MUSCLE PHYSIOLOGY

- muscle properties
- muscle contraction
- membrane potential
- spread of AP and neuromuscular transmission
- comparison of skeletal and cardiac muscle
- model organism
- experiment

MUSCLE PROPERTIES (ALL 3 TYPES)

- **Irritation** (nerve and muscle cells)
- **Conductivity** – action potential (AP)
- **Ability to contract**

muscle movement is based on ATP consumption →

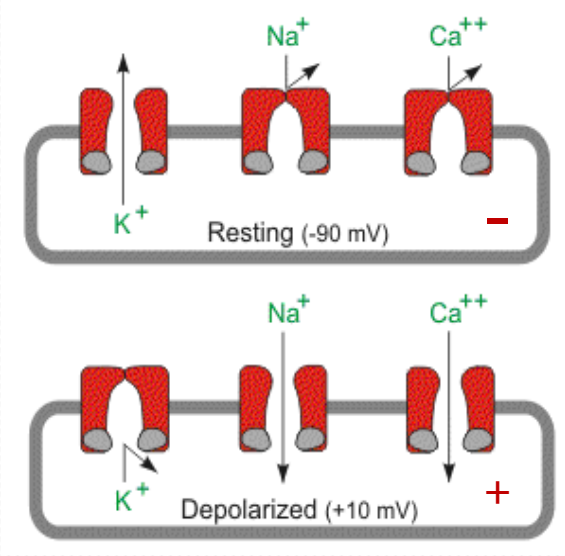
muscle cells specialize in the conversion of energy contained in ATP into contractile movement (by-product is heat)

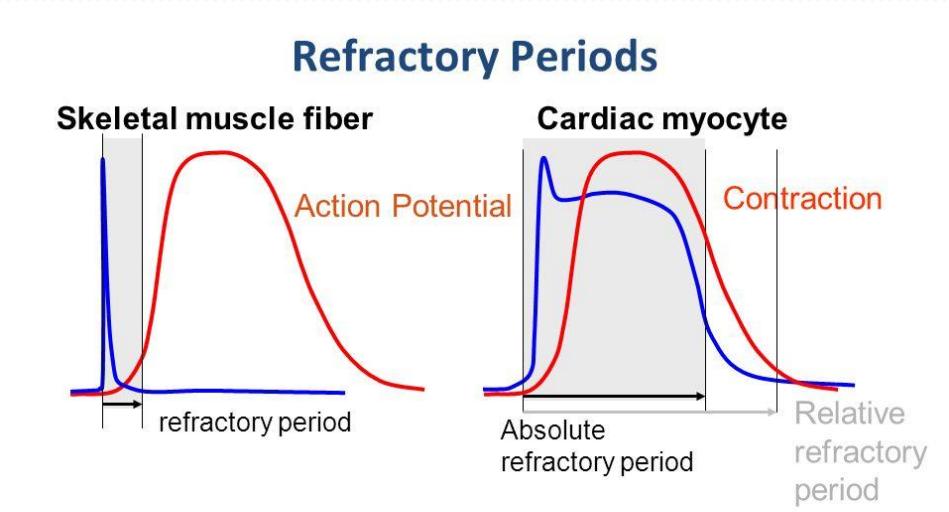
Heart:

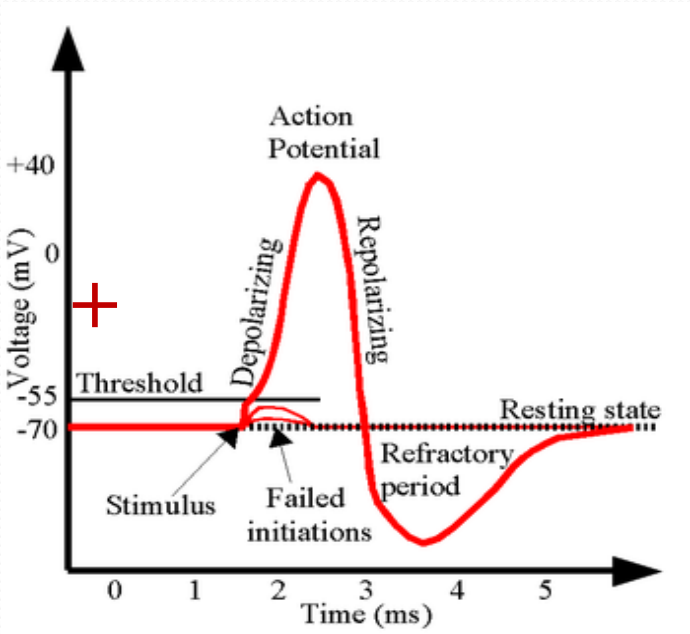
- **Automation** - independence from the CNS
- **Rhythmicity** - heart rate is controlled by pacemaker = source of excitations, AP generator

Rest and action potential

- **Polarization**- negative charge inside, positive charge outside
- When an electrical signal is received, Na flows inside the cell^{+}→**membrane depolarization**
- overshoot from - to + values =**origin of AP**
- Restoration, out flow K^{+}→**membrane repolarization (hyperpolarization)**
- **refractory period**



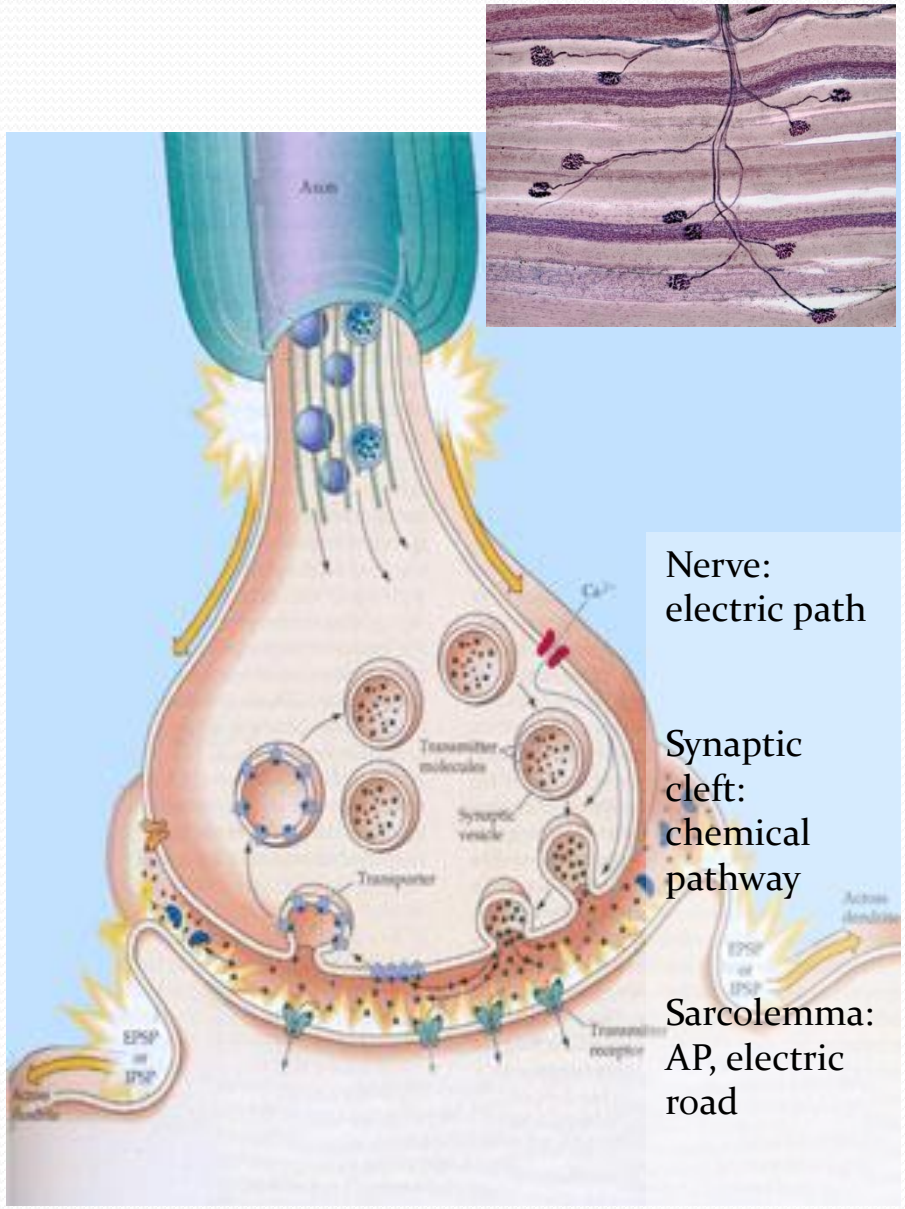




Neuromuscular disc

- Nerve - axon with myelin (Schwann) sheath, Ranvier incisions → AP spreads **saltatory** (after notches)

