

CARDIAC MECHANICS

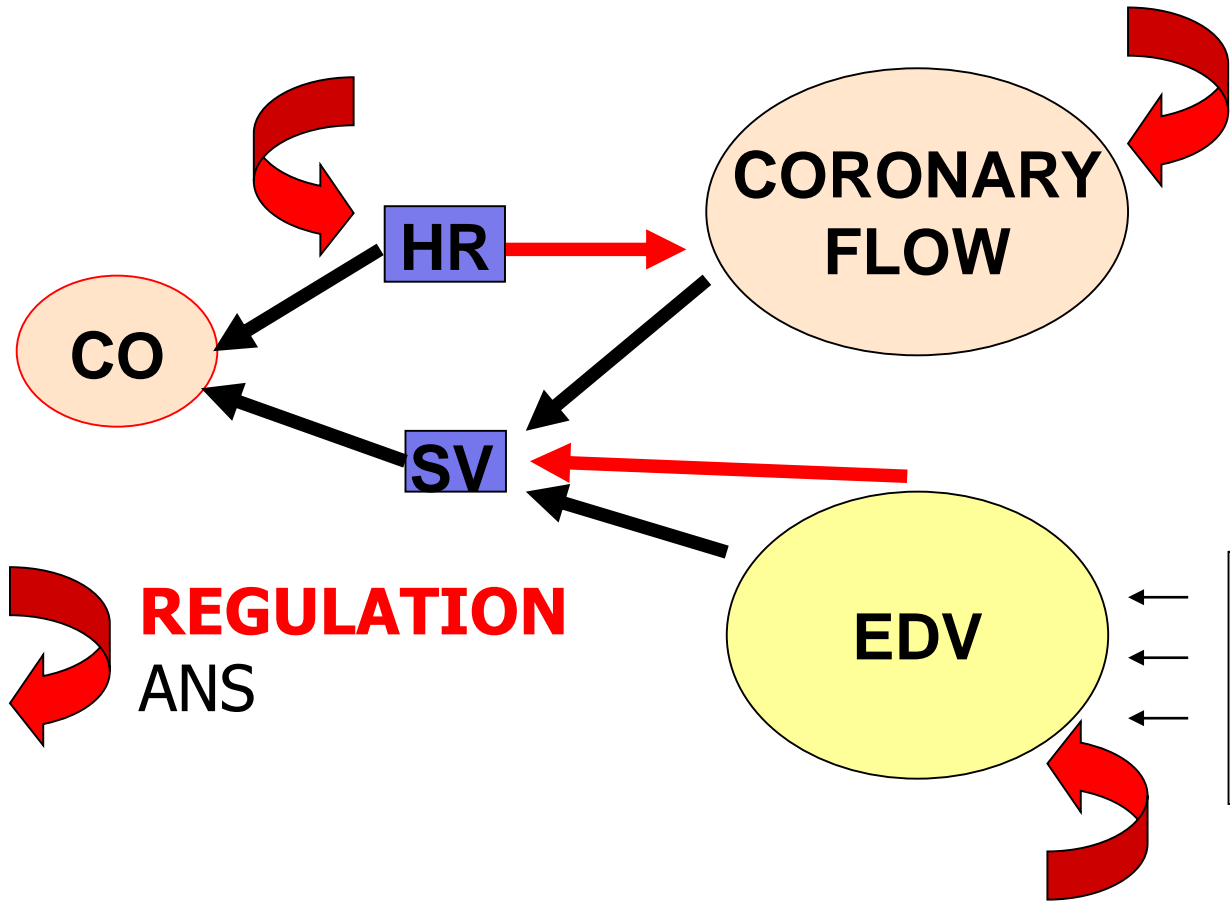
HEART AS A PUMP

CARDIAC CYCLE

HEART FAILURE

CARDIAC OUTPUT (CO)

$$LV = RV$$



$$CO = HR \times SV \quad 5l/min$$

$$SV = EDV - ESV \quad 70ml$$

$$\text{Ejection fraction} \quad EF = \frac{EDV - ESV}{EDV} \quad >60\%$$

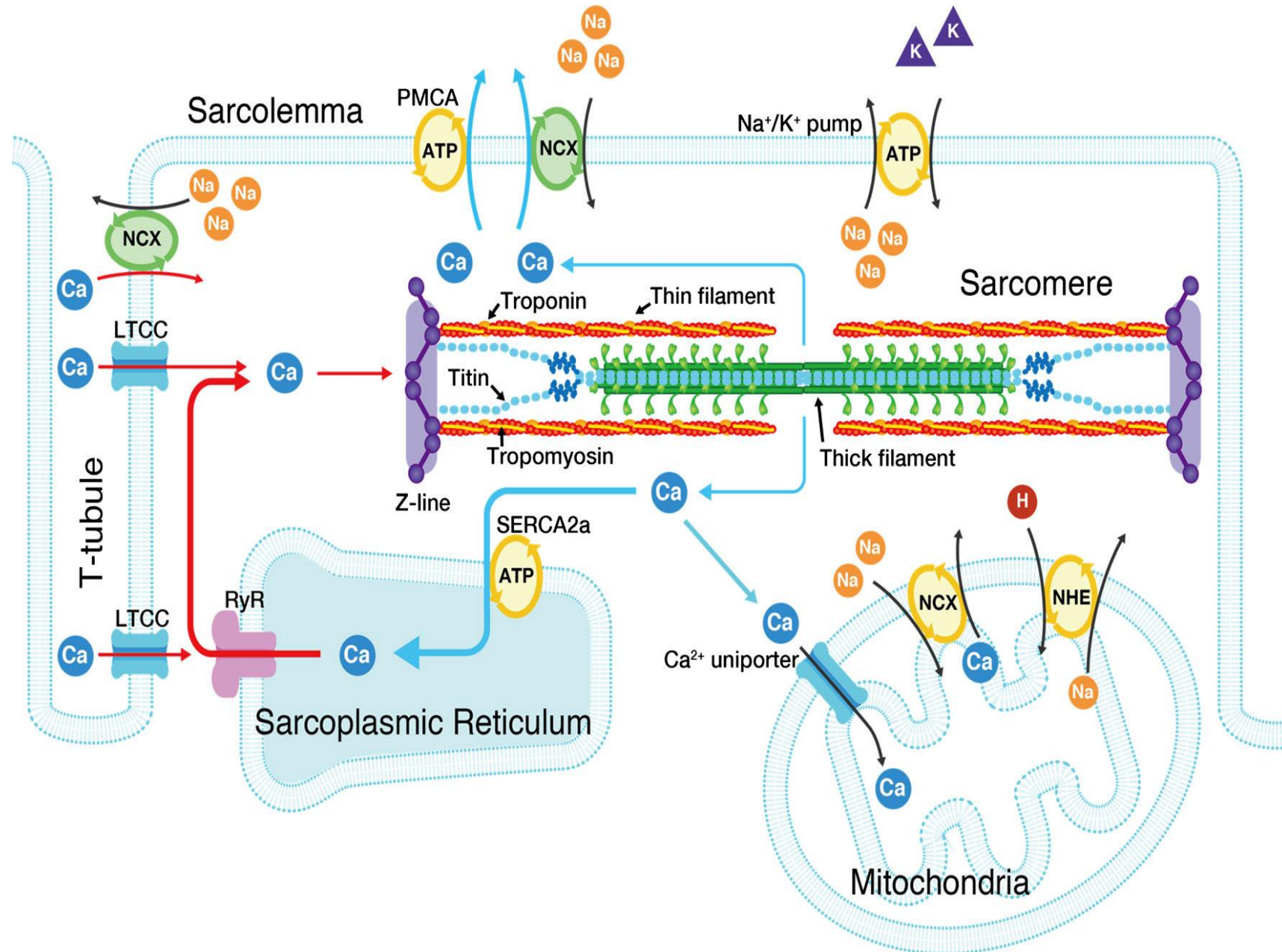
Venous return
Compliance
Aortal pressure

