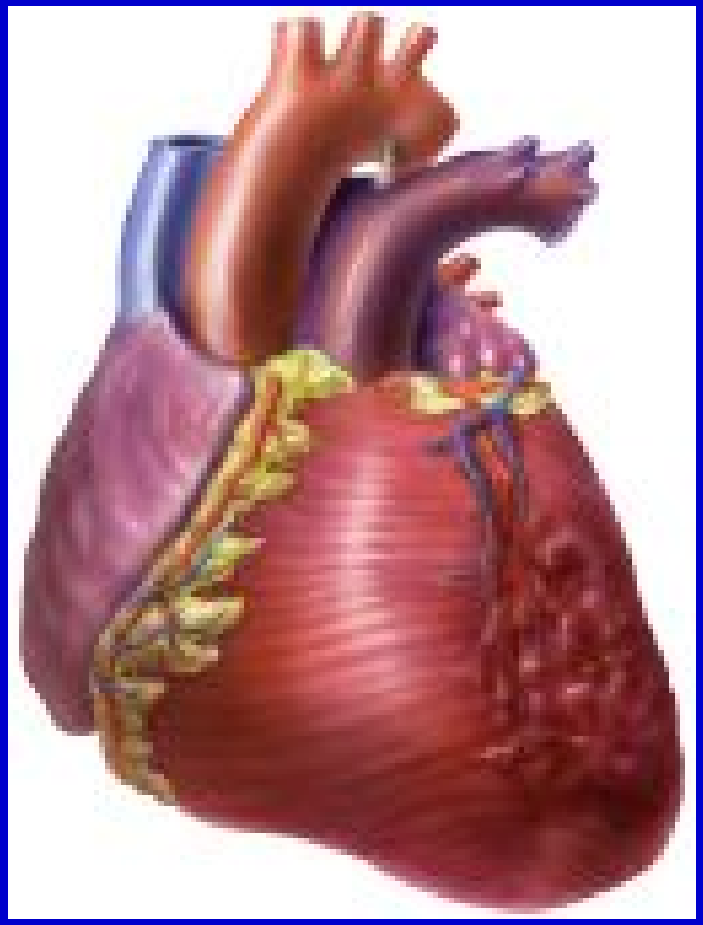
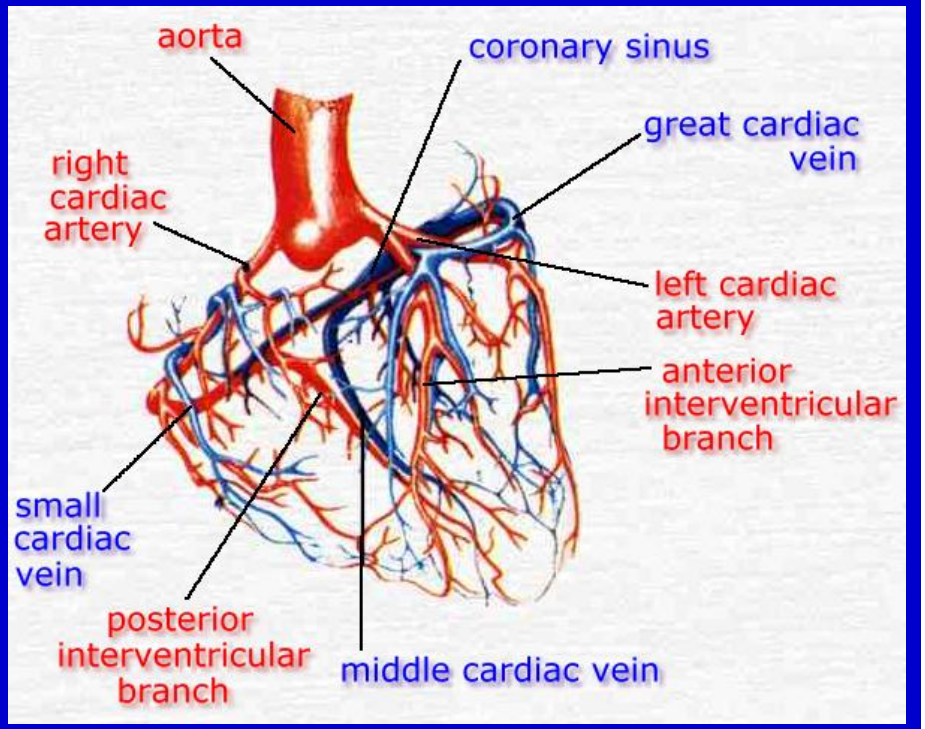
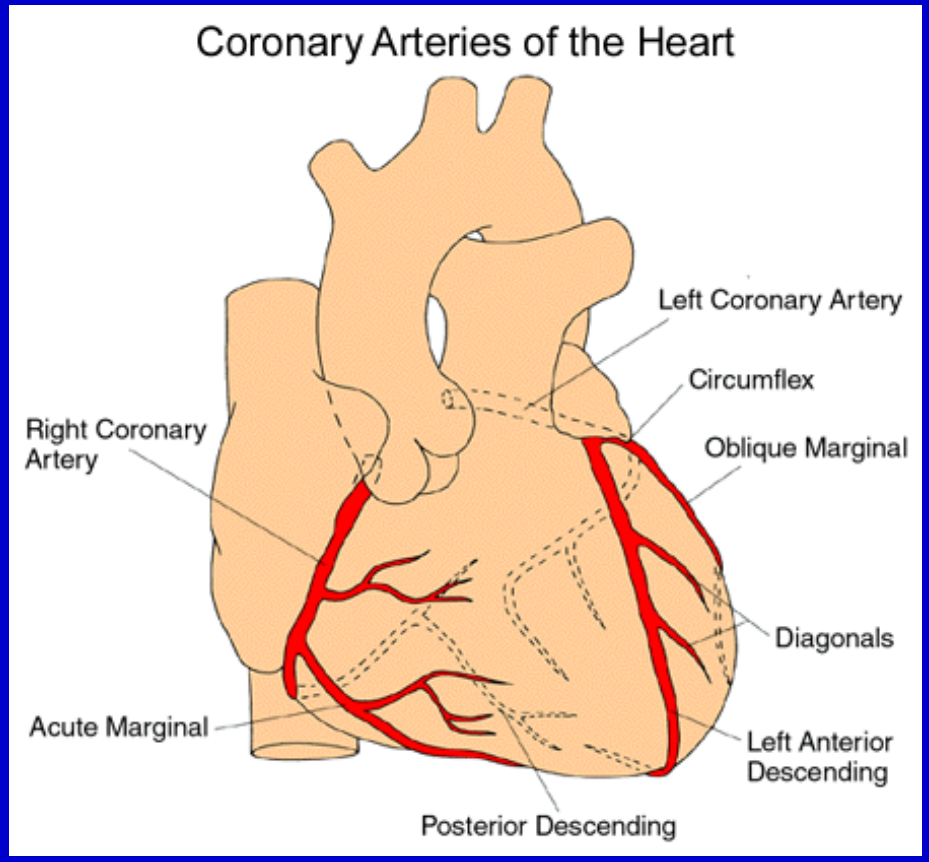