1. AP in the presynaptic nerve ending→ **release of Ach** from the vesicles to the synaptic cleft
2. **ACh binding to receptors**in the subsynaptic membrane (sarcolemma), degradation by acetylcholinesterase → release of receptors for other APs from the neuron
3. increased membrane permeability for Na + and K + ions (by opening ion channels), the emergence of so-called**plate potential**- it always triggers an AP that spreads **in both directions along the surface of the muscle fiber**



video

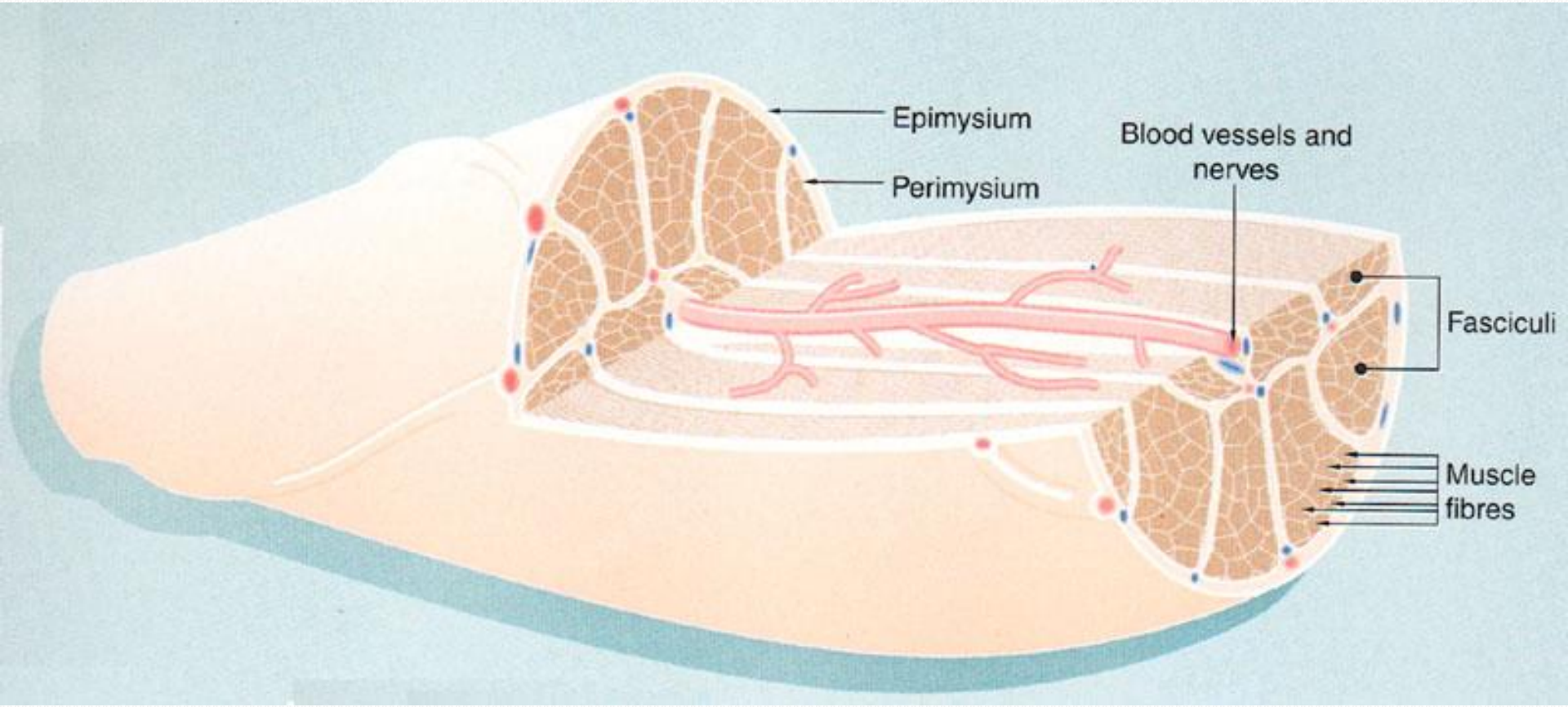
Block of neuromuscular transmission

- **Local anesthetics**- dampens Na^+channels;**muscle relaxants**- operations, etc.
- **Botulinum toxin**- blocks the release of acetylcholine
- **Kurare, snake venoms**(cobra) - binds to Ach receptors (more strongly than acetylcholine alone) but does not induce ion channel opening; death occurs by suffocation due to respiratory muscle arrest
- **Organophosphates (pesticides)**- they block cholinesterase, the disc is permanently depolarized, neuromuscular transmission is blocked

A. Structure and function of heart, skeletal and smooth muscle

Structure and function	Smooth muscle	Cardiac muscle (striated)	Skeletal muscle (striated)
Motor end-plates	None	None	Yes
Fibers	Fusiform, short (≤ 0.2 mm)	Branched	Cylindrical, long (≤ 15 cm)
Mitochondria	Few	Many	Few (depending on muscle type)
Nucleus per fiber	1	1	Multiple
Sarcomeres	None	Yes, length ≤ 2.6 µm	Yes, length ≤ 3.65 µm
Electr. coupling	Some (single-unit type)	Yes (functional syncytium)	No
Sarcoplasmic reticulum	Little developed	Moderately developed	Highly developed
Ca^{2+} "switch"	Calmodulin/caldesmon	Troponin	Troponin
Pacemaker	Some spontaneous rhythmic activity ($1s^{-1}$ – $1h^{-1}$)	Yes (sinus nodes ca. $1s^{-1}$)	No (requires nerve stimulus)
Response to stimulus	Change in tone or rhythm frequency	All or none	Graded
Tetanizable	Yes	No	Yes

