

Homeometric regulation of the heart

Characteristics

- the ability to change the force of contraction of the cardiac muscle even with a constant length of cardiomyocytes
 - does not depend on changes in sarcomere length
 - affects inotropy (contractility)
 - in contrast to heterometric control, more beats are required for the regulation to take effect
 - the goal is to change cardiac output

Means of homeometric control

Sympathetic

- Positively chronotropic, inotropic, dromotropic and batmotropic effect

Parasympathetic

- Negatively chronotropic, inotropic, dromotropic and batmotropic effect

Potassium cations

- At elevated extracellular concentrations, there is a slow depolarization of the resting membrane potential (values closer to zero) and a concomitant inactivation of some sodium channels (thus preventing the action potential), ^[1], net effect is:
 - the intensity of the action potential is reduced
 - heart dilated, flaccid
 - heart rate slows down
 - in severe hyperkalaemia –up to blockage of impulse transmission through the atrioventricular bundle

Calcium cations

- opposite effect to potassium cations
- increased concentration gradually leads to spastic contraction (calcium cations initiate contraction)
- at **reduced concentration** - flaccidity

Bowditch (Treppe) effect

- see Bowditch effect

Temperature

- heat increases the permeability of the membrane to ions
- **frequency**
 - Increased temperature significantly increases frequency
 - reduced temperature significantly decreases frequency
 - in near-death hypothermia (15-20 °C) frequency only a few beats per minute
 - frequency only a few beats per minute
- **force of contraction**
 - temporarily strengthened when the temperature rises, then the metabolic system is exhausted and the contraction is weakened

Links

Related articles

- Frank-Starling mechanism (heterometric regulation of the heart)

References

1. <https://en.wikipedia.org/wiki/Hyperkalemia#Pathophysiology>

Used literature

- GUYTON, Arthur C. – HALL, John E. *Textbook of Medical Physiology*. 11. edition. Elsevier, 2006. ISBN 978-0-7216-0240-0.