Coronary heart disease (CHD)

CORONARY CIRCULATION AND MYOCARDIAL METABOLISM


aorta
coronary sinus
great cardiac vein
right cardiac artery
left cardiac artery
anterior interventricular branch
small cardiac vein
posterior interventricular branch
middle cardiac vein

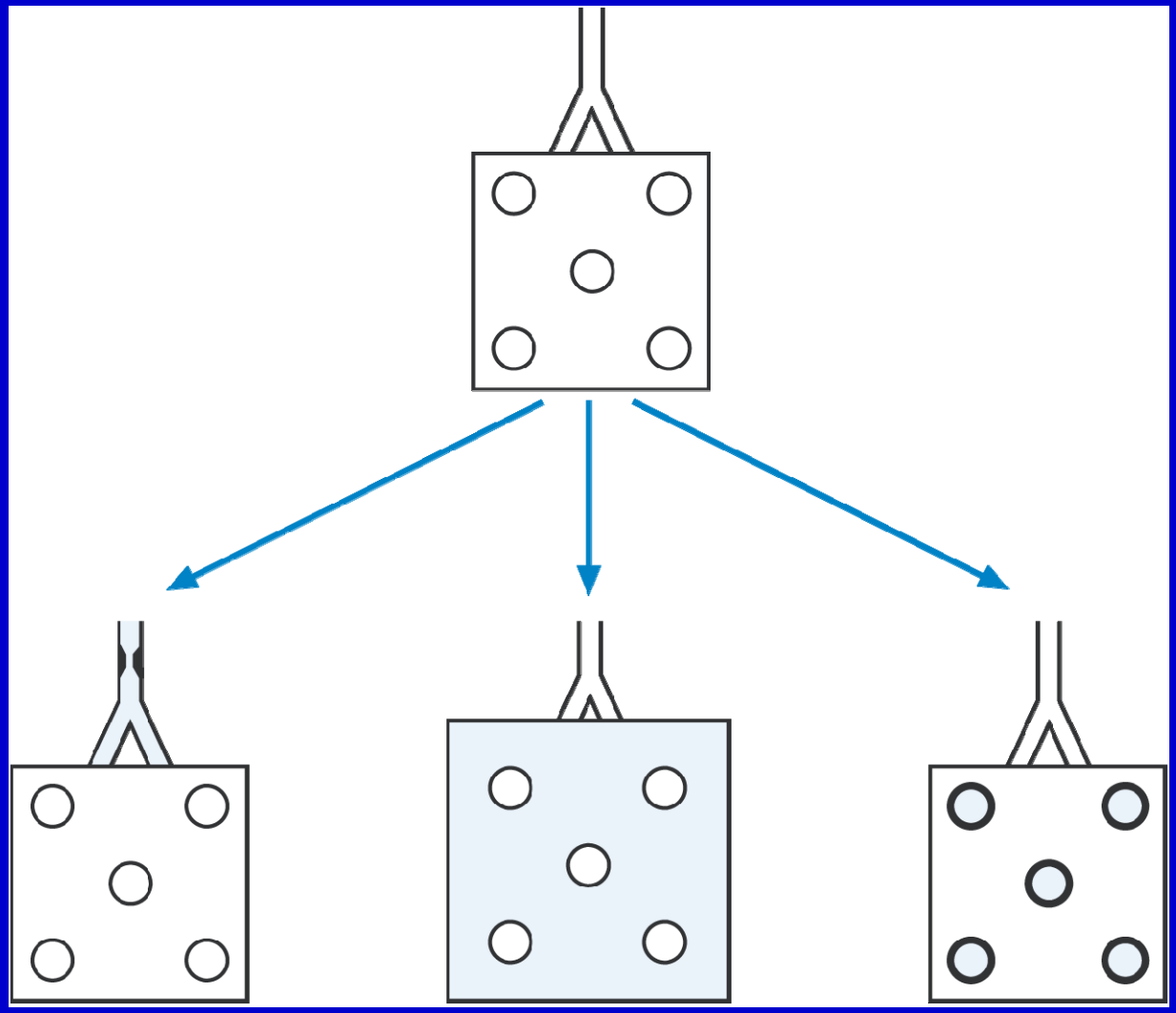

Coronary Arteries of the Heart
Left Coronary Artery
Circumflex
Right Coronary Artery
Oblique Marginal
Diagonals
Acute Marginal
Left Anterior Descending
Posterior Descending

Blood flow:
resting: 250 ml/min

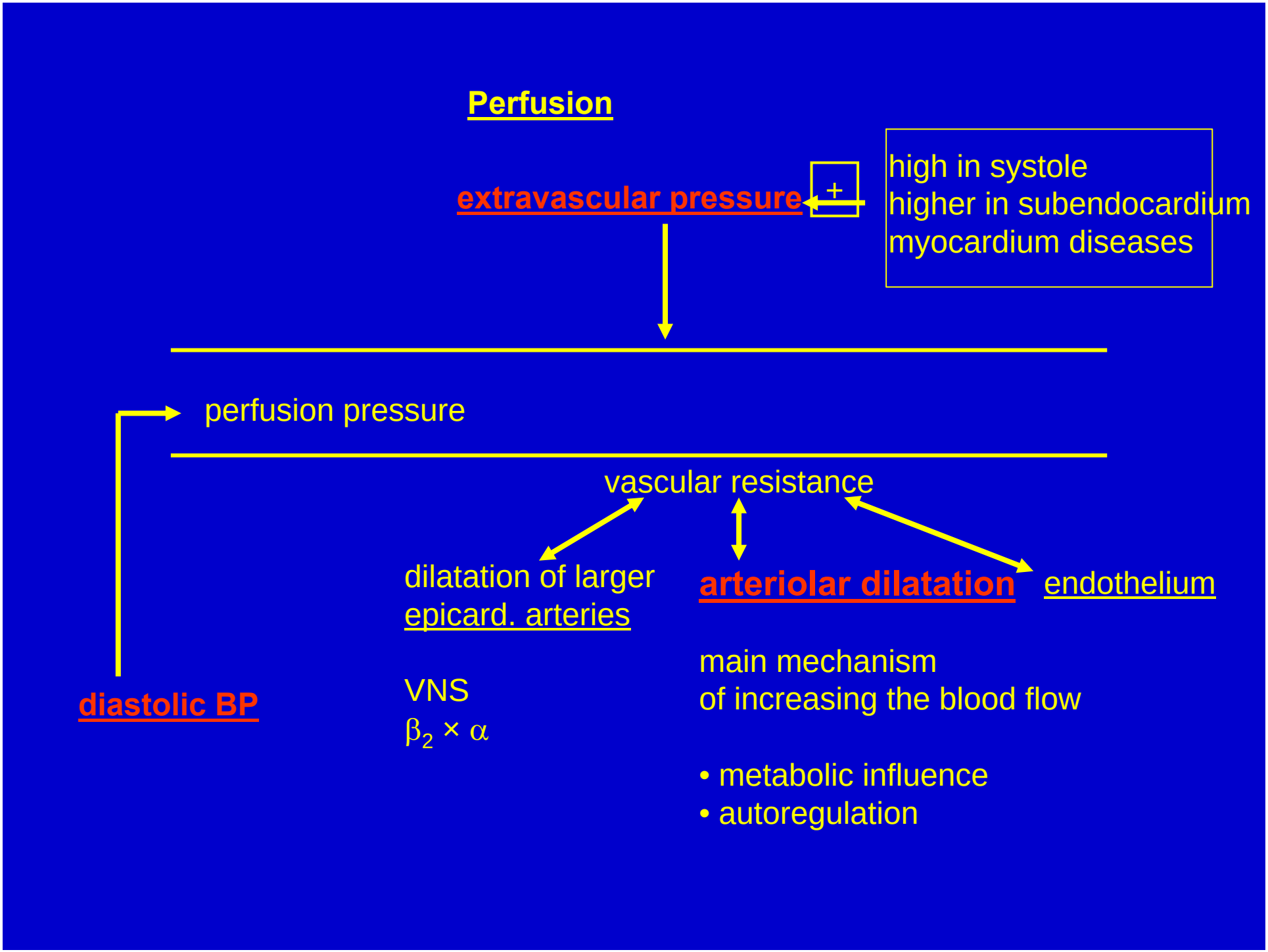
Main components:

- coronary arteries in epicardial part
- small coronary vessels
- myocardium

Perfusion pressure x resistance



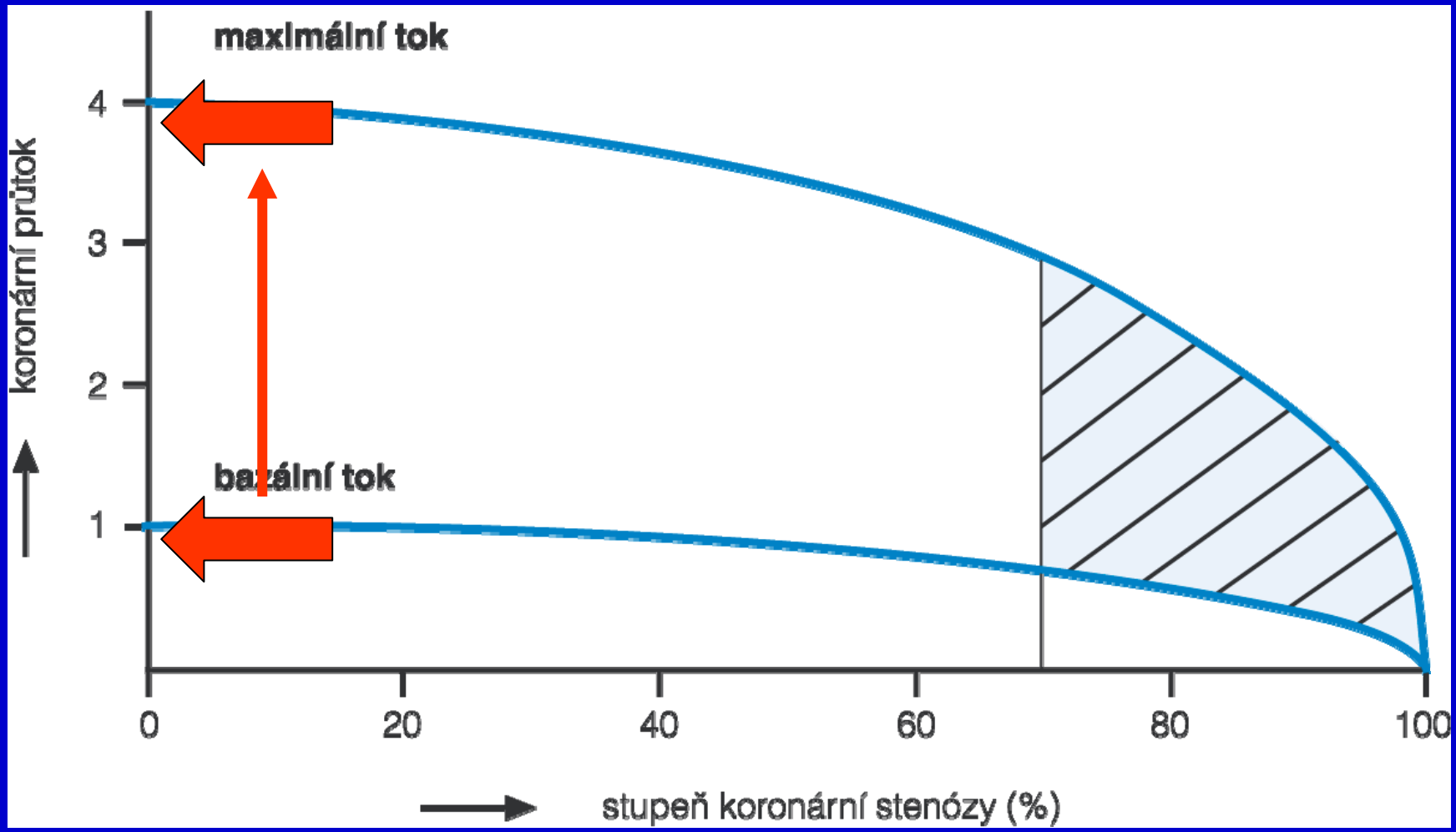
Zátěžová echokardiografie, Maxdorf


Perfusion
extravascular pressure
+
high in systole
higher in subendocardium
myocardium diseases
perfusion pressure
vascular resistance
dilatation of larger epicard. arteries
arteriolar dilatation
endothelium
diastolic BP
VNS
$\beta_2 \times \alpha$
main mechanism
of increasing the blood flow
• metabolic influence
• autoregulation

Perfusion pressure:
pressure difference between the beginning of coronary arteries and estuary of coronary sinus

Coronary reserve:
maximal increase of blood flow through myocardium – cca 4×
vasodiatation of small vessels

Differences in perfusion:
impaired perfusion of subendocardial parts



Zátěžová echokardiografie, Maxdorf

Blood flow through the subendocardial vessels
is less during systole
than in the outer coronary vessels.

