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MED

CARDIAC MECHANICS

HEART AS A PUMP

CARDIAC CYCLE

HEART FAILURE

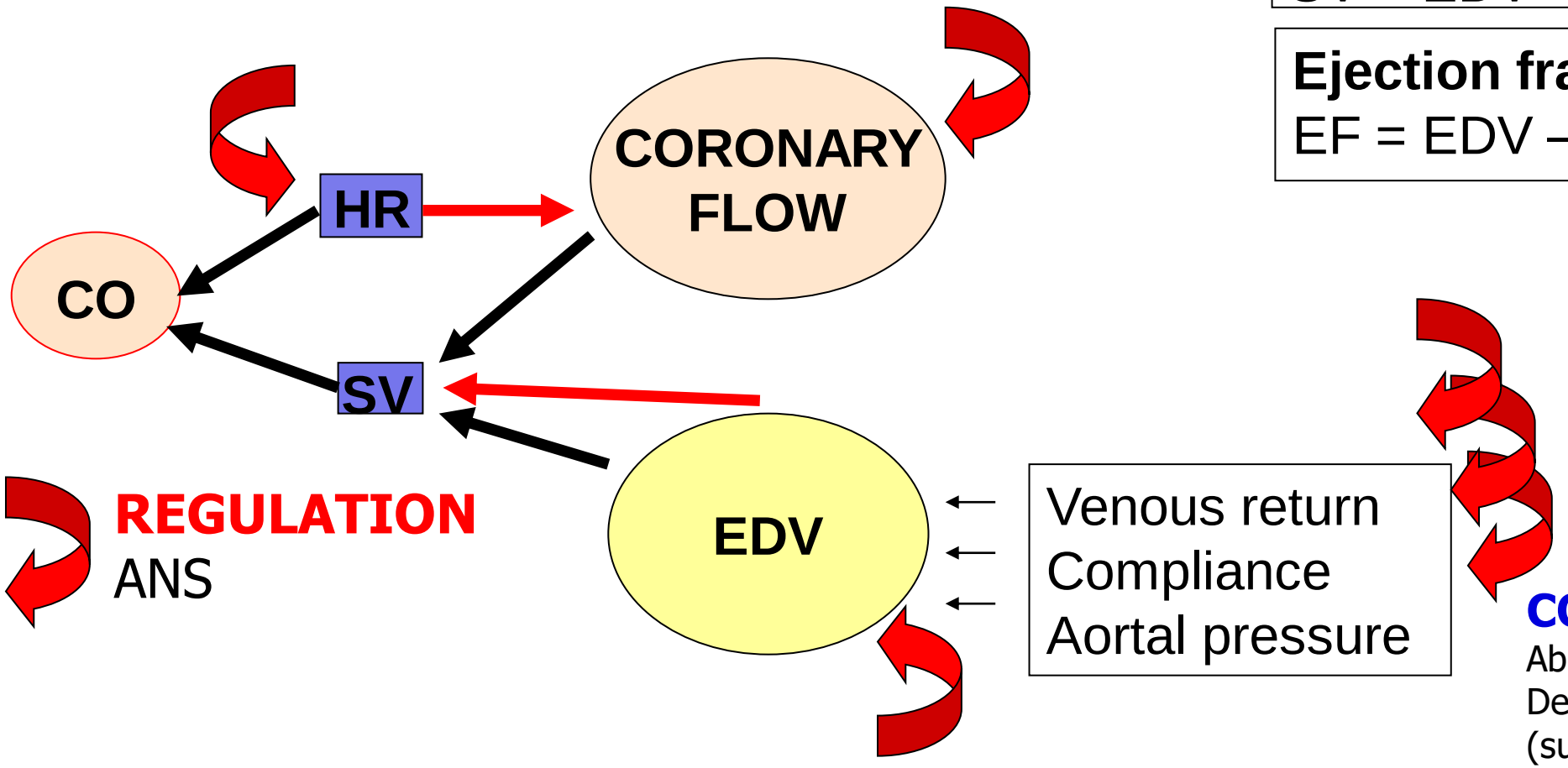
CARDIAC OUTPUT (CO)

LV = RV

CO = HR x SV 5l/min

SV = EDV - ESV 70ml

Ejection fraction >60%
EF = EDV – ESV / EDV



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

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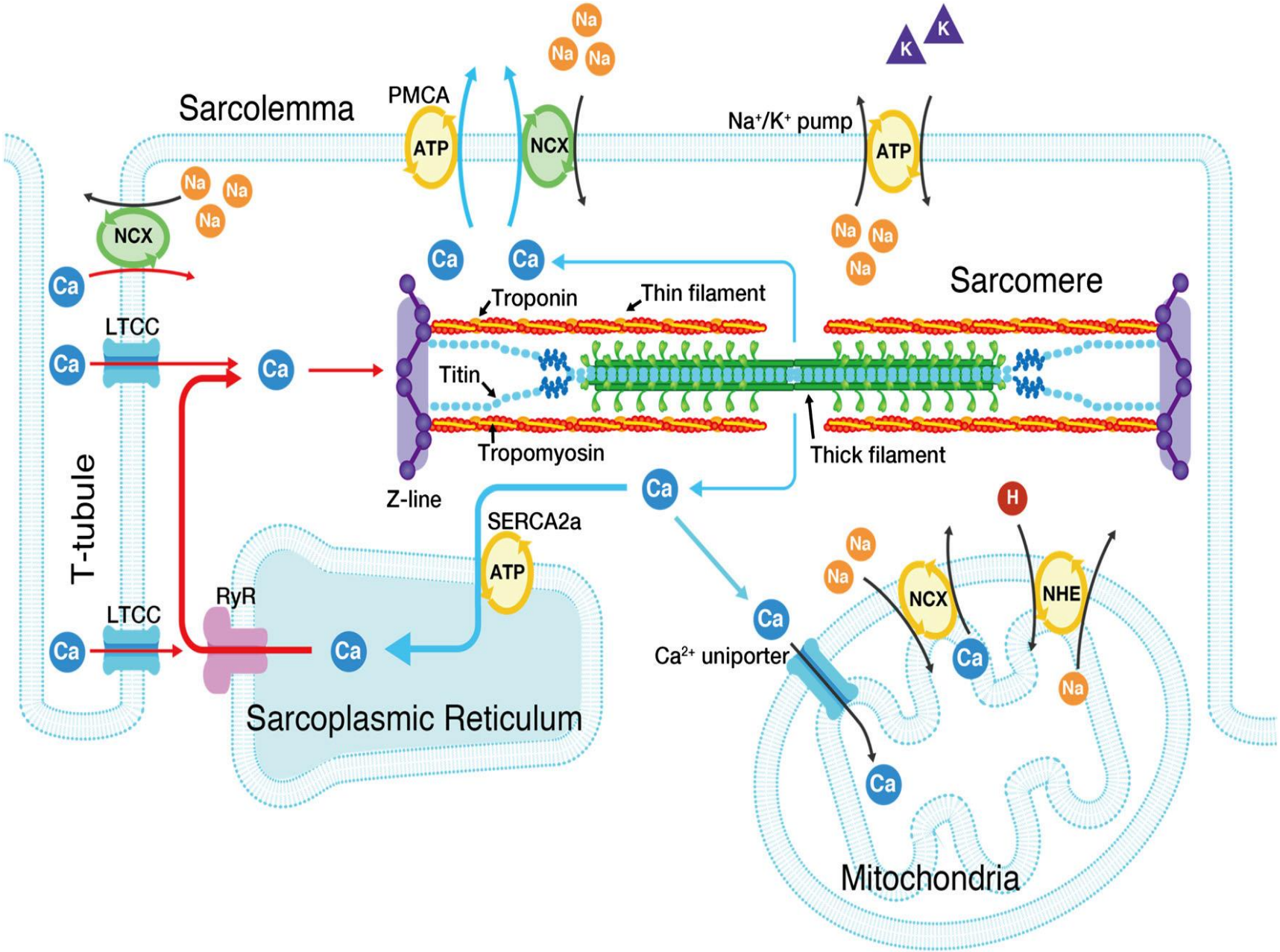
STARLING LAW