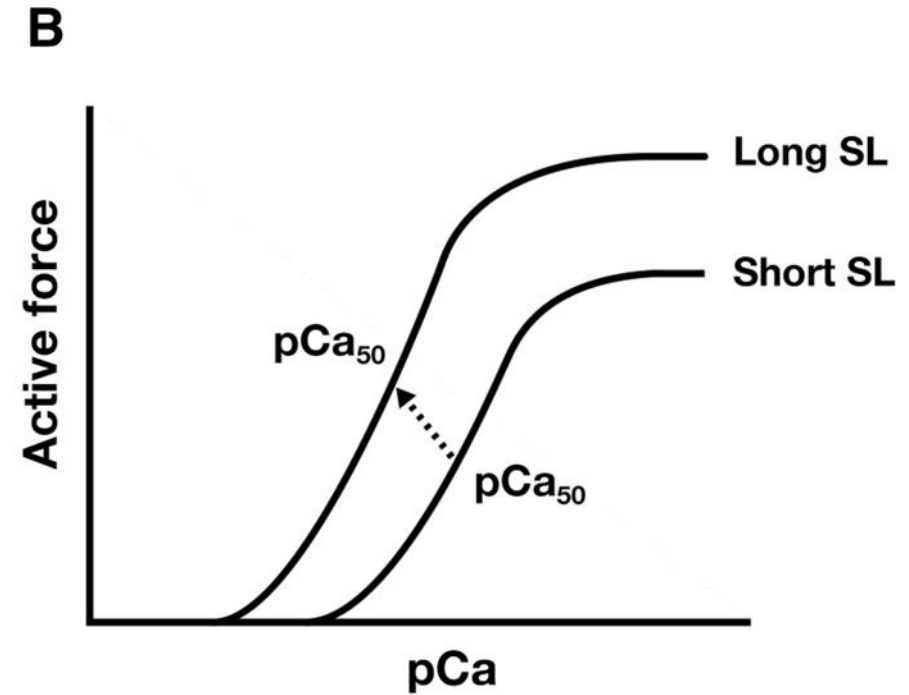
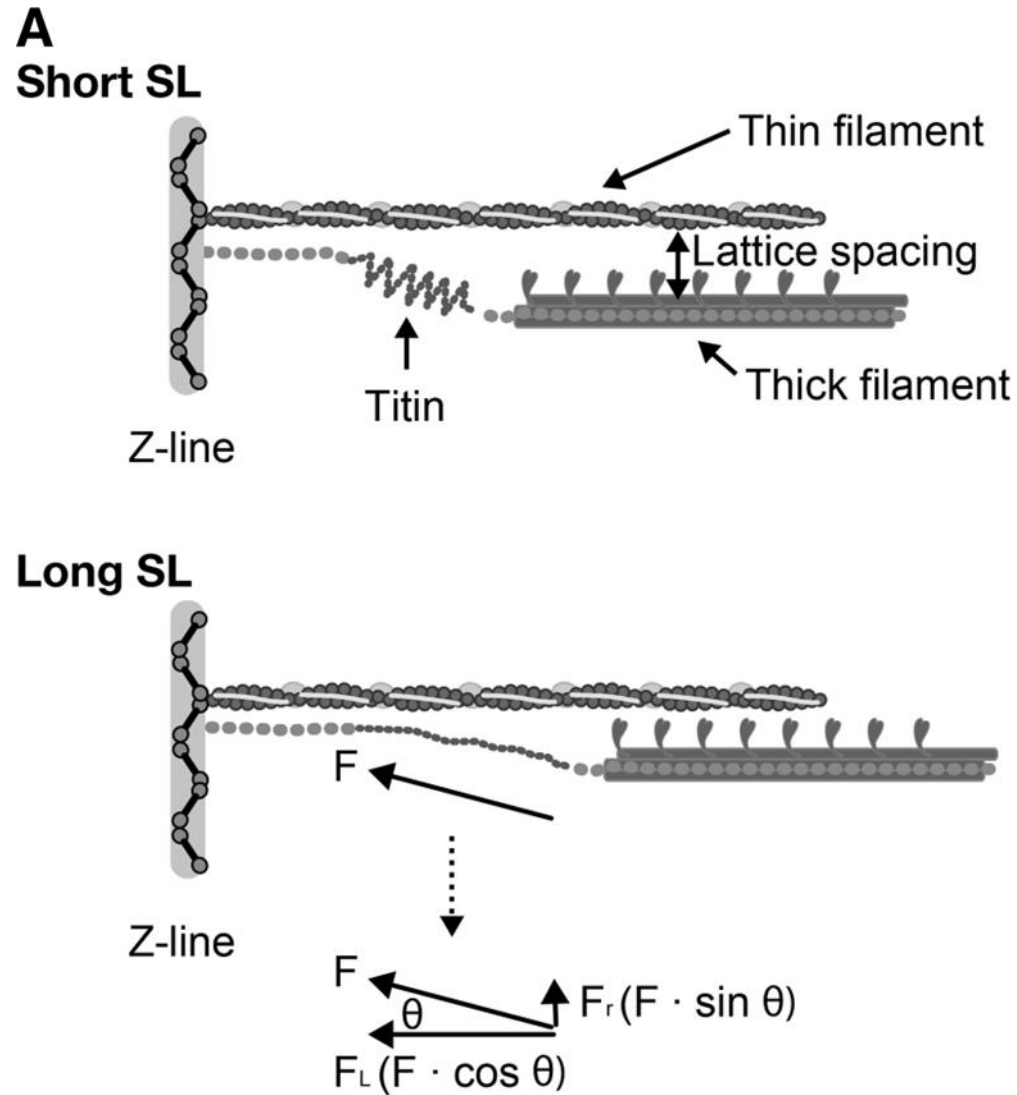
CONTRACTILITY
Ability to contract
Depends on tissue perfusion
(substrates and oxygen supply for
ATP production; Ca²⁺ availability)

AUTOREGULATION of cardiac contraction

Heterometric: Starling law
Homeometric: Frequency effect

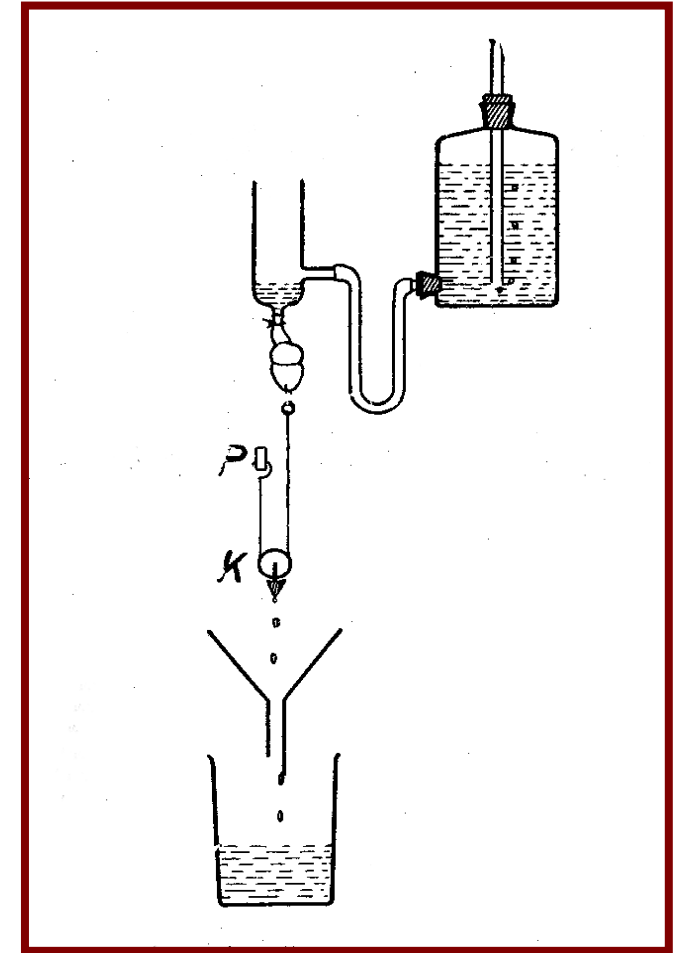
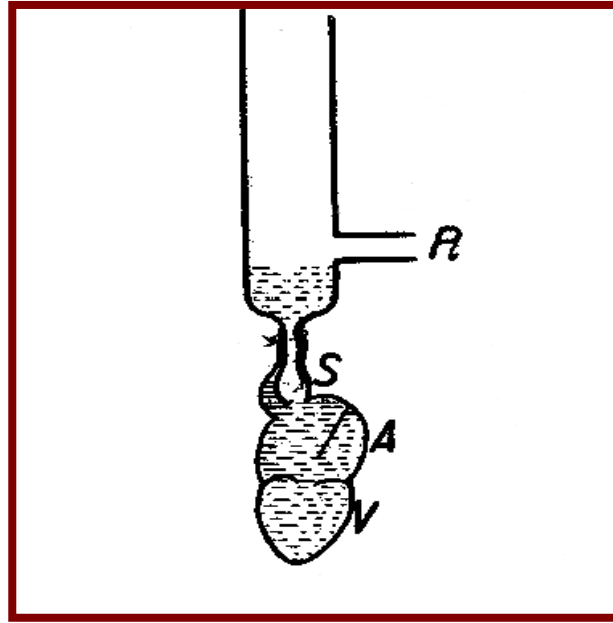
STARLING LAW







Henry Pickering Bowditch
(1840 – 1911)

