Cardiac muscle contraction

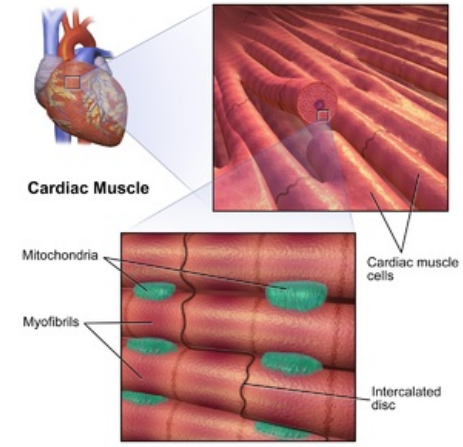
Cardiac muscle contraction produces the energy needed to maintain blood flow through the tissues. The basic mechanisms of heart and skeletal muscle contraction are based on the same principle – interaction of contractile elements – actin and myosin fibers.

Heart muscle structure

The basic building unit of striated cardiac muscle tissue is the **cardiomyocyte** (cardiac muscle cell) – an elongated branching cell with one or two nuclei and many mitochondria. Cardiomyocytes are filled with myofibrils, which are organized in parallel. The myofibril consists of thin actin filaments and thick myosin filaments.

Thin myofilaments

- 1 mm long, 8 nm diameter;
- composition: actin, tropomyosin, troponin;
- arrangement: **double helix of F-actin** (polymerization from G actin: globular (diameter: 5.6 nm), it has a binding site for myosin)-anchored to the telophragm;
- **tropomyosin** (thin, 40 nm long, 2 polypeptide chains (wrapped around each other), connects longitudinally) – turns around actin, has a binding place for troponin;
- **troponin** (protein complex: 3 subunits: TnT – binds the complex to tropomyosin, TnC – binds Ca^{2+}, TnI – inhibits actin + myosin binding) – binds to a specific site on each tropomyosin;
- ratio arrangement: 7 G-actins + 1 tropomyosin + 1 troponin.



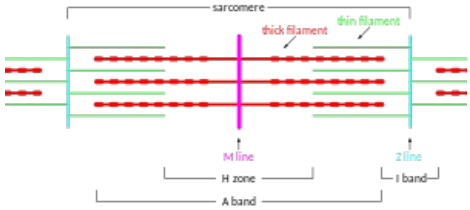
cardiac muscle

Thick myofilaments

- 1.6 mm long, diameter: 15 nm;
- composition: myosin (several hundred in one bundle);
- by proteolysis it is cleaved into: **light meromyosin** (stick-shaped part) and **heavy meromyosin** (curved stick-shaped part and globular part);
- globular part shows ATPase activity, contains place for ATP and place for actin.

Sarcomere

Each myofibril is divided lengthwise into sarcomeres (uncontracted is 2.5 mm long), which separate the **Z-line**. Actin myofilaments are anchored in these lines. The section between the Z-line and the onset of thick myofilaments is called I band (isotropic). The area of myosin filaments is called lane A (anisotropic). The center of the sarcomere forms a H zone, in the middle of which is the **M-line**. Into M-line are the myosin filaments anchored.



Sarcomere

Contraction mechanism

The direction for myocardium contraction is cell excitation. The AP must therefore "transform" into a muscle contraction in the cell. The mechanism that ensures this is called the excitation contraction coupling. It also ensures the connection of the electrical and mechanical activities of the heart. The transfer of excitation from the activated cell membrane to intracellular myofibrils is mediated by Ca^{2+}.

Synchronized activity of cardiomyocytes is ensured by specialized connections between cells, so called intercalated discs. Intercalated discs contain desmosomes (tight junction) and nexy (gap junction). Thanks to the intercalated discs, electrical excitations are allowed to spread fast from one cell to another, so that the heart muscle acts as a syncytium, even though it is made up of individual cells.

The wave of depolarization spread fast across the sarcolemma and enters the cell through the T-tubule system. During the plateau phase, Ca^{2+} channels (dihydropyridine receptors) open and Ca^{2+} ions flow in the direction of their concentration gradient into the cell. This calcium would not be enough to cause contraction, but an increase in the concentration of Ca^{2+} ions in the cytosol acts on Ca^{2+} dependent ryanodine receptors in the sarcoplasmic reticulum. Due to the flow of Ca^{2+} ions from the SR, the cytosolic calcium concentration increases approximately 100-fold and can cause muscle contraction.