Fuyu Kobirumaki-Shimozawa et al., J Physiol Sci (2014) 64:221–232

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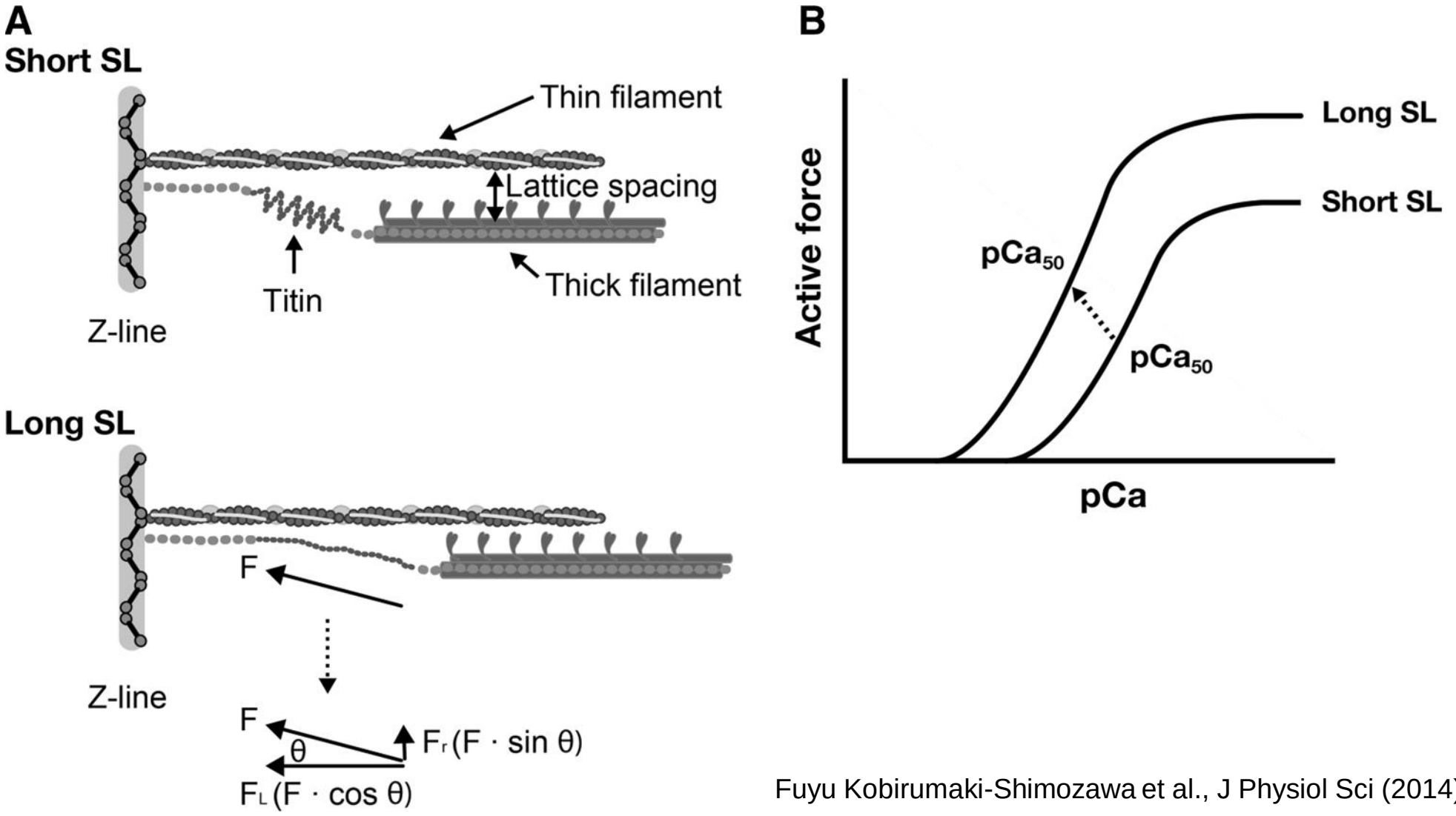

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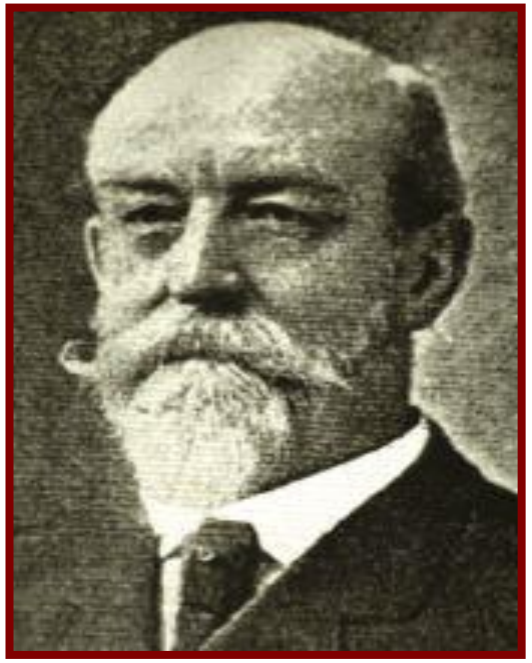
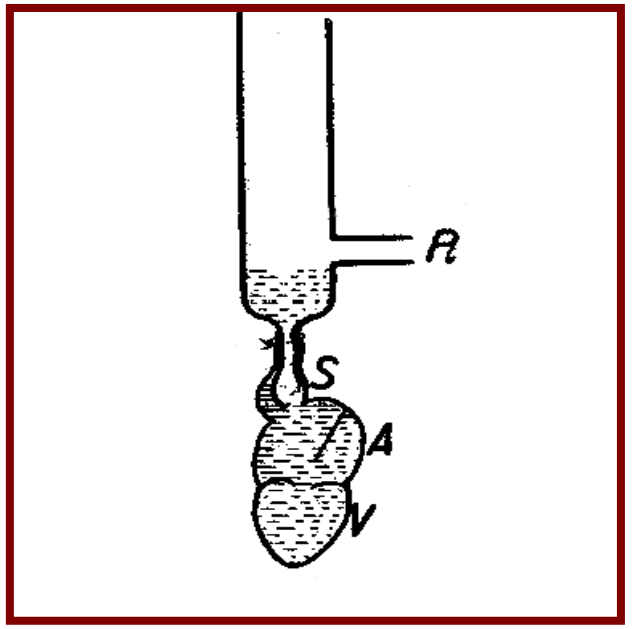
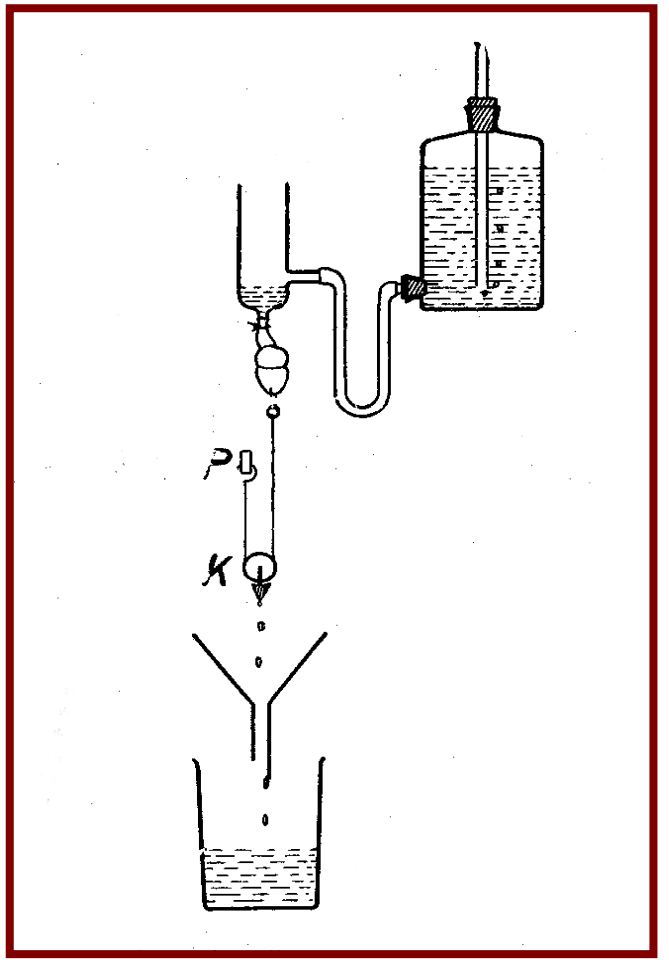


Henry Pickering Bowditch
(1840 – 1911)





HOMEOMETRIC AUTOREGULATION
(FREQUENCY EFFECT)

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

Ratio between intra- and extracellular calcium concentrations increases

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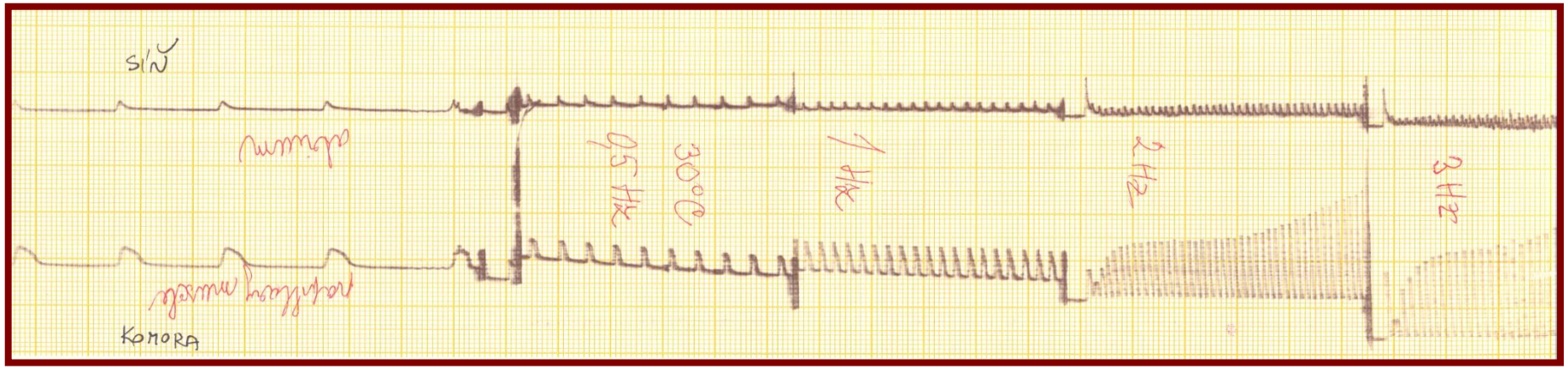