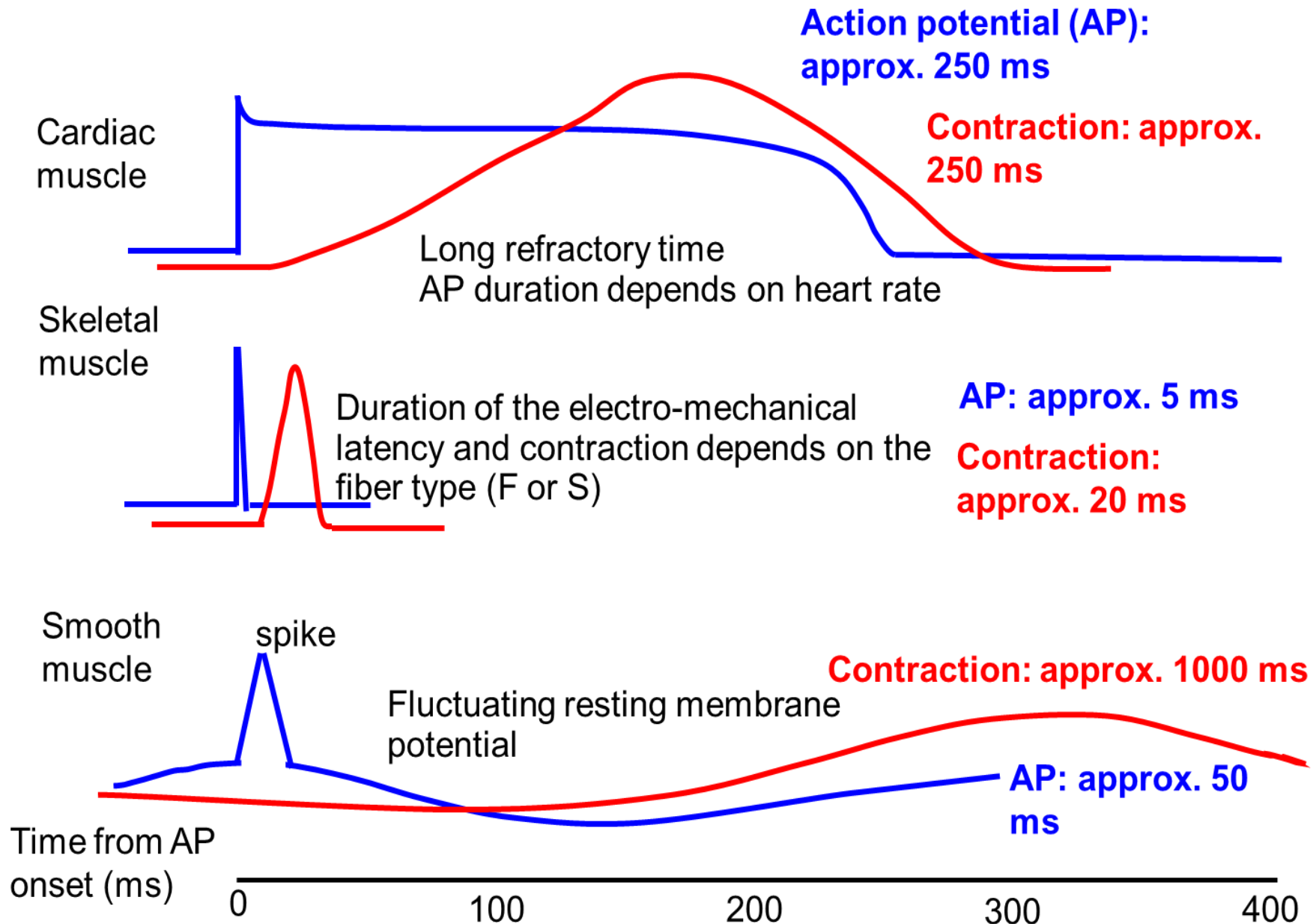


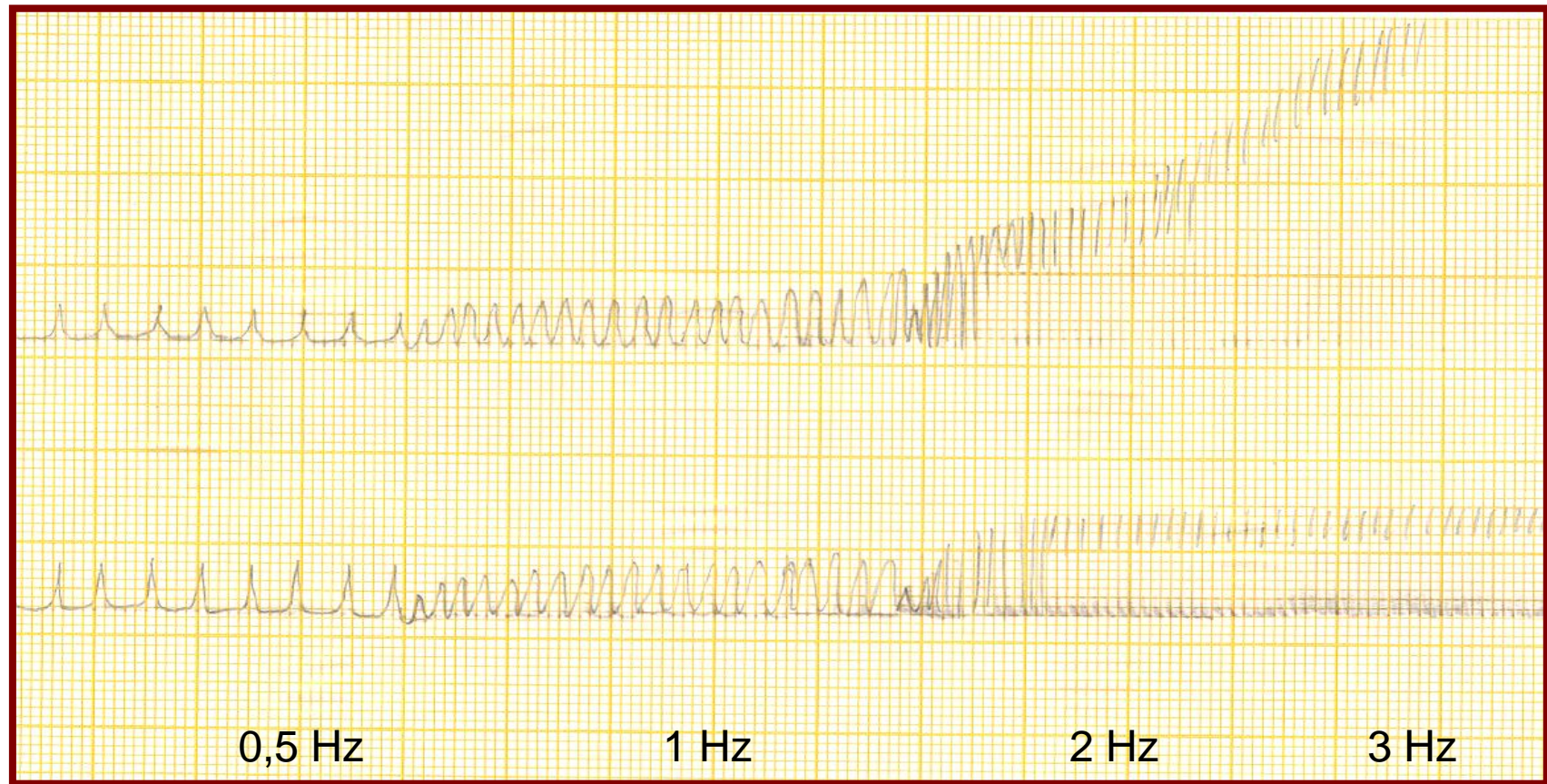
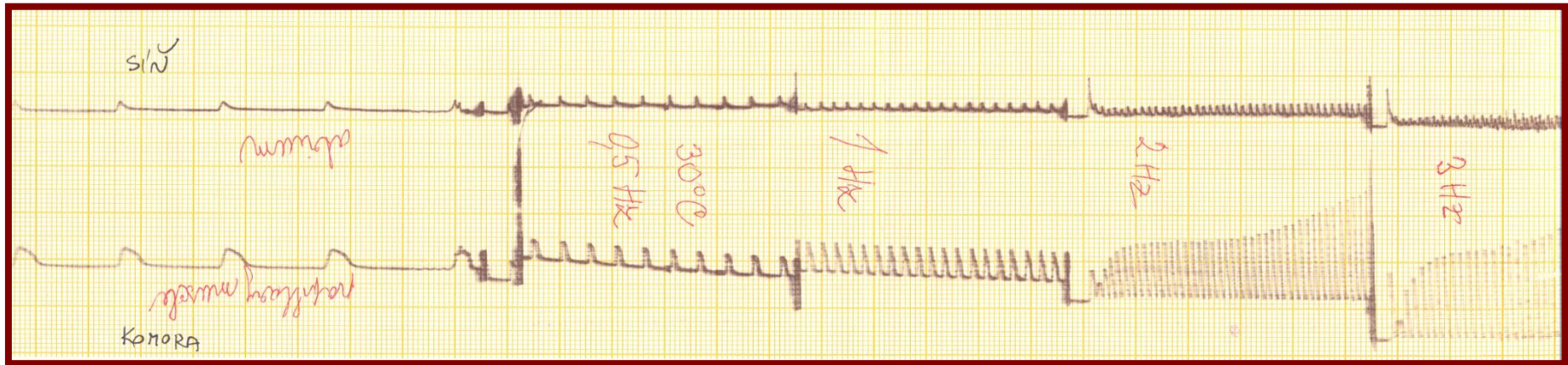
HOMEOMETRIC AUTOREGULATION (FREQUENCY EFFECT)

