CARDIAC MECHANICS

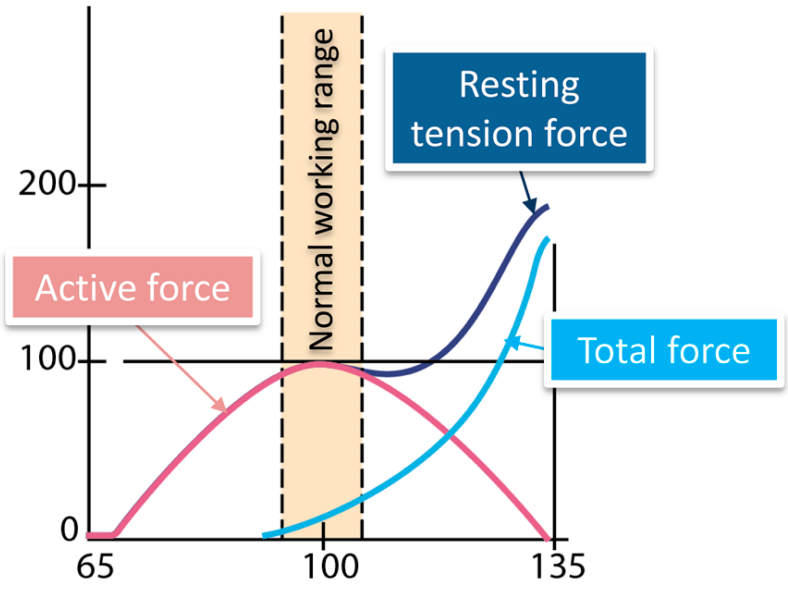
HEART AS A PUMP

CARDIAC CYCLE

HEART FAILURE

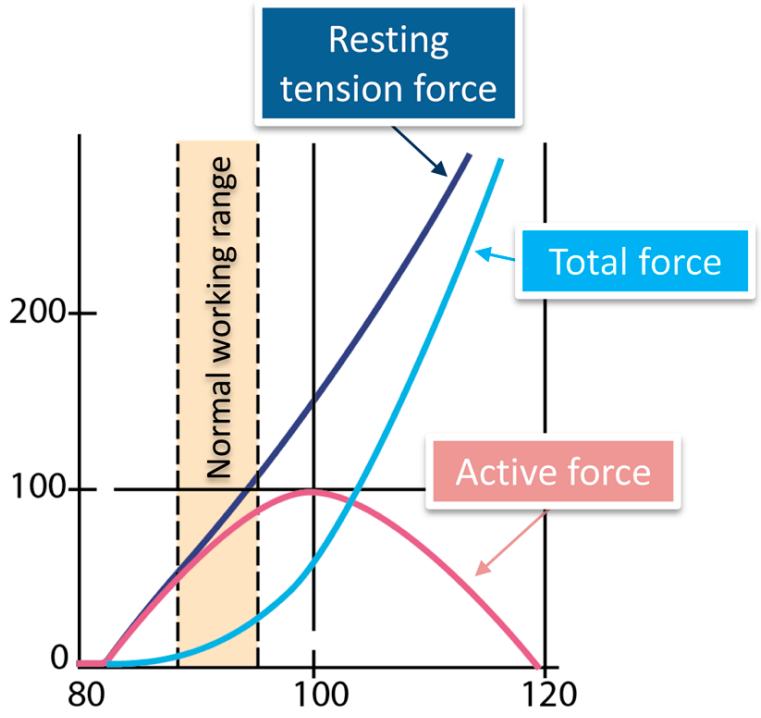
LENGTH – TENSION RELATIONSHIP

1. Striated muscle



Relative muscle lenght
(lenght at max. force, L_{max} = 100%)

2. Cardiac muscle

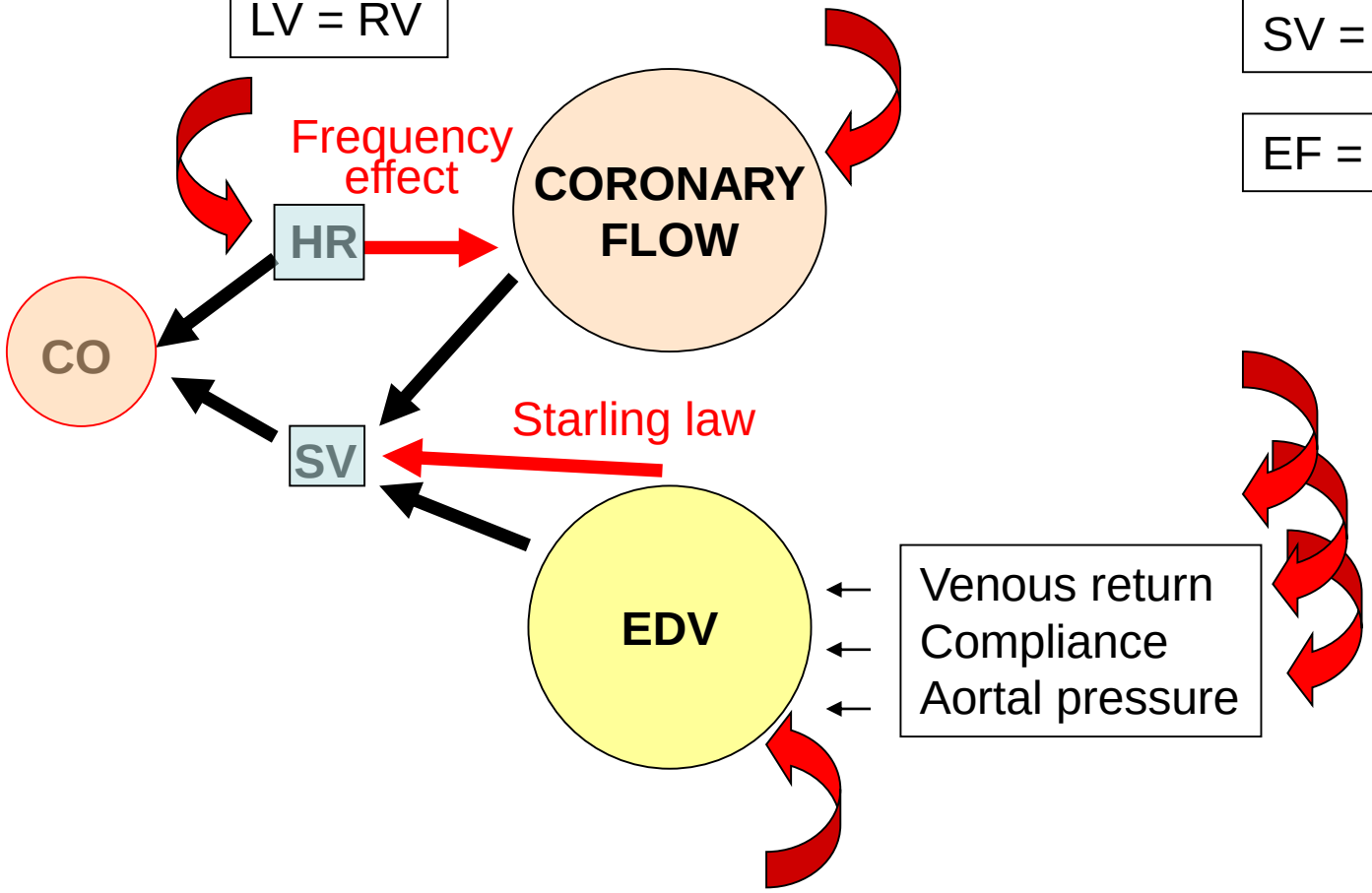


Relative muscle lenght
(lenght at max. force, L_{max} = 100%)