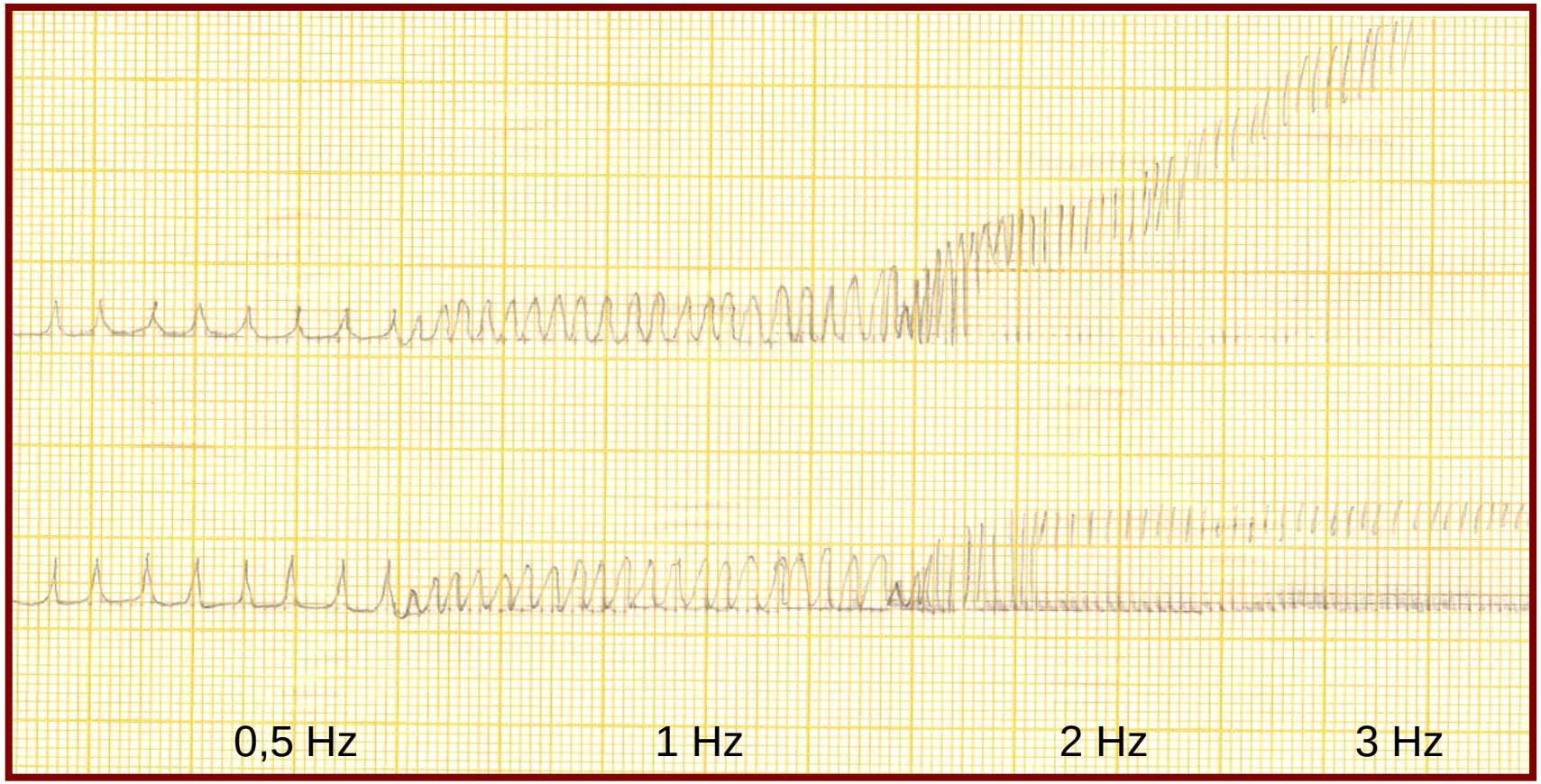

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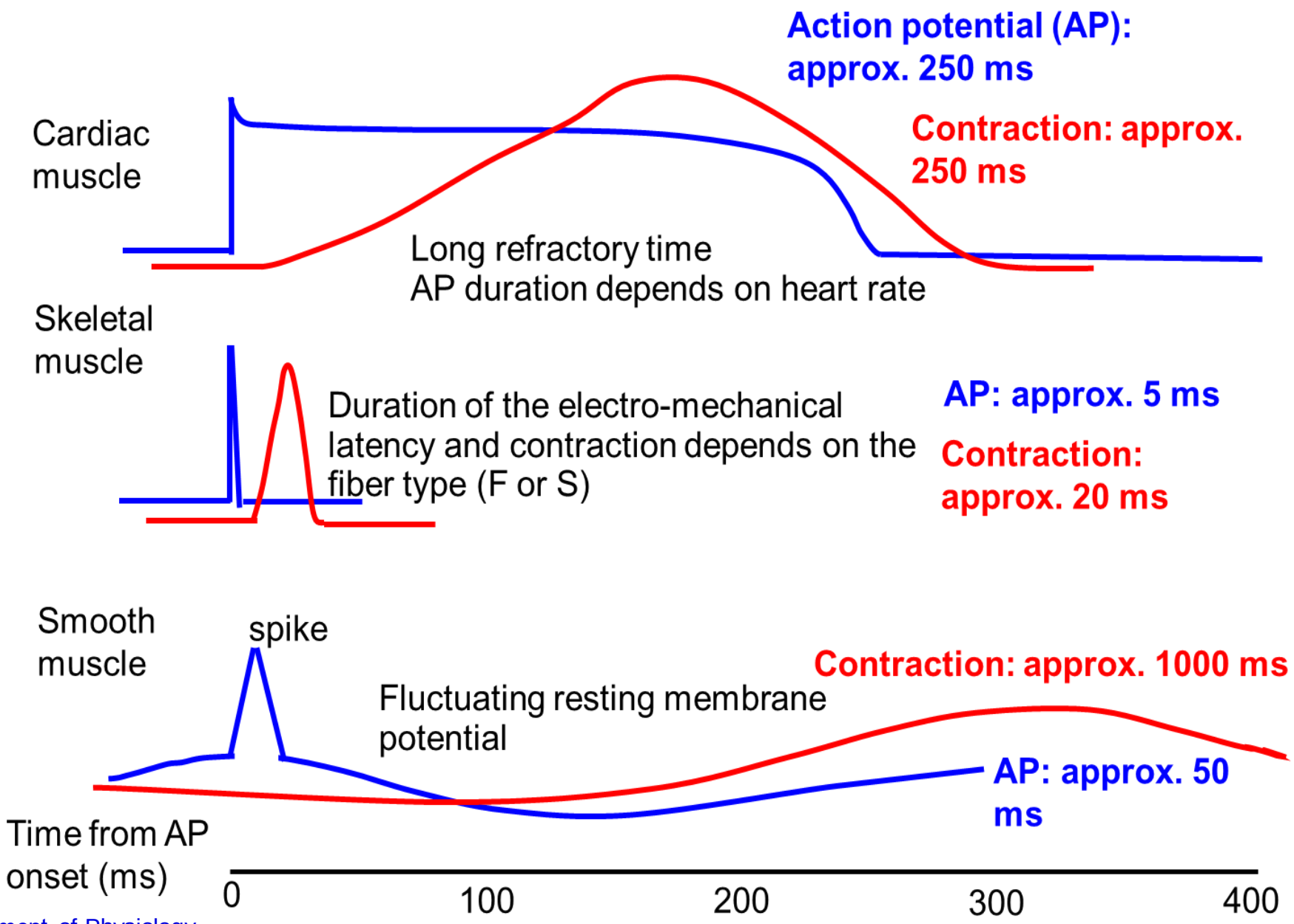
Skeletal, cardiac and smooth muscle – action potential and contraction



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CARDIAC RESERVE = maximal CO / resting CO **4 - 7**

