

15. Calculate the Nernst potential for Cl^- for a (mythical) creature with $[\text{Cl}^-]_o = 540 \text{ mM}$, $[\text{Cl}^-]_i = 60 \text{ mM}$ at $T = 27^\circ\text{C}$. $R = 8.32 \text{ V} \cdot \text{coul/mol} \cdot ^\circ\text{K}^{-1}$, $F = 9.65 \cdot 10^4 \text{ coul/mol}$.

T in Kelvin = 300 K

$R = 8.32 \text{ V} \cdot \text{coul} \cdot ^\circ\text{K}^{-1} \cdot \text{mol}^{-1}$

$F = 9.65 \cdot 10^4 \text{ coul/mol}$

$z = -1$

$R T / z F = -25.86 \text{ mV}$ multiplying by 2.303 gives -59.56 mV

$V_{\text{membrane}} = -59.56 \cdot \text{Log}_{10} ([\text{Cl}^-]_o / [\text{Cl}^-]_i) = -56.84 \text{ mV}$

16. Draw (sketch) a graph showing the Frank-Starling “law” of the heart – be sure to clearly label each label axis

“Frank-Starling” law describes the ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return.

As seen in the plot graph stretching the heart, the force increases, peaks and then falls. And the second plot you can see a relationship between the force and the free intracellular Ca^{2+} . If the heart is stretched as intracellular Ca^{2+} increases, more force is generated.

The greater the ventricular diastolic volume, the more the myocardial fibers are stretched during diastole. Within a normal physiologic range, the more the myocardial fibers are stretched, the greater the tension in the muscle fibers, and the greater force of contraction of the ventricle when stimulated.

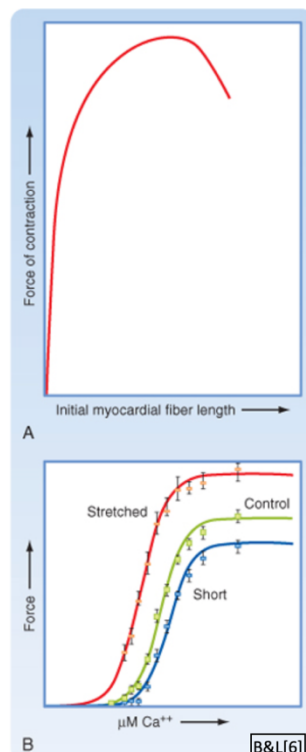


Figure 13-7 Stretching the heart increases the force of contraction (A). This is attributable to both an increase in the maximal force of contraction and an increase in the sensitivity of contraction to Ca^{2+} (B) and reflects an intrinsic regulatory process referred to as the Frank-Starling law of the heart.

17. Explain the mechanism(s) by which action potentials are conducted in myelinated and in unmyelinated nerve fibers. Briefly address relative speeds of conduction (of action potentials) as functions of myelination and of fiber diameter.

A myelinated nerve fiber, the nerve is wrapped in a Schwann cell which contains myelin acting like an electrical insulator. The myelinated nerve fiber has nodes of Ranvier where the myelin sheath stops. And the current in a myelinated nerve fiber jumps from node to node, it is a saltatory conduction. It “appears” that the action potential jumps from node to node. The conduction in an unmyelinated nerve fiber is an electrotonic conduction: the polarity is reversed in the depolarized region where the action potential occurs, and the two regions on the other sides will become depolarized, the depolarization will spread in both directions, away from the initial depolarized region. Every patch of cells needs to be depolarized on its turn. The electrotonic conduction is slow compared to the saltatory conduction. Even when the diameter of an unmyelinated axon is greater than a myelinated axon, (> 100 times), the conduction velocity is still greater in the myelinated axon. To give an idea (the plot would be more correct if it was comparing same species at same temperature) see below plot:

