

CARDIOVASCULAR PHYSIOLOGY

E-C coupling. Heart as a pump.

EXCITATION – CONTRACTION COUPLING

Events connecting electrical activity of the heart (depolarization of sarcolemma), **EXCITATION**

and its mechanical response, CONTRACTION

Link between these events is Ca²⁺

FUNCTION

- Triggering of contraction
- Regulation of contraction force



KEY STRUCTURES / ORGANELLES / RECEPTORS

- Transverse tubules of sarcolemma (T-tubules)
- Sarcoplasmic reticulum (SR)
- Calcium channels:

DHPR (dihydropyridine receptor) in T-tubules/sarcolemma

RYR (ryanodine receptor) in SR

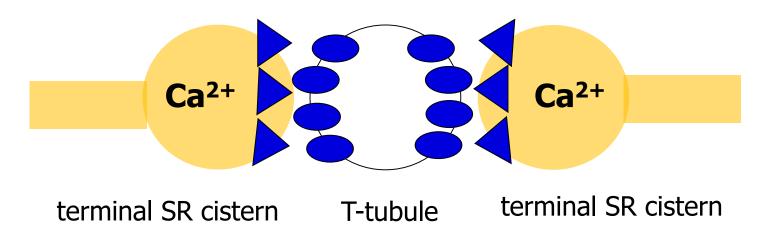




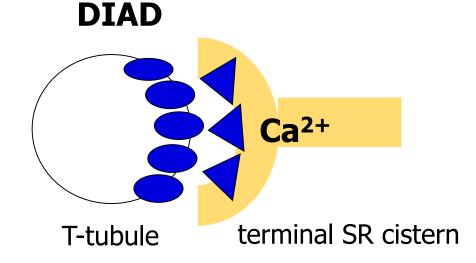
FUNCTIONAL ORGANISATION of SARCOTUBULAR SYSTEM