To compensate, *the subendocardial vessels are far more extensive* than the outermost arteries, allowing a disproportionate increase in subendocardial flow during diastole.

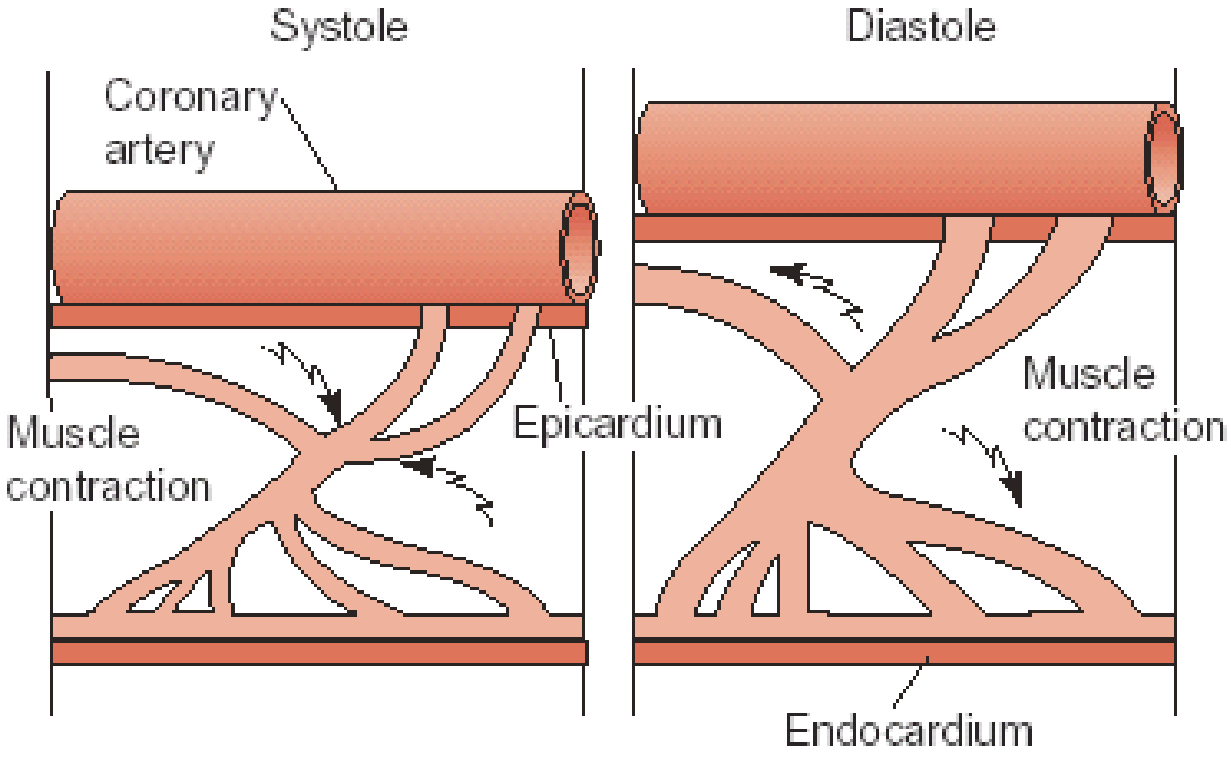
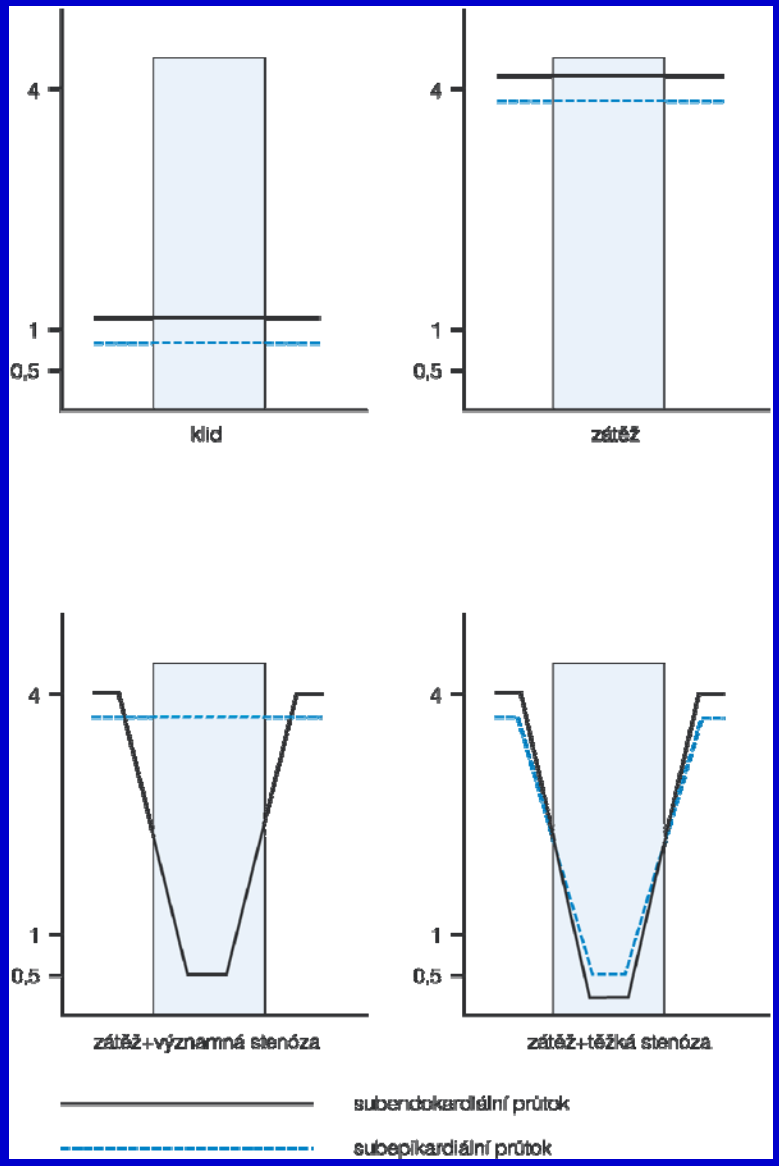