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

CARDIAC OUTPUT

LV = RV



CO = HR x SV 5l/min

SV = EDV - ESV 70ml

EF = EDV – ESV / EDV >60%

sympathetic, parasympathetic stimulation

CONTRACTILITY

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

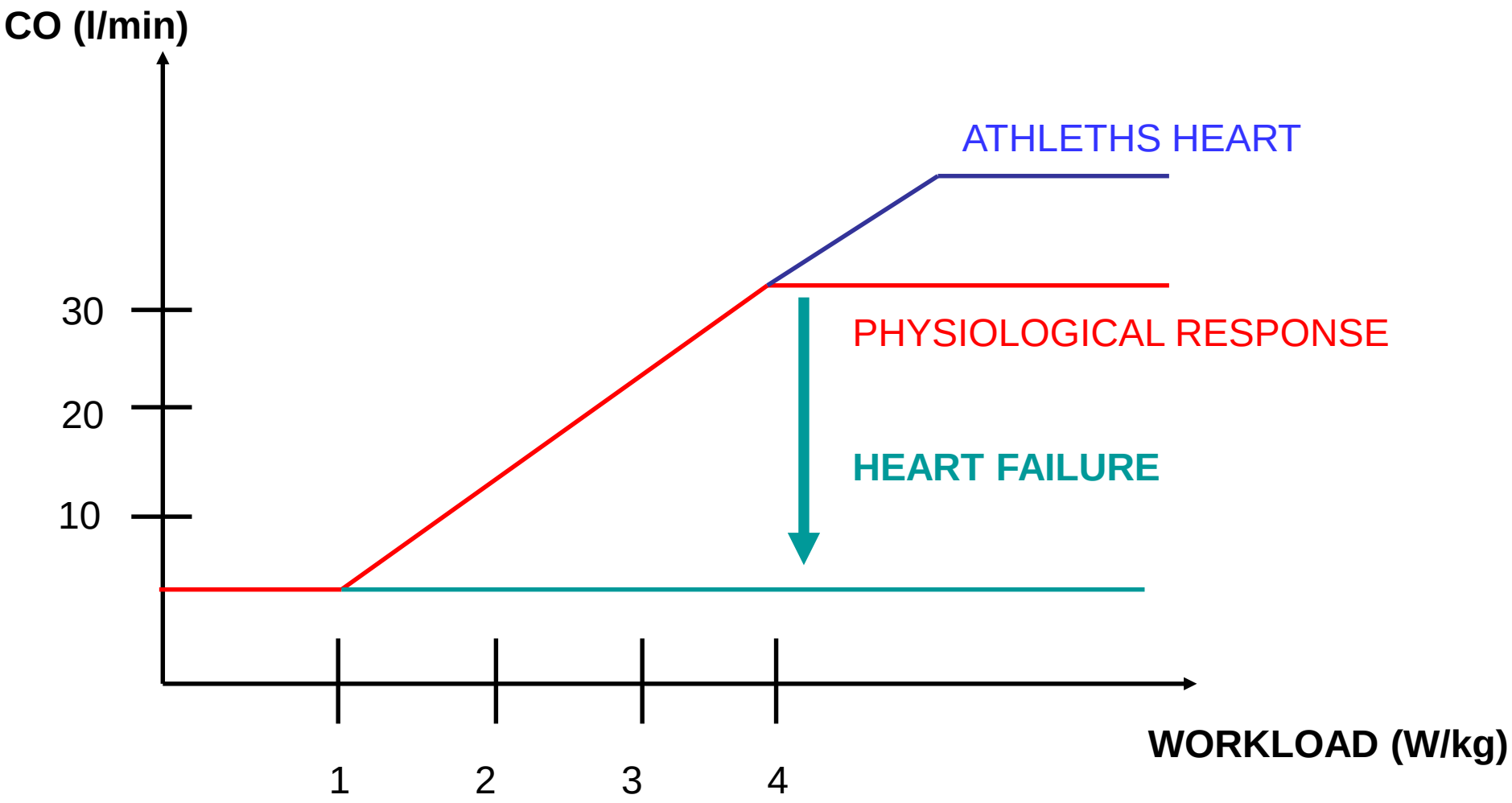
CORONARY RESERVE = maximal CF / resting CF **3,5**

CHRONOTROPIC RESERVE = maximal HR / resting HR **3 - 5**

VOLUME RESERVE = maximal SV / resting SV **1,5**

CARDIAC INDEX = CO / body surface

CARDIAC RESERVE

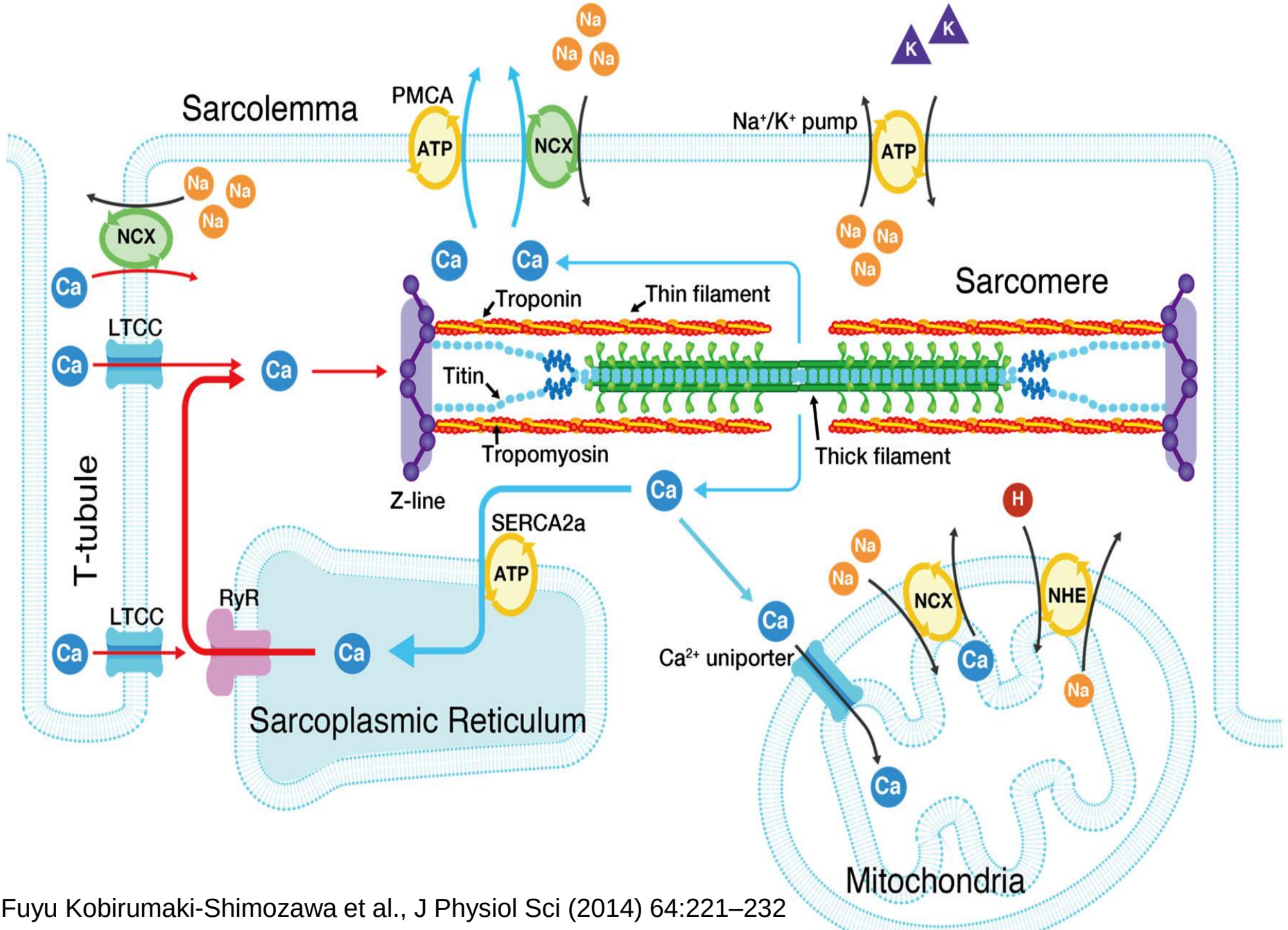


HEART AUTOREGULATION (FORCE)