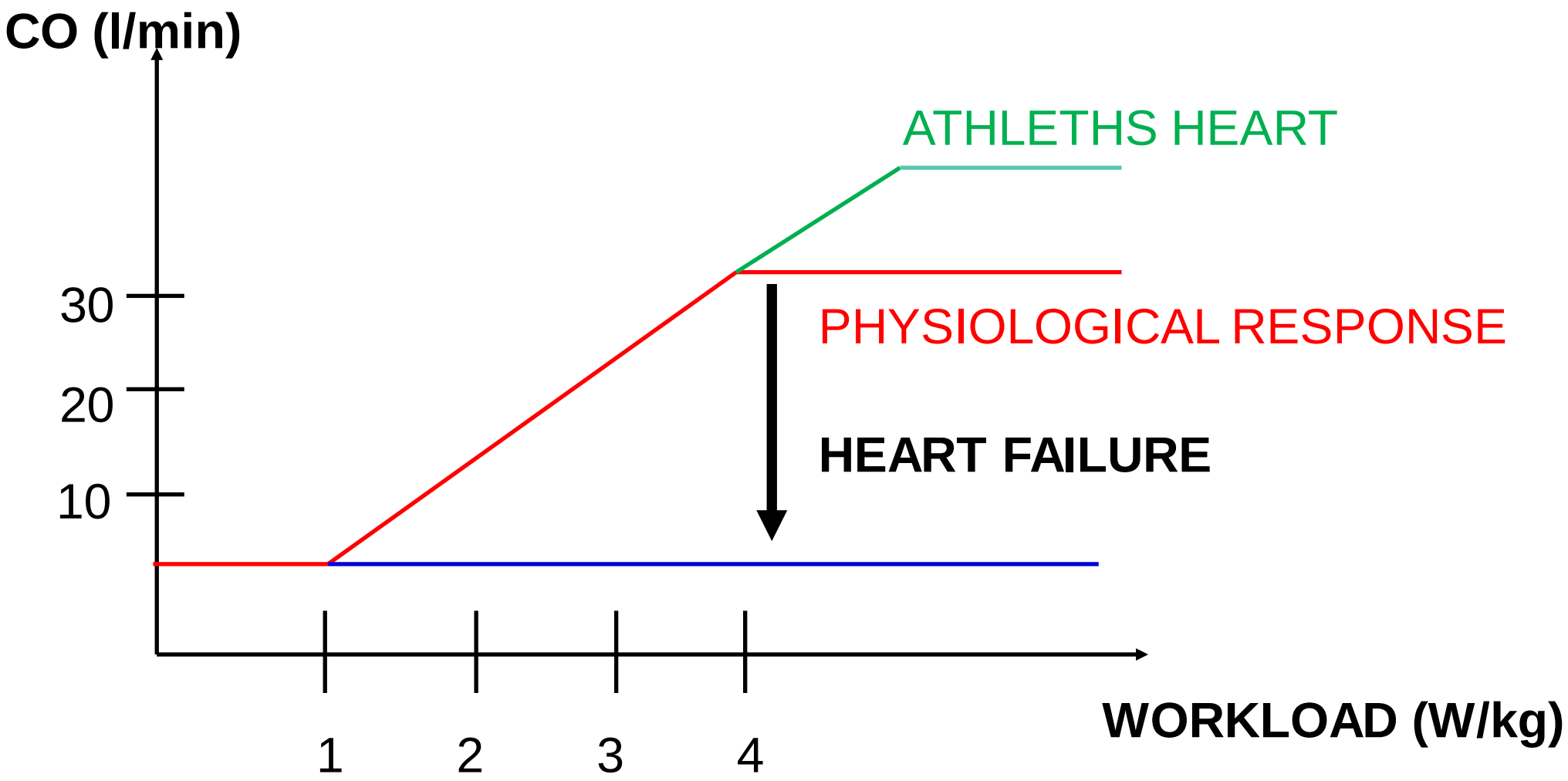
CORONARY RESERVE = maximal CF / resting CF **3,5**

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



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IMPORTANT TERMS

Length-tension relationship (curve)

Minimal length l_0

Passive, active, total force

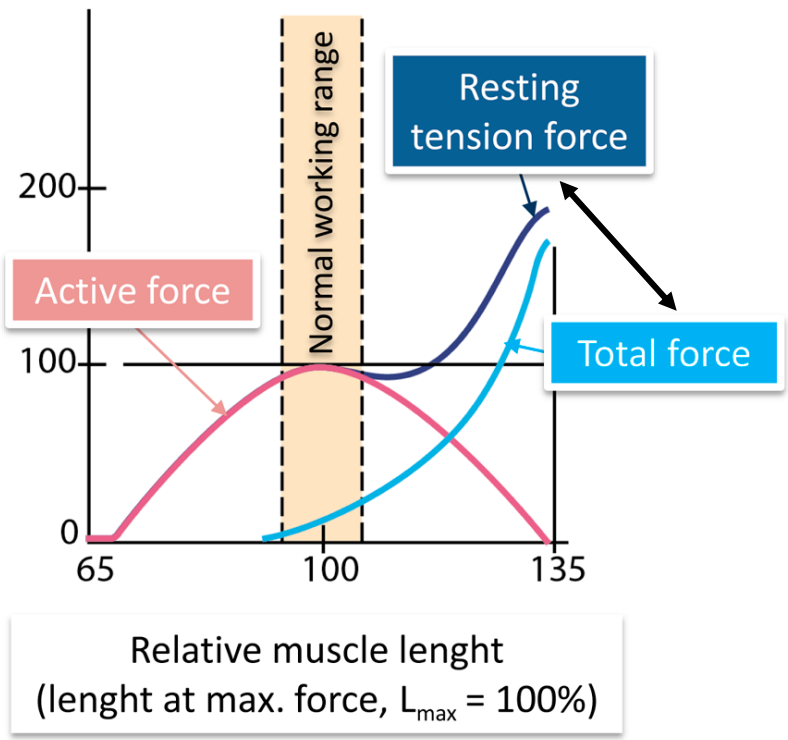
Optimal length

Isometric, **isotonic**, **auxotonic** contraction

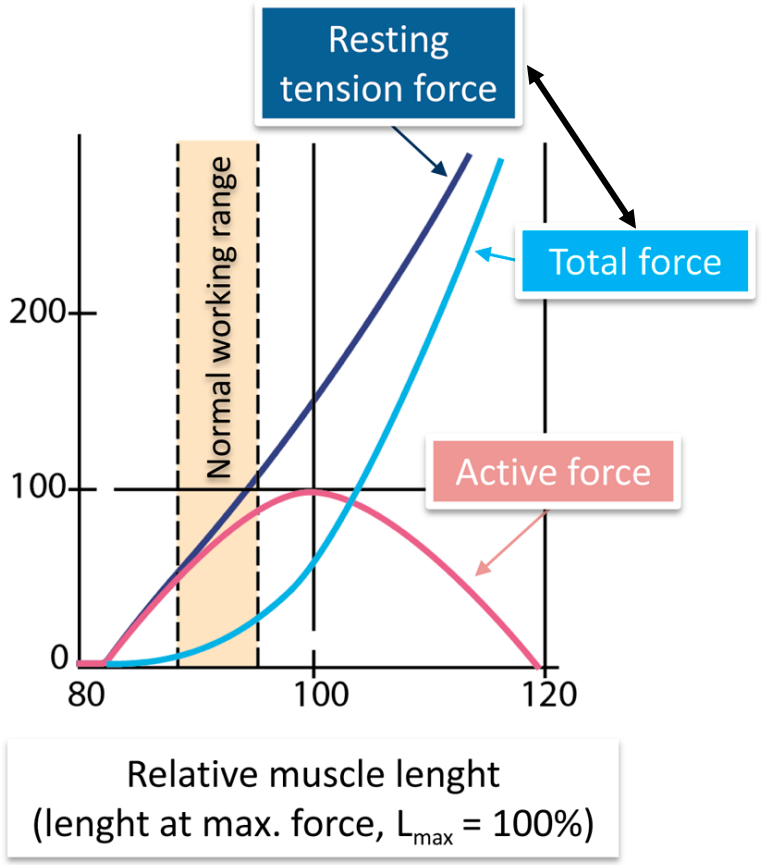
Autoregulation of contraction – **heterometric** (Starling)

Preload, **afterload**

1. Striated muscle



2. Cardiac muscle



Passive tension, active tension, isometric contraction, isotonic contraction, auxotonic contraction

