmic reticulum (SR). Together, these receptors function as voltage gated calcium channels which open and allow calcium ions to diffuse out of the sarcoplasmic reticulum and into the sarcoplasm.

- The initial diffusion of calcium ions into the sarcoplasm appears to open calcium sensitive ryanodine receptors (calcium gated calcium channels) and allow even more calcium to diffuse out of the SR.
- As shown in Figure 11.6, the calcium that enters the sarcoplasm binds to troponin and moves tropomyosin away from the binding sites on actin, allows the myosin heads to bind to actin, and causes muscle contraction.

Twitches, partial summation, and tetanus

Responses of skeletal muscle cells to slow and progressively more rapid activation are shown in Figure 11.8.

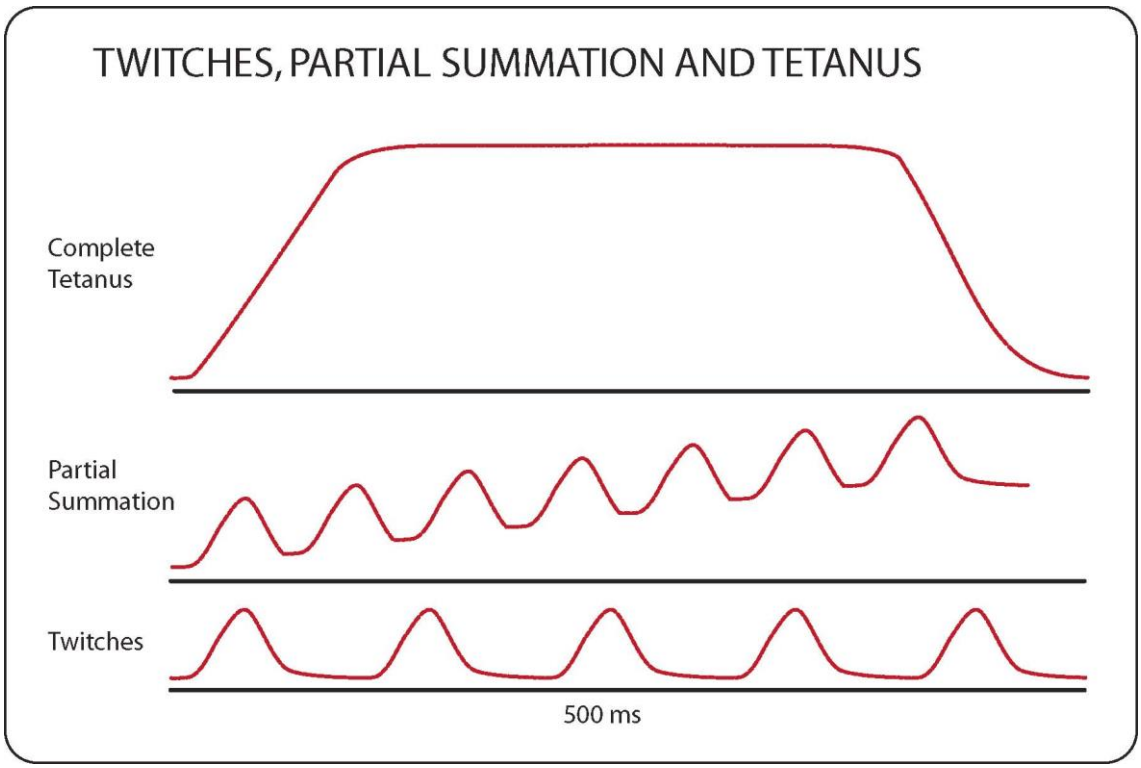


Figure 11.8 © 2007 David G. Ward, Ph.D.

Slow stimulation (bottom graph) causes twitches. A twitch is a single stimulus-contraction-relaxation sequence in a muscle cell. A single twitch in response to muscle cell stimulation will show a latent period, a contraction phase, and a relaxation phase.

- The latent period represents the time required for the action potential to sweep across the sarcolemma and initiate the release of calcium ions (about 2 msec).
- The contraction phase represents the time period that cross bridges are interacting with binding sites on the actin (about 5 to 40 msec).
- The relaxation phase represents the time period as the cross bridges detach from the actin (about 10 to 60 msec).

More rapid stimulation (middle graph) causes activation of the muscle cell before the relaxation phase ends and causes twitches that increase in tension, and reach a plateau. This leads to partial summation and movements that are moderately jerky.

Even faster stimulation (top graph) causes activation of the muscle cell before the relaxation phase begins and prevents the appearance of twitches. The tension will increase and reach a plateau. This cause tetanus and movements that are smooth.

Tension development

The tension developed in a muscle cell increases as the number of cross bridges increase. However, tension decreases when the sarcomeres shorten too much and the thick filaments run into the Z-line. Tension in a whole muscle is increased through a process of recruitment by increasing the number of muscle cells activated.

- In isotonic contraction the length of muscle shortens as tension is developed.
- In isometric contraction the length of muscle remains constant as tension is developed.

Classification of skeletal muscle

Skeletal muscle can be divided into three major classifications: slow; fast, fatigue resistant; and fast, fatigable, as shown in Table 11.1.

Table 11.1. **Classification of Skeletal Muscle**

Slow	Fast, Fatigue Resistant	Fast, Fatigable
Twitch slowly	Twitch rapidly	Twitch rapidly
Weak contractions (force-tension is low)	Intermediate contractions (force-tension is intermediate)	Strong contractions (force-tension is high)
Resist Fatigue	Resist Fatigue (intermediate)	Easily Fatigue
Rely on Aerobic Metabolism	Rely on Aerobic and Anaerobic Metabolism	Rely on Anaerobic Metabolism
Large quantity of Myoglobin	Large quantity of Myoglobin	Small quantity of Myoglobin
Small muscle cell (fiber) diameter	Intermediate muscle cell (fiber) diameter	Large muscle cell (fiber) diameter

Cardiac Muscle

General anatomical organization of cardiac muscle

Cardiac muscle cells are short, macaroni shaped, with a single nucleus, and arranged in a branching manner. A photomicrograph of cardiac muscle is shown in Figure 11.9.