RHABDOMYOCYTE

TRIAD



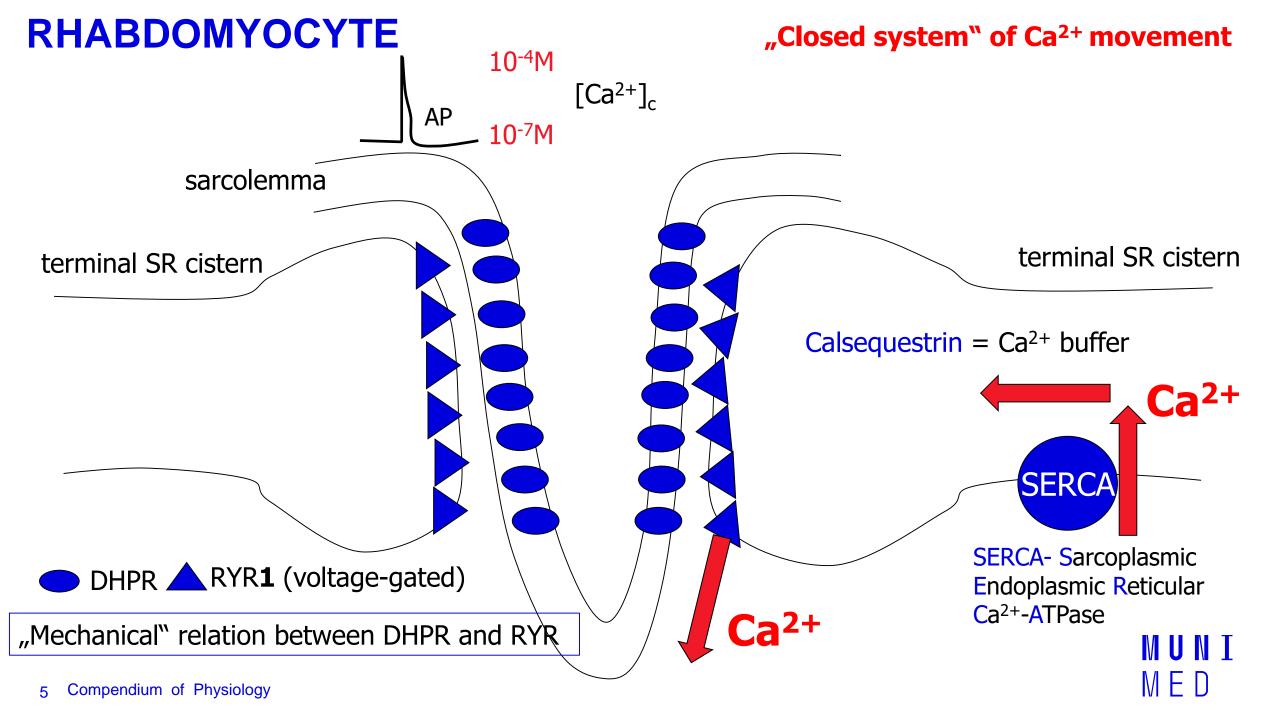
CARDIOMYOCYTE

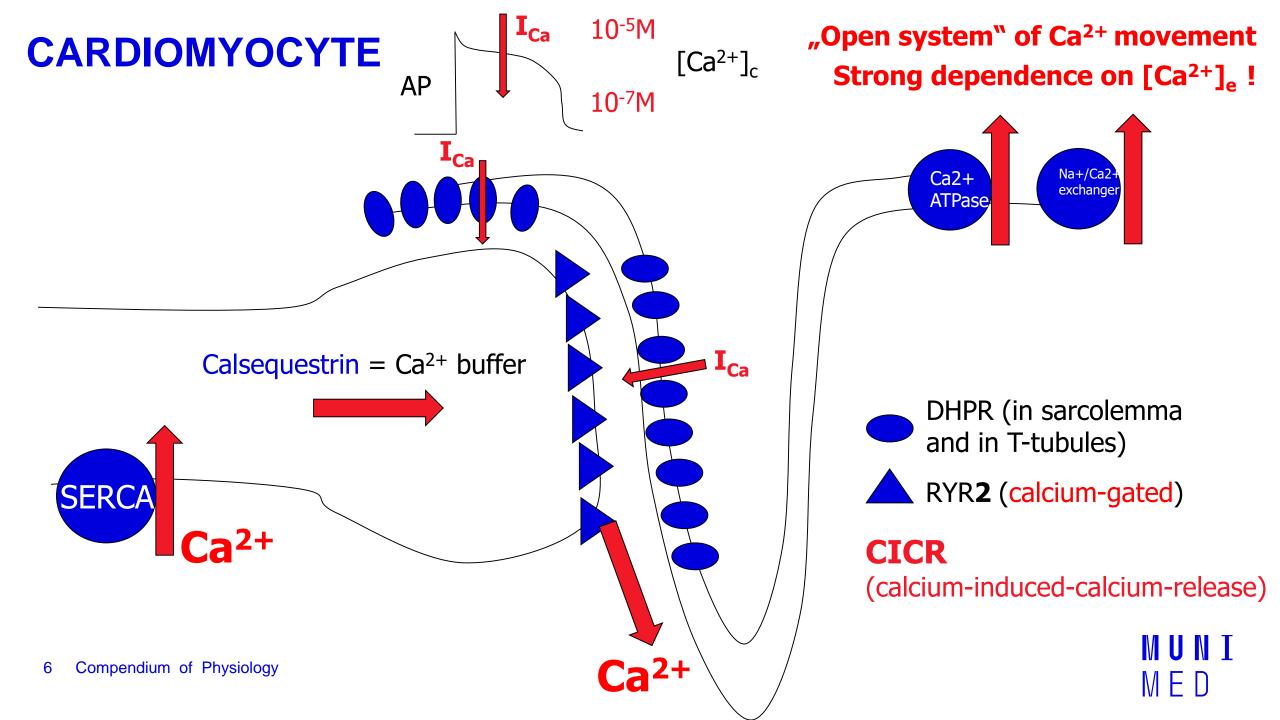










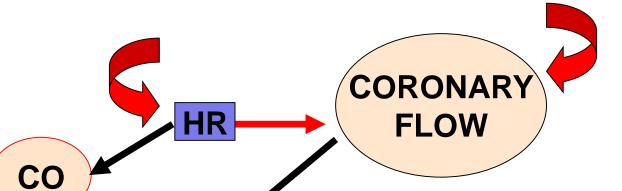


HEART AS A PUMP



CARDIAC OUTPUT (CO)