Muscle building



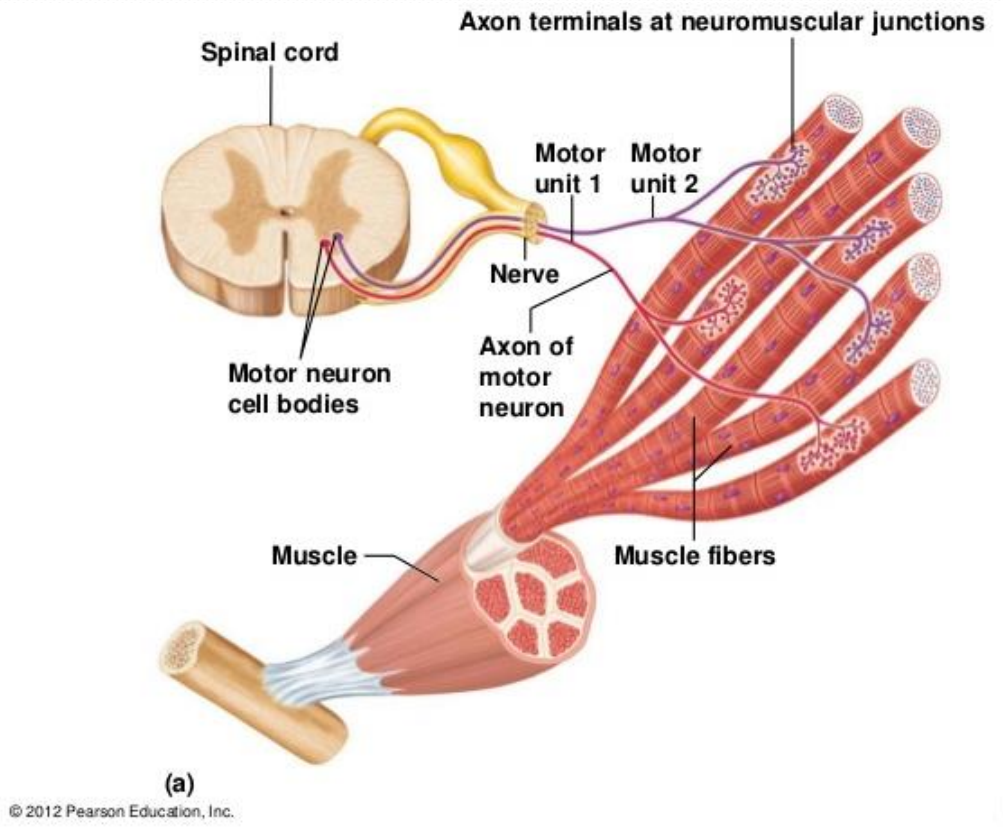
Muscle cell - myofibril

- length 1-400 mm; widthka 10-100 µm
- muscle fascia, membrane - sarcolemma,

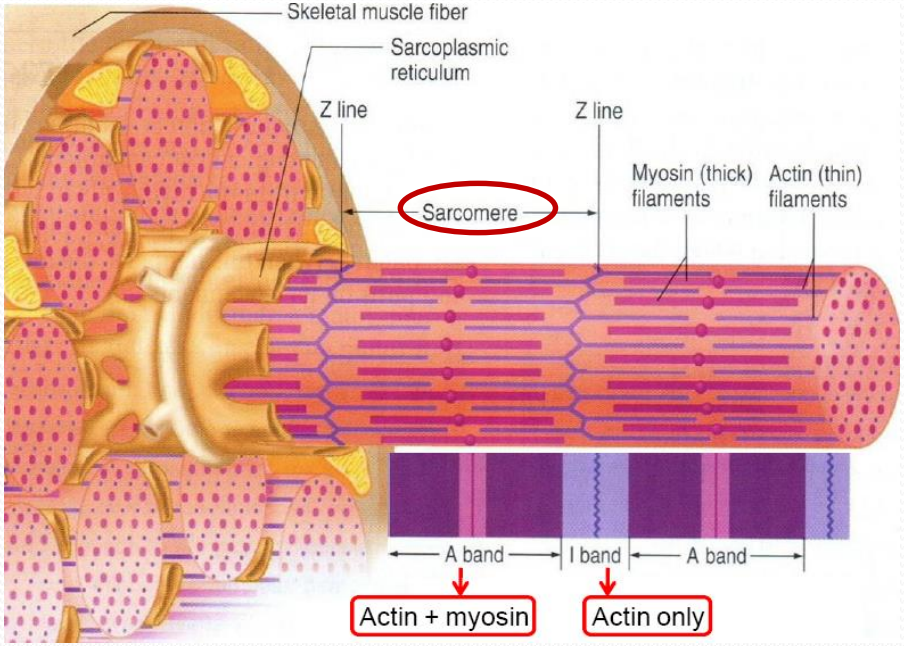
sarcoplasmic ER (sER), mitochondria - sarcosomes

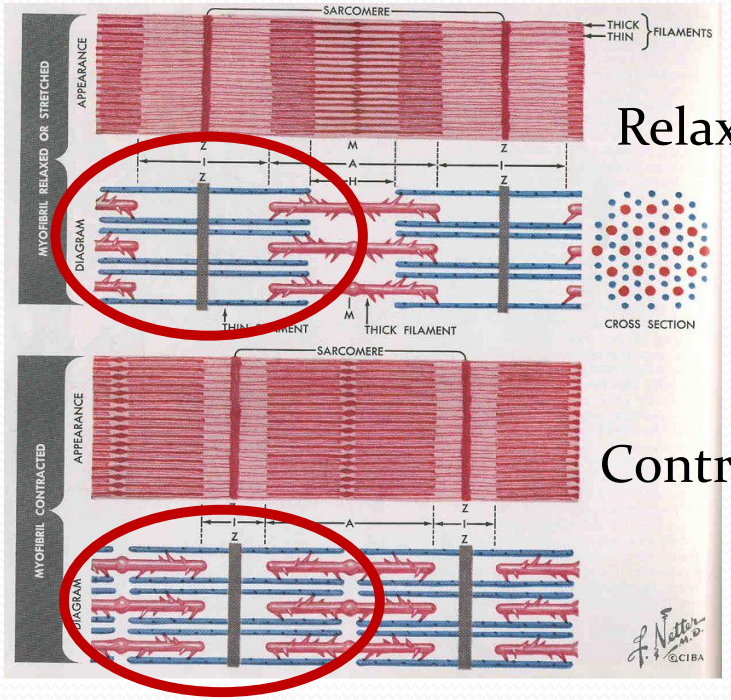
Innervation:

- **1 neuron** can innervate **more muscle fibers** (1 end for each muscle fiber)
- **motor unit** - muscle fibers + motoneuron, which innervates them, 1 muscle 10-1000 motor units
- short refractory period (ms)



- **myofibril** - own propulsion equipment
  - sarcometers - individual functional sections, Z-lines
  - actin, myosin, troponin, tropomyosin, ...