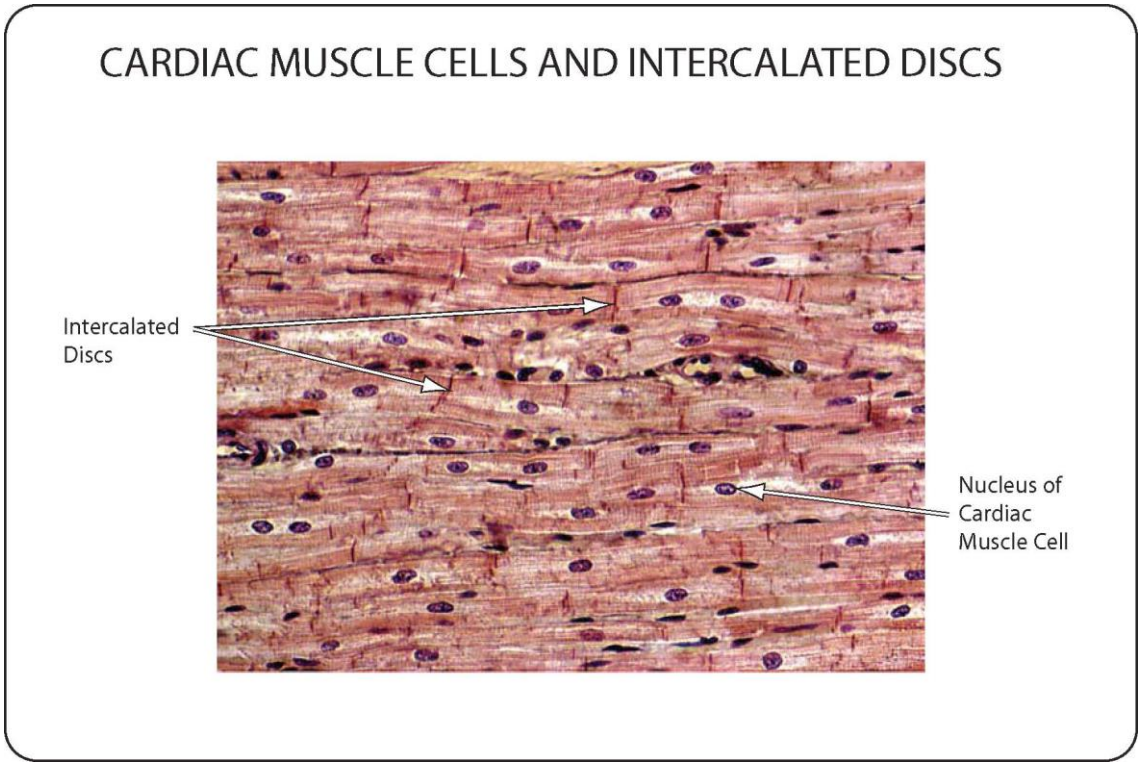


Figure 11.9 © 2007 David G. Ward, Ph.D.

- The cells are connected end to end by interdigitating of the cell membranes called the intercalated discs.
- The cells are connected side by side with fibrous connective tissue called the endomysium.

Organization of individual cardiac muscle cells

Cardiac muscle cells do not contain motor end plates and thus are not controlled by motor neurons in the same manner as skeletal muscle cells. In contrast, cardiac muscle cells are connected at the intercalated discs by gap junctions that allow action potential to spread rapidly by direct flow of ions.

Other than these differences, cardiac muscle is similar anatomically to skeletal muscle.

- Both cardiac muscle and skeletal muscle have a sarcomere structure.
- Both cardiac muscle and skeletal muscle have troponin and tropomyosin.
- However, cardiac action potentials are broad and last for hundreds of milliseconds

Cardiac muscle contraction

The general mechanisms responsible for cardiac muscle contraction are very similar to those illustrated in Figure 11.5 and 11.6. However, as shown in Figure 11.10, there are

considerable differences in the time course of the action potentials, of the flow of calcium into the sarcoplasm, and of the contraction.