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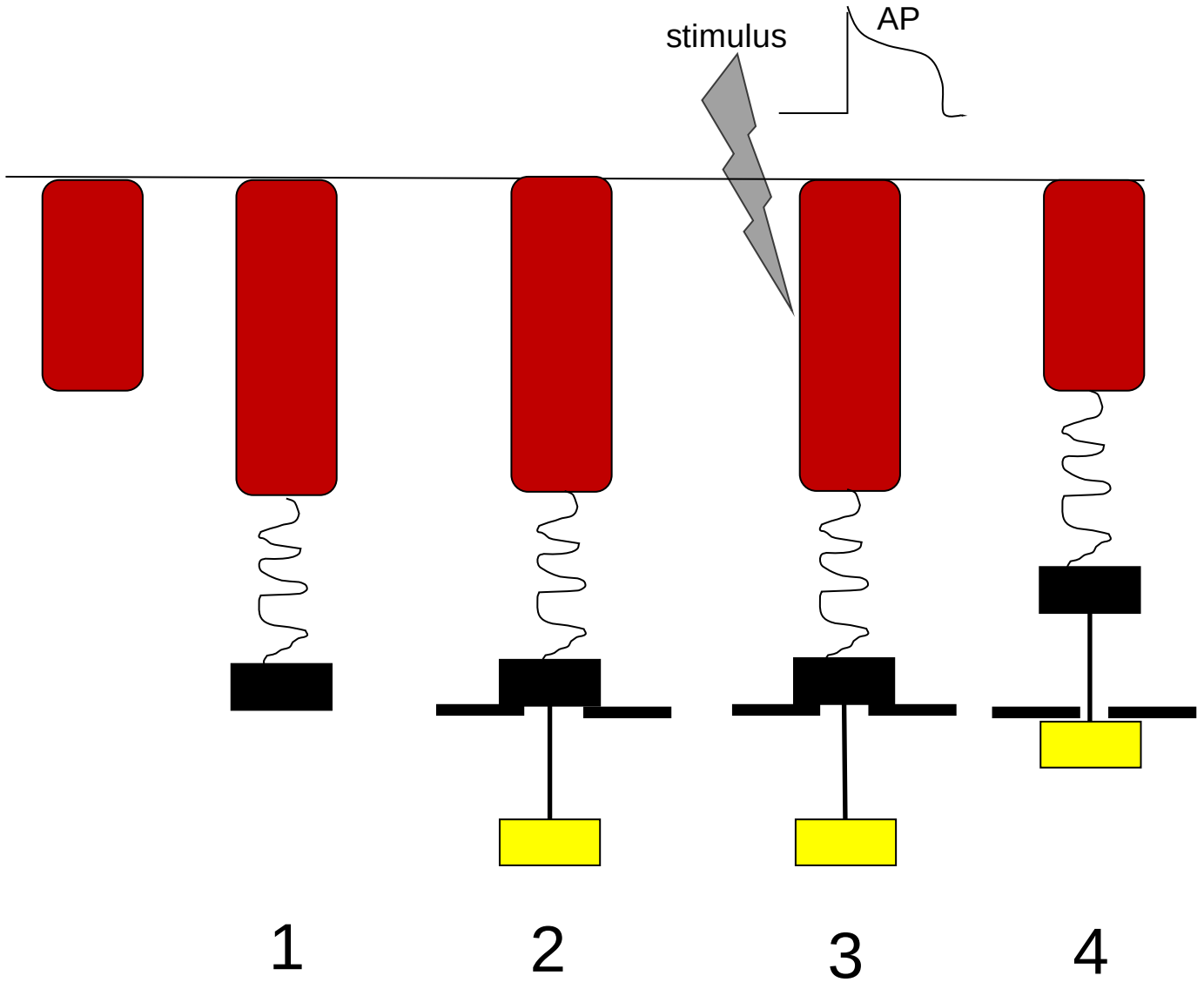
AFTERLOADED CONTRACTION



PRELOAD, AFTERLOAD

PRELOAD
(~ enddiastolic filling)

AFTERLOAD
(~ pressure which must be developed)

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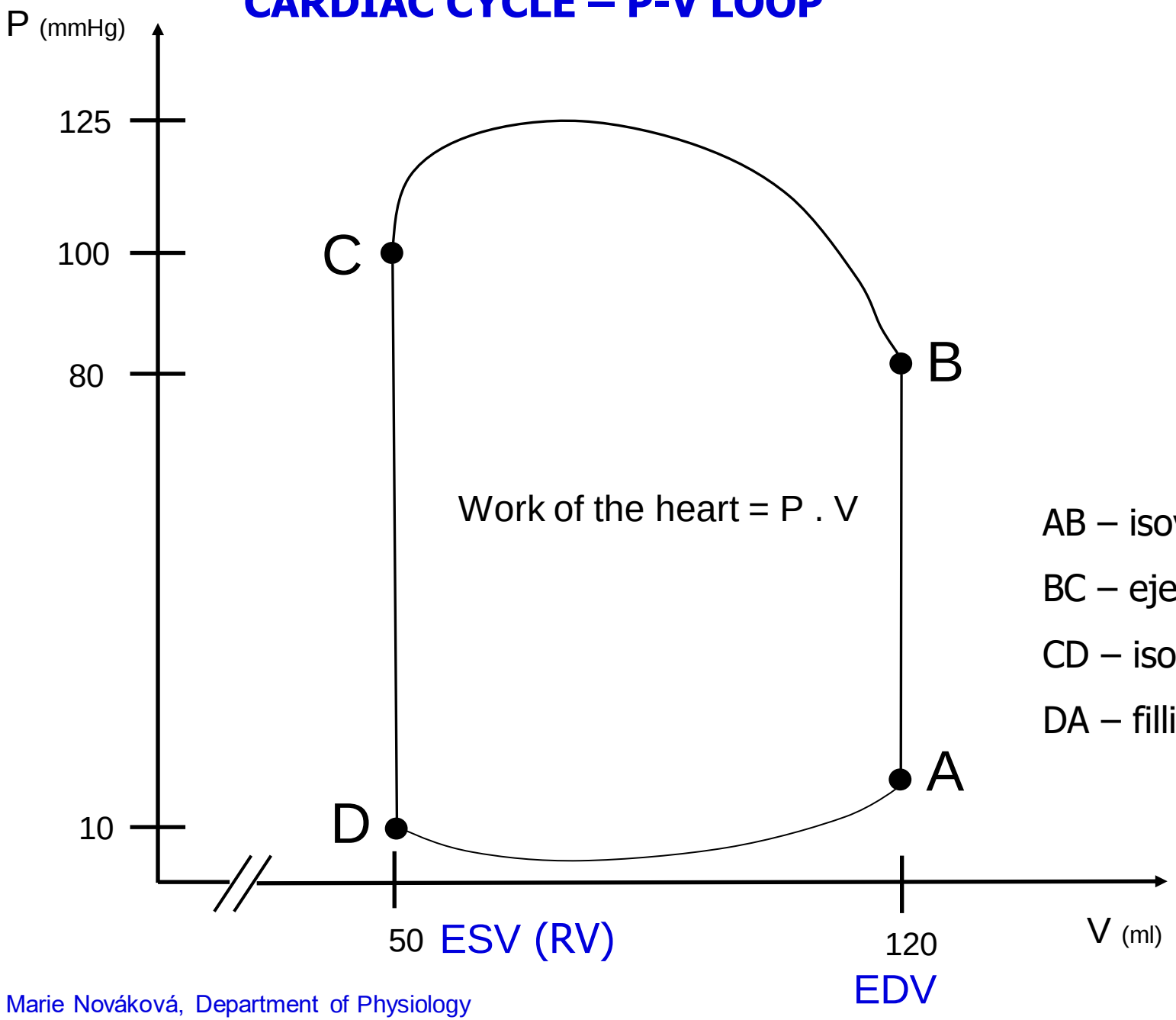
CARDIAC CYCLE – P-V LOOP



LAPLACE law:

$T = P \cdot r / 2h$

$\uparrow P = T \cdot \uparrow 2h / \downarrow r$

AB – isovolumic contraction

BC – ejection

CD – isovolumic relaxation

DA – filling

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$P = T \cdot 2h \cdot r^{-1}$ **Isovolumic contraction**: T rises up, valves closed – increase in P