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

Ratio between intra- and extracellular calcium concentrations increases

Skeletal, cardiac and smooth muscle – action potential and contraction





CARDIAC RESERVE = maximal CO / resting CO

4 - 7

CORONARY RESERVE = maximal CF / resting CF

3,5

CHRONOTROPIC RESERVE = maximal HR / resting HR

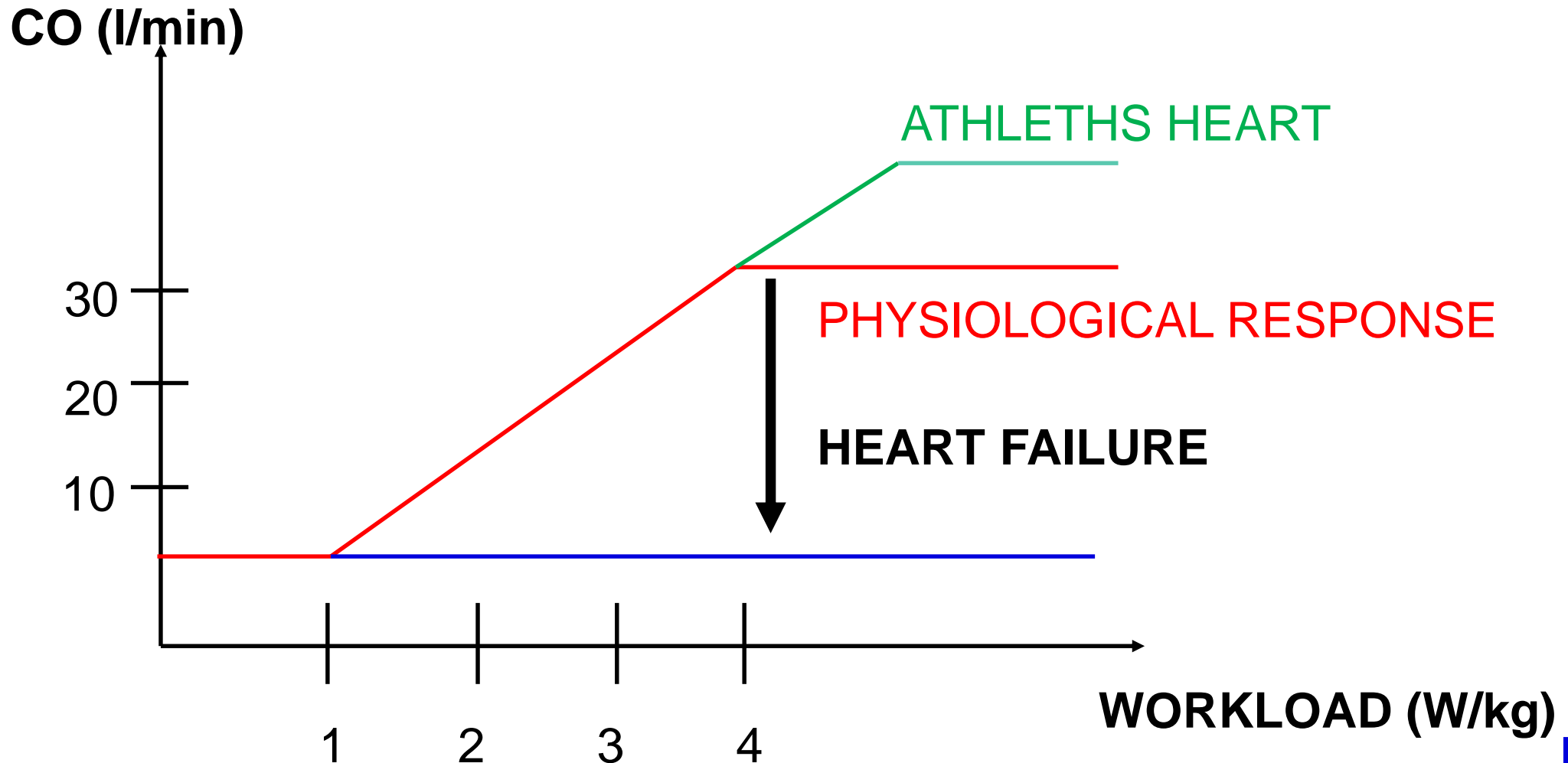
3 - 5

VOLUME RESERVE = maximal SV / resting SV

1,5

CO = cardiac output
CF = coronary flow
HR = heart rate
SV = stroke volume

CARDIAC RESERVE



IMPORTANT TERMS

Length-tension relationship (curve)

Minimal length l_0

Passive, active, total force

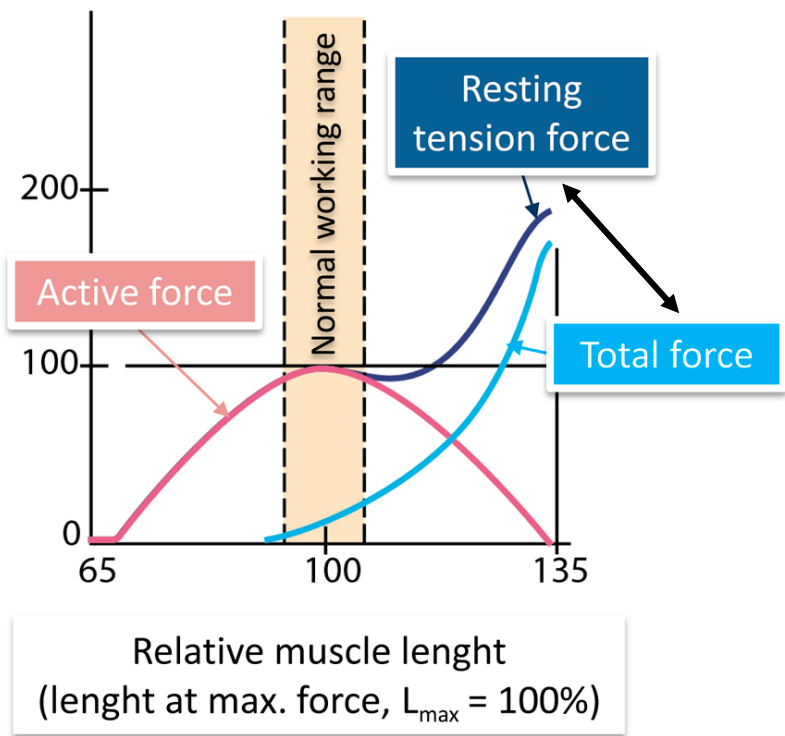
Optimal length

Isometric, isotonic, auxotonic contraction

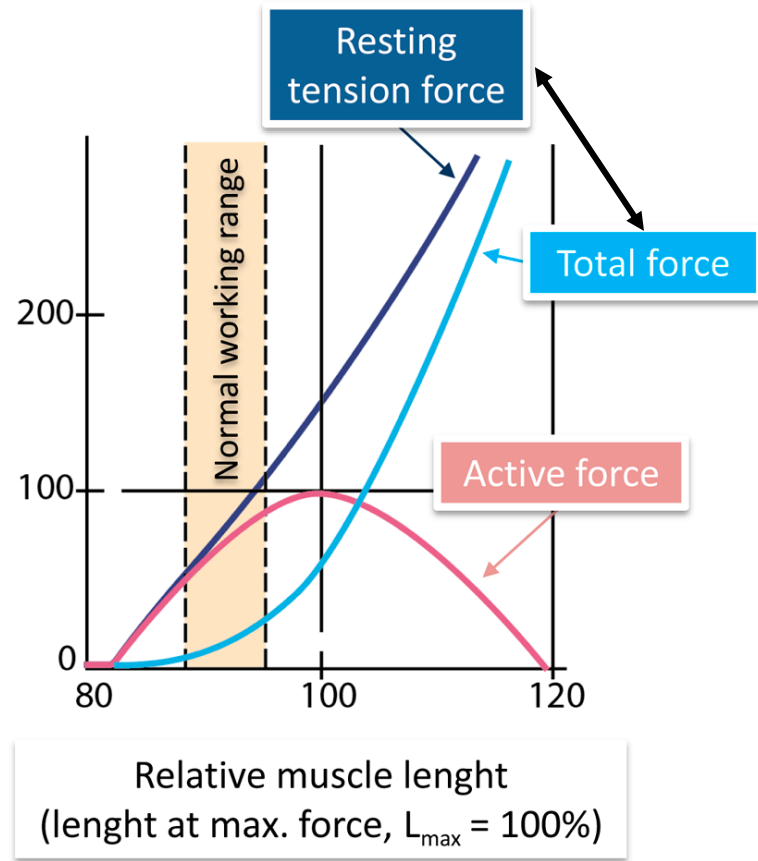
Autoregulation of contraction – **heterometric** (Starling)