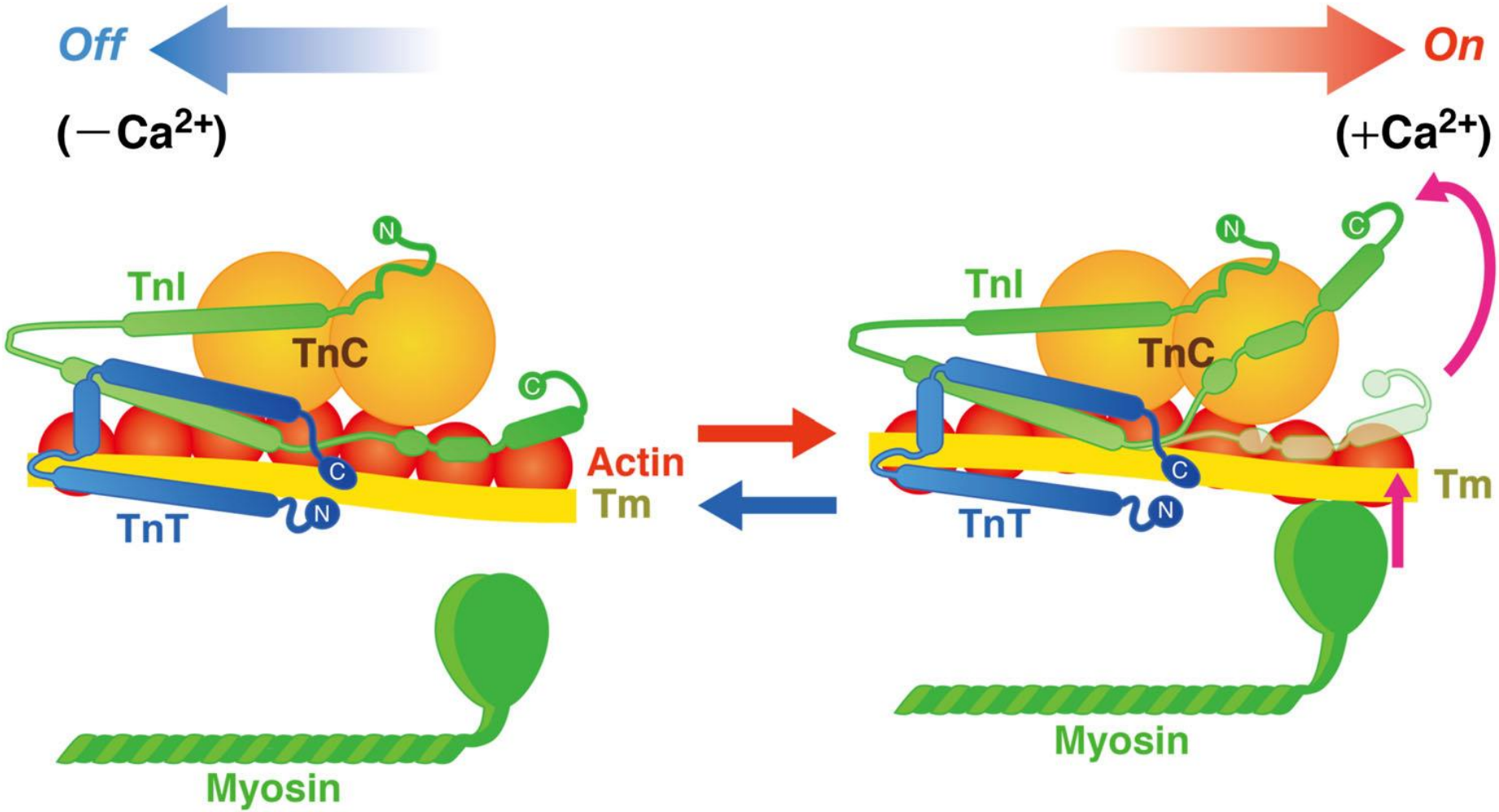
HETEROMETRIC – STARLING LAW

HOMEOMETRIC – FREQUENCY EFFECT

STARLING LAW



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



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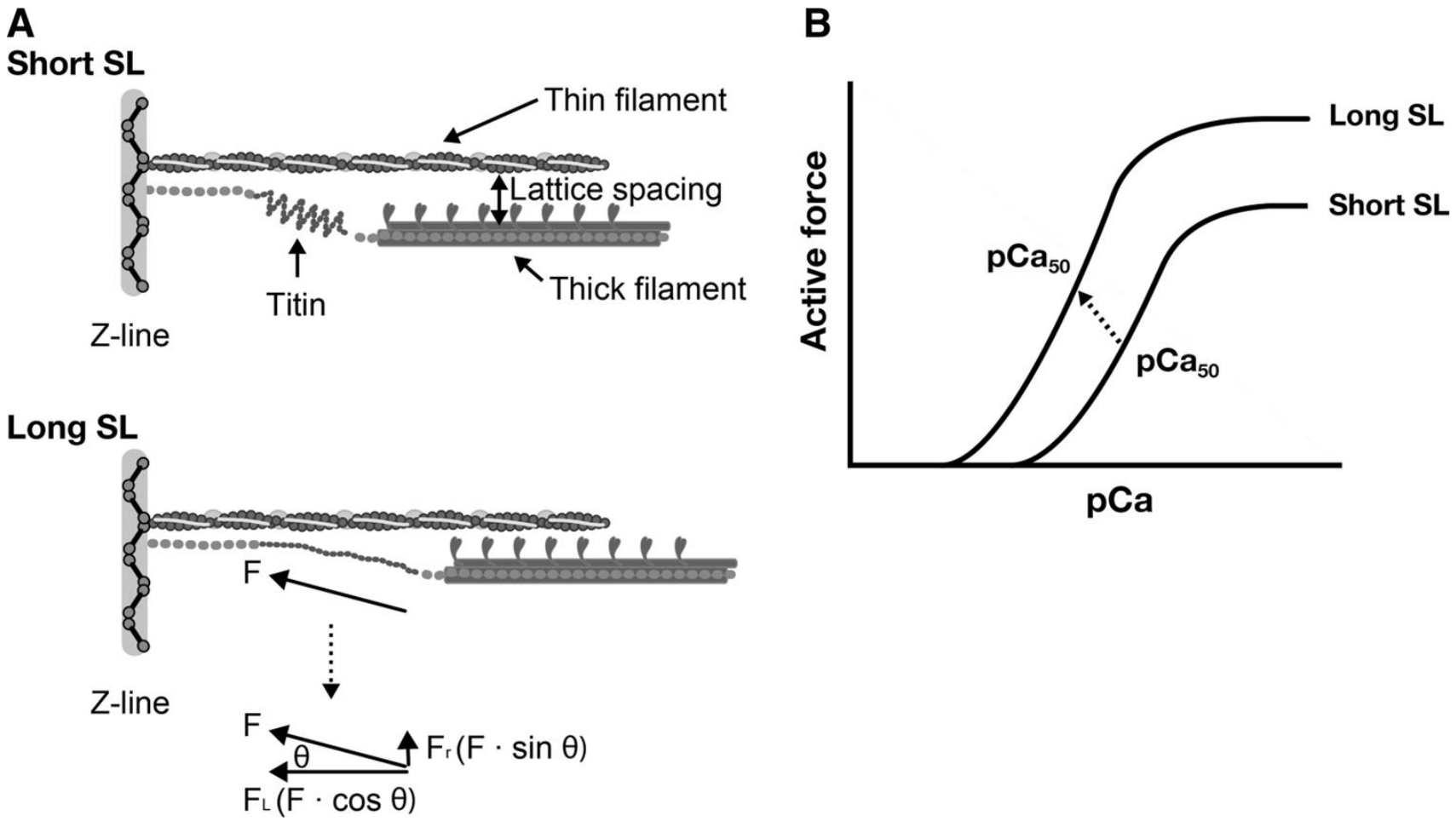