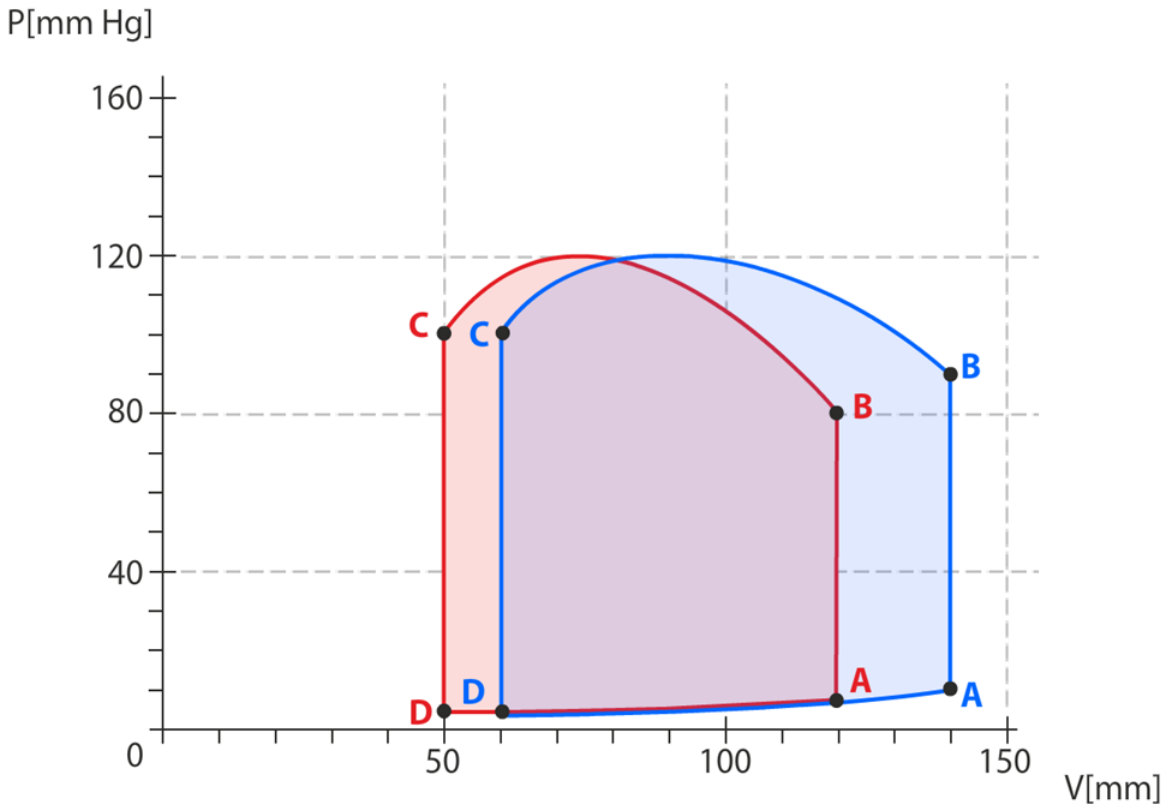
$P = T \cdot 2h \cdot r^{-1}$ **Ejection**: r decreases, h rises, thus P increases (even at the same T)

$P = T \cdot 2h \cdot r^{-1}$ **Isovolumic relaxation**: T decreases, valves closed – decrease in P

$P = T \cdot 2h \cdot r^{-1}$ **Ventricular filling:** r and T rise, P first falls down, then rises up (length/tension relationship)

INCREASED PRELOAD

MODEL



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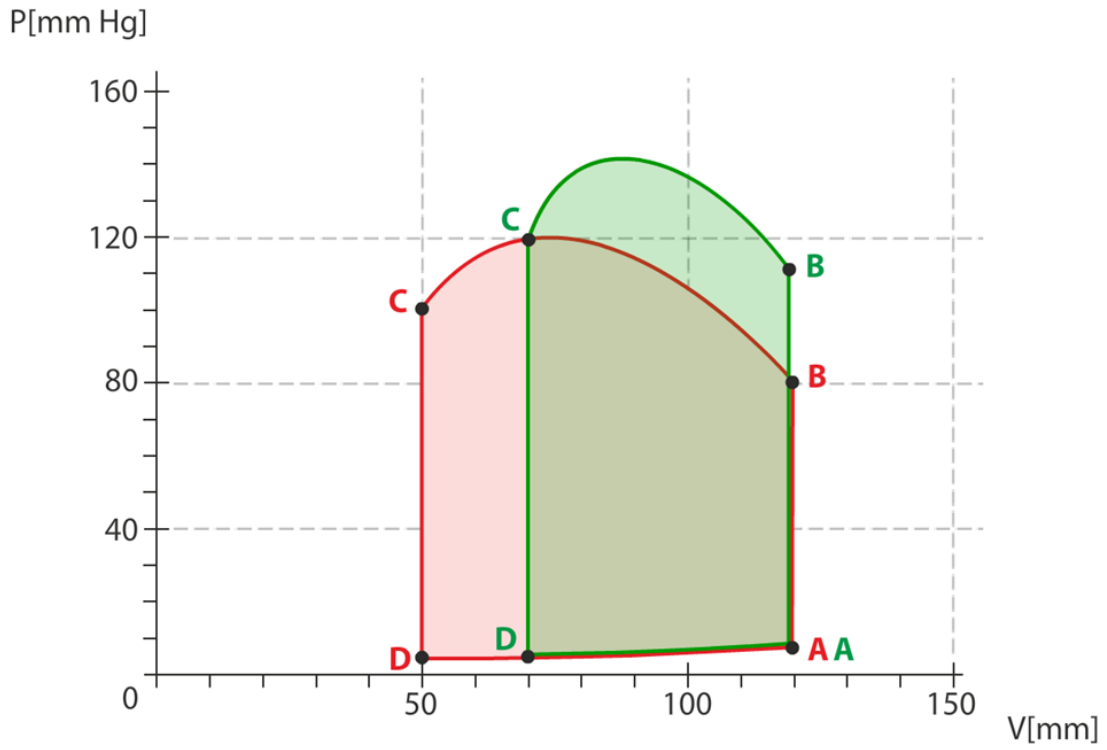
INCREASED AFTERLOAD

MODEL



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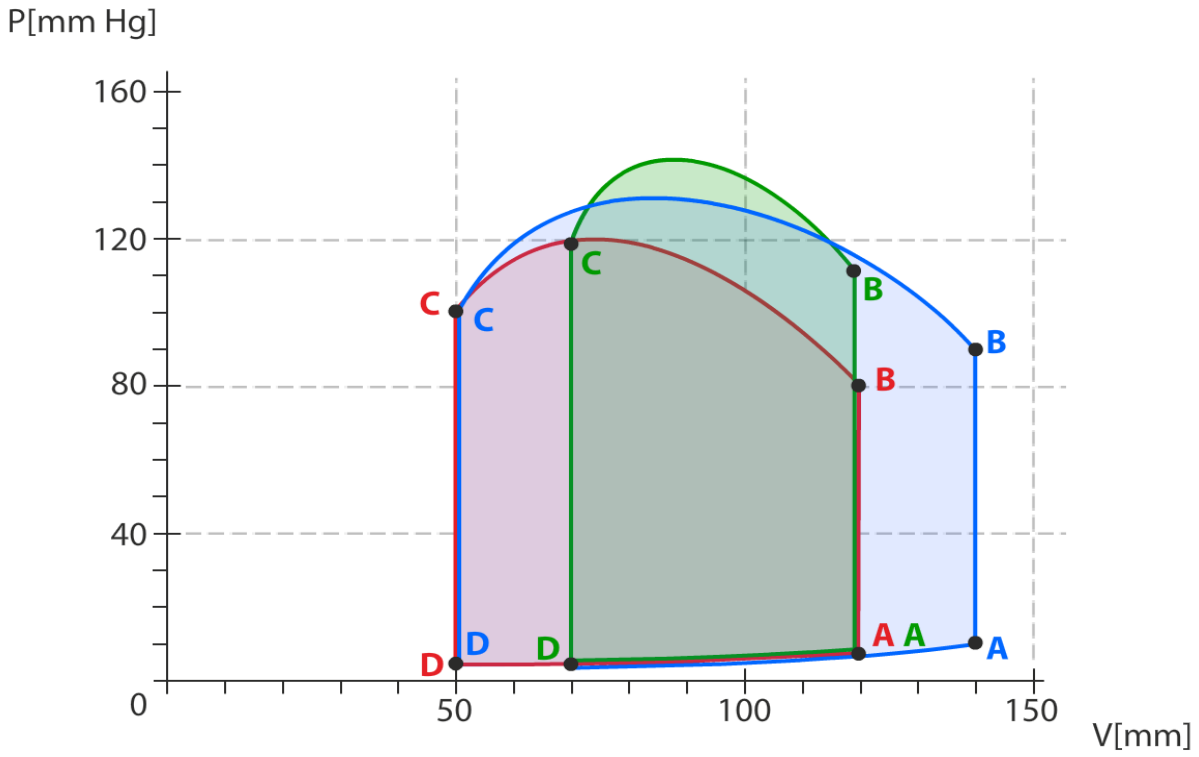
INCREASED PRELOAD AND AFTERLOAD

MODEL



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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