Preload, afterload

1. Striated muscle



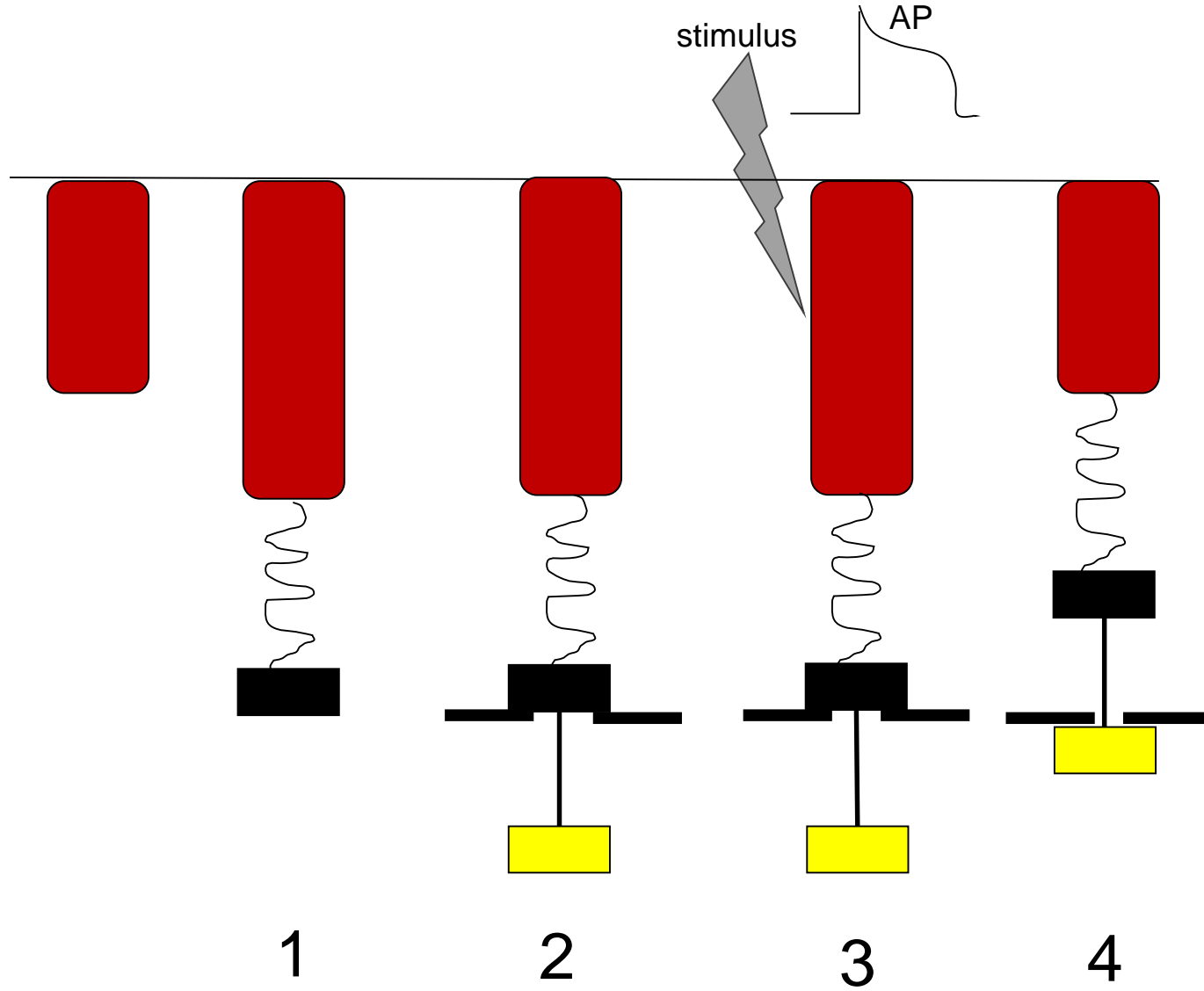
2. Cardiac muscle



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

AFTERLOADED CONTRACTION

PRELOAD, AFTERLOAD



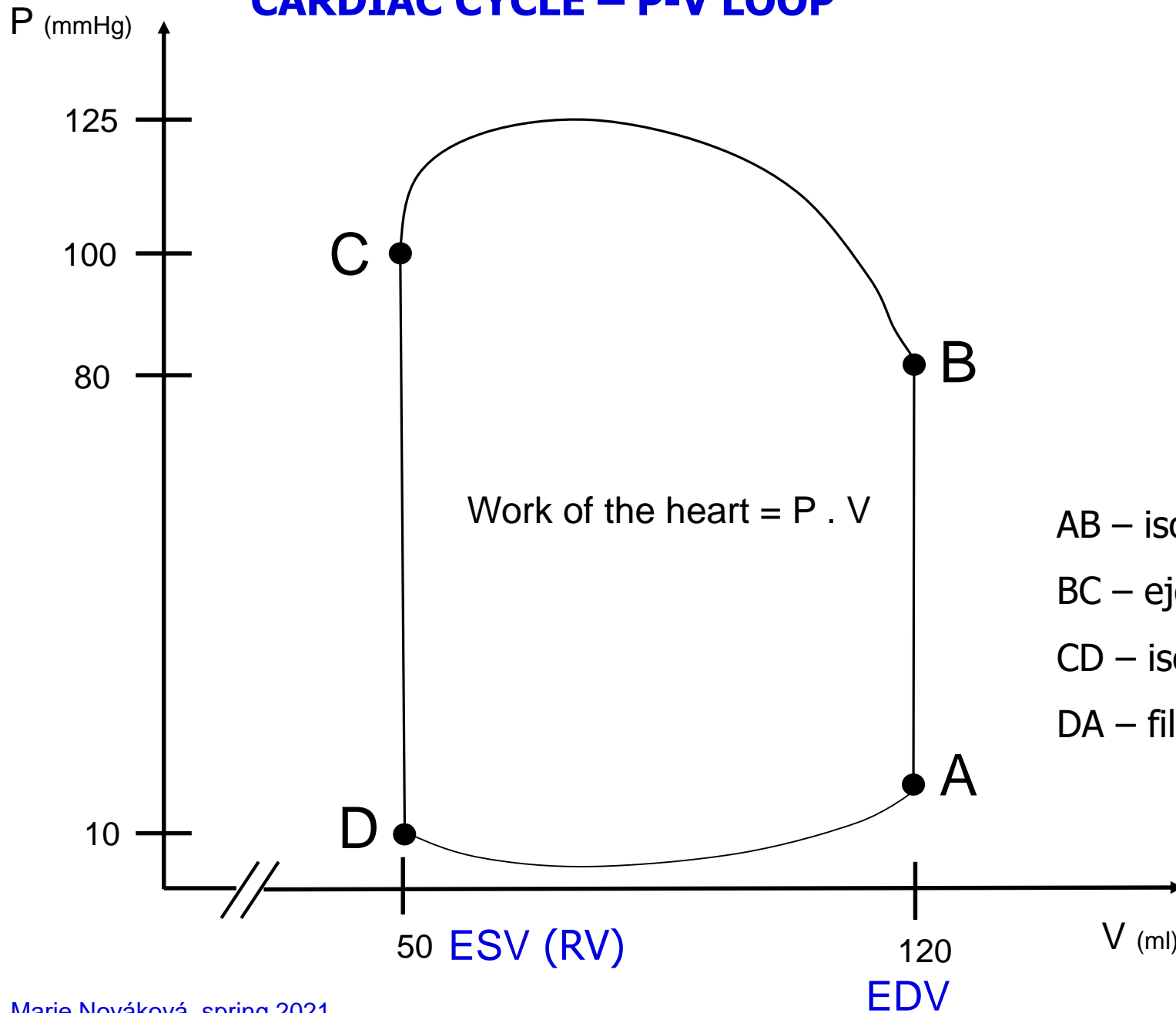
PRELOAD
(~ enddiastolic filling)



AFTERLOAD
(~ pressure which must be developed)