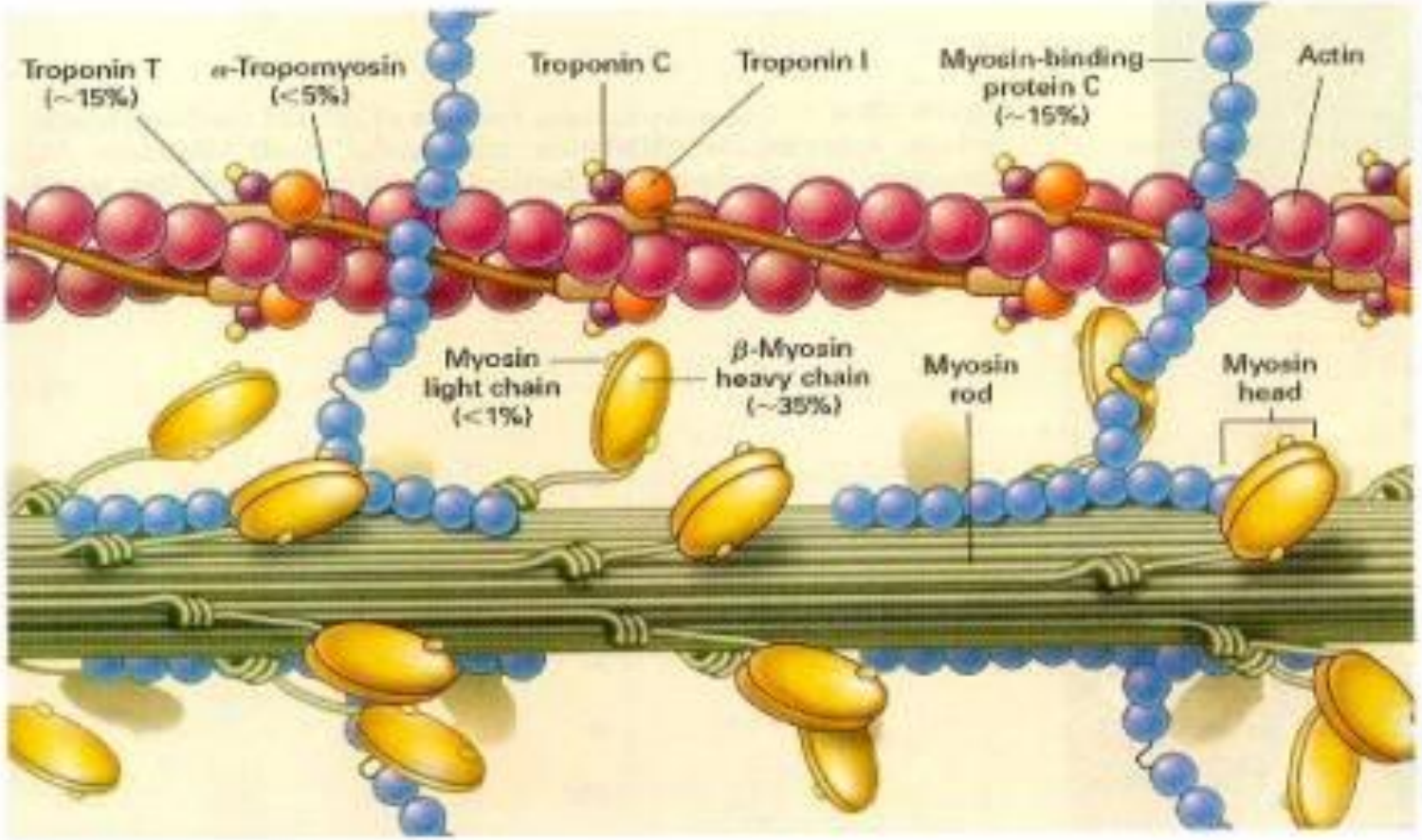




Relaxation

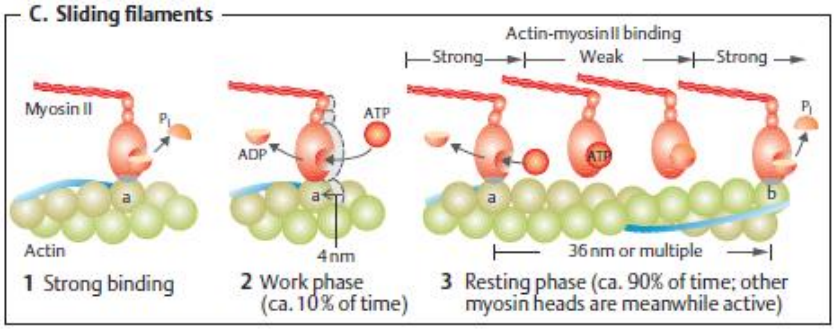
Contraction

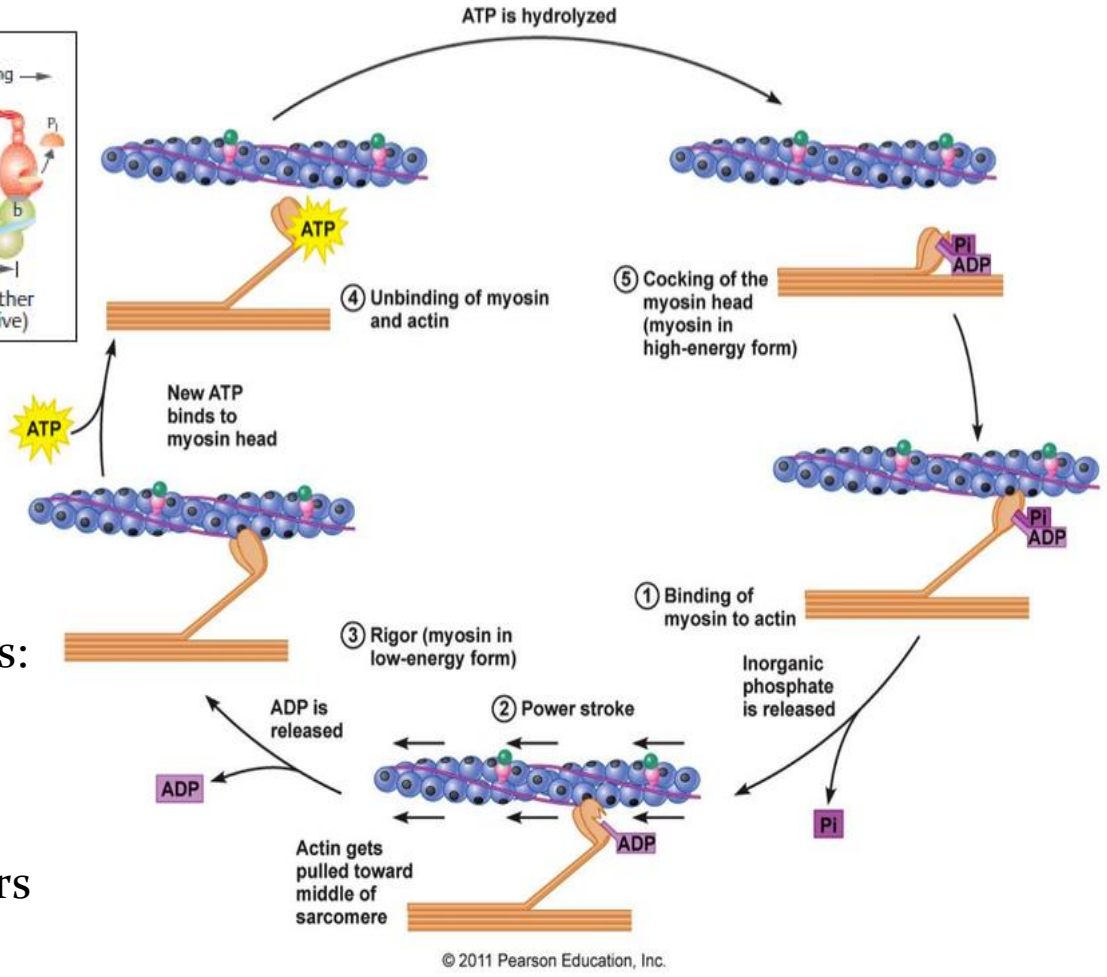
MUSCLE MULTIPROTEIN COMPLEX



http://www.edcenter.sdsu.edu/cso/paper/image005.jpg

Muscle contraction cycle

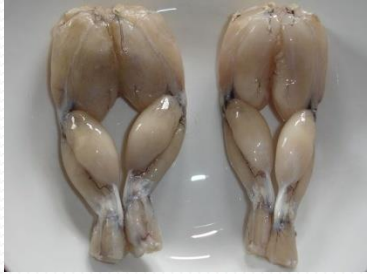




[video]

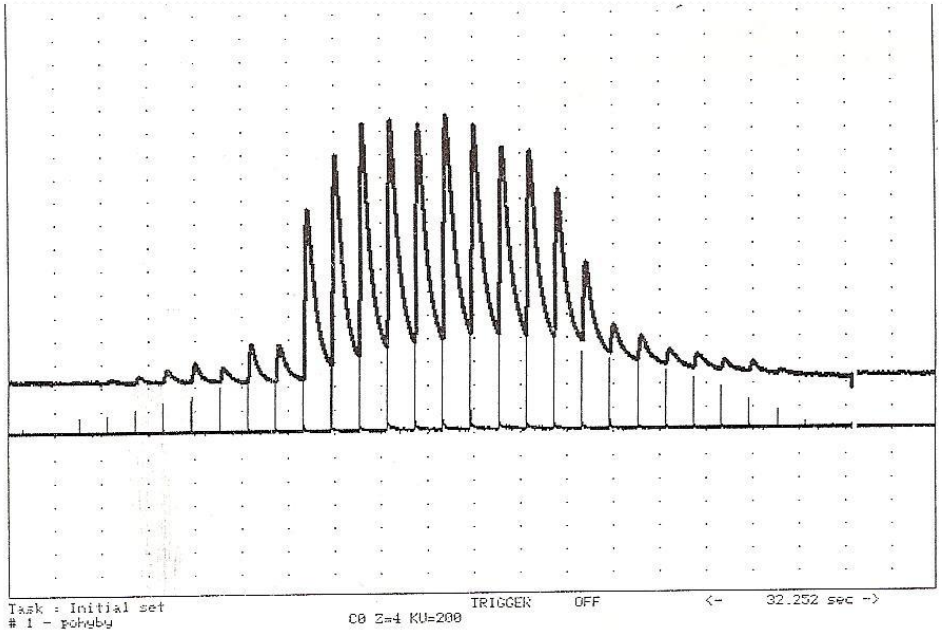
rigor mortis - postmortem stiffness: depletion of ATP stores (actin-myosin binding and Ca release^{2+} from the sarcoplasmic reticulum) about 3-6 hours after the end of delivery O_2, muscle relaxation occurs only after decomposition of myofibrils (48-60h)

