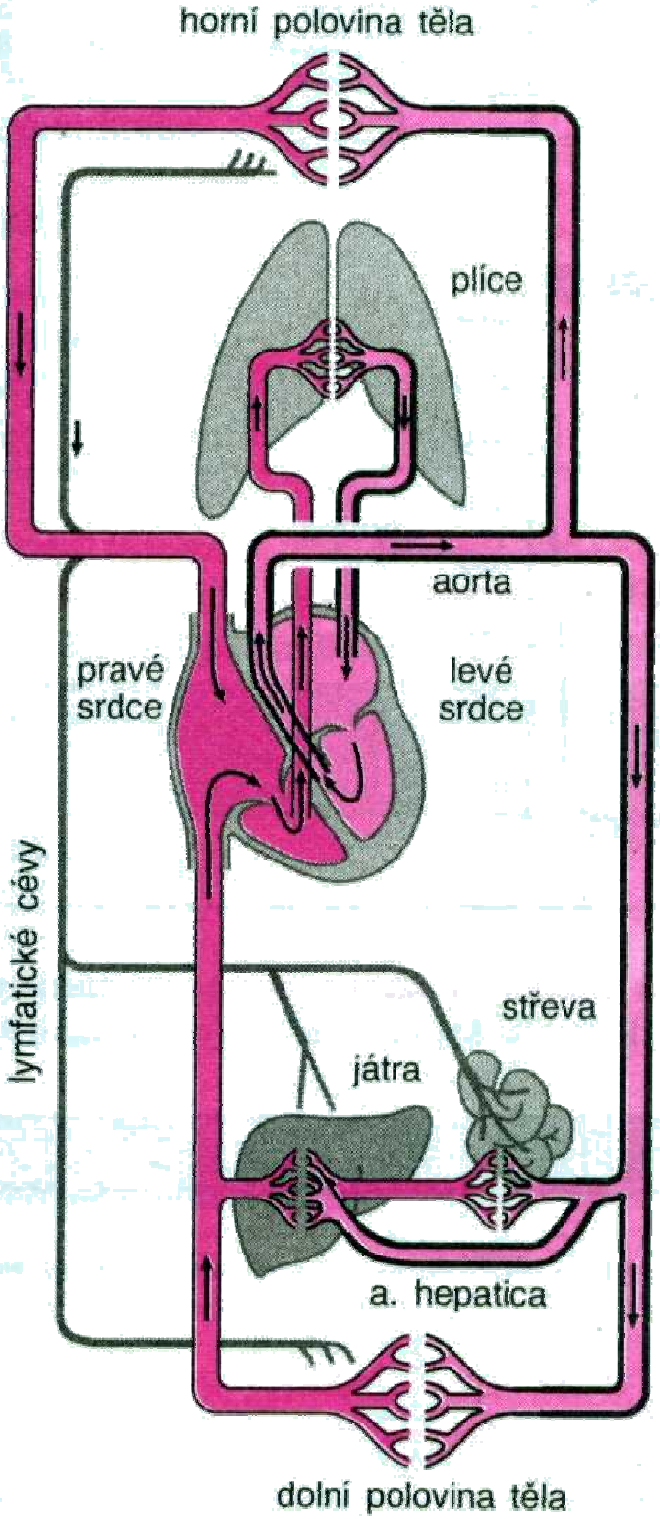
EXERCITATIO.