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

$$P = T \cdot 2h \cdot r^{-1}$$

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)

$$P = T \cdot 2h \cdot r^{-1}$$

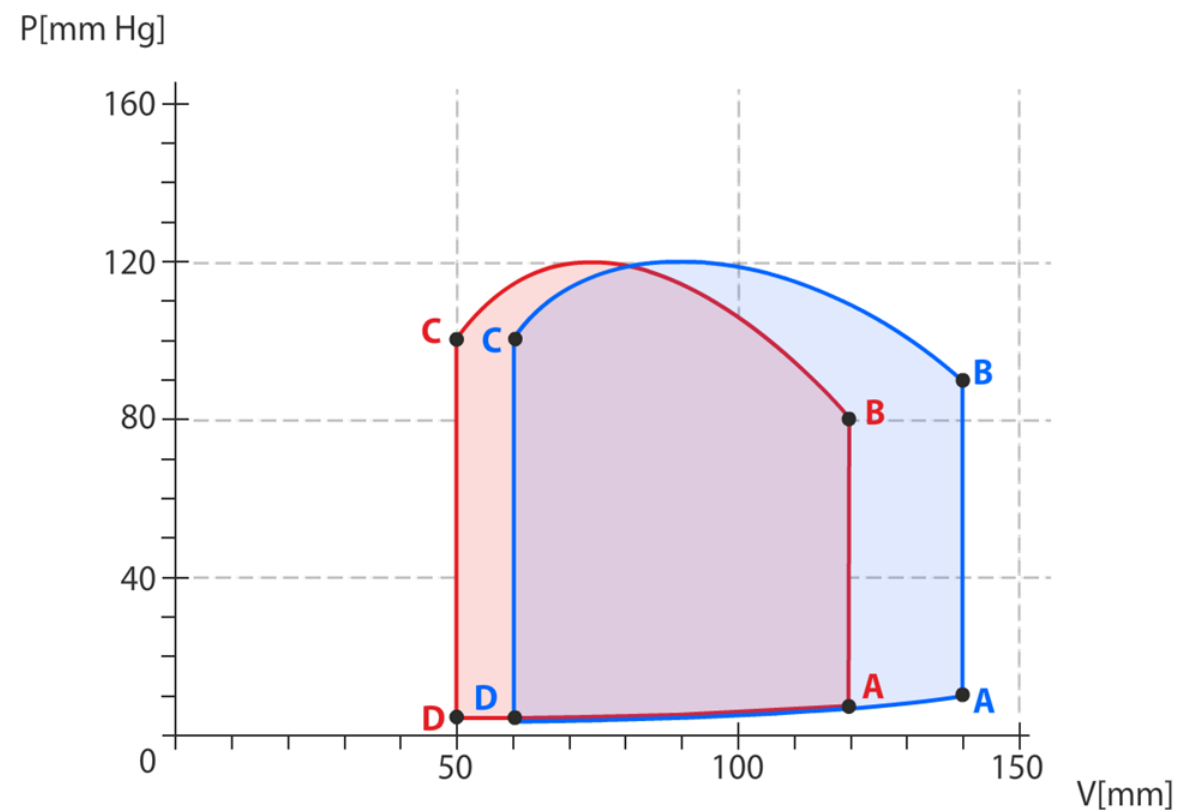
Isovolumic relaxation: T decreases, valves closed – decrease in P

$$P = T \cdot 2h \cdot r^{-1}$$

Ventricular filling: r and T rise, P first falls down, then rises up (length/tension relationship)

