

# Contraction of the heart muscle

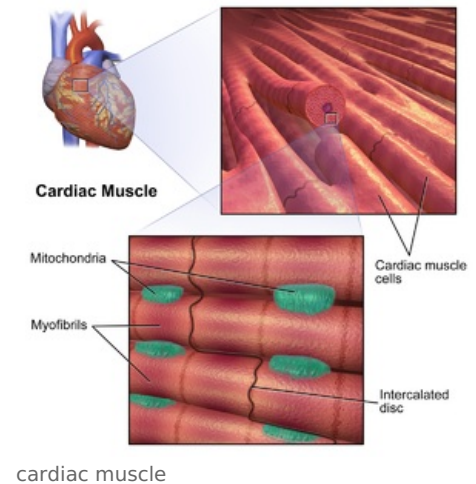
Contraction of the heart muscle creates the force needed to maintain blood flow through the tissues. The basic mechanisms of cardiac and skeletal muscle contraction are based on the same principle. Their essence is the interaction of contractile elements - actin and myosin fibers

## Structure of heart muscle

The basic structural unit of striated cardiac muscle tissue is the *cardiomyocyte* (cardiac muscle cell) - an elongated, branching cell with one or two nuclei and a large number of mitochondria. Cardiomyocytes are filled with myofibrils, which are arranged in parallel. A myofibril consists of thin actin filaments and thick myosin filaments.

### Thin myofilaments

- 1  $\mu\text{m}$  long, 8 nm in diameter;
- composition: actin, tropomyosin, troponin;
- arrangement: **F-actin double helix** (polymerization from G actin: globular (diameter: 5.6 nm), has a myosin binding site) - anchored in the telophragm;
- **tropomyosin** (thin, 40 nm long, 2 polypeptide chains (twisted around each other), joins longitudinally) - wraps around actin, has troponin binding site;
- **troponin** (protein complex: 3 subunits: TnT - binds the complex to tropomyosin, TnC - binds  $\text{Ca}^{2+}$ , TnI - inhibits actin+myosin binding) - binds to a specific site on each tropomyosin;
- relative arrangement: 7 G-actins + 1 tropomyosin + 1 troponin.



### Thick myofilaments

- 1.6  $\mu\text{m}$  long, diameter: 15 nm;
- composition: myosin (several hundred in one bundle);
- by proteolysis it is split into: **light meromyosin** (rod-like part) and **heavy meromyosin** (bent rod part and globular part);
- the globular part shows ATPase activity, contains a site for ATP and a site for actin.

## Sarcomere

Each myofibril is divided lengthwise into sarcomeres (uncontracted measuring 2.5  $\mu\text{m}$ ), which are separated by **Z-lines**. Actin myofilaments are anchored to these lines. The section between the Z-line and the beginning of the thick myofilaments is called the I band (isotropic). The region of myosin filaments is referred to as band A (anisotropic). The center of the sarcomere is the H band, in the middle of which is the **M-line**, into which the myosin filaments are anchored.

## Mechanism of contraction

The instruction for the contraction of the myocardium is cell excitation. Thus, the AP must be "converted" into a muscle contraction in the cell. The mechanism that ensures this is called excitation-contraction coupling and ensures the coupling of the heart's electrical and mechanical activity.  $\text{Ca}^{2+}$  mediates the transfer of excitation from the activated cell membrane to the myofibrils inside the cell.

Synchronized activity of cardiomyocytes is ensured by means of specialized junctions between cells, intercalary discs. Intercalary discs contain desmosomes (tight junctions) and nexae (electrical junctions). Thanks to the intercalary discs, the rapid spread of electrical impulses from one cell to another is enabled, so the heart muscle acts functionally as a syncytium, even though it is composed of individual cells.

The wave of depolarization quickly spreads along the sarcolemma and reaches the interior of the cell via the T-tubule system. During the plateau phase,  $\text{Ca}^{2+}$  channels (dihydropyridine receptors) open and  $\text{Ca}^{2+}$  ions flow in the direction of their concentration gradient into the cell. This calcium would not be enough to cause contraction, but the rise in the concentration of  $\text{Ca}^{2+}$  ions in the cytosol acts on the  $\text{Ca}^{2+}$  dependent ryanodine receptors in the sarcoplasmic reticulum and due to the influx of  $\text{Ca}^{2+}$  ions from the SR, the cytosolic calcium concentration increases about 100-fold and can induce muscle contraction.

## Mechanism

ATP binds to myosin and is cleaved, but no cleavage products are released. As soon as  $\text{Ca}^{2+}$  ions reach the contractile elements, they begin to bind to troponin and the conformational change of the troponin-tropomyosin complex unblocks the active sites on the actin filaments to form a bond with the myosin heads (specifically, the filamentous tropomyosin sinks deeper into grooves between the two actin filaments so that it stops overlapping the

active sites). The result is the release of energy (actin acts as a cofactor for the release of fission products) and subsequent bending of the myosin head, which causes the actin filaments to move along the myosin filaments. Binding of another ATP molecule to myosin weakens the actinomyosin bridge, the cycle repeats as long as  $\text{Ca}^{2+}$  is available.

The entire process of flushing  $\text{Ca}^{2+}$  into the cytosol and the subsequent initiation of muscle contraction is very fast, from the beginning of depolarization to the start of contraction, only about 60 ms elapse.

At the end of the plateau phase, the flow of  $\text{Ca}^{2+}$  ions into the cell stops and  $\text{Ca}^{2+}$  channels in the SR close, simultaneously  $\text{Ca}^{2+}$  ATP-ase the pump in the SR wall (SERCA 2) begins to actively pump calcium from the cytosol back into the reticulum. Part of the  $\text{Ca}^{2+}$  ions is removed from the cell via the sarcolemma  $\text{Ca}^{2+}$ - $\text{Na}^{+}$  antipode (secondary active transport). The result is a decrease in the cytosolic concentration of  $\text{Ca}^{2+}$  and the release of  $\text{Ca}^{2+}$  ions from binding to troponin. This is followed by blocking of the active sites with tropomyosin and relaxation (requires energy!).

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

## Links

### Related Articles

- Heart
- Heart/Histology
- Frank-Starling Mechanism
- Homeometric regulation of the heart

### Used literature

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- BROŽEK, Gustav – HERGET, Jan – VÍZEK, Martin. *Lecture notes in Physiology : Body Fluids - Blood - Digestive Tract - Nutrition and Metabolism - Endocrinology - General Neurophysiology - Special Neurophysiology - Fetal Development*. 1. edition. Jinočany : H+H, 1993. 325 pp. ISBN 80-85787-16-4.