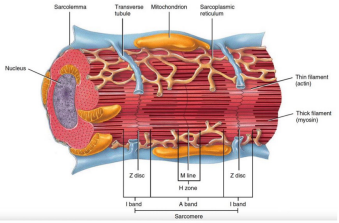
Physiology Lecture 3



	T tubules	Mitochondria	SR	ATP production
Cardiac muscle	Wider 5 times · Less abundent ↳ located at the z disc.	Larger More numerous	Less well developed ↳ then Smaller intracellular reserve of Ca^{2}	Little by anaerobic cellular respiration and depends on aerobic respiration
Skeletal muscle	More abundent	Smaller	More Developed	

* Cardiac muscles need oxygen diffuses from blood in the coronary circulation and is released from myoglobin inside cardiac muscle fibers.

* In person at rest, the heart's ATP mainly comes from oxidation of FA (60%) and glucose (35%) with smaller contributions from lactic acid, amino acids, and ketone bodies.

* During exercise, the heart use of lactic acid , produced by actively contracting skeletal muscles, rises.

Important note
Cardiac and skeletal muscles produces some ATP from creating phosphate

Intercalated Disc

1) Contain desmosomes and gap junctions.
2) Desmosomes are specialized adhesive protein complexes that localize to intercellular junctions and are responsible for maintaining the mechanical integrity of tissues.

3) At each intercalated disc the cell membranes fuse with one another to form permeable communicating junctions (gap junctions) that allow rapid diffusion of ions.

No gap junctions between atria and ventricles.

Refractory period

The period during which a normal cardiac impulse cannot reexcite an already excited area of cardiac muscle , The normal refractory period of the ventricle is 0.25 to 0.30 second, which is about the duration of the prolonged plateau action potential, There is an additional relative refractory period of about 0.05 second during which the muscle is more difficult to excite than normal but can be excited by a very strong excitatory signal (early premature contraction).

The refractory period of atrial muscle is much shorter; about 0.15 second.

During excitation–contraction coupling, calcium ions are released into the sarcoplasm from both the sarcoplasmic reticulum (SR) and T tubules when an action potential opens voltage–dependent calcium channels in the T tubule membrane. The calcium entering from the T tubules further activates ryanodine receptor channels in the SR, releasing additional calcium into the sarcoplasm. This surge in calcium triggers muscle contraction.

The T tubules contain negatively charged mucopolysaccharides that bind and store calcium ions, keeping them ready for release into the cardiac muscle fiber during an action potential.

Relaxation

At the end of the cardiac action potential plateau, calcium influx into the muscle fiber stops suddenly, and calcium ions already in the sarcoplasm are rapidly pumped back into the sarcoplasmic reticulum (SR) through the SERCA2 pump. Additional calcium is removed from the cell via the Na–Ca exchanger, which utilizes the inward sodium gradient. The sodium that enters during this exchange is then expelled from the cell by the Na–K ATPase pump. These transport processes reduce the intracellular calcium concentration, causing calcium to dissociate from troponin C, which prevents actin–myosin interactions. As a result, the muscle fiber relaxes until a new action potential initiates another cycle of contraction.

Contractility

Contractility, or inotropism, is the heart muscle's ability to generate force at a set muscle length. Positive inotropic agents increase the rate and peak of muscle tension, while negative agents reduce them. Contractility is directly related to intracellular Ca^{2+} concentration, influenced by the amount of Ca^{2+} released from the sarcoplasmic reticulum (SR) during excitation–contraction. Ca^{2+} release depends on the inward Ca^{2+} current and SR stores. Higher Ca^{2+} levels lead to greater contractility. Tension in myocardial cells is proportional to Ca^{2+} levels, and agents affecting Ca^{2+} currents or SR stores impact tension produced by the heart.