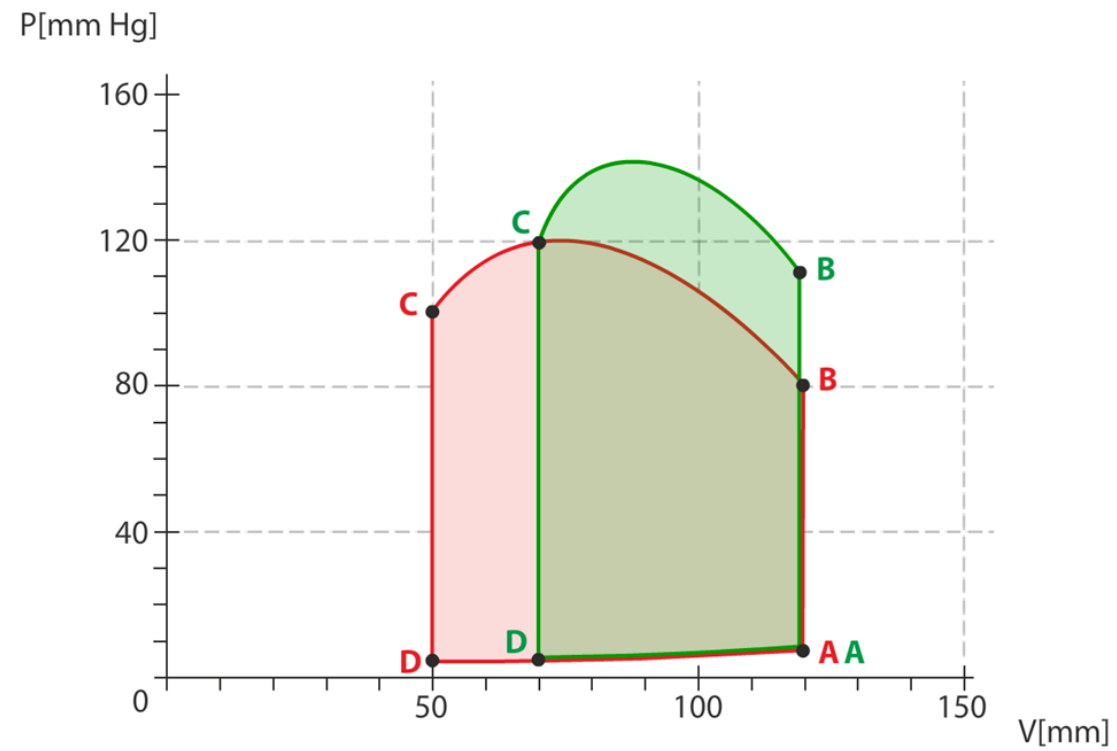
INCREASED PRELOAD

MODEL



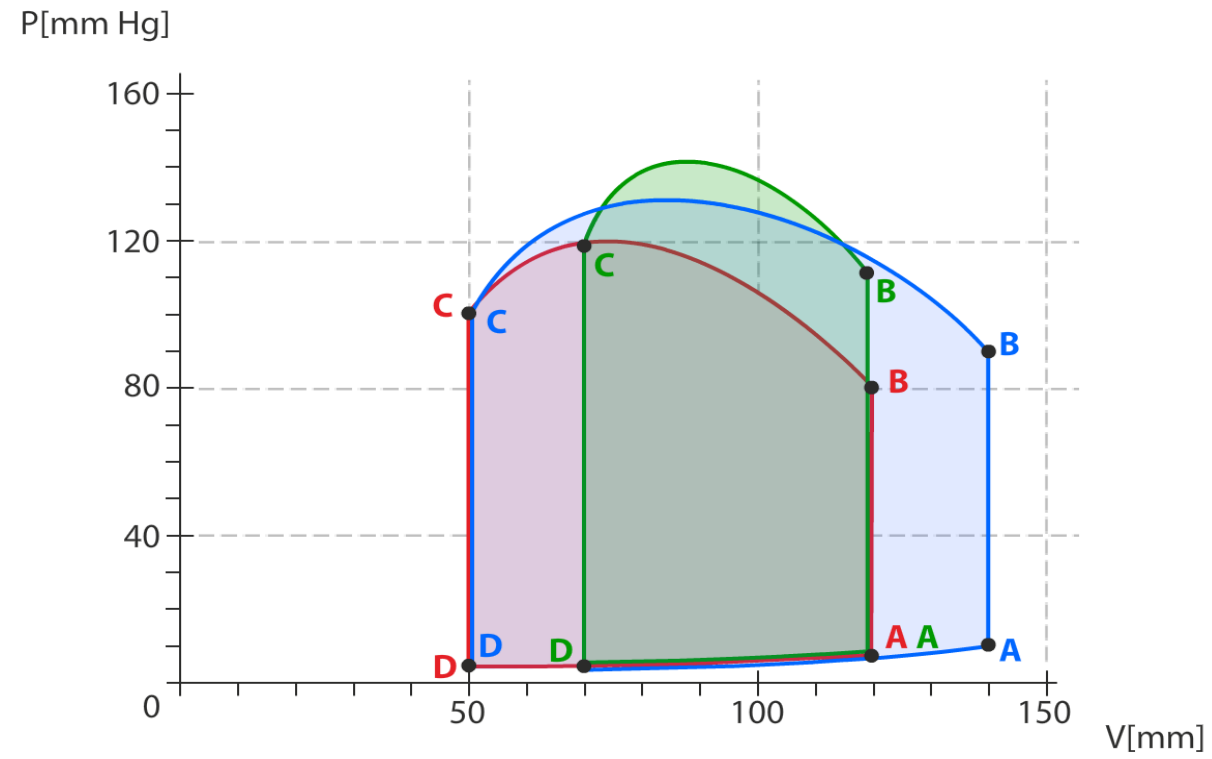
INCREASED AFTERLOAD

MODEL



INCREASED PRELOAD AND AFTERLOAD

MODEL



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

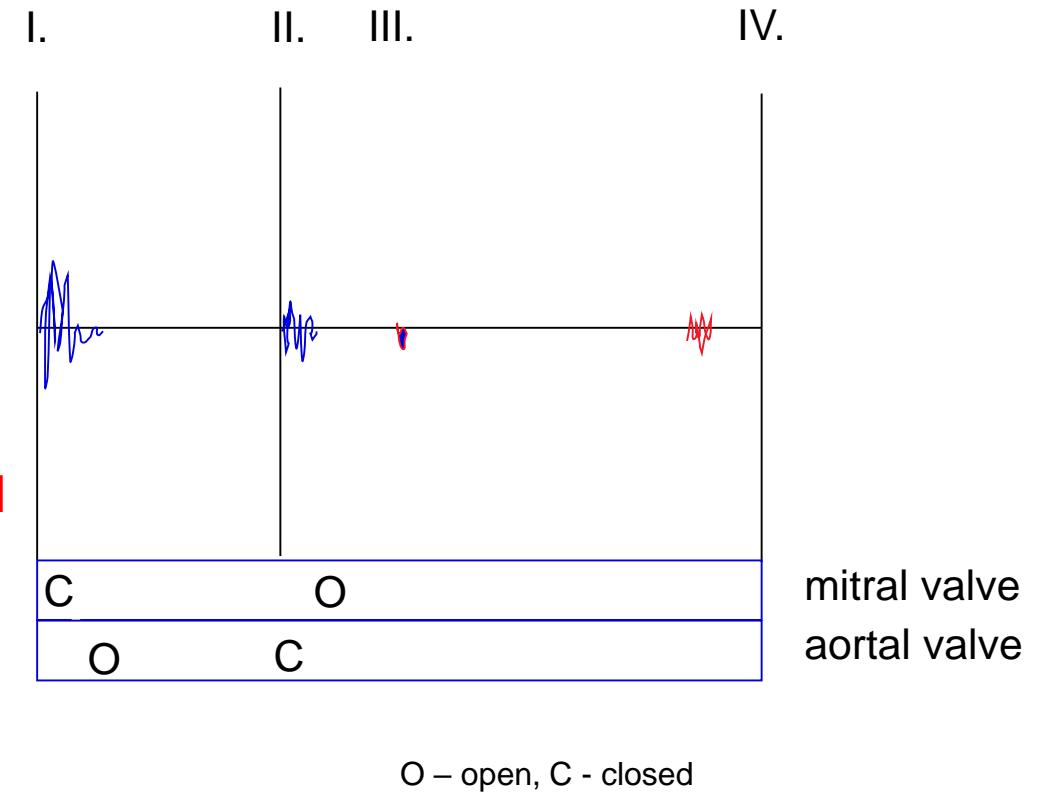
II. - aortal (+ pulmonary) valve closure

III. - fast filling of ventricles - **pathological**

IV. - contraction of atria – **mostly pathological**

→ **Vibration of ventricular wall**

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



MURMURS – pathological phenomena based on turbulent blood flow

1. SYSTOLIC

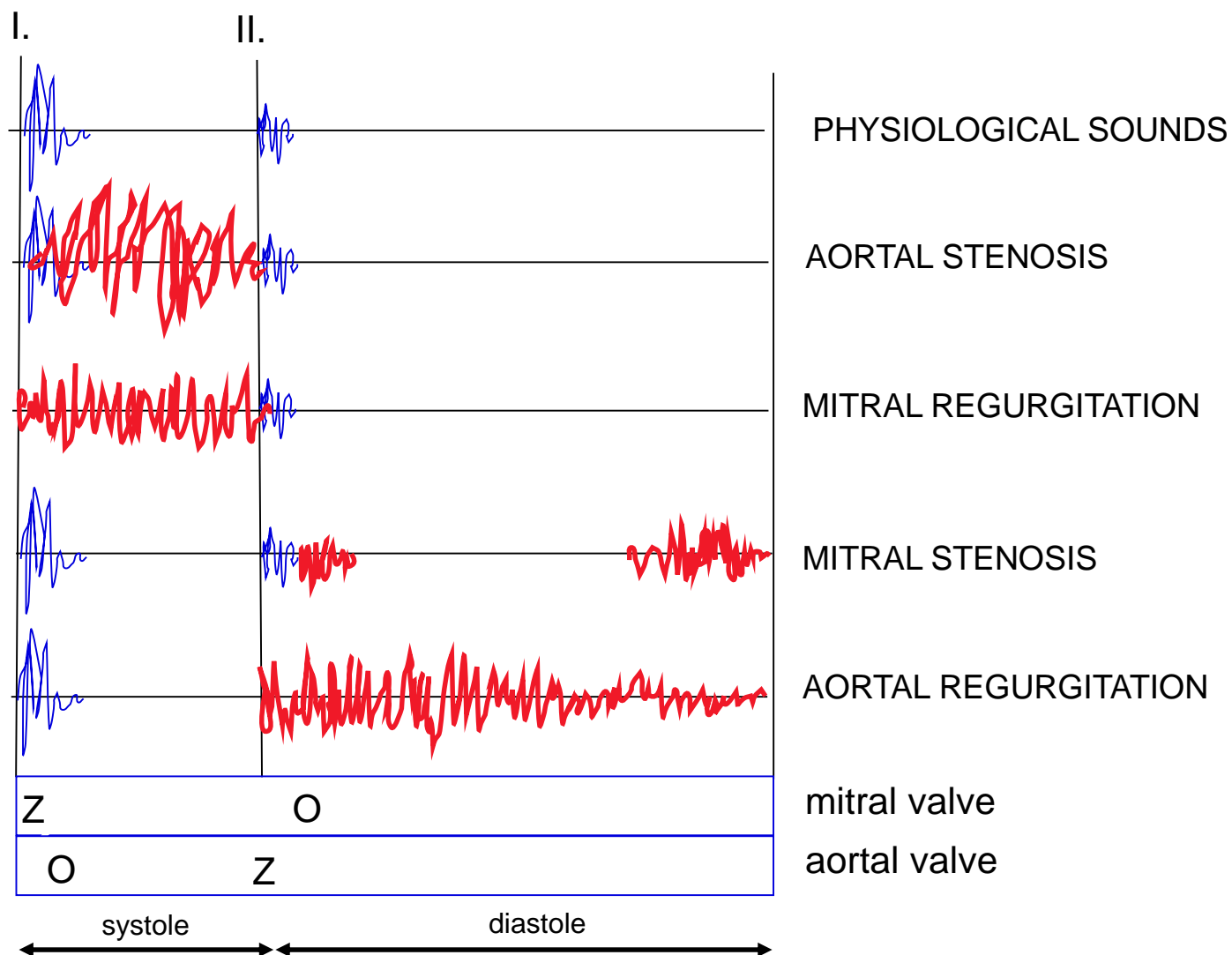
- Stenosis – aortal, pulmonary (1)
- Regurgitation – mitral, tricuspidal (2)