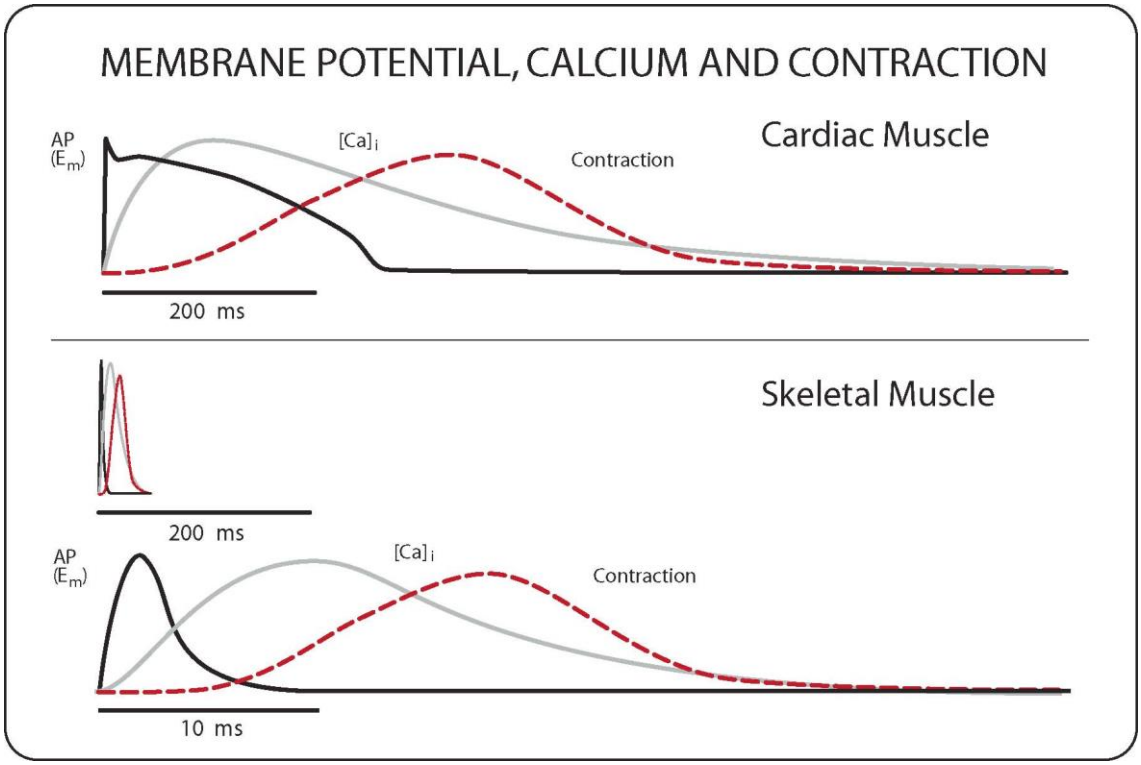


Figure 11.10 © 2007 David G. Ward, Ph.D.

- In both skeletal muscle and cardiac muscle contraction is initiated by the appearance of an action potential in the sarcolemma. However,
  - In skeletal muscle the duration of the action potential is usually much less than 10 msec and the refractory period is about 10 msec which permits rapid tetanic contraction.
  - In cardiac muscle the duration of the action potential is usually about 250 msec and the refractory period is about 300 msec which permits only individual twitches.
- In both skeletal muscle and cardiac muscle contraction is triggered by the appearance of calcium ions in the sarcoplasm. However,
  - In skeletal muscle sarcomere activation is more dependent on Ca+ from the sarcoplasmic reticulum (SR).
  - In cardiac muscle sarcomere activation is more dependent on Ca+ from the extracellular fluid. The action potential opens slow calcium channels in the sarcolemma and transverse tubules. Ca+ from the sarcoplasmic reticulum (SR) is also involved.

Cardiac muscle activation

Cardiac muscle cells are mainly activated by diffusion of cations through gap junctions in the intercalated discs, as shown in Figure 11.11.

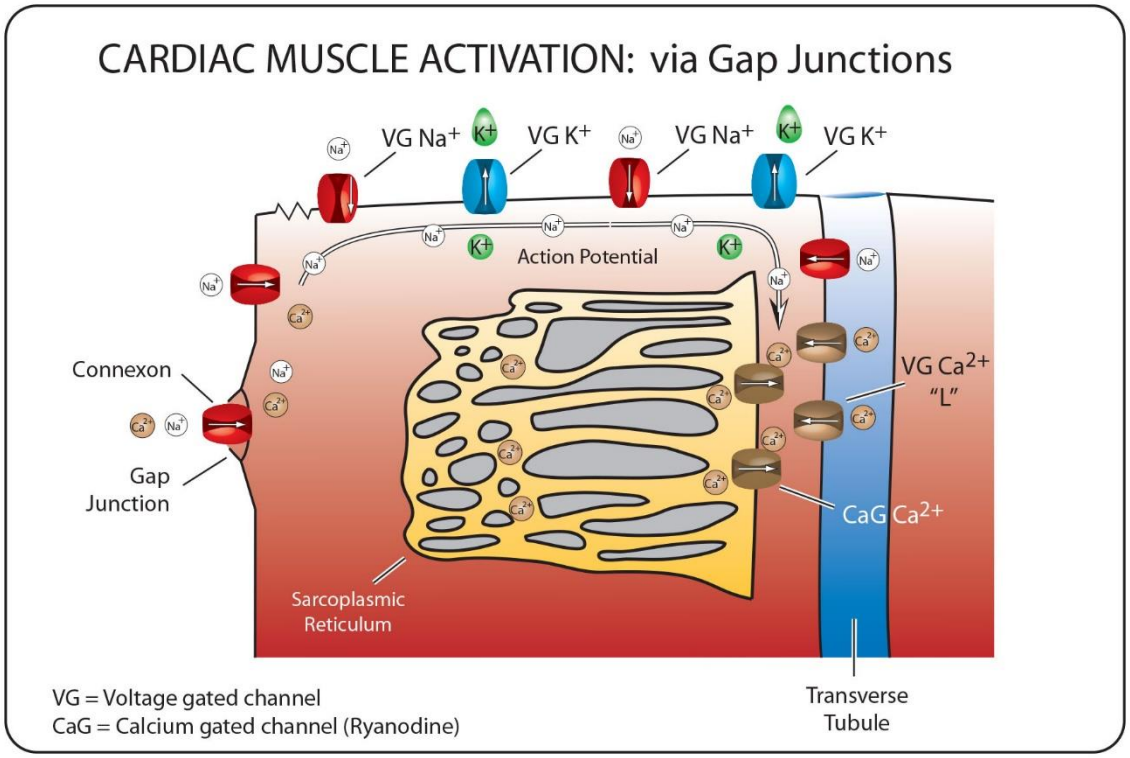


Figure 11.11 © 2014 David G. Ward, Ph.D.

