Physiology of the heart II.

The Frank-Starling law of the heart
Cardiac electrophysiology

(Learning objectives 37-38.)

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Blood volumes during the cardiac cycle

- end-diastolic volume (EDV) : 110-160 ml
- stroke volume (SV) : 70 - 80 ml
- end-systolic volume (ESV) : 40 - 80 ml
- ejection fraction: SV / EDV ~ 50 - 70%

increasing stroke volume:
  increasing the EDV and/or decreasing the ESV
The heart as a pump

- stroke volume x heart rate = cardiac output

- **cardiac output** = blood volume leaving the ventricle in 1 minute
  \[ \sim 5.5 \text{ l/min} \]

- **cardiac index** = cardiac output/body surface
  \[ \sim 3.1 \text{ l/m}^2/\text{min} \]

*Figure 9-1. Structure of the heart and course of blood flow through the heart chambers.*
**Figure 9–7.** Relationship between left ventricular volume and intraventricular pressure during diastole and during systole. Also shown by the heavy red lines is the “volume-pressure diagram” that illustrates the changes in intraventricular volume and pressure during the cardiac cycle.

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**Preload and Afterload**

Preload: volume entering ventricles

Afterload: resistance left ventricle must overcome to circulate blood
Adaptation to:

increased venous return (preload):
  change in body position, respiration...

increased peripheral resistance (afterload)
  pressure increases in the aorta

sympathetic stimulation (positive inotropic effect)

diastolic and systolic reserve volume

Frank Starling law of the heart

- stroke volume of the heart increases in response
to an increase in the volume of blood filling the heart (the end diastolic volume)
  or
- the strength of the heart's systolic contraction is directly proportional to its diastolic expansion (end diastolic volume)
resting length (diastolic volume) determines the contraction force
„Frank-Starling mechanism“
Ca++ sensitivity increases (Ca++ channels?)
works also in isolated heart

contraction force may increase without change in length
„positive inotropic effect“
background: intracellular Ca++ increases
effect of sympathetic stimulation

Rhythmic excitation of the heart

• special features: automatic and rhythmic
• where is it coming from?
  Stannius, the ligatures and the frog heart
The Stannius ligature

![Diagram of heart with labels: ventricle, right atrium, left atrium, truncus arteriosus, sinus venosus.]

Rhythmic excitation of the heart

- special features: automatic and rhythmic
- Stannius and the frog heart
- nomotop (natural) pacemaker (SA node)
- heterotopic (ectopic) pacemakers
- extrasystole
• fast AP (atrium, ventricle, Purkinje fibers), more negative, steeper,
  larger amplitude → Na channels

• slow AP (SA and AV node), less negative, less steep,
  smaller amplitude → no voltage dep. Na channels

• conduction speed of fast APs: 0.3-4 m/s,
• conduction speed of slow APs: 0.02-0.1 m/s
plateau phase
repolarization
fast depolarization
time (ms)
potential (mV)
membrane
intracellular
extracellular
permeability

Na⁺ 12 mmol/l
Ca²⁺ <0.015 mmol/l
K⁺ 150 mmol/l

0 145 mmol/l
1.25 mmol/l
4 mmol/l
time (ms)

Na⁺ Ca²⁺ K⁺
0 145 300
0 1,0 10

slow AP
actual pacemaker
maximal diastolic potential
slow diastolic depol. (pacemaker pot.)
threshold

what ions ???

fast AP
myocardium
resting pot.
threshold

resting pot.
sympathetic β1 receptor stimulation

heart rate decreases during vagal nerve stimulation

ACh
Regulation of the cardiac pumping function

1. Frank - Starling mechanism: intrinsic regulation
2. Neural regulation
   - parasympathetic- (n. vagus – ACh - muscarinergic)
     - negative chronotropy, dromotropy, bathmotropy
     - inotropy (not in the ventricles)
   - sympathetic- (NA- β1 receptor)
     - positive chronotropy, dromotropy, bathmotropy
     - inotropy, lusitropy + vasodilation
3. Humoral: Adrenalin, NA
4. Effect of Ca²⁺ and K⁺
Effect of membrane resting potential on depolarisation speed