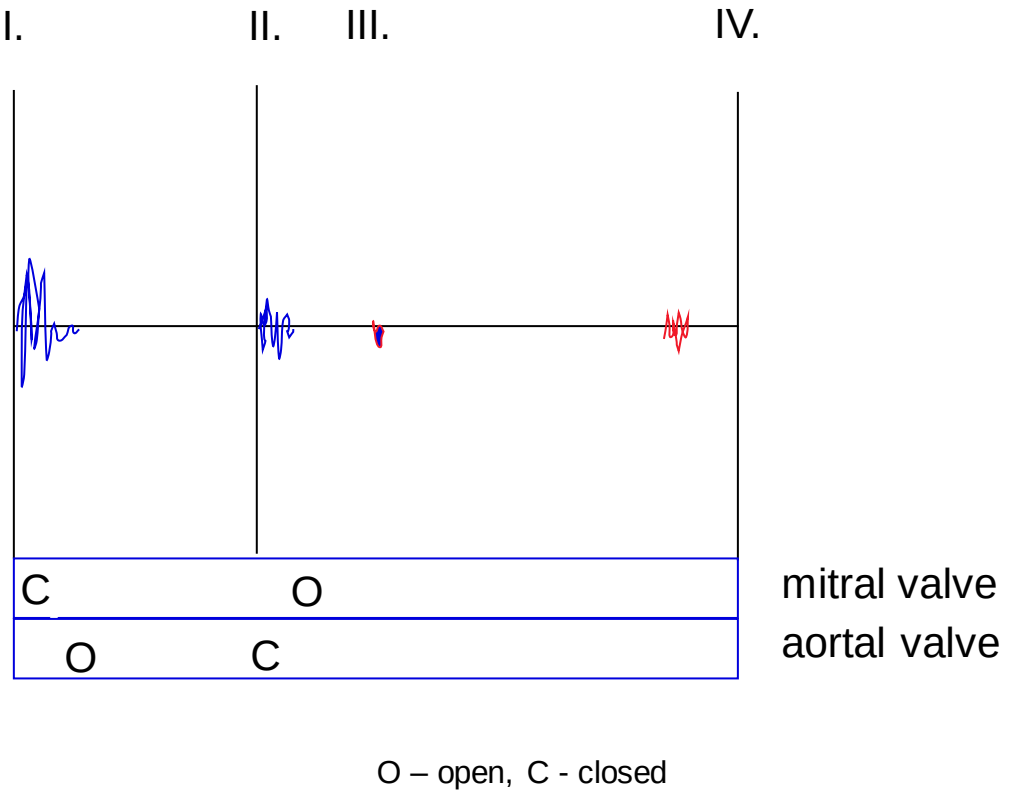
I. – mitral (+ tricuspidal) valve closure

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

Splitting of I. or II. sound:
asynchronous closure of M - T valve (I.)
or Ao - P valve (II.)
(inspiration, hypertension….)

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MURMURS – pathological phenomena based on turbulent blood flow

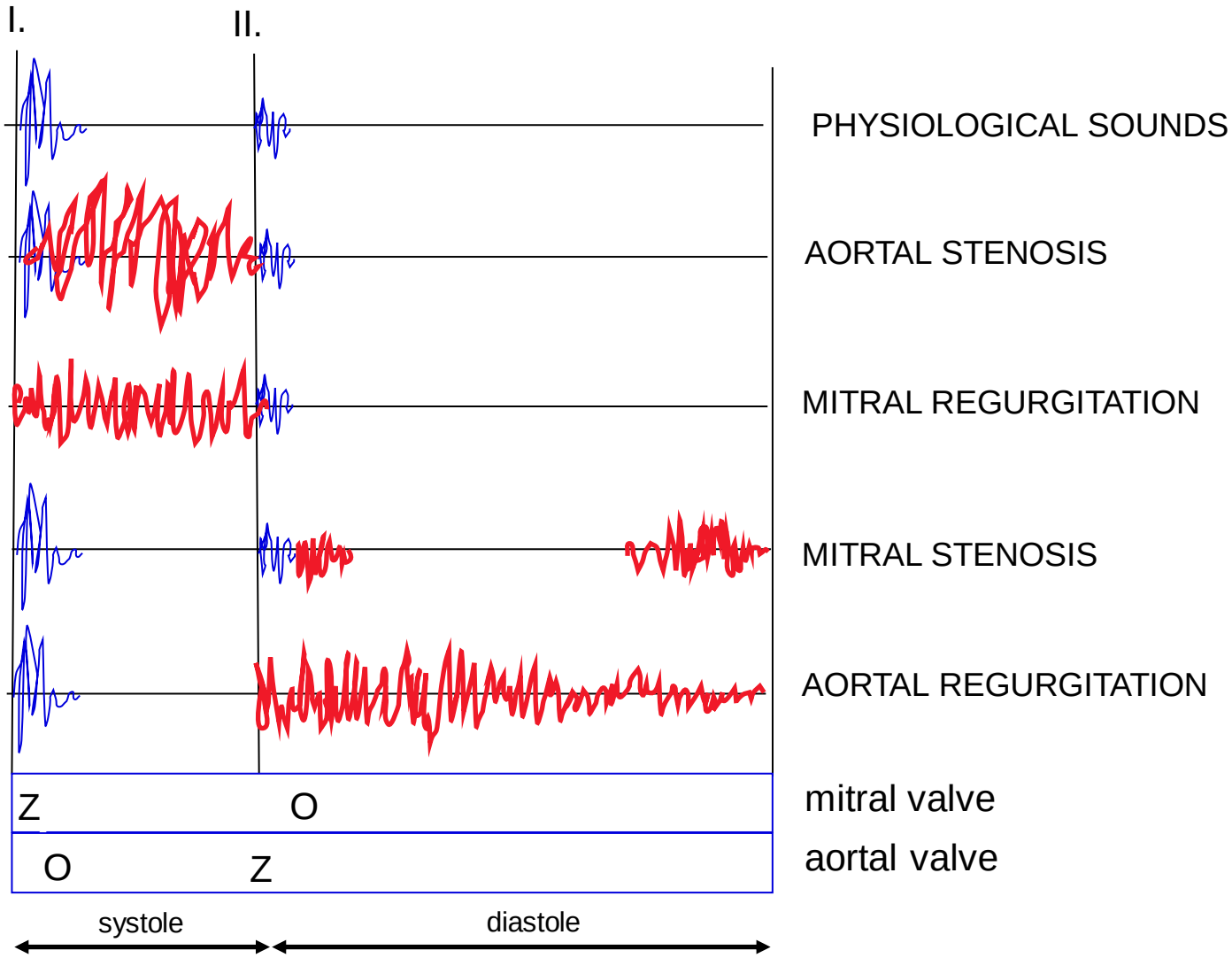
1. **SYSTOLIC**

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

2. **DIASTOLIC**

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

3. **SUSTAINED**:
- Defects of septum



O – open, C - closed

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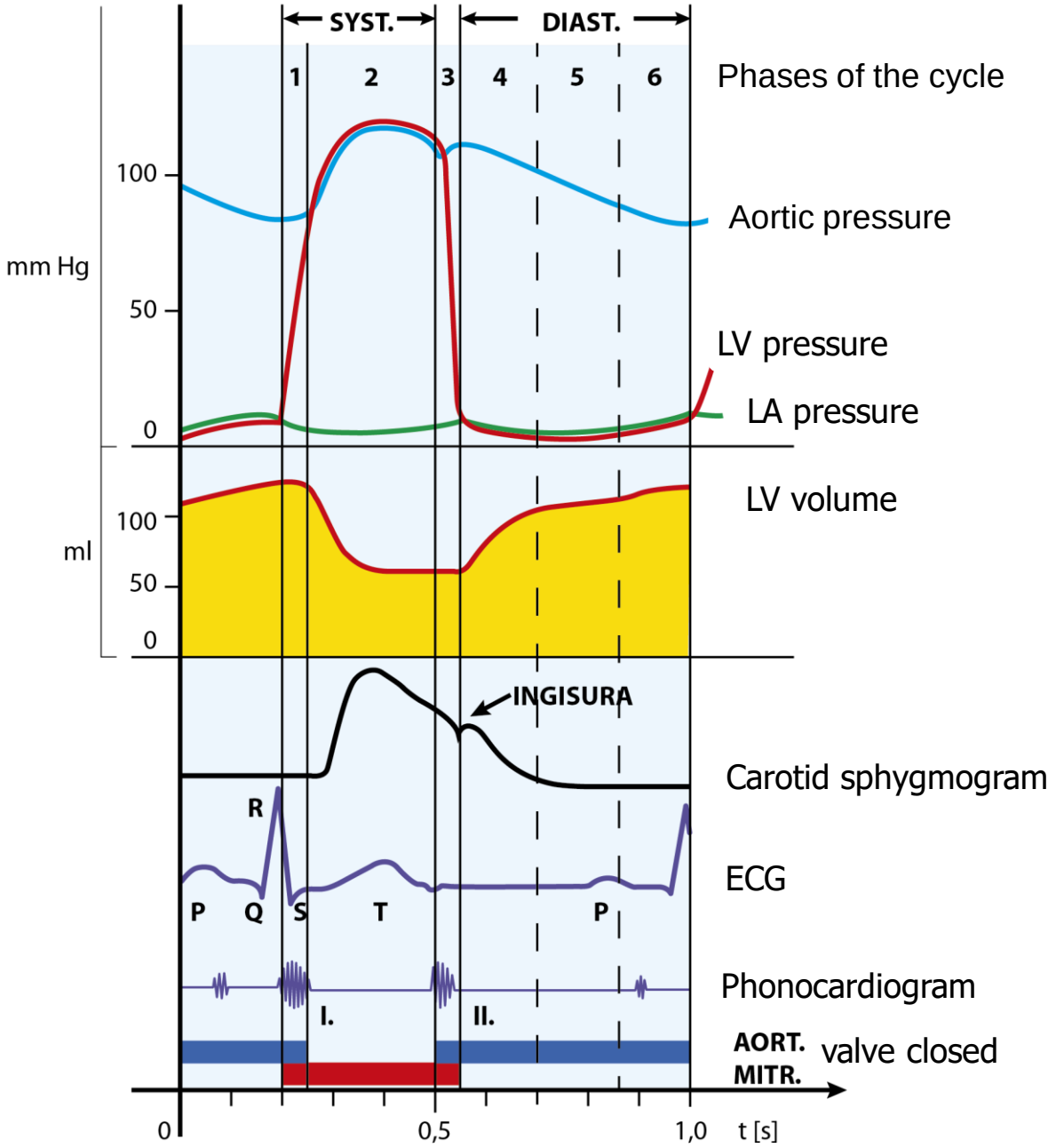
POLYGRAPHY (polygram)