- The influx of sodium through the **connexon** of the gap junction leads to the opening of voltage gated sodium channels in the sarcolemma.
- The sequential opening and closing of sodium channels and potassium channels along the membrane produces an action potential like that seen in axons (refer to Chapter 8).
- The action potential is conducted across the sarcolemma and down each of the transverse tubules.
- The action potential activates voltage gated slow L-type calcium channels (dihydropyridine sensitive) in the transverse tubules and allows calcium to diffuse from the extracellular fluid into the sarcoplasm.
- The entry of calcium seems to activate some of the calcium gated calcium channels (ryanodine sensitive) in the sarcoplasmic reticulum and allows calcium to diffuse out of the sarcoplasmic reticulum (SR) and into the sarcoplasm.
- Voltage gated potassium channels open in response to the prolonged depolarization, and subsequently close.
- As shown in Figure 11.6, the calcium that enters the sarcoplasm binds to troponin and moves tropomyosin away from the binding sites on actin, allows the myosin heads to bind to actin, and causes muscle contraction.

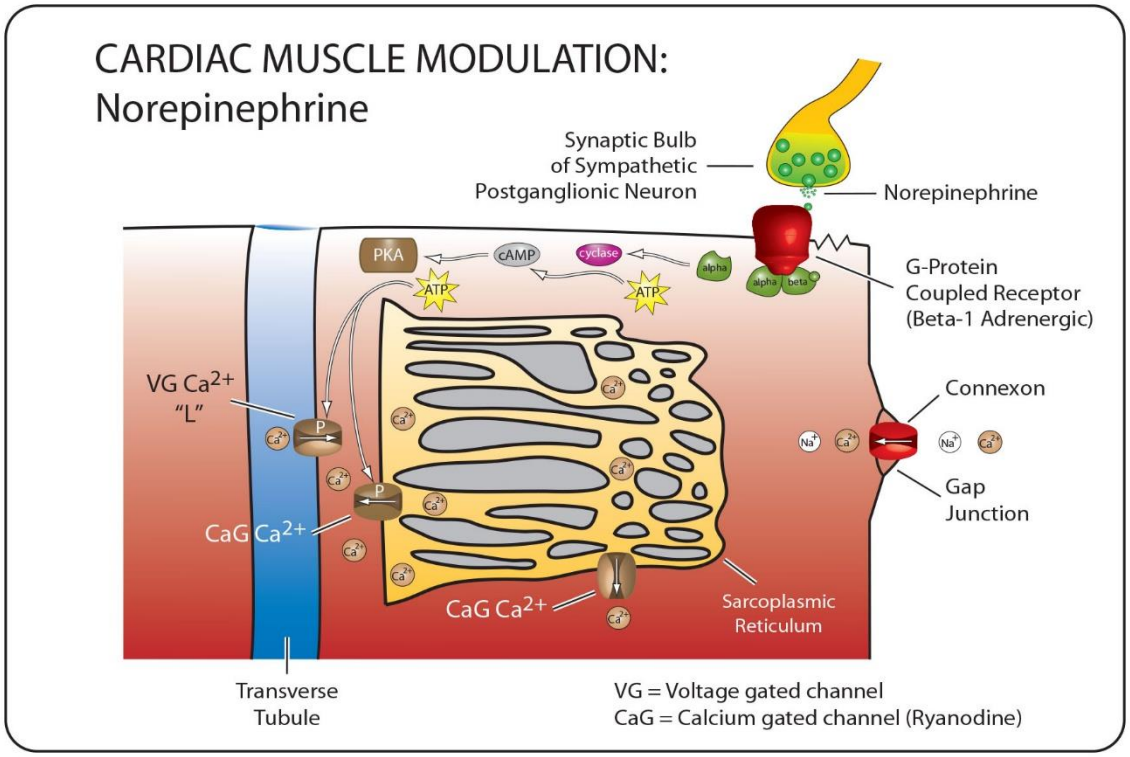


Figure 11.12 © 2014 David G. Ward, Ph.D.

Cardiac muscle modulation

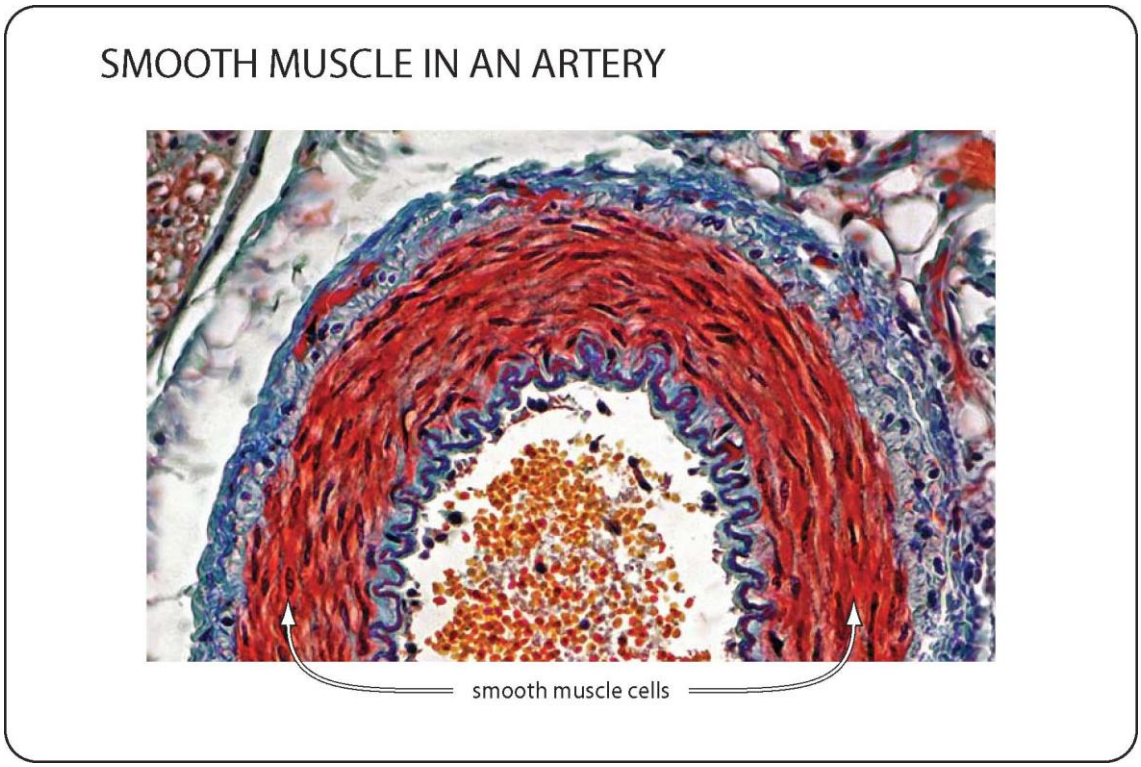
- Cardiac muscle cells are modulated by various hormones, for example by norepinephrine, as shown in Figure 11.12. Norepinephrine binds to G-protein coupled Beta-1 adrenergic receptors.
- G-protein alpha is activated (releases ADP and binds to ATP).
- Activated G-protein alpha activates adenylyl cyclase.
- Adenylyl cyclase catalyzes the conversion of ATP to cAMP.
- cAMP activates protein kinase A (PKA).
- Protein kinase A phosphorylates (P) the slow L-type calcium channels (dihydropyridine sensitive) in the transverse tubules and allows calcium to diffuse from the extracellular fluid into the sarcoplasm.
- Protein kinase A also phosphorylates (P) the calcium gated calcium channels (ryanodine sensitive) in the sarcoplasmic reticulum and allows calcium to diffuse out of the sarcoplasmic reticulum (SR) and into the sarcoplasm.
- As shown in Figure 11.6, the calcium that enters the sarcoplasm binds to troponin and moves tropomyosin away from the binding sites on actin, allows the myosin heads to bind to actin, and causes muscle contraction.