LV = RV



 $CO = HR \times SV$

5l/min

SV = EDV - ESV

70ml

>60%

Ejection fraction

EF = EDV - ESV / EDV

Venous return Compliance Aortal pressure

CONTRACTILITY

Ability to contract
Depends on tissue perfusion
(substrates and oxygen supply for
ATP production; Ca2+ availability)

AUTOREGULATION of cardiac contraction

Heterometric: Starling law

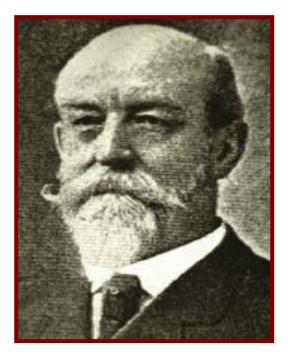
Homeometric: Frequency effect

EDV

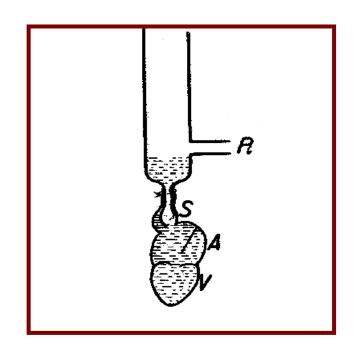


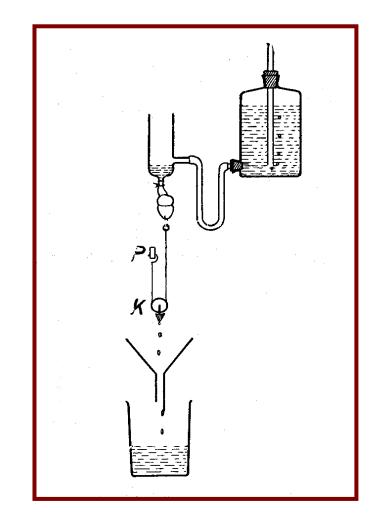
ANS

REGULATION



Henry Pickering Bowditch (1840 – 1911)





HOMEOMETRIC AUTOREGULATION

(FREQUENCY EFFECT)

During increasing HR (stimulation frequency) the force of developed contraction rises

