Coupling of excitation and contraction in cardiac muscle

The principle of the mechanism

The excitation-contraction coupling mechanism ensures the connection of electrical and mechanical activity of the heart. **Electrical events** in the myocardium represent the regular rhythmic generation of impulses and their distribution throughout the myocardium. Excitement is only an instruction for the mechanical work of the heart (to contract). The main role here is played by Ca 2+ ions , which ensure the transfer of excitation to the structures performing the contraction. The wave of **depolarization** quickly spreads along the **sarcolemma** of the cells of the working myocardium and reaches the interior of the cell via **T-tubules** (**T-tubules are projections of the sarcolemma into the cell, which contain the extracellular space**). During the **plateau phase** , **L-type Ca-channels** openand Ca 2+ ions begin to flow into the cell in the direction of the concentration gradient. Subsequently, their concentration in the cytosol increases, which causes the opening of **Ca-channels** in the **sarcoplasmic reticulum** and the cytosolic concentration of Ca 2+ increases about a hundred times. This causes **muscle contraction**.

Structure of the contractile apparatus

The contractile apparatus consists of **actin** filaments and **myosin** molecules

Actin filamenta

- 1 mm long, 5–7 nm wide;
- made up of 2 strands wrapped around each other (like two strings of pearls);
- at regular intervals (40 nm) between the actin chains, spherical molecules of **troponin** connected to the fibrous **tropomyosin** , which lies between the two actin chains, are stored.

The myosin molecule

- it is shaped like a thin rod with a spherical flare at one end;
- one myosin filament contains several hundred myosin molecules (rod-shaped with a head;
- the filament has a helix shape, periodically appearing extensions projecting against the actin filament.

Calcium cycling during cardiac action

- Under **normal conditions** , tropomyosin filaments prevent the binding of actin and myosin (low Ca2+ level).
- **As soon as the concentration of Ca^{2+}** in the cytosol rises , calcium ions begin to bind to **troponin**.
- The change in conformation of the troponin-tropomyosin complex leads to the **unblocking of the active sites on the actin** molecule for the formation of actin binding to the myosin head.
- After binding, **the ATPase activity of the myosin head is activated** and the myosin head bends
- Actin fibers move and **muscle contraction** occurs.
- In this event, **ATP is split**.
- **60 ms** elapses from the start of depolarization to the start of contraction.
- At the end **of the plateau phase** , the flow of Ca 2+ into the cell ceases and the Ca-channels in the sarcoplasmic reticulum close.
- **The Ca 2+ -ATPase pump** begins to pump Ca from the cytosol back into the sarcoplasmic reticulum and there is a rapid decrease in the cytosolic Ca concentration.
- This is followed by the release of Ca from binding to troponin, which **blocks the active sites** again - **muscle relaxation** occurs
- Part of the Ca 2+ is removed from the cell by the activity of the sarcolemma **Ca 2+ - Na +** antiport
- Ca pumping by the ATPase pump requires energy in the form of **ATP**.
- If Ca ions do not reach the myofibrils, the myocardium is permanently relaxed.
- Myocardial cells contain at least 4 types of Ca channels - the most clinically significant are **L-type channels** (they are easily influenced by a number of blockers).

Types of Ca channels

1. **Type L** - located on the sarcolemma, responsible for conduction of the impulse in the AV node, depolarization of pacemaker cells, coupling of excitation and contraction
2. **Type T** - also located on the sarcolemma, plays a major role in spontaneous diastolic depolarization

3. **Ryanodine receptor-gated channel** - in sarcoplasmic reticulum, major role in Ca release from SR
4. **A channel controlled by the IP3 receptor** - in the sarcoplasmic reticulum, modulates the release of Ca from the SR

Links

Related articles

- Heart
- Myocardium
- Cardiac conduction system
- Pacemaker potential

References

- KITTNAR, Otomar. *Lékařská fyziologie.* 1. edition. Praha : Galén, 2011. pp. 790. ISBN 978-80-247-3068-4.

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