Smooth Muscle

General anatomical organization of smooth muscle

Smooth muscle cells are short with a single nucleus, are tapered towards the ends, and are arranged in an interlocking manner. A photomicrograph of smooth muscle in an artery is shown in Figure 11.13.

- The cells are connected on all sides with fibrous connective tissue.



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Organization of individual smooth muscle cells

Smooth muscle cells do not contain defined motor end plates. Receptors for neurotransmitter and hormones are distributed throughout the sarcolemma. Smooth muscle cells are often connected by gap junctions that allow action potential to spread rapidly by direct flow of ions.

Smooth muscle cells are distinctly different than either cardiac muscle or skeletal muscle.

- Smooth muscle does not have a classic sarcomere structure.
    - Actin and myosin are arranged in parallel bundles that run obliquely in various directions.
    - Thin filaments (actin) attach to dense bodies that are scattered throughout the sarcoplasm. (Dense bodies are analogous to Z-lines).
- Smooth muscle does not have transverse tubules.
- Smooth muscle does not have troponin.

Smooth Muscle Contraction

Calcium, largely under the control of nerves and hormones, regulates contraction of smooth muscle. Contraction of smooth muscle does not involve troponin as the factor controlled by calcium. Rather, calcium is involved in controlling myosin light chain kinase (MLC kinase) which controls the activity of the ATPase of the myosin heads, as shown in Figure 11.14.

1) Myosin light chain kinase (MLC kinase) is activated by calcium-calmodulin. MLC kinase activates the ATPase of the myosin head by a process of phosphorylation (attached to phosphate, Pi).
2) Once phosphorylated, the ATPase splits ATP, causing ADP and Pi to bind to the myosin, putting the myosin head in a high energy conformation, ready to bind to exposed actin.
3) The myosin head binds to actin. During the binding process, the second Pi is released.
4) The myosin head bends, and pulls the actin toward the center of the sarcomere. During the bending process, ADP is released.
5) As long as the ATPase of the myosin head is phosphorylated (Pi), the ATPase splits more ATP. This splitting of ATP causes ADP and Pi to bind to the myosin, putting the myosin head in a high energy conformation, ready to bind to exposed actin. (See step 2.)
   The cycle of re-energizing the myosin head can be slowed or stopped by dephosphorylating the myosin with phosphatase (to remove the Pi).