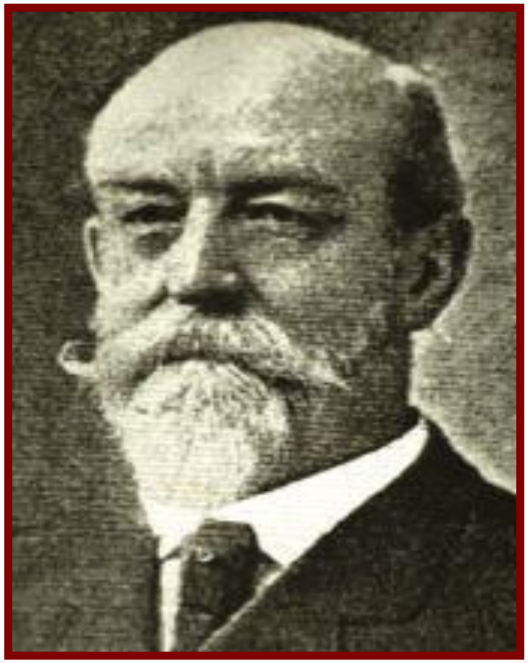
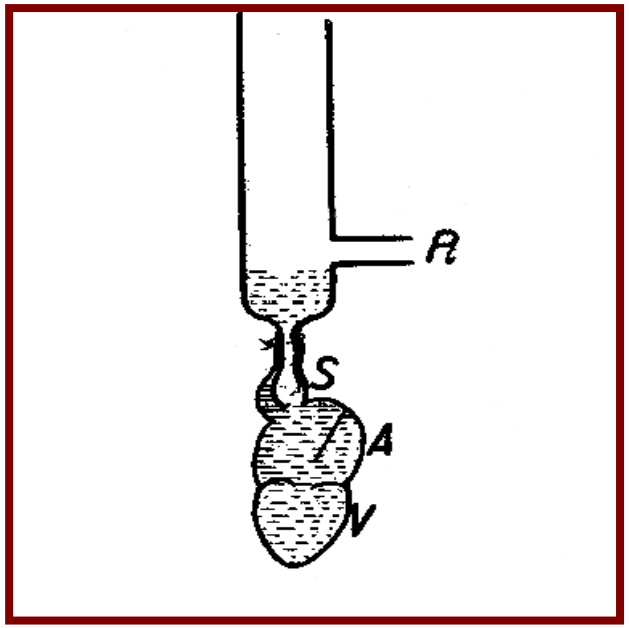
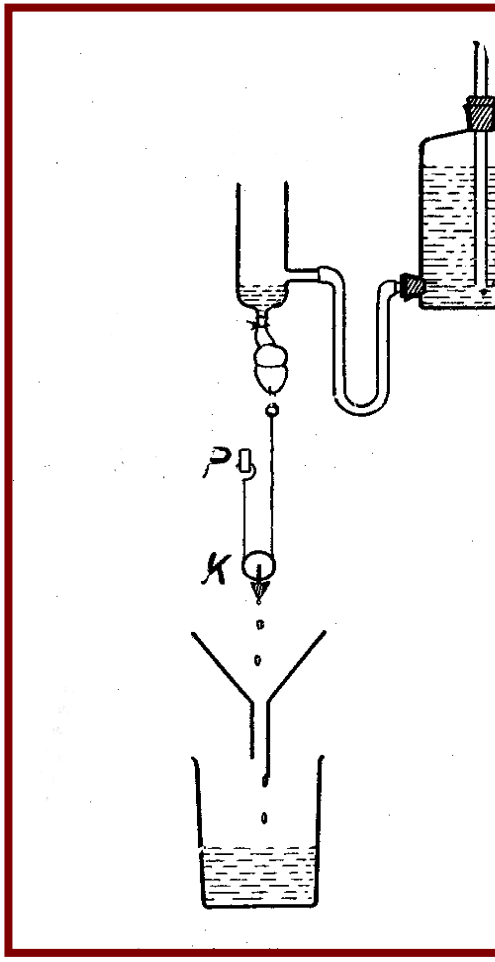


Fuyu Kobirumaki-Shimozawa et al., J Physiol Sci (2014) 64:221–232



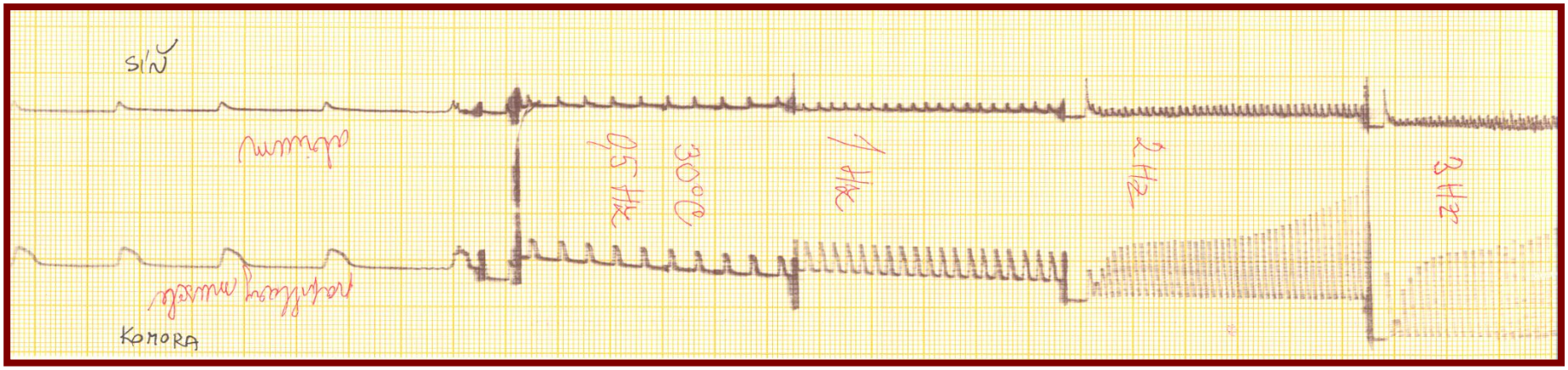
Henry Pickering Bowditch
(1840 – 1911)