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HEART FAILURE = loss of cardiac reserve

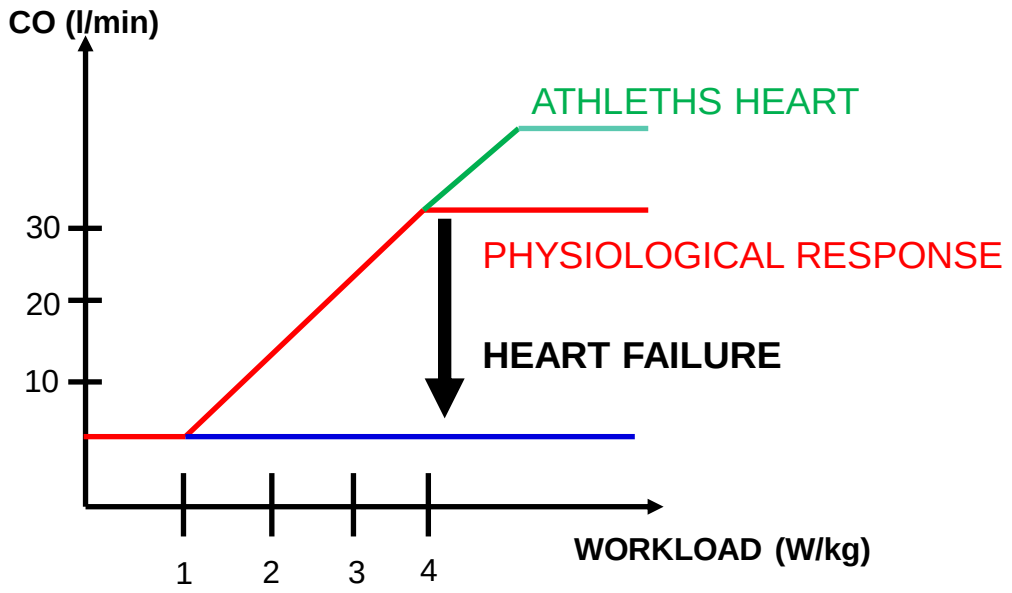
The heart is not able pump sufficient amount of blood into periphery at normal venous return.

SYMPTOMS
fatigue, oedemas, venostasis, dyspnoea, cyanosis

ACUTE x CHRONIC.

COMPENSATED x DECOMPENSATED.

MOST FREQUENT CAUSES:
- Severe arrhythmias
- Overload – *volume* (aortal insufficiency, a-v shunts) or *pressure* (hypertension and aortal stenosis – left overload, pulmonary hypertension and stenosis of pulmonary valve – right overload)
- Cardiomyopathy



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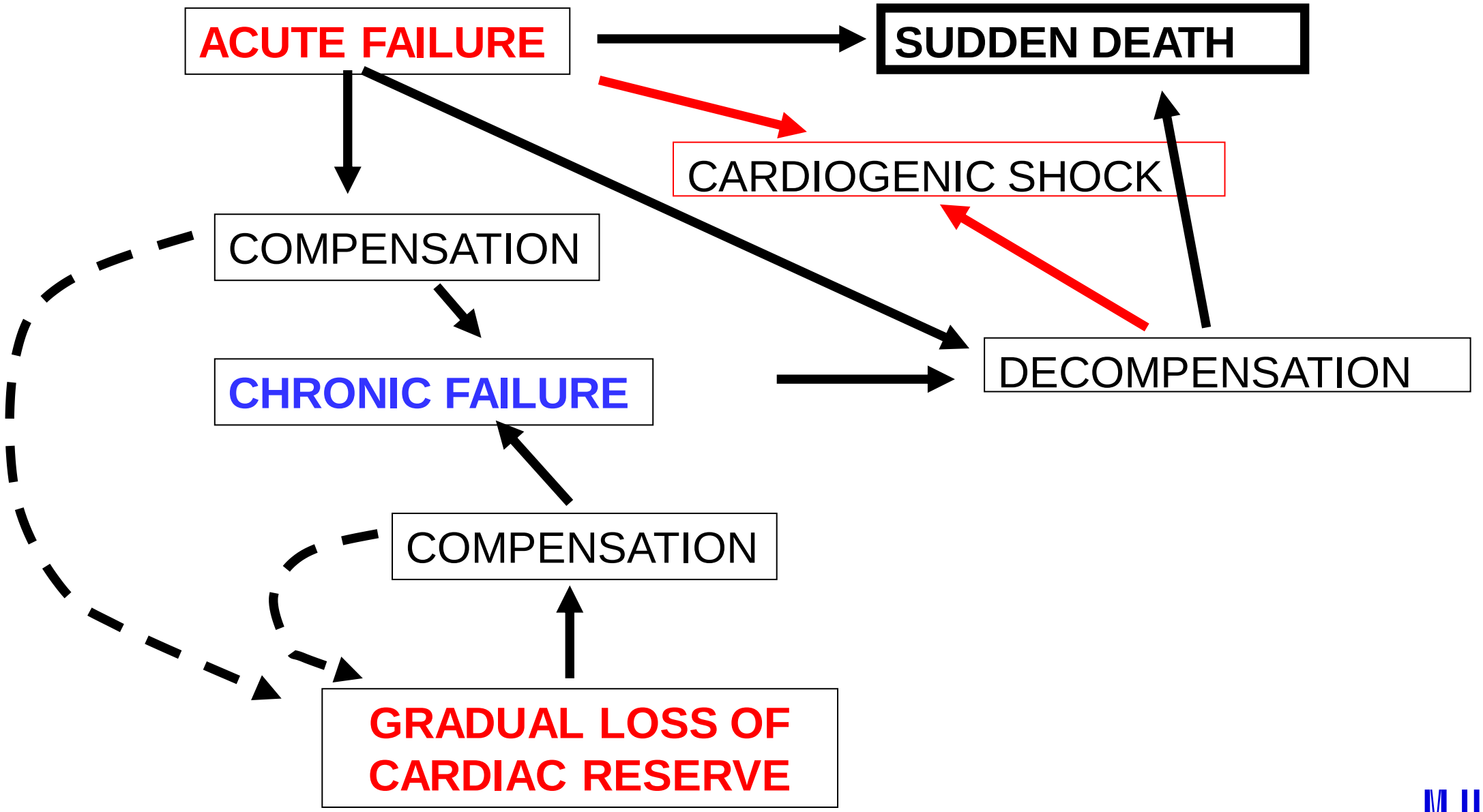
ACUTE FAILURE
SUDDEN DEATH
CARDIOGENIC SHOCK
COMPENSATION
CHRONIC FAILURE
DECOMPENSATION
COMPENSATION
GRADUAL LOSS OF CARDIAC RESERVE

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HEART FAILURE - COMPENSATION

BAROREFLEX

Physiological role: compensation of decrease in minimal volume of circulating fluids

Signal: BP decrease (orthostase, work vasodilatation)

Sensor: baroreceptors

Response: activation of SAS (increased HR, inotropy, BP)

Pathological signal: long-lasting decrease of BP due to heart insufficiency

Results: increased energy outcome – **vicious circle**

Ca^{2+} - antagonists

β – sympatolytics

ACTIVATION OF RAAS

Physiological role: compensation of loss of circulating fluids (bleeding)

Signal: decrease in renal perfusion

Sensor: juxtaglomerular system of kidney

Response: BP increase (angiotenzin II.), water retention (aldosteron)

Pathological signal: decrease in renal perfusion due to heart insufficiency

Results: increased preload and afterload, increased energy outcome – **vicious circle**

angiotenzin-converting enzyme inhibitors (AT II. receptors)

DILATATION (STARLING PRINCIPLE)

Physiological role: compensation of momentary right-left differences

Signal: orthostase, deep breathing, beginning of exercise

Pathological signal: continual blood stasis in the heart

Results: increased energy outcome – **vicious circle**

HYPERTROPHY

Physiological role: preservation of energetically demanding tension of ventricular wall

Signal: P = s . 2 h / r, intermittent BP increase (athletes heart)

Response: concentric remodelling

Pathological signal: continual increase of preload or afterload

Results: worsening of oxygenation, fibrotisation – **vicious circle**

diuretics

cardiac glycosides (digitalis)