EXPERIMENT



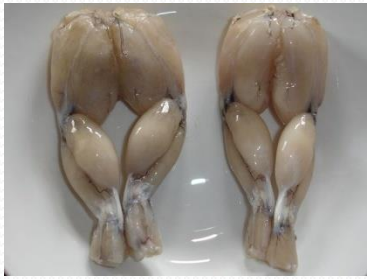
spatial summation (addition of individual pulses, change of pulse size)

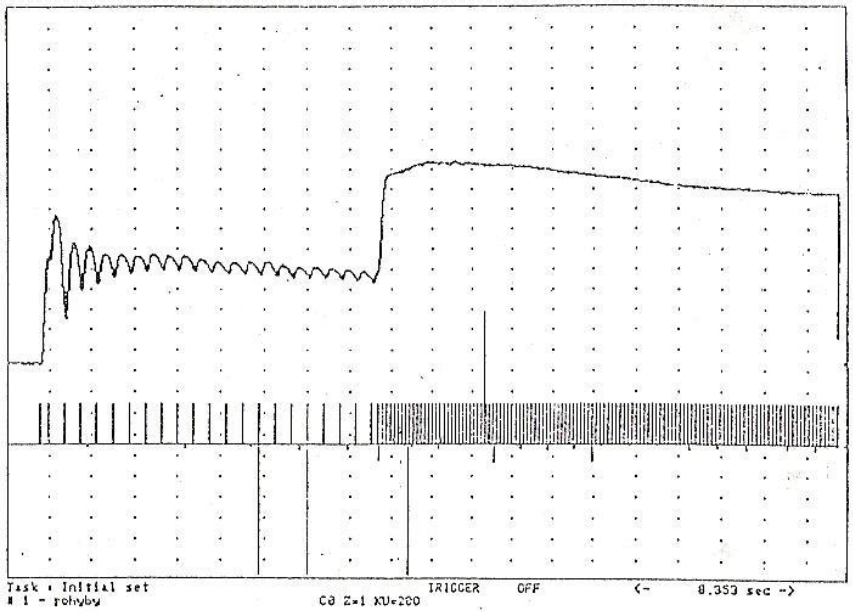
- with increasing tension (irritation of more muscle fibers) the contraction of the muscle increases → graduated answer at the stimulus (at the heart "all or nothing")

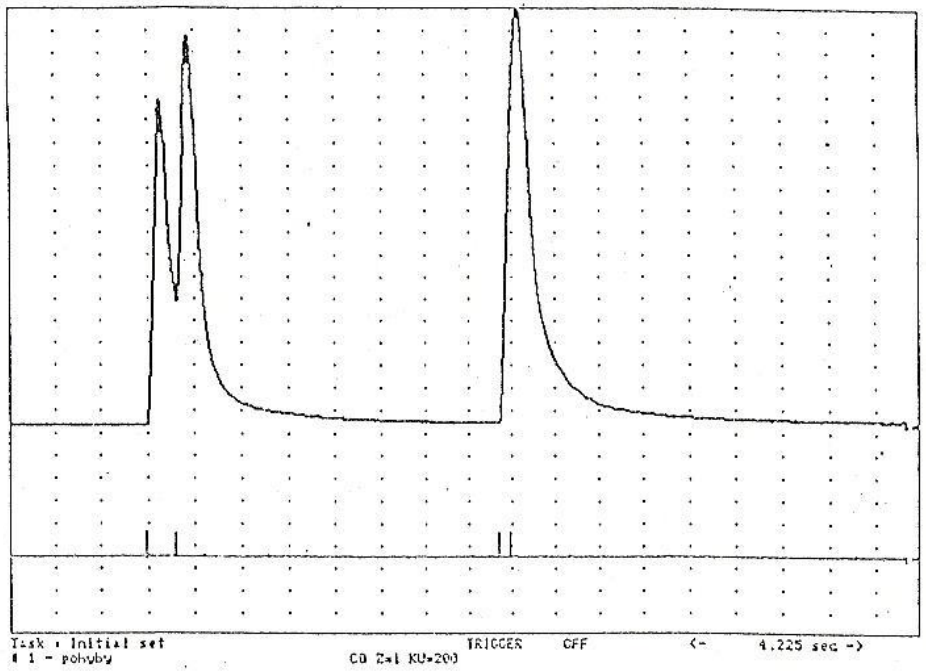


subthreshold, threshold and above-threshold stimuli

- **time summation** (2 stimuli in quick succession) - pulse frequency changes; the muscle cannot return to its original position and vibrates at the top wavy and corrugated and smooth tetanus (**tetanic contraction** = permanent muscle contraction)
- **superposition** - two stimuli later in a row