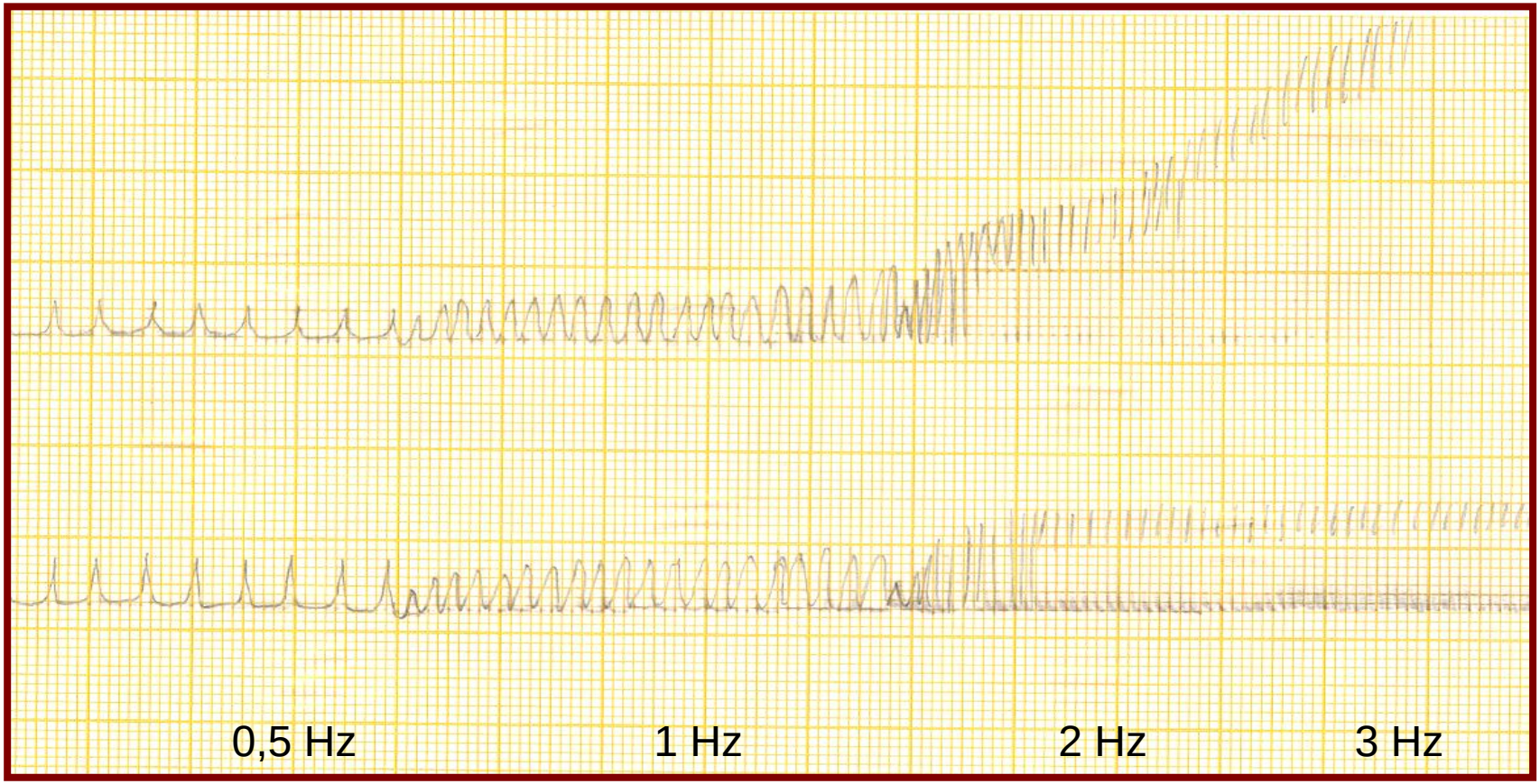




HOMEOMETRIC AUTOREGULATION (FREQUENCY EFFECT)

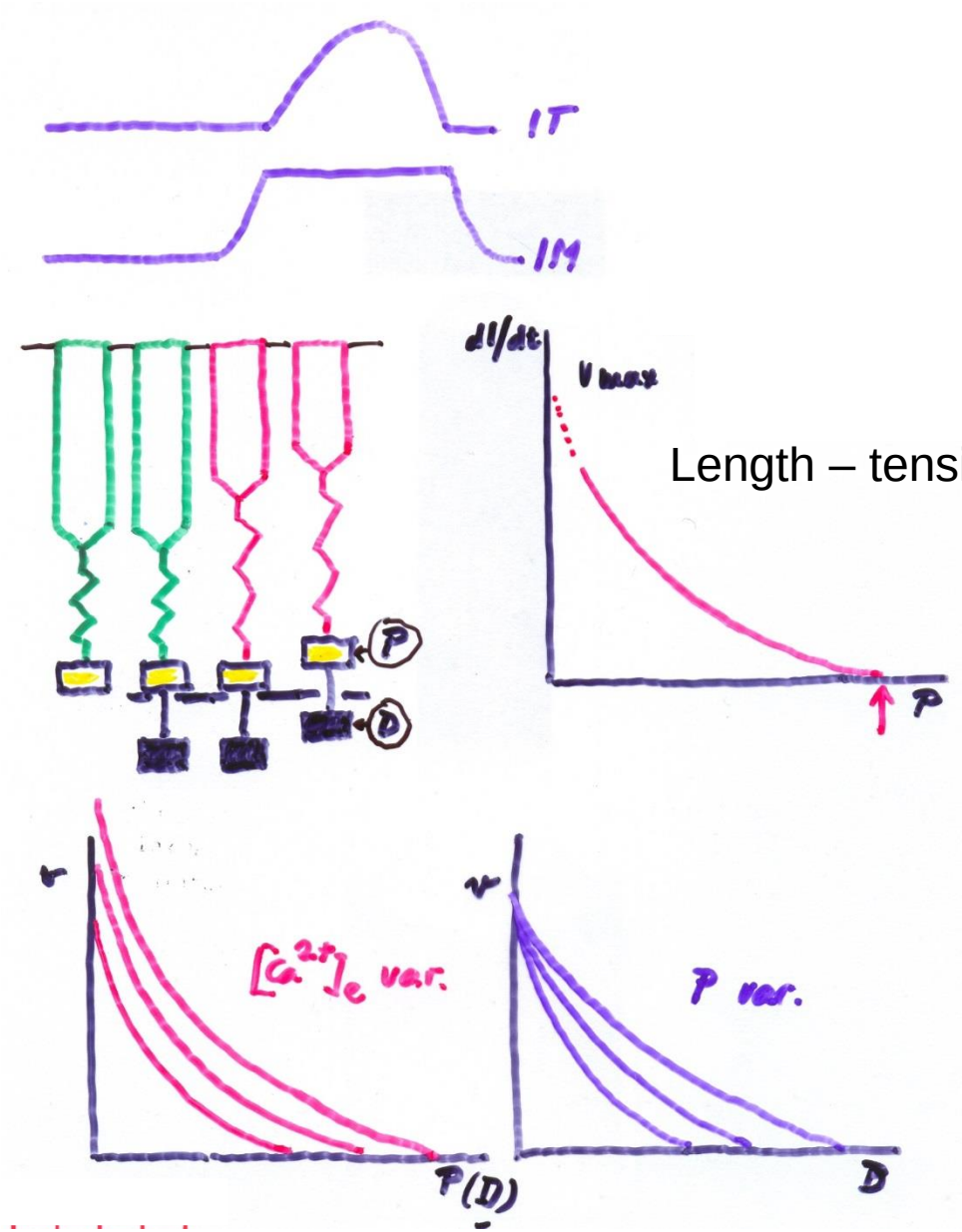
During increasing HR (stimulation frequency) the force of developed contraction increases


