**Terms you should understand:** smooth muscle, L-type Ca\(^{++}\) channels, actin, myosin, sarcoplasmic reticulum (SR), myosine phosphatase, IP\(_3\) receptors, calmodulin (CaM), myosin light chain kinase (MLCK), Ca\(^{++}\)/Na\(^{+}\) ATPase, vasopressin (ADH), dense bodies, mechanical junctions, gap junctions, unitary (single-unit) muscles, multi-unit (multi-fiber) muscles, neurogenic, myogenic, Ca\(^{++}\) spark, tetanus, latch state (aka ‘catch’).

I. **Smooth muscles** are generally found in the walls of hollow organs and control the movement of material, such as blood or food, through those organs.

A. Smooth muscle fibers are single, mononuclear cells, not multinucleate like skeletal muscle fibers.

B. Smooth muscle contraction is slower and more sustained, but can generate more force per cross sectional area than most striated muscle contraction.

1. Smooth muscle contraction is **tonic**: tension can be maintained for long periods of time.

   ![Fig. 10.1. Smooth (left) and striated (right) muscle.](image)

2. Contraction is based on the interaction of actin and myosin filaments, but the highly-organized array of filaments found in the sarcomere is lacking (**Fig. 10.3**).

   a. Most Ca\(^{++}\) for smooth muscle contraction enters through **L-type Ca\(^{++}\) channels** (blocked by DHP) in the cell membrane during the action potential, not from the sarcoplasmic reticulum.

   b. In some smooth muscle cells there is intracellular Ca\(^{++}\) release by:

      i. **Ca\(^{++}\)-induced Ca\(^{++}\) release**: opens ryanodine receptors; produces “Ca\(^{++}\) sparks”.
      ii. Ligand-gated: metabotropic receptor activates IP\(_3\); receptor on the ER opens Ca\(^{++}\) channels.
      iii. Stretch-activated (e.g., some blood vessels): is a myogenic contraction.
      iv. Hormone-activated: e.g., vasopressin on blood vessels without changing membrane potential.

   c. Ca\(^{++}\) binds to **calmodulin (CaM)**, activates **myosin light chain kinase (MLCK)**, which phosphorylates light chains in myosin heads, which activates myosin ATPase activity, producing the sliding of actin past the myosin (**Fig. 10.3**).

   d. Is much more efficient: contraction of smooth muscle requires much less ATP per unit of work.

![Fig. 10.2. Twitch durations in smooth muscle compared to skeletal and cardiac muscles. (Fig. 12-24 in Silverthorn, 5\(^{th}\) edition.)](image)
3. Relaxation reverses contraction: **myosin phosphatase** removes $P_i$ from the myosin heads; the **MLCK** and **CaM** steps reverse; a **Ca$^{++}$/Na$^+$-ATPase** pumps Ca$^{++}$ back into the SR (**Fig. 10.4**).
4. Each smooth muscle fiber can contract further than a single skeletal muscle because smooth muscles have no Z disks and a different arrangement of myosin heads.
   a. The heads on the two sides of the thick filament point in opposite directions.
   b. The heads are all along the thick filament (there is no empty space in the middle).
   c. Smooth muscle fibers can contract to 50% or less of their rest length, whereas skeletal muscle contracts to only about 85% of its rest length.

Fig. 10.5. Arrangement of the thick and thin filaments, along with their attachment to dense bodies, allows smooth muscle fibers to make large contractions. (Fig. 12-27 in Silverthorn, 5th edition.)

5. Mechanical junctions couple the contractions of adjacent smooth muscle fibers.

Fig. 10.6. Dense bodies and mechanical junctions provide a way for the tension generated by sliding filaments to produce directed forces.
B. Some smooth muscles have cells that are active together, whereas others act independently (Fig. 10.7).

1. **Single-unit smooth muscles** (aka “unitary”): fibers in these muscles are electrically coupled (by gap junctions), so that contraction tends to spread throughout a muscle.

2. **Multi-unit smooth muscles**: fibers in these muscles are not electrically coupled and act independently of one another (e.g., the iris, muscles in arterial walls, piloerecter muscles.)
   a. Contraction in these muscles is **neurogenic**.
   b. The activity of smooth muscle is not readily under voluntary control, and therefore smooth muscle has been called **involuntary** muscle.
   c. Some smooth muscle reflexes (e.g., control of heart rate or blood pressure) can apparently be conditioned, however, suggesting that the control can be complex.

![Fig. 10.7. Single-unit (left) and multi-unit (right) smooth muscles.
(Fig. 12.23 in Silverthorn, 7th edition.)](image)

C. Initiation and control of contraction in smooth muscle (Fig. 10.8).

![Fig. 10.8. Summary of the relationships between membrane potential changes and contractions of different kinds of smooth muscles. (Like Fig. 12.28 in Silverthorn, 7th ed.)](image)

A. Repeated **neurogenic** action potentials in the muscle cause summation of contraction (e.g., iris).

B. The membrane potential depolarizations and hyperpolarizations may be **myogenic** (stomach).

C. Contraction of some smooth muscle does not require APs (e.g., arteries, intestines, uterus, ureter).

D. Either positive-going or negative-going changes in \( V_{m} \) produce changes in the tension produced by the muscle; action potentials do not occur (e.g., in some blood vessels).
a. Ligand-gated receptors (e.g., hormone receptors) can change \([\text{Ca}^{++}]_{\text{in}}\) without affecting \(V_m\); the \(\text{Ca}^{++}\) changes the force of contraction.

b. The amplitude of the contractions in these muscles is modulated by:
   i. the degree of stretch.
   ii. neurotransmitters of the autonomic nervous system.
   iii. hormones (Fig. 10.9) (Ca\(^{++}\) sparks are bursts of \(\text{Ca}^{++}\) through the RyR in the SR).

![Fig. 10.9. Example of a contraction of smooth muscle without a change in its resting potential, induced by substance (an arachidonic acid metabolite).](image)

D. Summary of the mechanisms that control smooth muscle activity (Fig. 10.10):

![Fig. 10.10. Smooth muscles respond both to autonomic and hormonal signals.](image)

a. Intrinsic (myogenic) rhythms.

b. Sympathetic and/or parasympathetic nerves.

c. Hormones.

6. Mechanical properties of many smooth muscles make them ideal for producing slow, sustained contractions.
   a. Less \(\text{Ca}^{++}\) and energy is required for prolonged contractions than for twitches.
b. The velocity of smooth muscle contractions is slow, especially when the muscle produces force.

Fig. 10.11. The Ca^{++} levels and cross-bridge phosphorylation (a measure of energy required) decreases as a twitch (upper trace) is prolonged into a tetanus.

Fig. 10.12. Smooth muscles contract much more slowly than skeletal muscles, even slow skeletal muscles.
6. Summary of the ways in which smooth muscle contractions can be modified:

*Fig. 10.13. Factors that influence the magnitude of smooth muscle contraction. (Fig. 12-29 in Silverthorn, 7th edition.)*