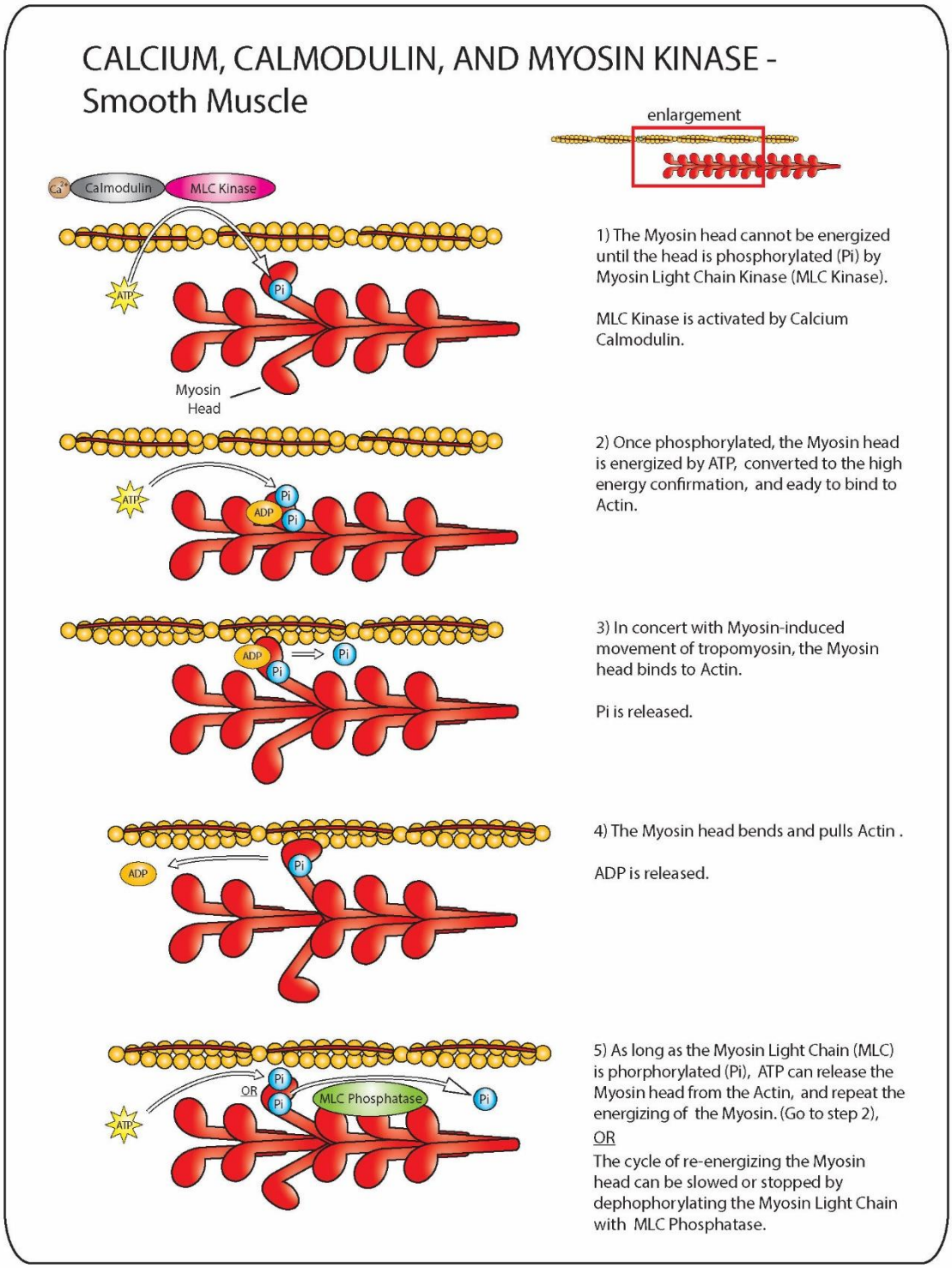

Figure 11.14 © 2018 David G. Ward, Ph.D.

Smooth muscle activation

Smooth muscle cells may be activated by diffusion of cations through gap junctions, by various neurotransmitters, hormones or paracrines acting through G-protein coupled receptors, and by various other means. Smooth muscle cells respond to stimulation in a graded fashion. The activation of smooth muscle via gap junctions and by acetylcholine is shown in Figure 11.15.

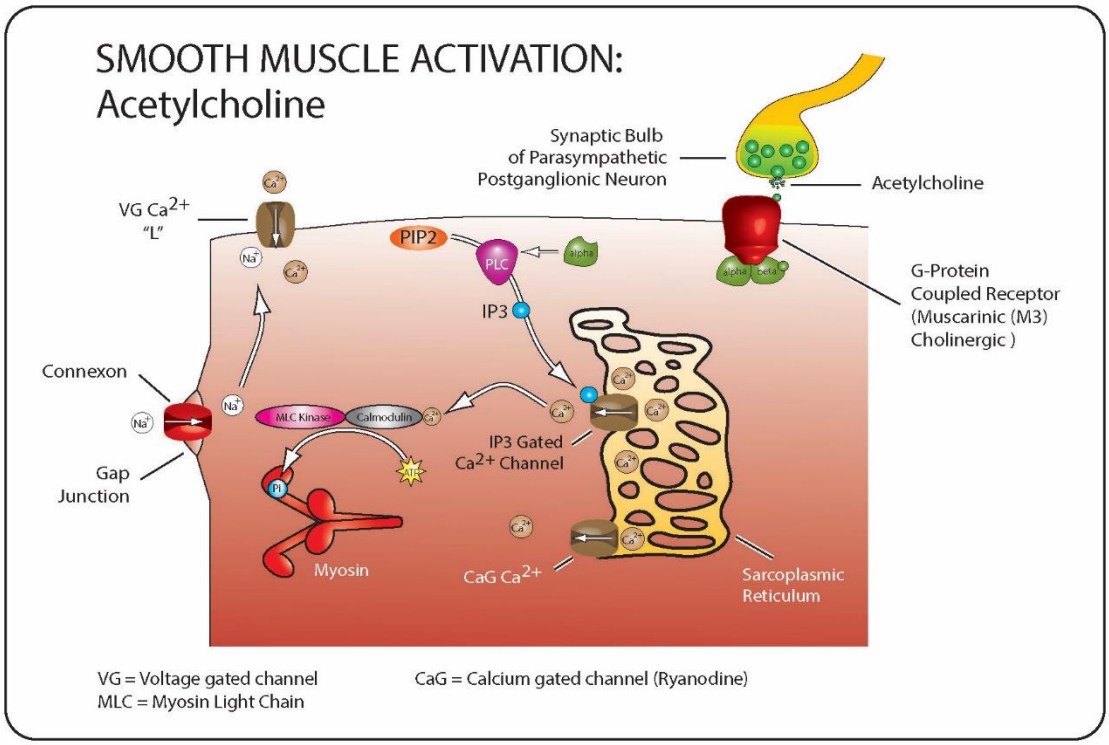

Figure 11.15 © 2014 David G. Ward, Ph.D.

- The influx of sodium through the **connexon** of the gap junction leads to the opening of voltage gated slow L-type calcium channels (dihydropyridine sensitive) in the sarcolemma.
- and/or
- Acetylcholine binds to G-protein coupled M-3 cholinergic receptors.
  - Activated G-protein alpha activates phospholipase C.
  - Phospholipase C catalyzes the conversion of PIP2 to IP3.
  - IP3 opens IP3 gated calcium channels in the sarcoplasmic reticulum (SR) and allows calcium to diffuse out of the sarcoplasmic reticulum (SR) and into the sarcoplasm.