FIGURE 26-5 The compressing effect of the contracting myocardium on intramyocardial blood vessels and subendocardial blood flow during systole and diastole.

Because blood flow mainly occurs during diastole, there is a risk for subendocardial ischemia

- **diastolic pressure is low**
- **elevation in diastolic intraventricular pressure sufficient to compress the vessels in the subendocardial plexus**
- **rapid heart rates, the time spent in diastole is greatly reduced**



Zátěžová echokardiografie, Maxdorf

Oxygen extraction: almost maximal (as in intensively working skeletal muscles)

AV difference: 140–160 ml O_2/L blood

Oxygen consumption (AV difference × flow):

rest – 140 × 0,25 = 35 ml

exercise – 160 × 1,00 = 160 ml

Mainly achieved by increase of flow – vessel parameters are crucial for oxygen delivery to the myocardium during exercise

Energy consumption:

- 90 % mechanical activity (contraction, relaxation)
- 9,5 % proteosynthesis
- 0,5 % electrical activity

- tension in the wall of LV ~ blood pressure
- inotropy
- heart rate

Energy sources in the myocardium:
rest – FFA, glucose, lactate
exercise – increase of lactate up to 2/3

Factors infuencing oxygen consumption:

- heart work
- contractility
- heart rate
- myocardium properties: wall tension (dilataation, afterload – hypertension), hypertrophy
- adrenergic stimulation

Factors infuencing oxygen delivery to the myocardium:

- parcial tension of oxygen in the environment
- respiratory functions
- hemoglobin
- blood flow through myocardium

ISCHEMIA

Ischemia

insufficient delivery of oxygen and nutrients and insufficient outflow of metabolites from the tissues due to the impaired perfusion

Imbalance between metabolic requirements and perfusion:

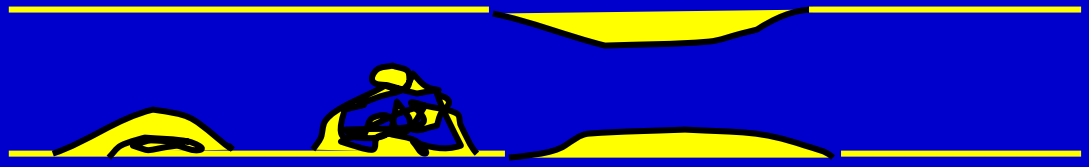
1. **increased requirements – *simulation by exercise tests***
2. **impaired perfusion**
3. **combination**