wavy tetanus smooth tetanus

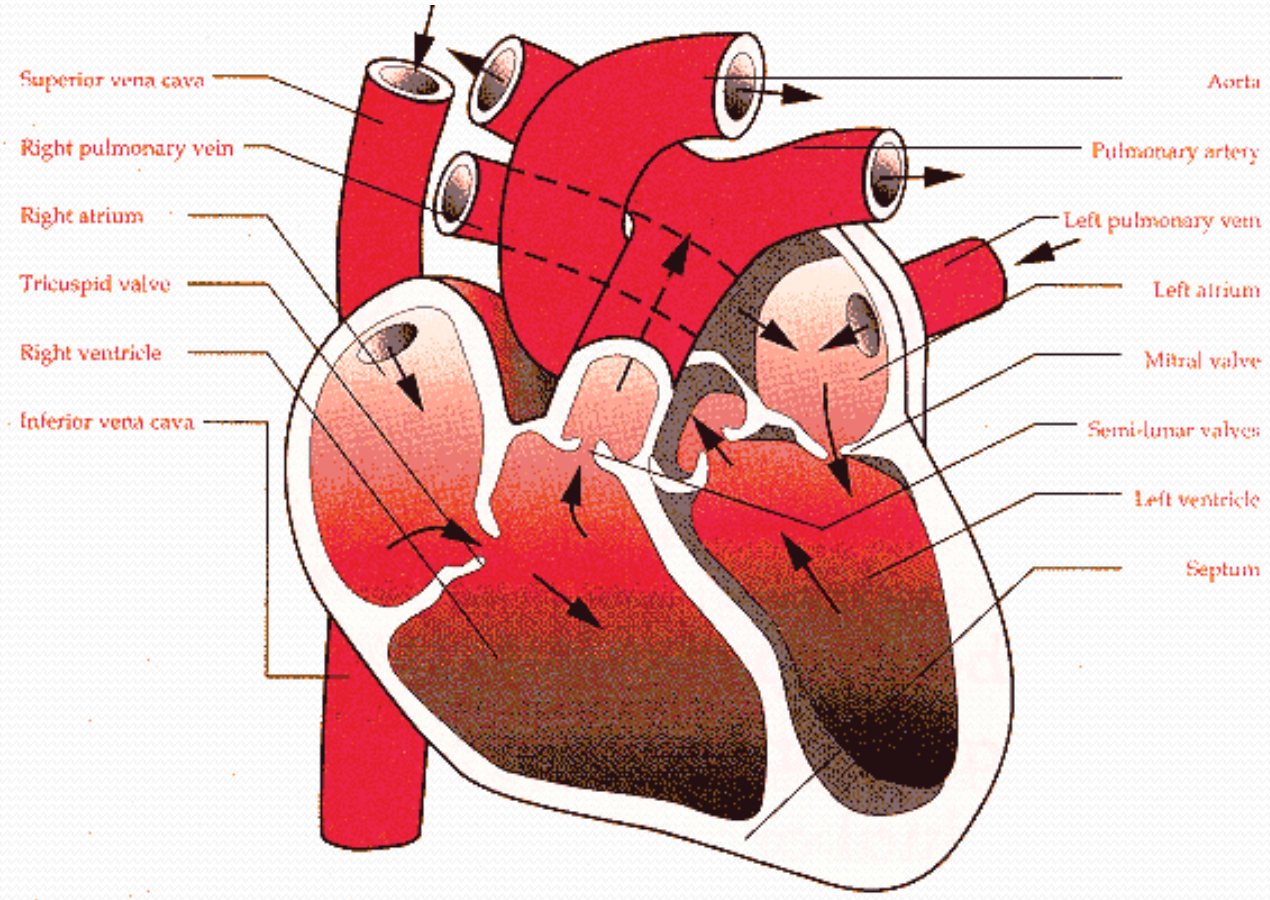
calf skeletal muscle irritation

superposition after second stimulus, refractery period

Conclusion: Demonstration of phenomena characterizing skeletal muscle.

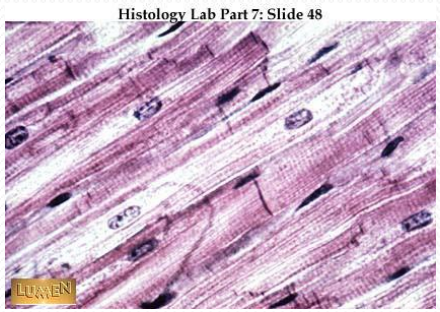
HEART

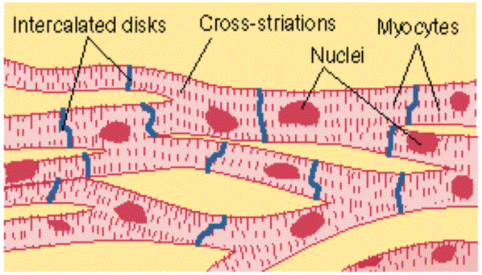
- **endocardium** (inner membrane)
- **myocardium** (own muscle layer of the heart)
- **epicardium** (outer membrane)
- **pericardium** - covers the heart



Heart muscle

- the mechanism of contraction is the same as for skeletal muscle
- of **mononuclear** forked **cells, functional syncytium**
- cells separated **intercalary discs** (stair partitions)
- spreading irritation through nexus (**gap junctions**), responds to hormones (eg adrenaline)
- many sarcosomes (Ca^{2+}) and less myofibrils
- **automation**(contains natural pacemakers - SA and AV nodes) - HF Stannius
- **long refractory period** (min. 250 msec)