- The entry of calcium seems to activate some of the calcium gated calcium channels (ryanodine sensitive) and also allows calcium to diffuse out of the sarcoplasmic reticulum (SR) and into the sarcoplasm.
- As shown in Figure 11.14, calcium that enters the sarcoplasm forms a complex with calmodulin that binds to myosin light chain kinase to phosphorylate the ATPase of the myosin heads and thus, to allow ATP to re-energize the myosin head and cause muscle contraction.

The role via gap junctions and norepinephrine in activation of smooth muscle is shown in Figure 11.16. Note that the mechanisms for the activation of smooth muscle by alpha-1 adrenergic receptors responding to norepinephrine are nearly identical to the mechanism for the activation of smooth muscle by M-3 cholinergic receptors responding to acetylcholine.

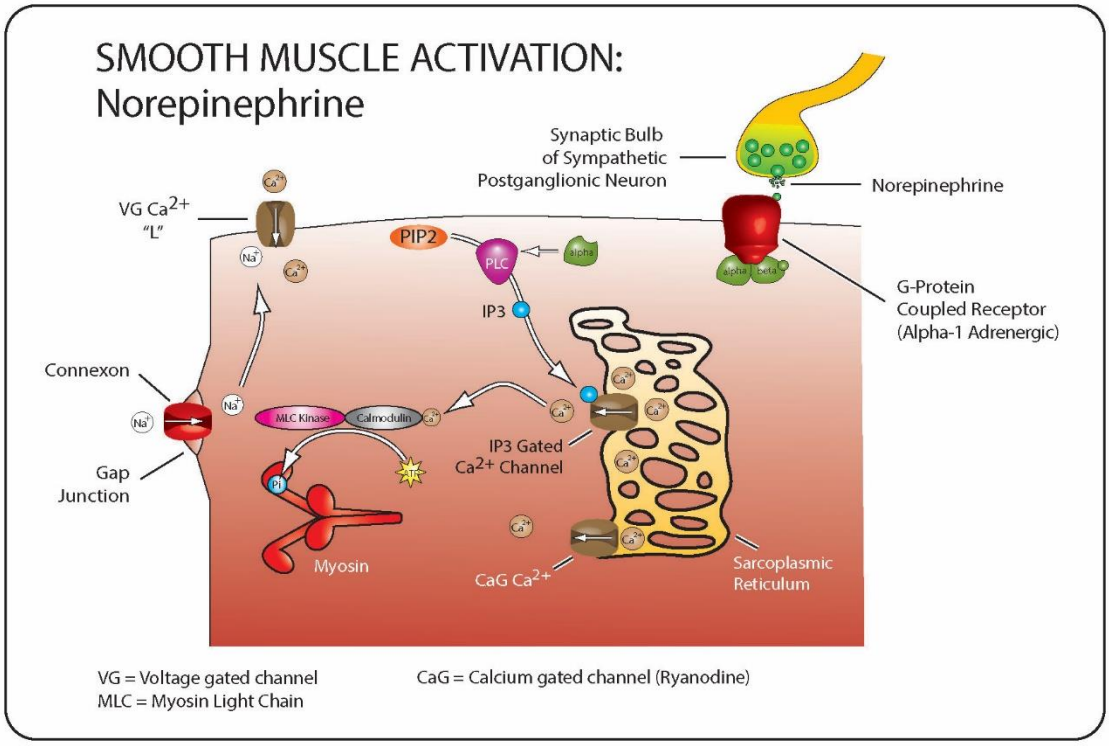

Figure 11.16 © 2014 David G. Ward, Ph.D.

- The influx of sodium through the **connexon** of the gap junction leads to the opening of slow L-type voltage gated calcium channels (voltage sensitive dihydropyridine receptors) in the sarcolemma.
- and/or
- Norepinephrine binds to G-protein coupled alpha-1 adrenergic receptors.
    - Activated G-protein alpha activates phospholipase C.
    - Phospholipase C catalyzes the conversion of PIP2 to IP3.
    - IP3 opens IP3 gated calcium channels in the sarcoplasmic reticulum (SR).and allows calcium to diffuse out of the SR and into the sarcoplasm.

- The entry of calcium seems to activate some of the calcium sensitive ryanodine receptors (SR calcium channels) and also allows calcium to diffuse out of the sarcoplasmic reticulum (SR) and into the sarcoplasm.
- As shown in Figure 11.14, the calcium that enters the sarcoplasm forms a complex with calmodulin that binds to myosin light chain kinase to phosphorylate the ATPase of the myosin heads and thus, to allow ATP to re-energize the myosin head and cause muscle contraction.

Smooth muscle modulation

Smooth muscle cells are modulated by various hormones, for example by epinephrine, as shown in Figure 11.17. This mechanism allows for such things as bronchodilation and vasodilation.