SIN
0,5 Hz
30°C
1 Hz
2 Hz
3 Hz
KOMORA


0,5 Hz
1 Hz
2 Hz
3 Hz

AFTERLOADED CONTRACTION

PRELOAD, AFTERLOAD



Length – tension relationship

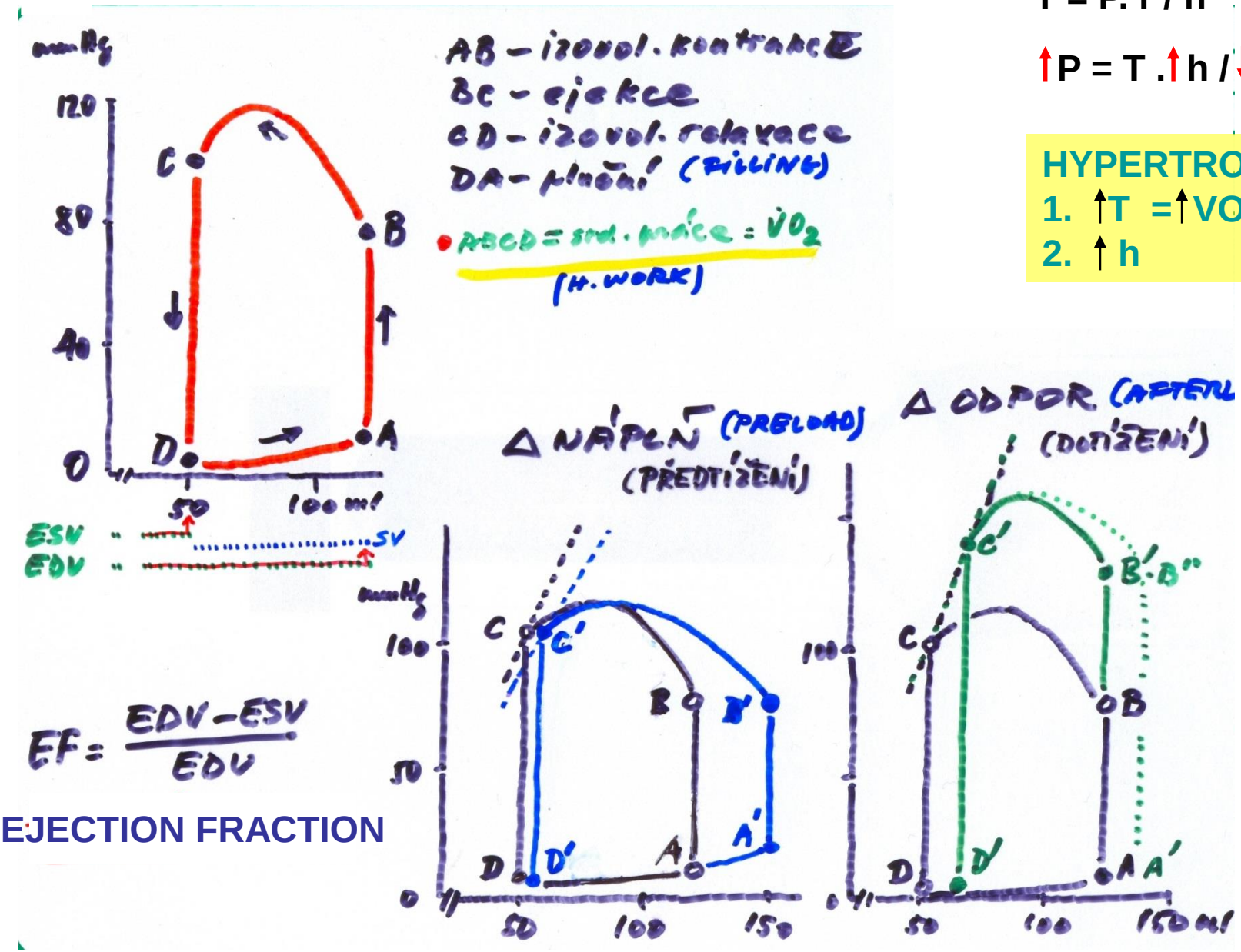
LAPLACE LAW

T = P. r / h

↑P = T .↑h /↓r

HYPERTROPHY

1. **↑T =↑VO_2**
2. **↑h**



EJECTION FRACTION

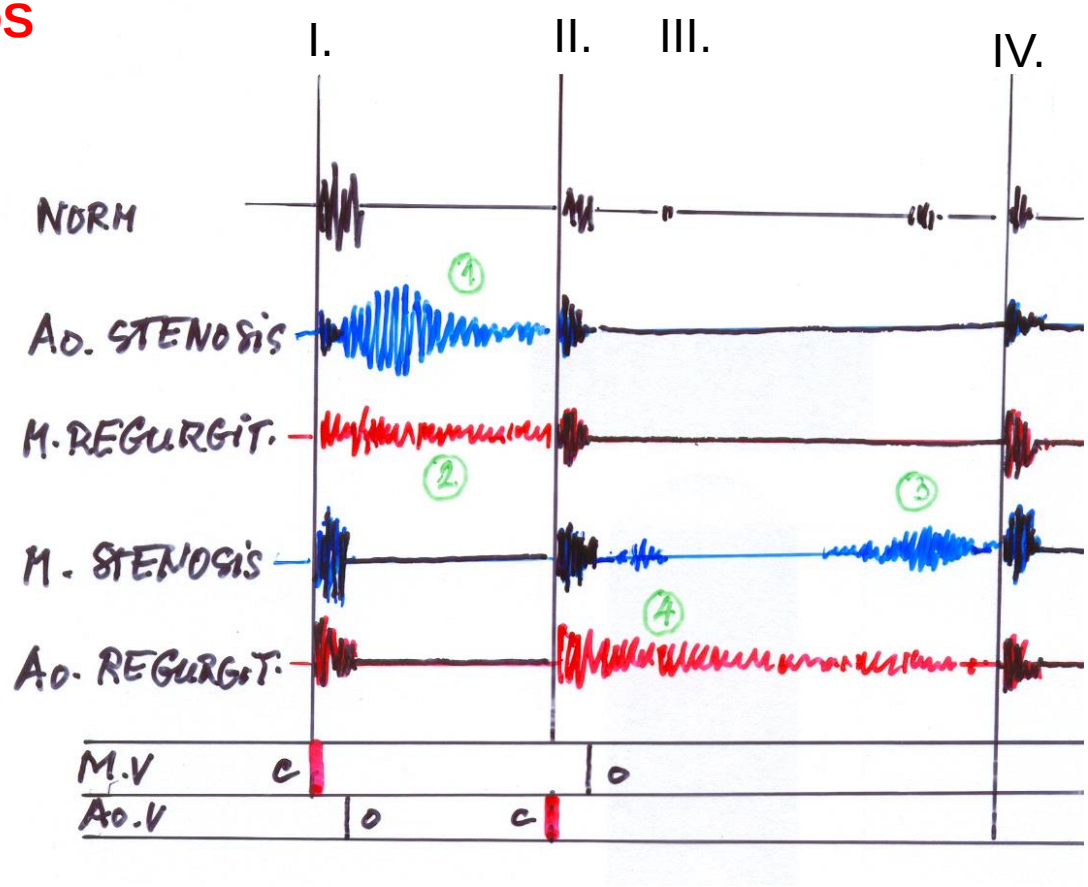
$P = \frac{T}{r^{-1}} \cdot 2h$ **Ventricular filling:** r and T rise, P first falls down, then rises up (length/tension relationship)

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

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

HEART SOUNDS



I. – mitral (+ tricuspidal) valve closure

II. - aortal (+ pulmonary) valve closure

III. - fast filling of ventricles - pathological

IV. - contraction of atria – mostly pathological

Caused by vibration of:

- **Closure and stretching of valves**
- Izovolumic contraction of heart muscle (papill. muscles, tendons)
- Turbulent blood flow