Vessel narrowing

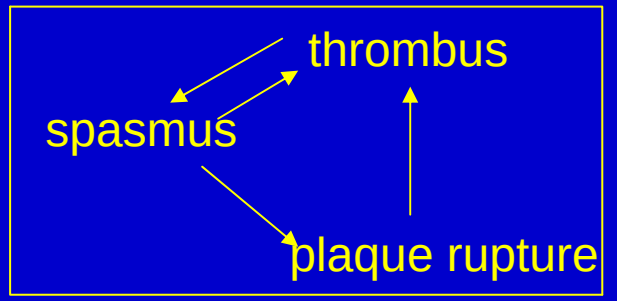
- organic
- functional
- combined

- fixed
- dynamic

plaque + thrombus + spasmus



atherom. plaque **thrombus** **spasmus**



<u>localisation</u>
- concentric
- excentric

<u>stability</u>
- fibrotisation
- lipids
- inflammation

platelets:
vasoconstr. factors
growth factors

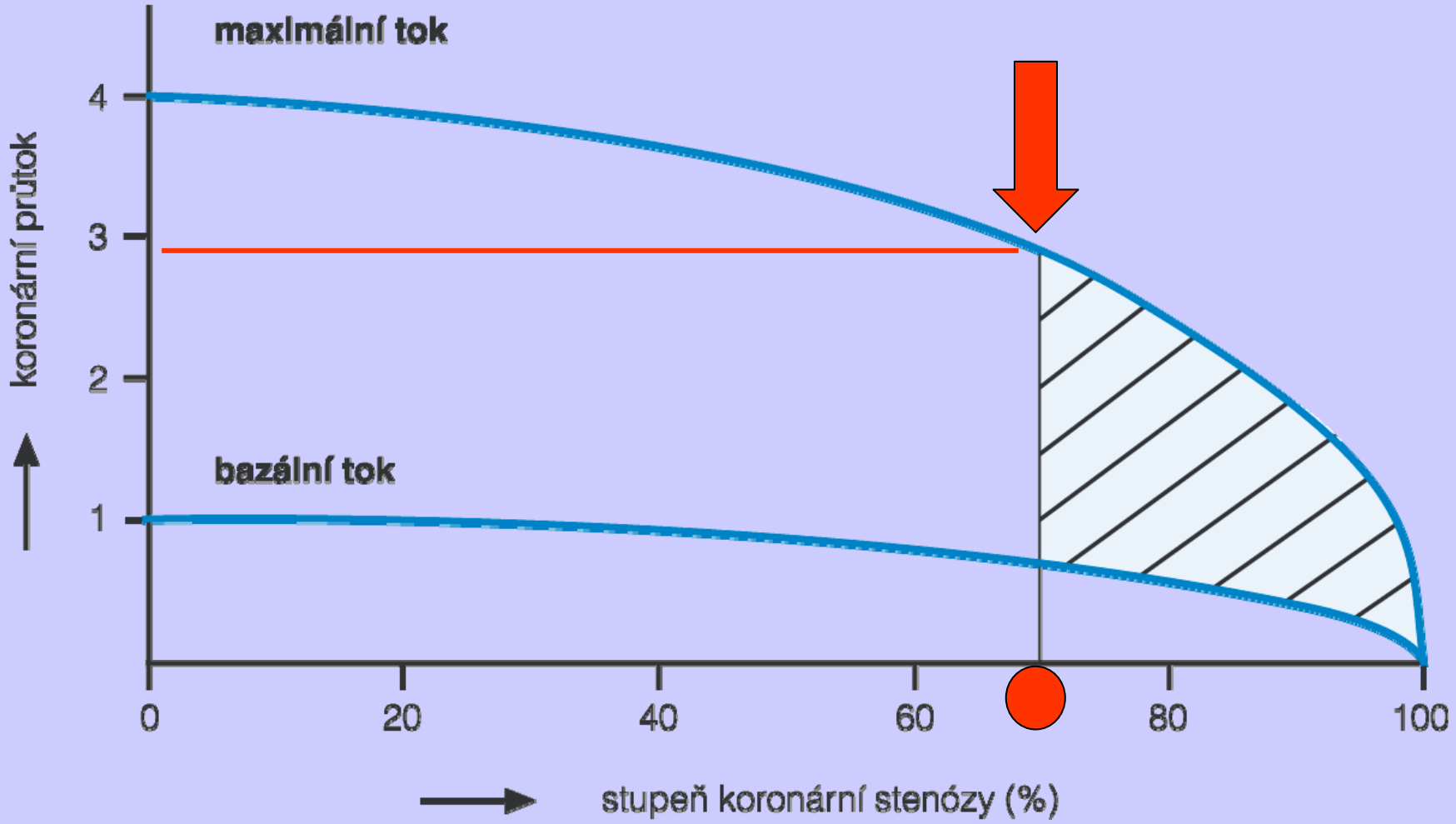
diurnal rhytm (morning!)
cold
smoking
psychic influences

Impact of the size of stenosis on hemodynamics of coronary blood flow:

- do 40 % – without influence
- 40–70 % – ischemia not apparent in physiological exercise
- 70–90 % – ischemia not in resting, distinct in exercise
- over 90 % – ischemia even in rest

Hemodynamically unimportant atherosclerotic plaque can be the cause of life-threatening myocardial infarction:

rupture ⇒ thrombus + spasmus ⇒ necrosis ⇒ arrhytmia (ventricular fibrillation) ⇒ death



Zátěžová echokardiografie, Maxdorf

Consequences of ischemia:

- metabolic changes: ATP depletion, lokal acidosis, increased inflow of *calcium* to the cells
- impaired contractility (*decrease of stroke volume*):
- impaired relaxation (*diastolic dysfunction*)
- impaired electrical events (*arrhytmias, ECG*)
- morphological changes (*myocytes, necrosis, fibrotisation, steatosis etc.*)
- clinical symptoms (*pain, arrhytmia, heart failure*)