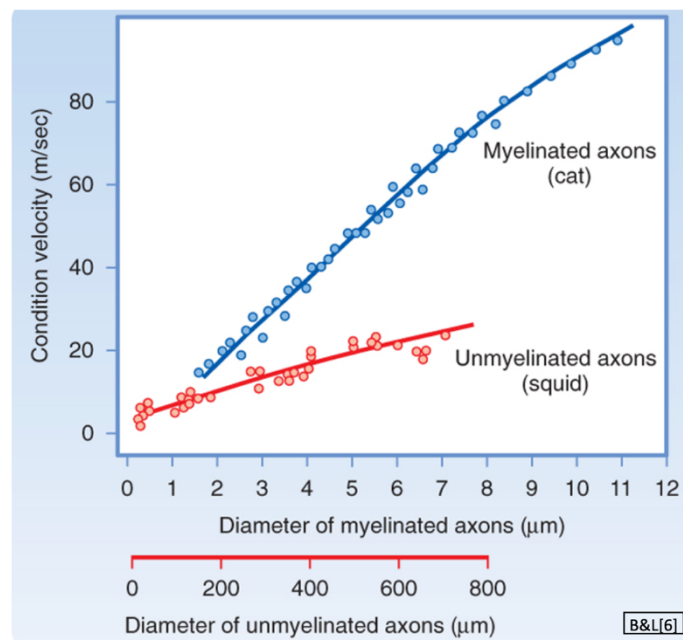


Figure 5-10 Conduction velocities of myelinated and unmyelinated axons as functions of axon diameter. Myelinated axons are from cat saphenous nerve at 38° C. Unmyelinated axons are from squid at 20° C to 22° C. Note that myelinated axons have greater conduction velocities than unmyelinated axons that are 100 times greater in diameter. (Based on data from Gasser HS, Grundfest H: *Am J Physiol* 127:393, 1939 [myelinated axons]; and Pumphrey RJ, Young JZ: *J Exp Biol* 15:453, 1938 [unmyelinated axons].)

18. Describe/explain the mechanism/process of calcium induced release of calcium (CIRC) in cardiac muscle.

When an action potential depolarizes the cell membrane, L-type calcium voltage-gated Ca^{2+} channels are activated. CICR occurs when the resulting intracellular Ca^{2+} , goes through the L-type voltage gated Ca^{2+} channels, and activates the ryanodine receptors on the SR membrane which causes more free intracellular Ca^{2+} to be released into the cytosol. In cardiac muscle the result of CICR could be observed as a bolous release of intracellular calcium. Then the free intracellular calcium binds to TnC allowing muscle contraction.

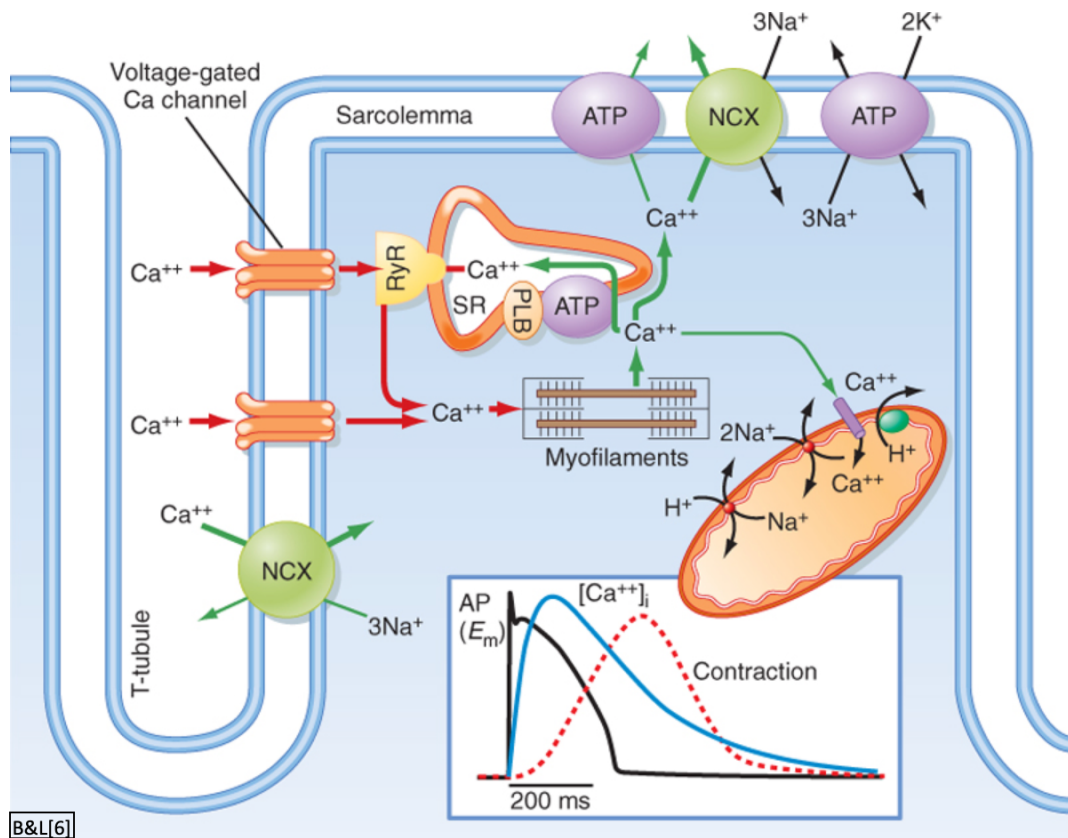


Figure 13-3 Excitation-contraction coupling in the heart requires Ca^{2+} influx through L-type Ca^{2+} channels in the sarcolemma and T tubules. See text for details. (Redrawn from Bers DM: *Nature* 415:198-205, 2002.)