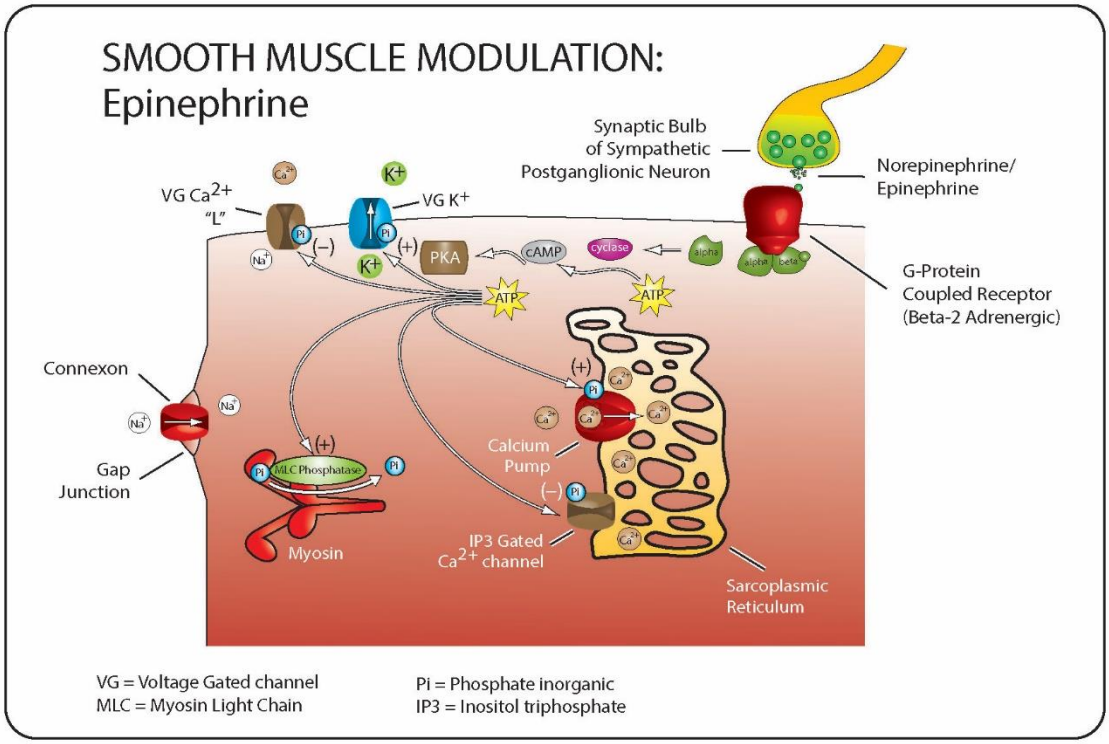

Figure 11.17 © 2018 David G. Ward, Ph.D.

- Epinephrine binds to G-protein coupled Beta-2 adrenergic receptors.
- Activated G-protein alpha activates adenylyl cyclase.
- Adenylyl cyclase catalyzes the conversion of ATP to cAMP.
- cAMP activates protein kinase A, that in turn.
    - Phosphorylates, and activates myosin light chain phosphatase, which removes phosphates from the myosin light chain and prevents the myosin from energizing.

  - Phosphorylates and inhibits L type Calcium channels in the sarcolemma, which prevents movement of Calcium from the extracellular fluid into the sarcoplasm.
  - Phosphorylates and inhibits IP3 receptor Calcium channels in the SR, which prevents movement of Calcium from the SR into the sarcoplasm.
  - Phosphorylates and activates calcium pumps in the sarcoplasmic reticulum (SR) which removes calcium from the sarcoplasm and stores it in the SR.
  - Phosphorylates and opens Potassium channels in the sarcolemma, which allow potassium ions to diffuse out of the sarcoplasm, hyperpolarizing the cell.

- Protein kinase A also may activate some proteins that interfere with the binding of myosin to actin.
- Together these intracellular responses will reduce muscle contraction and make the cell less sensitive to depolarizing stimuli.

Quiz Yourself

1-5. Matching

A)	Skeletal muscle	Contain(s) troponin	1) _____
B)	Cardiac muscle	Contain(s) tropomyosin	2) _____
C)	Smooth muscle	Contain(s) actin and myosin	3) _____
D)	A, B, and C	ATPase of myosin heads is intrinsically active	4) _____
E)	A and B	ATPase of myosin heads must be activated by light-chain kinase	5) _____

6-10. Place the following in order as they would occur in a skeletal or cardiac muscle cell.

A)	Muscle cell contracts	first	6) _____
B)	Myosin heads bind to actin	second	7) _____
C)	Ca^{2+} diffuses into intracellular fluid	third	8) _____
D)	Myosin head bends and pulls actin toward M-line	fourth	9) _____
E)	Ca^{2+} binds to troponin and moves tropomyosin away from actin	fifth	10) _____

11-15. Place these steps in the order causing activation of skeletal muscle cell by a neuron.

A)	Action potential propagates along sarcolemma	first	11) _____
B)	Calcium ions are released from sarcoplasmic reticulum (SR)	second	12) _____
C)	Acetylcholine binds to nicotinic-m receptors in motor end plate	third	13) _____
D)	Acetylcholine is released from motor neuron bulb into synaptic cleft	fourth	14) _____
E)	Action potential opens voltage gated Ca2+ channels in T-tubules and SR	fifth	15) _____

16-20. Place these steps in order for initiating smooth muscle contraction.

A)	Myosin light chain (MLC) kinase is activated by calcium-calmodulin	first	16) _____
B)	MLC kinase activates (Pi) the ATPase of myosin head	second	17) _____
C)	ATPase splits ATP and energizes the cross bridges	third	18) _____
D)	Myosin head bends and pulls actin toward M-line	fourth	19) _____
E)	Myosin head bind to actin	fifth	20) _____

Fill in (MLC = myosin light chain)

21. In skeletal and cardiac muscle myosin cross bridges are phosphorylated and activated by _____.

22. In skeletal and cardiac muscle actin must be _____ to allow cross bridge cycling.

23. In skeletal and cardiac muscle calcium binds to _____ and uncovers actin.

24. In smooth muscle calcium is involved in _____ MLC kinase.

25. In smooth muscle MLC kinase is necessary to _____ the ATPase of the myosin heads and allow cross bridge cycling.

Study Questions

1. Explain how nervous stimulation will cause the contraction of skeletal muscle.
2. Explain the role of calcium in muscle contraction. Include differences between skeletal, cardiac, and smooth muscle.
3. Compare and contrast the mechanisms for control of contraction of skeletal muscle, cardiac muscle, and smooth muscle.