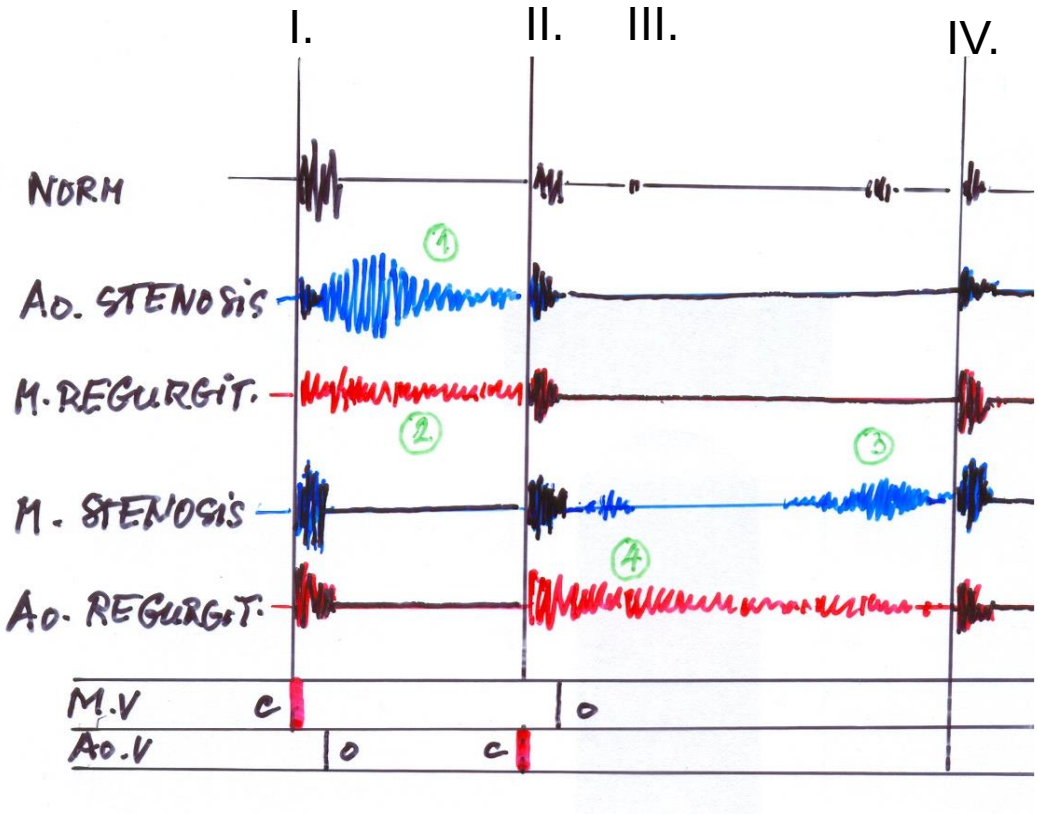
Vibration of ventricular wall

MURMURS – pathological phenomena

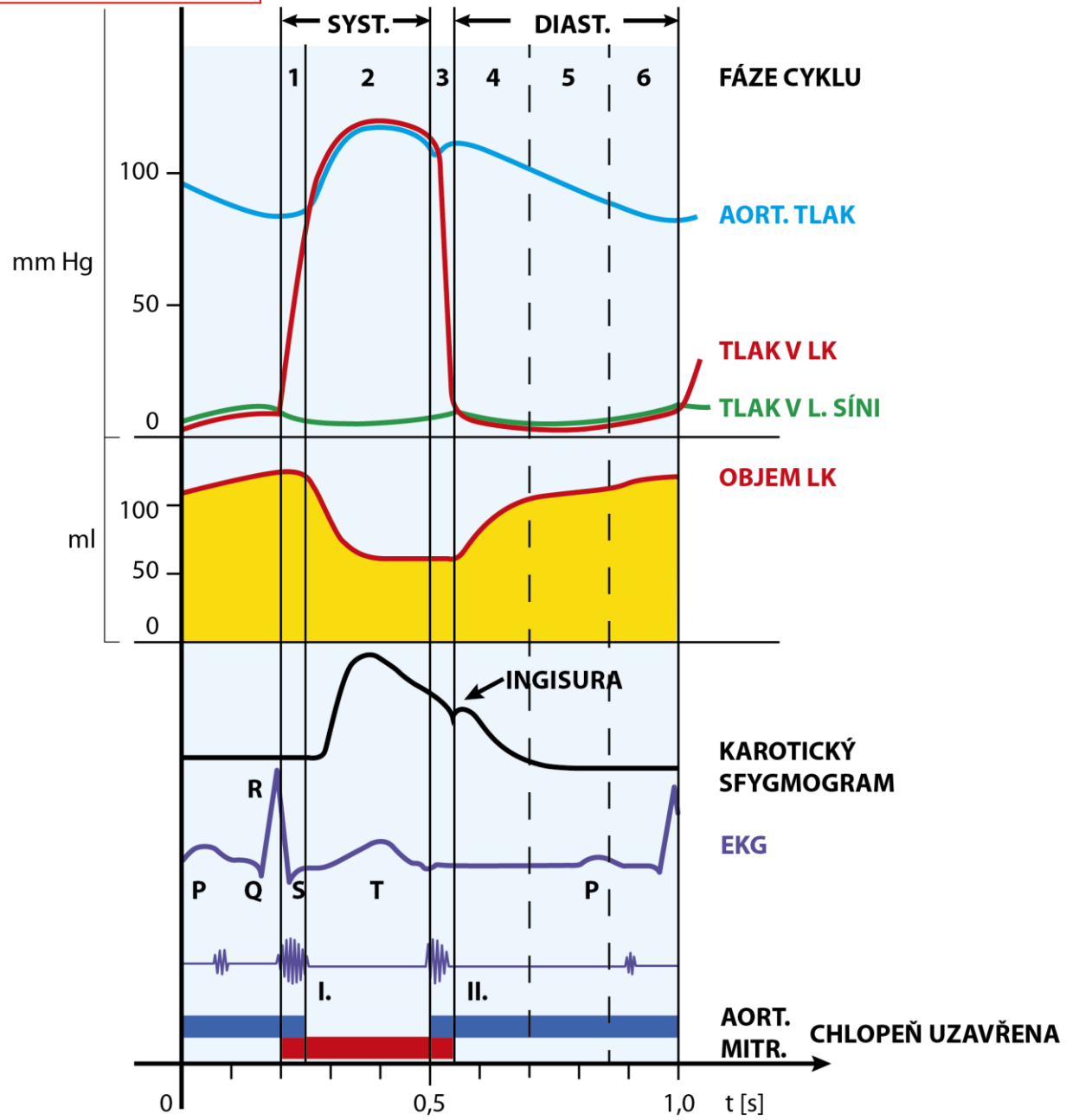
TURBULENT BLOOD FLOW

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

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



POLYGRAPHY (polygram)



HEART FAILURE

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

MOST OFTEN CAUSES:

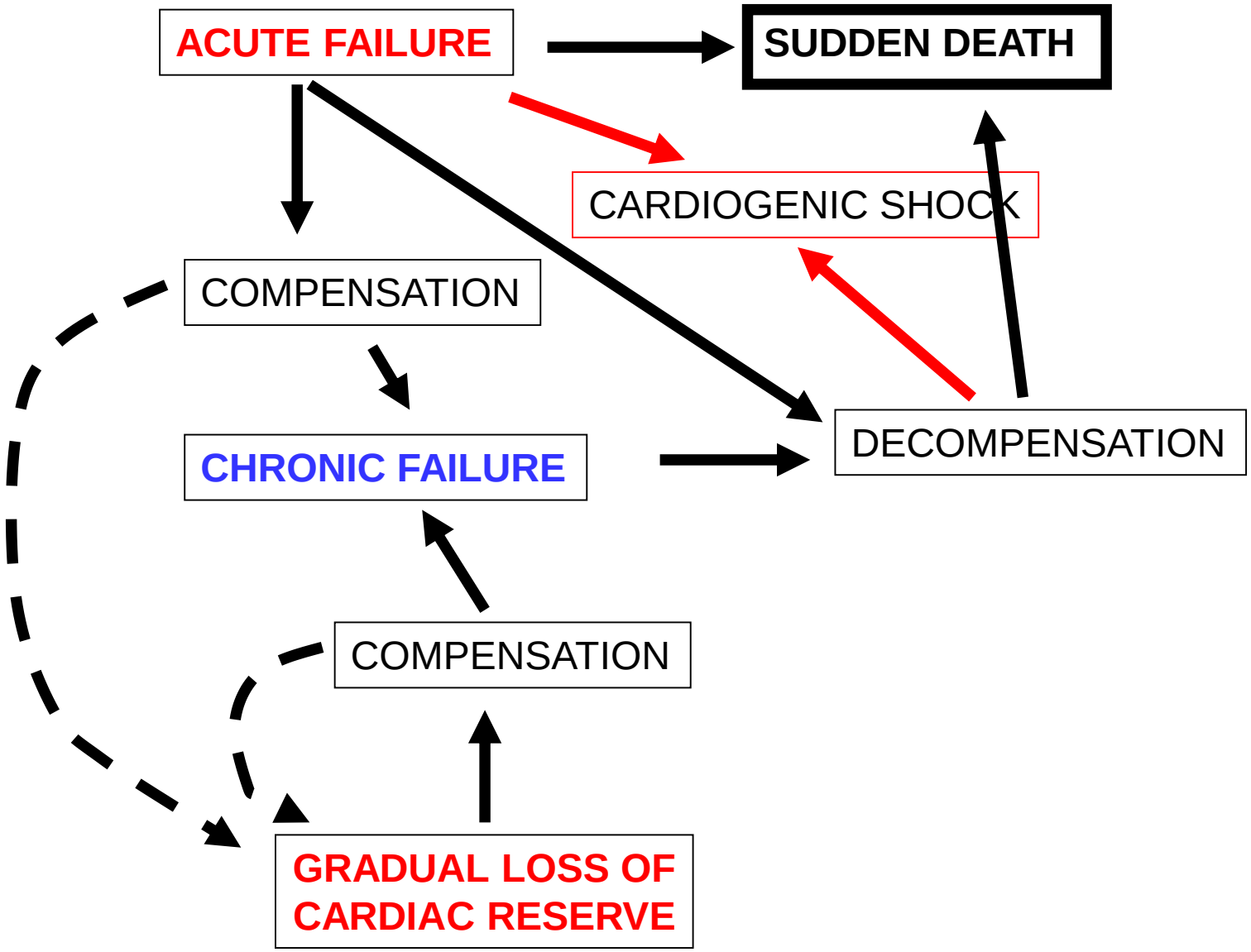
• Severe arrhythmias

• 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

SYMPTOMS: fatigue, oedemas, venostasis, dyspnoea, cyanosis

ACUTE x CHRONIC. COMPENSATED x DECOMPENSATED.


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

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**

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**

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: $P = \sigma \cdot 2\,h / r$, intermittent BP increase (athletes heart)

Response: concentric remodelling

Pathological signal: continual increase of preload or afterload

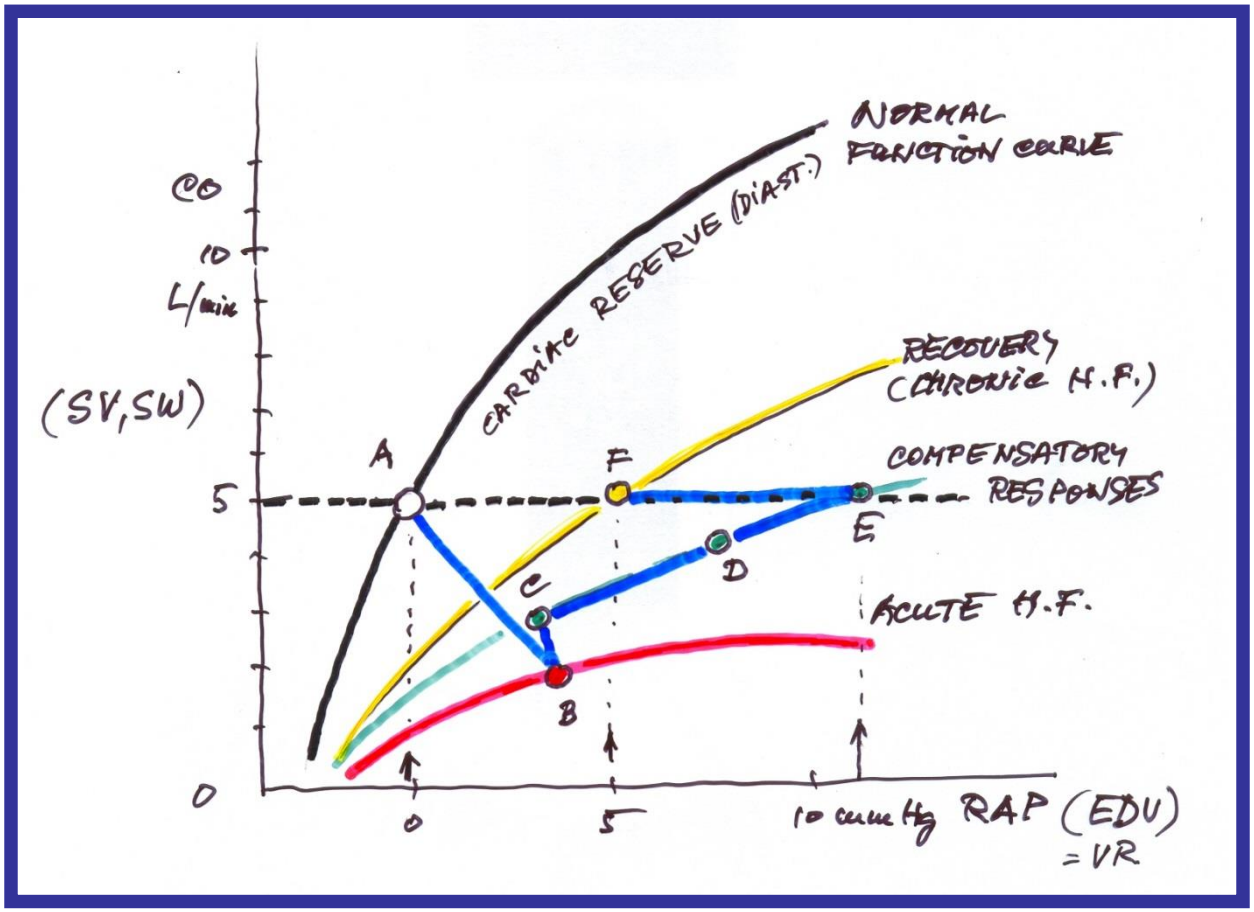
Results: worsening of oxygenation, fibrotisation – **vicious circle**

THERAPY OF CHRONIC HEART FAILURE

SPLITTING OF VICIOUS CIRCLES

- angiotenzin-converting enzyme inhibitors (AT II. receptors)
- β – sympatolytics
- diuretics
- cardiac glycosides (digitalis)
- Ca^{2+} - antagonists

COMPENSATION OF ACUTE HEART FAILURE



AB – acute heart failure (ventricular depression)

BC – acute sympathetic stimulation I. (increased contractility)

CD - acute sympathetic stimulation II. (venoconstriction, increased venous return)

DE – retention of fluids – COMPENSATION

EF – increased contractility – COMPENSATION OF CHRONIC HEART FAILURE