Postischemic changes

* ischemia duration
* reperfusion

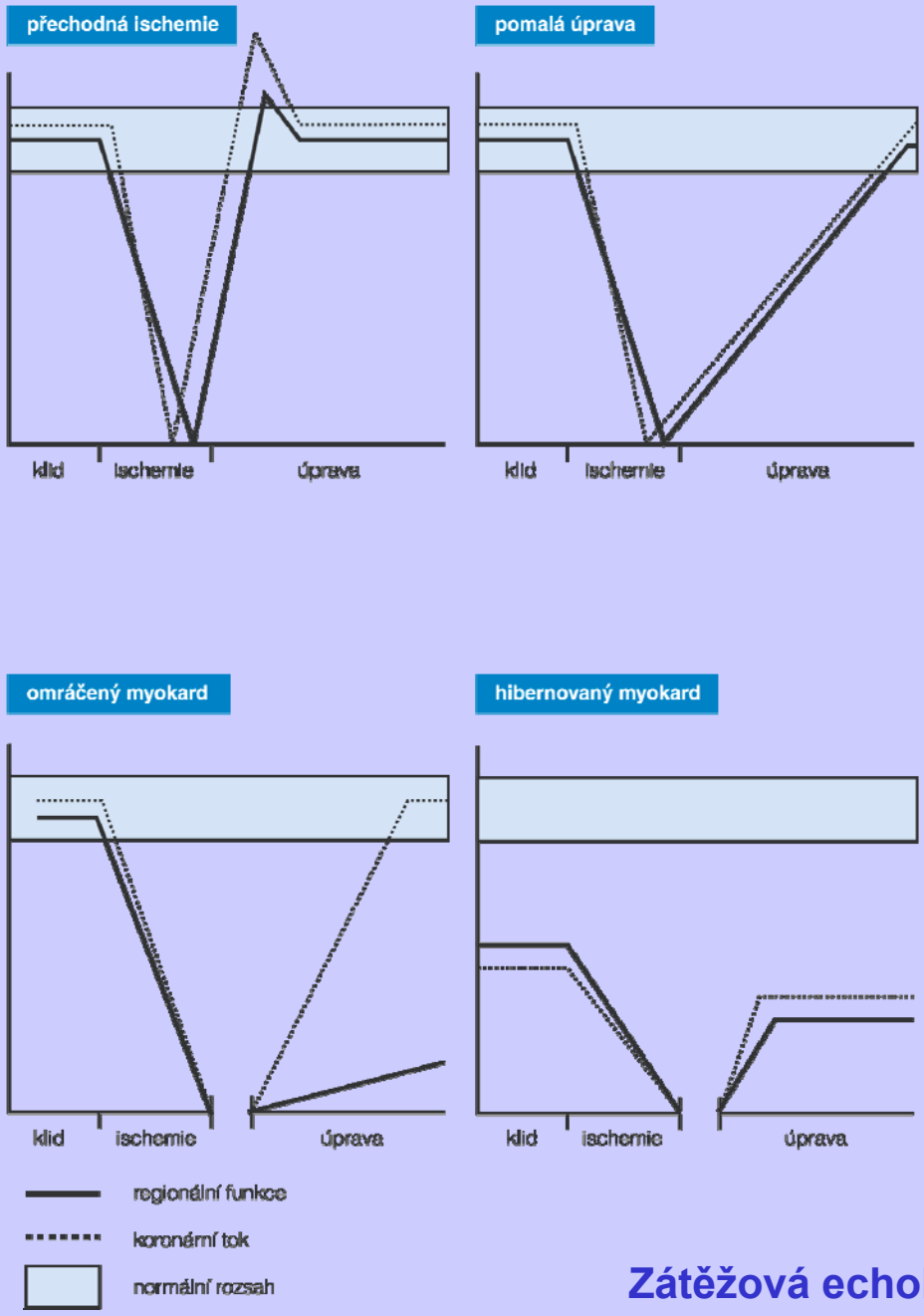
Stunned myocardium

perfused but not functioning
reversible continuous dysfunction of myocardium after reperfusion without apparent changes

Hibernating myocardium

chronically hypoperfused and functionally impaired
situation with continuously decreased blood flow accompanied by impaired contractility

adaptation of cells to decreased energy delivery



Zátěžová echokardiografie, Maxdorf

Ischemic preconditioning

increased resistence of myocardium against damage due to ischemia

caused by preceding ischemia and reperfusion

Reperfusion

Collaterals

Angiogenesis
VEGF (vascular endothelial growth factor)
FGF (fibroblast growth factor)
Angiopoetin
and others...

Therapeutical angiogenesis
gene therapy: direct intramyocardial aplicatioon of plasmid or use of vector (adenovirus) VEGF or FGF

Revascularization by invasive treatment
- PTCA (percutanneous transluminal coronary angioplastic)
- stents
- bypass

Reperfusion damage

* oxygen radical species: source in mitochondria, or leukocytes,
xanthinoxidase (less important in myocardium)
* increased amount of intracellular calcium
* neutrophils: radical formation, mechanical plugging of capillaries, proteolytic enzymes

clinically - ***arrhytmias***