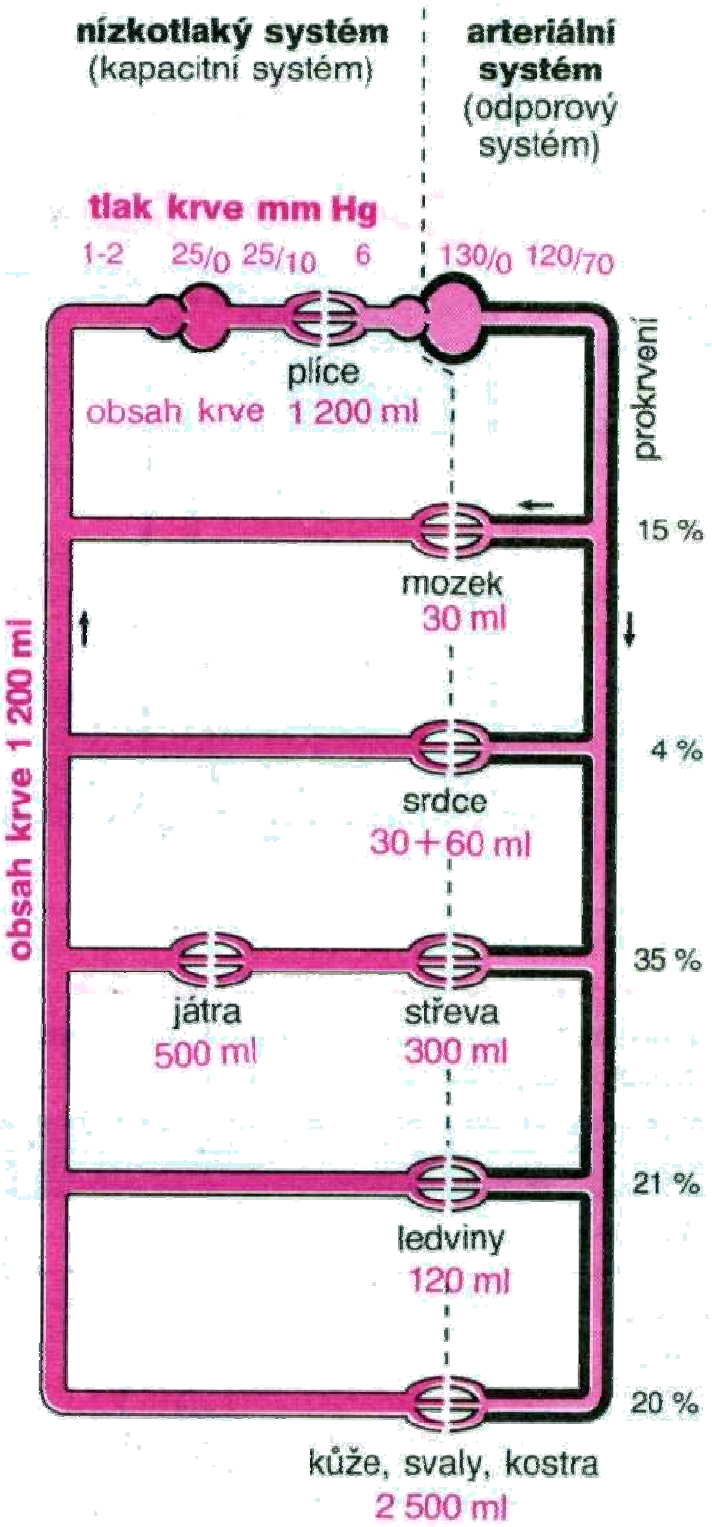
ANATOMICA DE MOTV CORDIS ET SANGVINIS IN ANIMALIBVS,

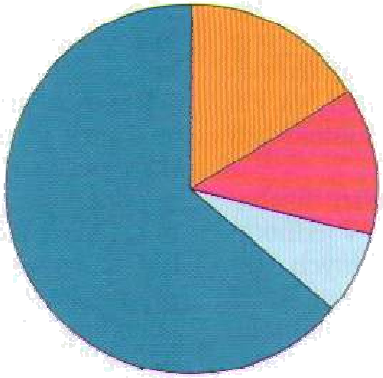
GVILIELMI HARVEI ANGLI,

Medici Regii, & Professoris Anatomiæ in Collegio Medicorum Londinensi.

FRANCOFVRTI,
Sumptibus GVILIELMI FITZERI.
ANNO M. DC. XXVIII.







srdce a plicní oběh
(7 a 9%)

tepny
(7%)

tepénky a kapiláry
(7%)

žíly
(64%)

CÉVY

tepny *(arterie)*	**- pružníkové cévy**
tepénky *(arterioly)*	**- odporové cévy**
vlásečnice *(kapiláry)*	**- výměnné cévy**
žíly *(vény)*	**- kapcitní cévy**