2. DIASTOLIC

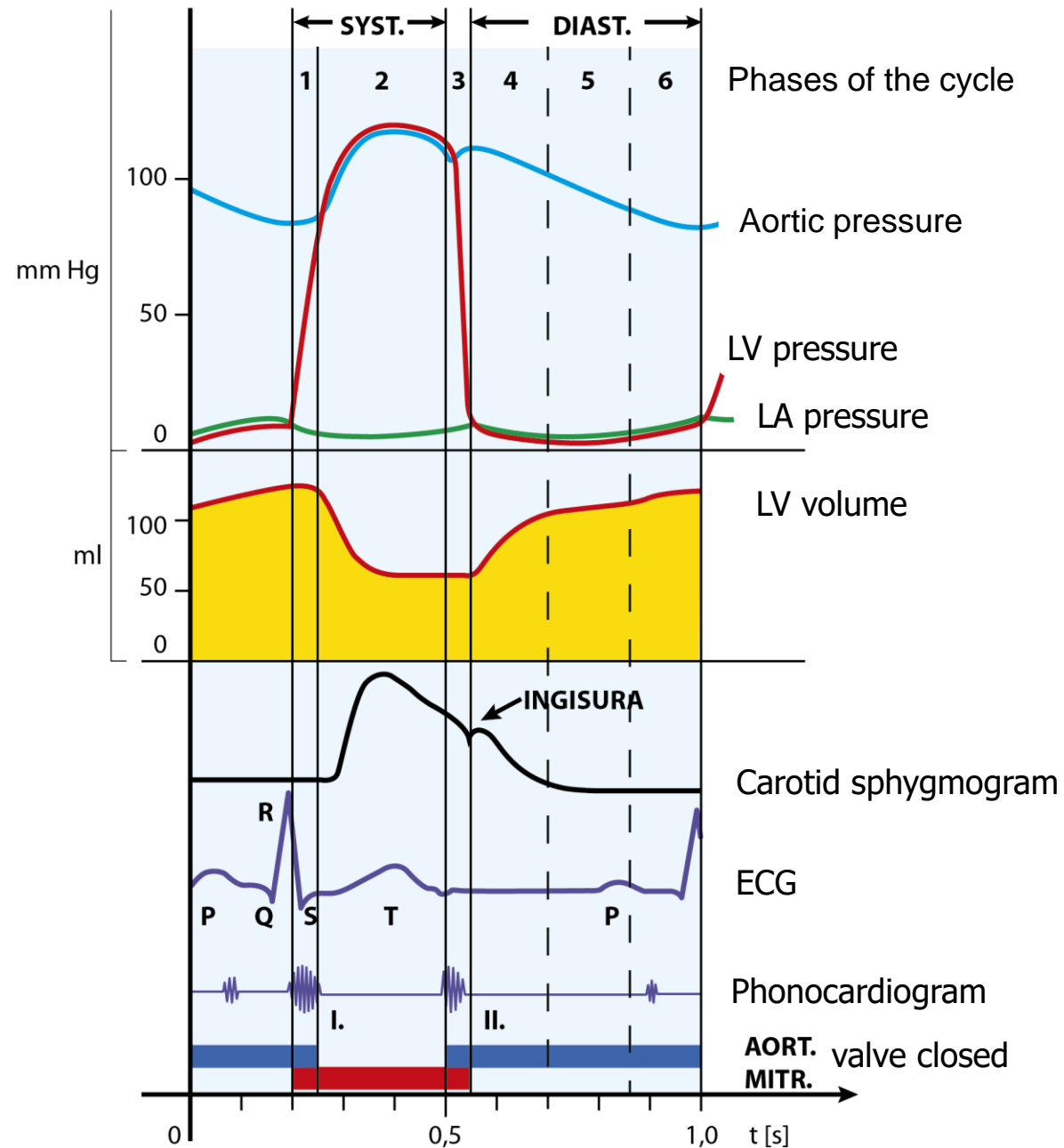
- Stenosis – mitral, tricuspidal (3)
- Regurgitation – aortal, pulmonary (4)

3. SUSTAINED:

- Defects of septum



POLYGRAPHY (polygram)



HEART FAILURE = loss of cardiac reserve

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

SYMPTOMS

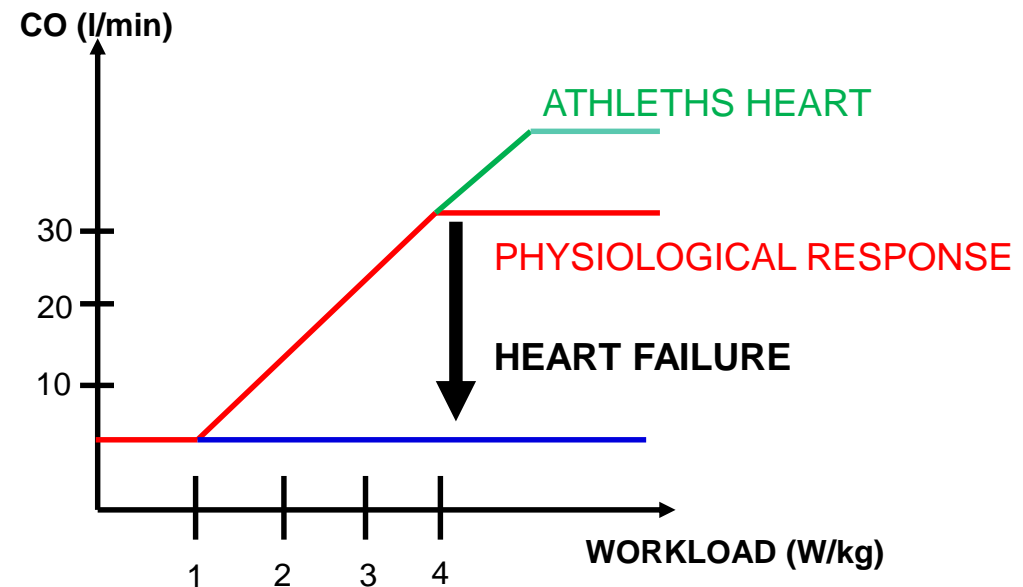
fatigue, oedemas, venostasis, dyspnoea, cyanosis

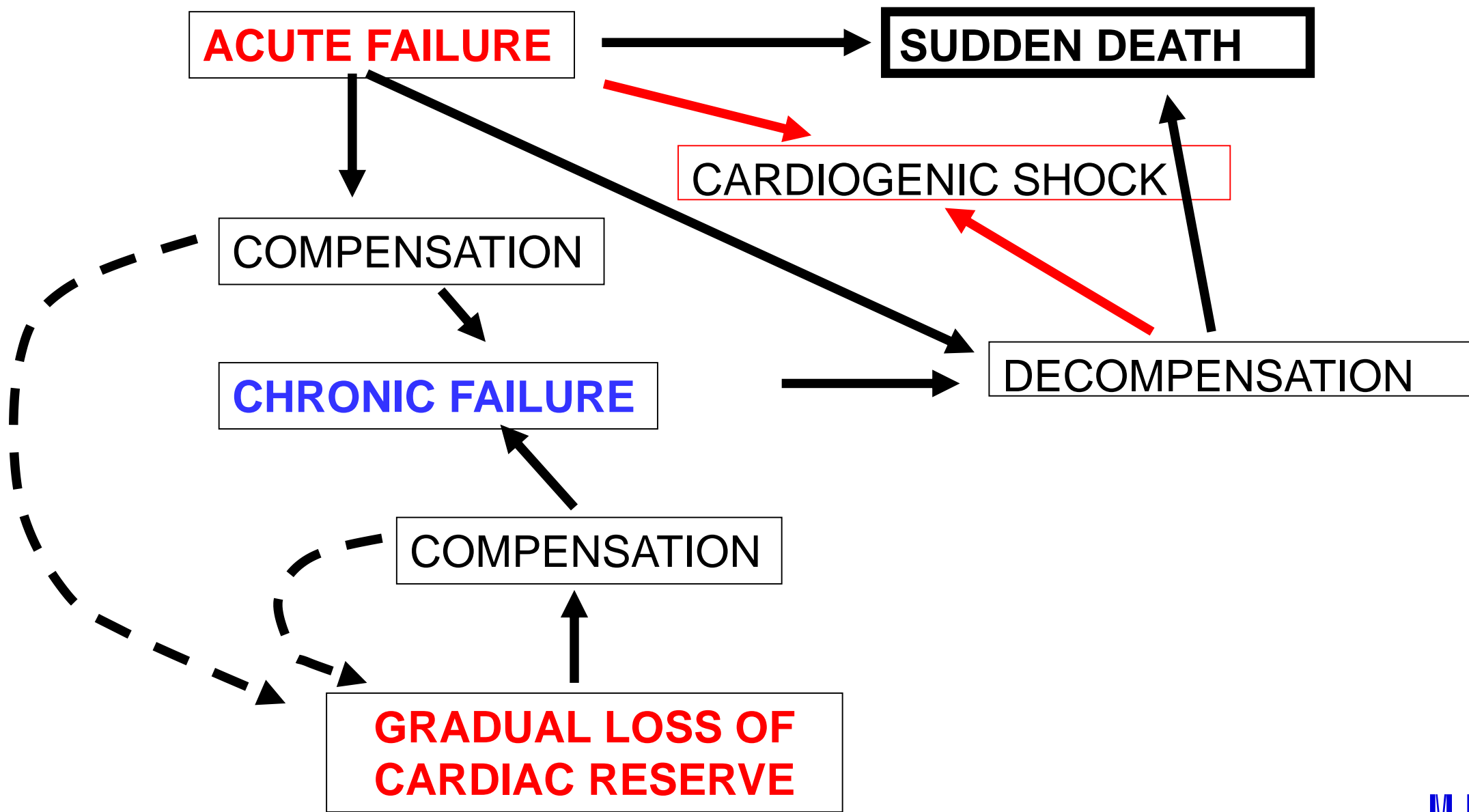
ACUTE x CHRONIC.

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](#)





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**

Ca²⁺ - antagonists

β – sympatholytics

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: $P = s \cdot 2 h / r$, 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)