Ratio between intra- and extracellular calcium concentrations increases



CARDIAC RESERVE = maximal CO / resting CO

4 - 7

CORONARY RESERVE = maximal CF / resting CF

3,5

CHRONOTROPIC RESERVE = maximal HR / resting HR

3 - 5

VOLUME RESERVE = maximal SV / resting SV

1,5

CO = cardiac output

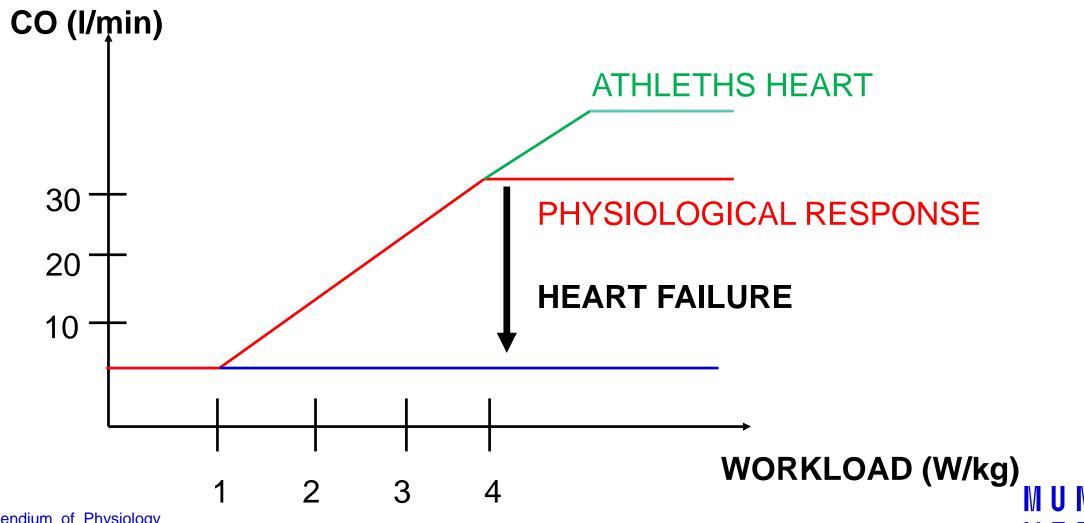
CF = coronary flow

HR = heart rate

SV = stroke volume



CARDIAC RESERVE



AFTERLOADED CONTRACTION AP

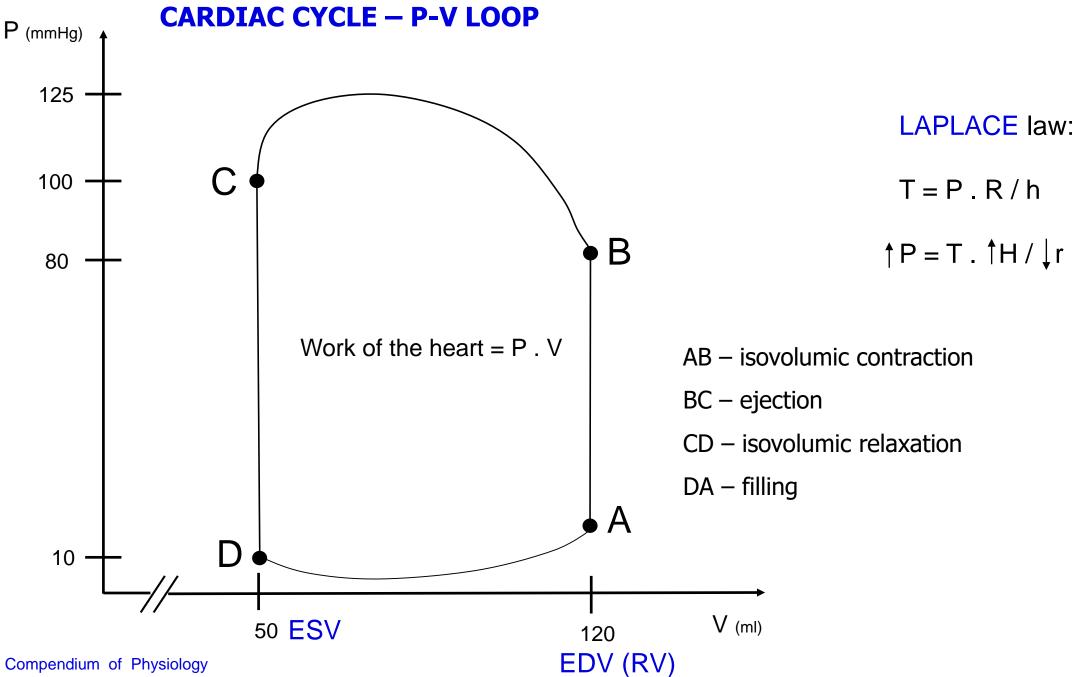
PRELOAD, AFTERLOAD

PRELOAD
(~ enddiastolic filling)

AFTERLOAD

(~ pressure which must be developed)





HEART SOUNDS

Caused by vibration of various anatomical structures and event. blood:

- Closure and stretching of valves
- Isovolumic contraction of heart muscle (papillary muscles, tendons)
- Turbulent blood flow

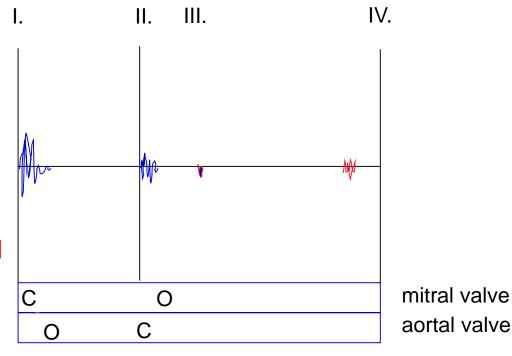
I. – mitral (+ tricuspidal) valve closure

II. - aortal (+ pulmonary) valve closure

III - fast filling of ventricles - pathological

IV. - contraction of atria – mostly pathological

Vibration of ventricular wall



O - open, C - closed



MURMURS – pathological phenomena based on turbulent blood flow

1. SYSTOLIC

- Stenosis aortal, pulmonary (1)
- Regurgitation mitral, tricuspidal (2)

2. DIASTOLIC

- Stenosis mitral, tricuspidal (3)
- Regurgitation aortal, pulmonary (4)

3. SUSTAINED:

Defects of septum