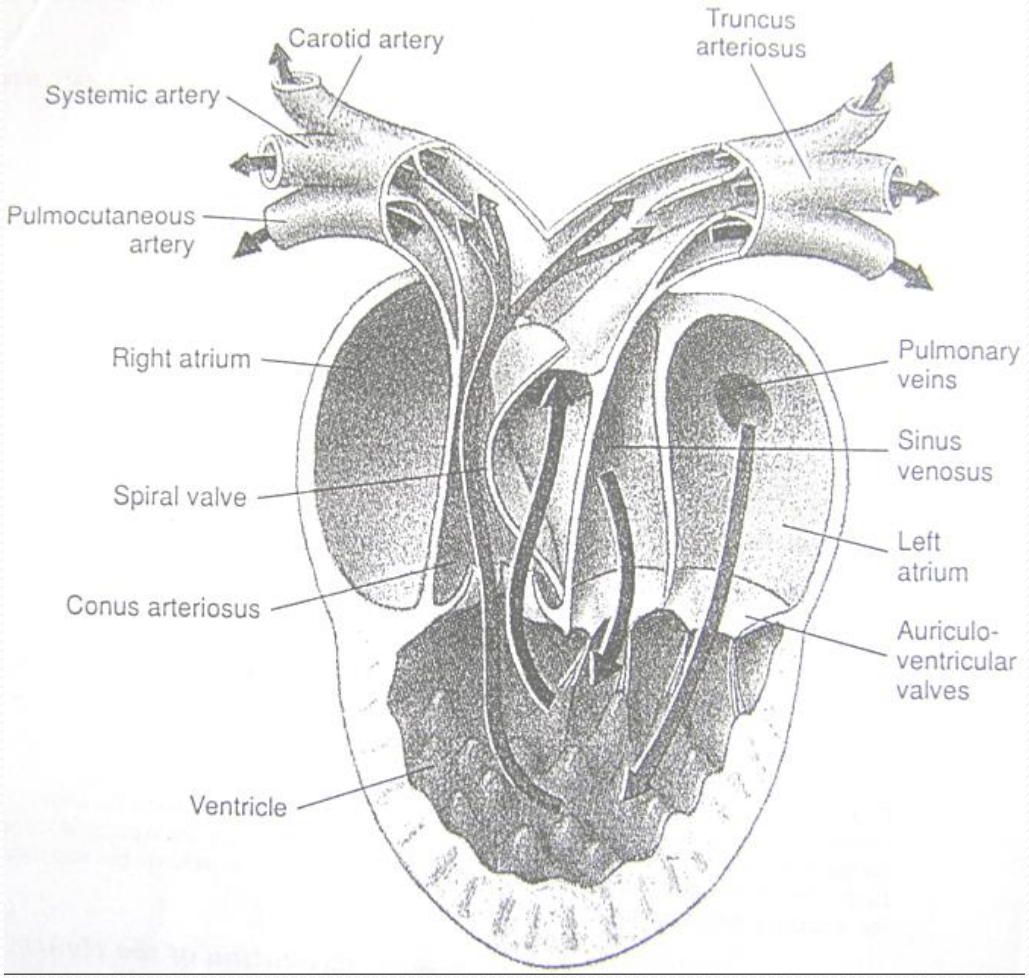
Cardiac muscle innervation

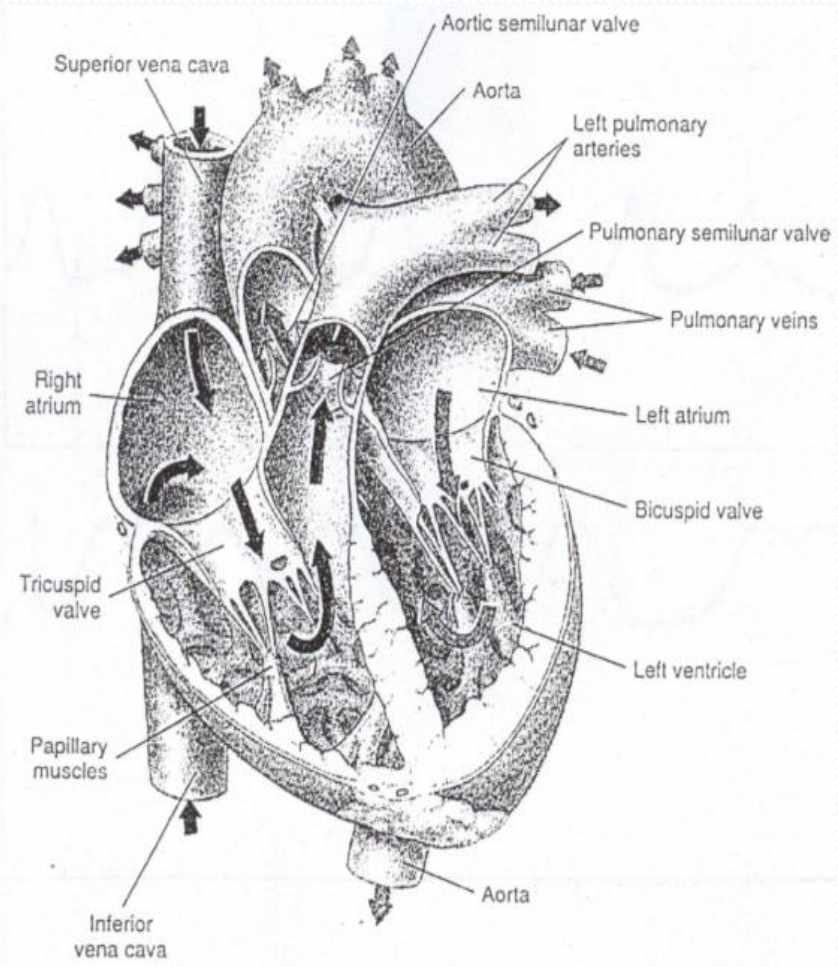
- **afferent innervation**- nerves perceive contraction or filling of the heart compartments; pain (during a heart attack)
- **efferent innervation**- controls the frequency and strength of contractions, the speed of conduction in the heart, the irritability of the heart:
  - **parasympathetic** (*n. vagus*- innervates the SA- and AV-nodes) mediator acetylcholine, which **muffles** heart activity
  - **sympathetic**(*accelerating nerve cordis,*innervates the whole heart) mediator norepinephrine, **encourages** heart activity; adrenaline from the adrenal medulla has a similar effect

- **tachycardia**- accelerated heart rate
- **bradycardia**- slower heart rate
- **fibrillation**- rapid chaotic superficial contractions of the heart muscle, the heart does not pump blood

XENOPUS LAEVIS

- laboratory amphibian (jumpers, toads - law protected)
- females 10-15 cm, males 1/3 smaller
- flattened body without neck and tail, strong hind legs, no front membranes on the front, smooth skin, pale belly
- comes from South and South Africa
- stagnant waters, ponds, swamps, limestone lakes
- exclusively aquatic animal
- hormone **choriogonadotropin** causes egg laying in the claws - embryology, pregnancy tests (early human embryo and placenta produce hCG, it is then in the blood and urine - blood is injected into the frogs, they lay eggs in the morning = pregnancy)
- hibernation for up to 10 months

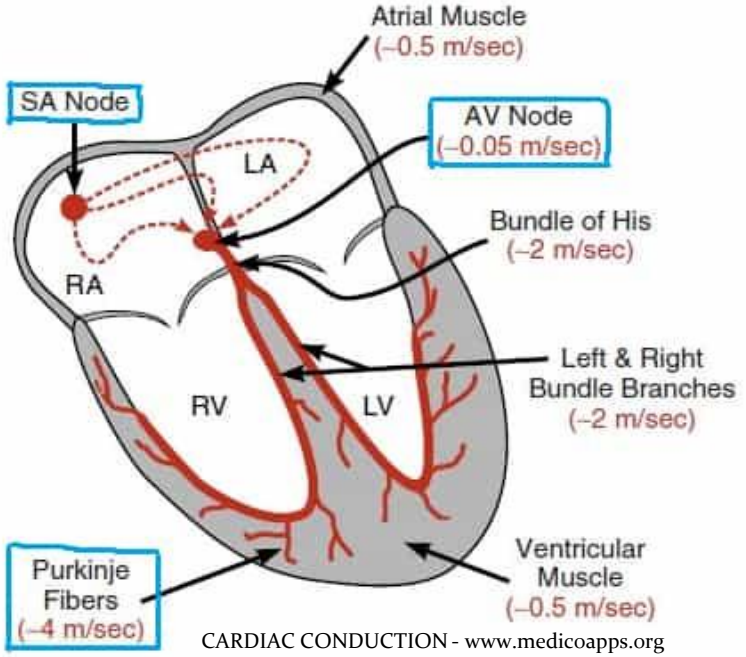

Carotid artery
Truncus arteriosus
Systemic artery
Pulmocutaneous artery
Right atrium
Pulmonary veins
Sinus venosus
Spiral valve
Left atrium
Conus arteriosus
Auriculo-ventricular valves
Ventricle


Aortic semilunar valve
Superior vena cava
Aorta
Left pulmonary arteries
Pulmonary semilunar valve
Pulmonary veins
Right atrium
Left atrium
Bicuspid valve
Tricuspid valve
Left ventricle
Papillary muscles
Aorta
Inferior vena cava

NATURAL HEART AUTOMATION CENTERS

- contain cells that spontaneously depolarize with a certain frequency

- sino-atrial node = Keith-Flack = **SA node** (*sinus venosus* - venous rafting); the main pacemaker, is the superior, responsible for rhythmicity, controls mainly the activity of the xxxxxxxx atriums
- atrio-ventricular node = Aschoff-Tawar = **AV node** = atrial node (*atrium cordis* - atrium, *ventriculum cordis* - ventricular chamber) - it would not keep the rhythm alone, it mainly controls the activity of the ventricles

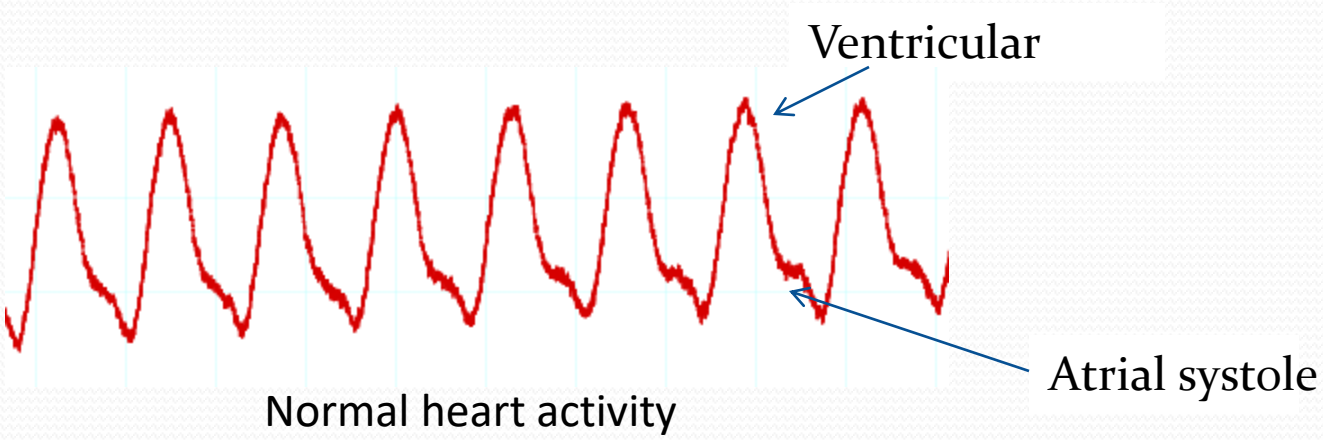


Experiment - heart muscle

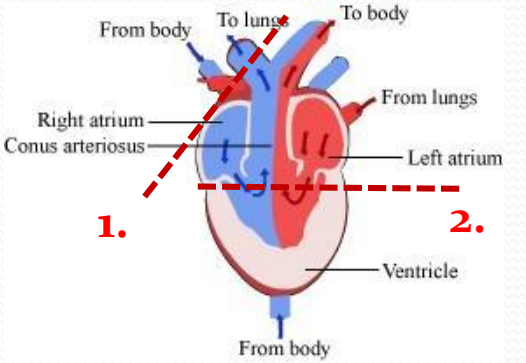
In situ
- stunning, decapitation, brain removal (CNS) and spinal cord disruption (reflexes), **the heart beats independently of the CNS**