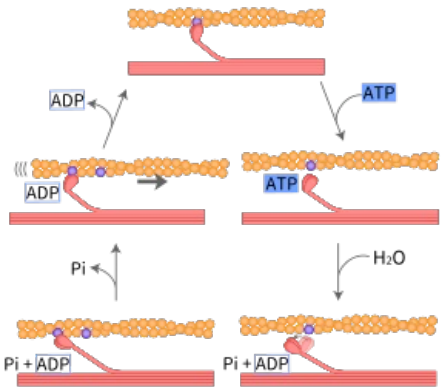
Mechanism

ATP binds to myosin and dissociates, but no cleavage products are released. Once Ca^{2+} ions reach the contractile elements, they begin to bind to troponin. That cause the conformational change of the troponin-tropomyosin complex that unblocks the active place on the actin filaments to form a bond with the myosin heads (concretely the fibrous tropomyosin sinks deeper into the groove between the two actin fibers, so that it no longer overlapses the active sites). The result is the release of energy (actin acts as a cofactor to release cleavage products) and the subsequent bending of the myosin head, which causes the actin fibers to move along the myosin ones. Binding of another ATP molecule to myosin weakens the actinomyosin bridge, the cycle is repeated until Ca^{2+} is available.

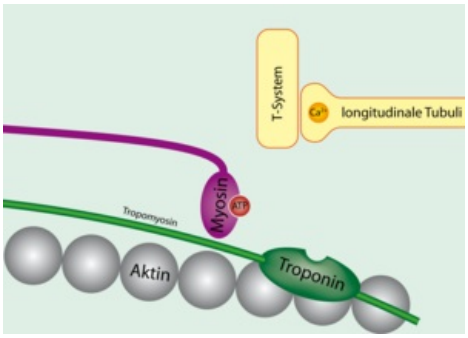
The whole process of pouring Ca^{2+} into the cytosol and start of muscle contraction is very fast, from the beginning of depolarization to the start of contraction it takes only about 60 ms.

At the end of the plateau phase, the flow of Ca^{2+} ions into the cell ceases and Ca^{2+} channels in SR are closing, at the same time Ca^{2+} the ATPase pump in the SR wall (SERCA 2) begins to actively pump calcium from the cytosol back to the reticulum. Part of the Ca^{2+} ions is removed from the cell via a sarcolemma Ca^{2+} –Na^{+} antipode (secondarily active transport). The result is a decrease in the cytosolic concentration of Ca^{2+} and the release of Ca^{2+} ions from troponin binding. This is followed by blockage of the active places by tropomyosin and relaxation (requires energy!).

It is obvious that a cycle of calcium ions must function in cardiomyocytes, which are alternately supplied to and pumped to myofibrils. If the ions did not reach the fibrils, the myocardium would be permanently relaxed, if they were not drained, it would be permanently contracted.



Mechanism of skeletal muscle contraction

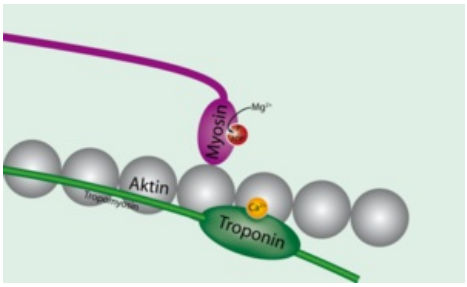


Tropomyosin bound to actin

Links

Related links

- Heart
- Frank-Starling mechanism



Tropomyosin unbound to actin

Bibliography

- TROJAN, Stanislav. *Lékařská fyziologie.* 4. edition. Praha : Grada, 2004. ISBN 80-247-0512-5.
- KONRÁDOVÁ, Václava. *Funkční histologie.* 2. edition. H + H, 2000. ISBN 978-80-86022-80-2.
- GANONG, William F. *Přehled lékařské fyziologie.* 20. edition. Galén, 2005. ISBN 80-7262-311-7.
- BROŽEK, Gustav – HERGET, Jan – VÍZEK, Martin. *Poznámky k přednáškám z fyziologie : Tělesné tekutiny - Krev - Trávicí trakt - Výživa a metabolismus - Endokrinologie - Obecná neurofysiologie - Speciální neurofysiologie - Vývoj plodu.* 1. edition. H+H, 1993. ISBN 80-85787-16-4.