- **Heart muscle contractions**

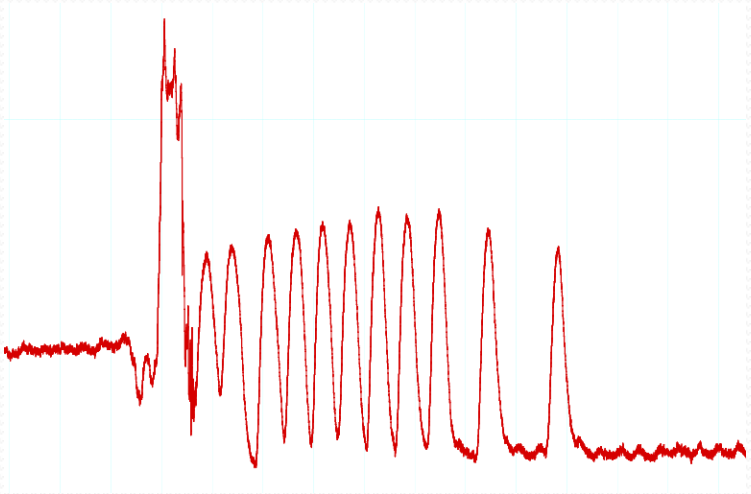
systole (contraction), diastole (release) of the heart muscle
1. SA node → atrium → 2. AV node → chamber ventriculum

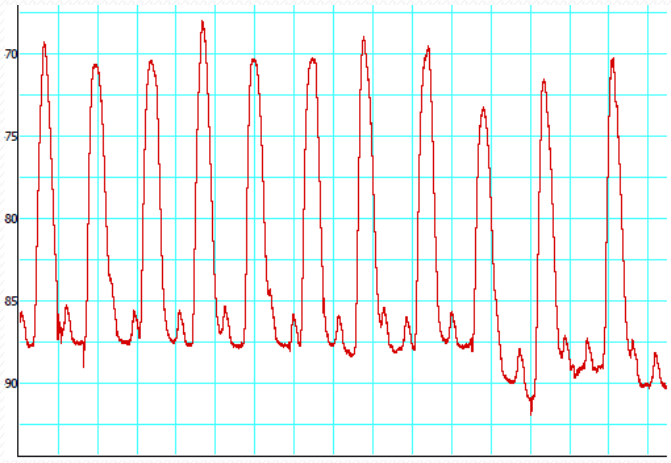


Experiment - heart muscle



- **1. Stannius's ligature**(ligation, strangulation) = interruption of conduction from the SA node by strangulation of the hall immediately behind the node, the heart stops, or we register only weak contractions *sinu venosu*(**SA node → cardiac arrest**)
- **2. Stannius's ligature**- constriction at the site of the AV node, mechanical irritation of the AV node (**AV node → the chamber is retracted**) ventriculum
- after a while the heart stops beating, external stimulation el. current



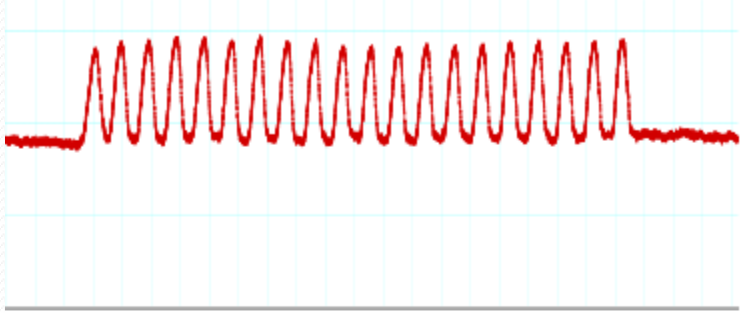


Normal - two pacemakers = two rhythms

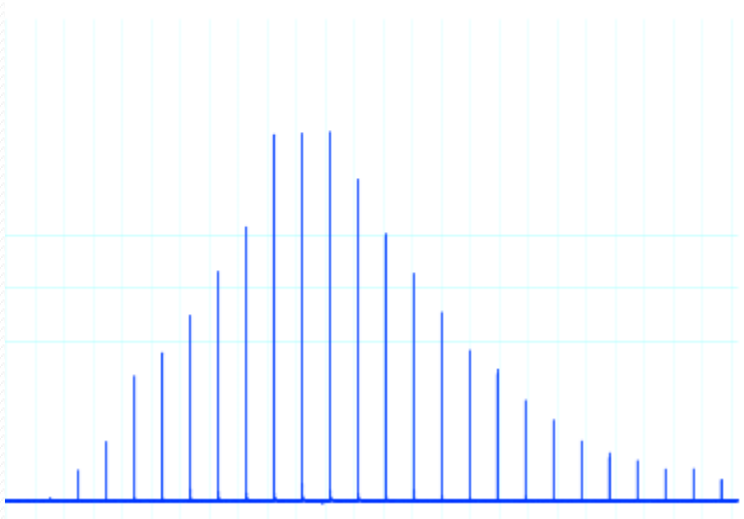
Is it possible to change the force of the contraction?

- **1. spatial summation** - change the size of the pulses - the heart is either contracted or relaxed - regardless of the increase in the intensity of the stimulus → answer "**All or nothing**"

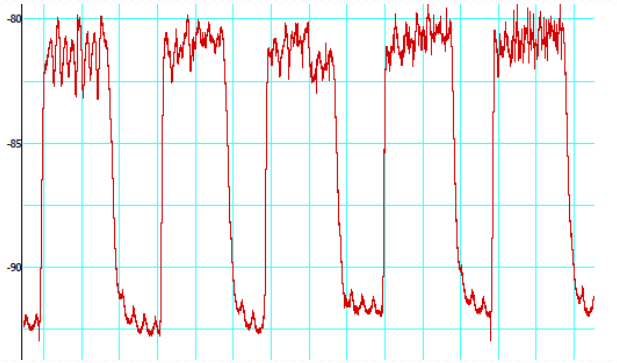
Cardiac activity



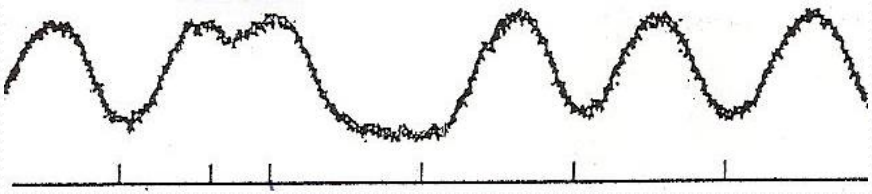
Stimulus intensity

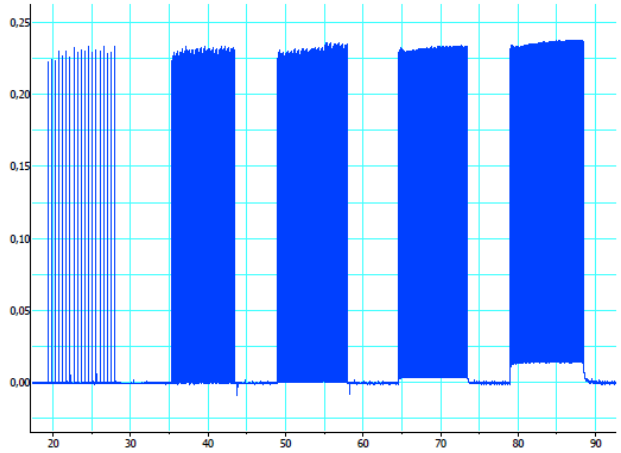


- **2. time summation**- pulse frequency change → wavy tetanus (fibrillation) - period 0.5 s; smooth tetanus cannot be induced in the heart - long absolute refractory period



- **extra systoly** - two stimuli in a row





Conclusion: Demonstration of phenomena characterizing the heart muscle

What are the functions and use of myostimulator?

Transcutaneous Electrical Nerve Stimulator. The electrical impulses generated by the device penetrate the body through adhesive electrodes placed on the surface of the skin.