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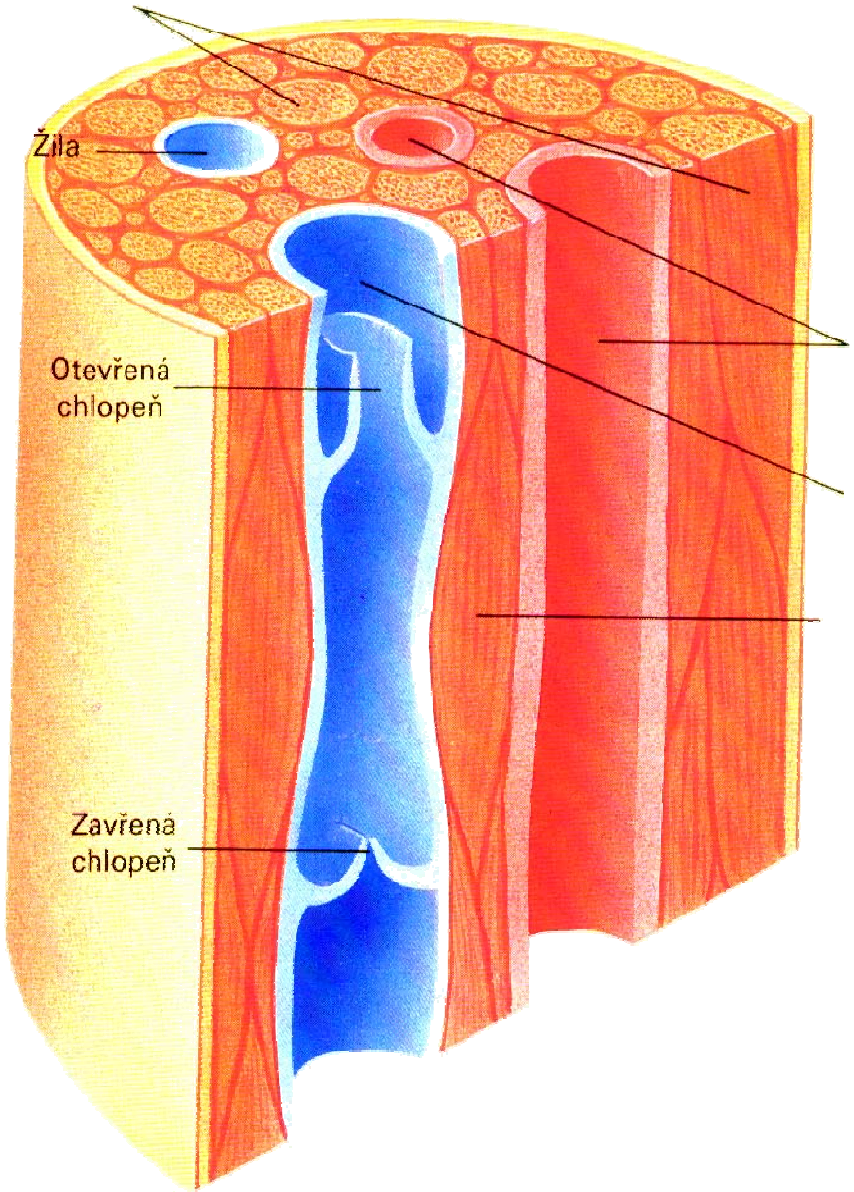
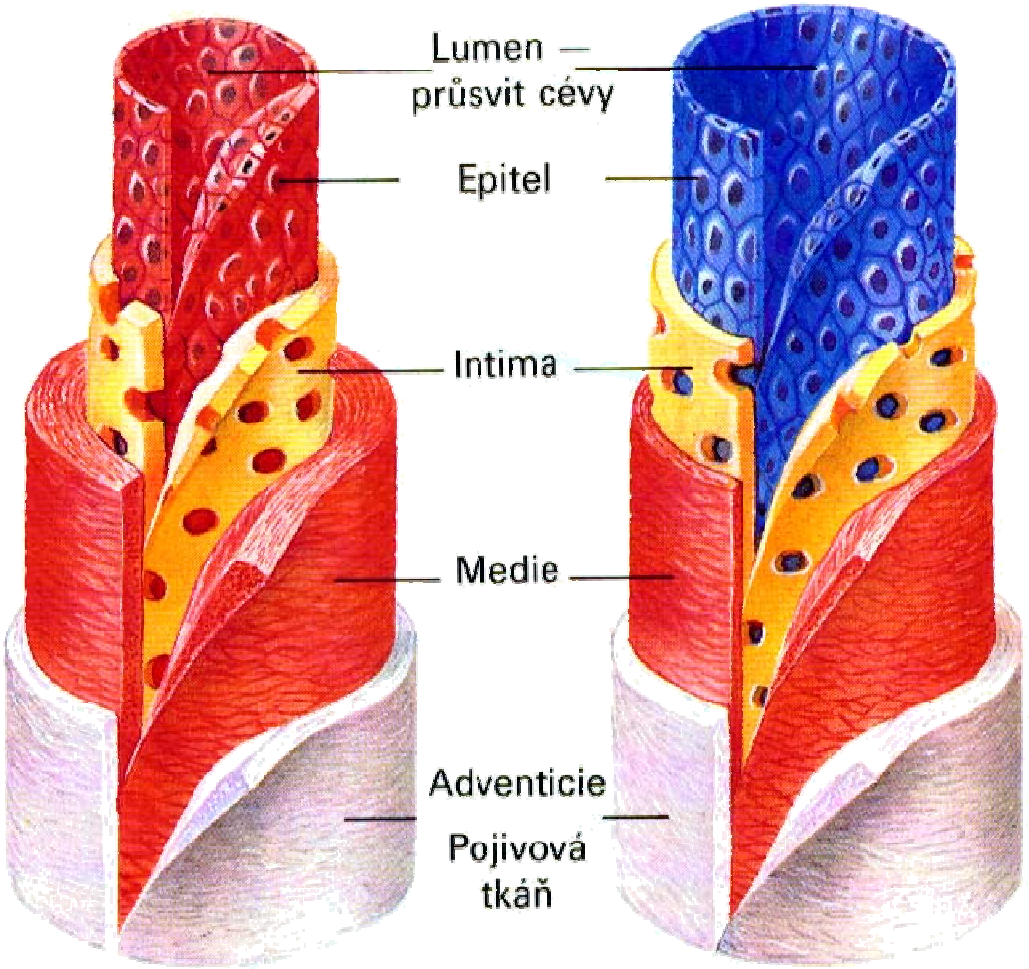
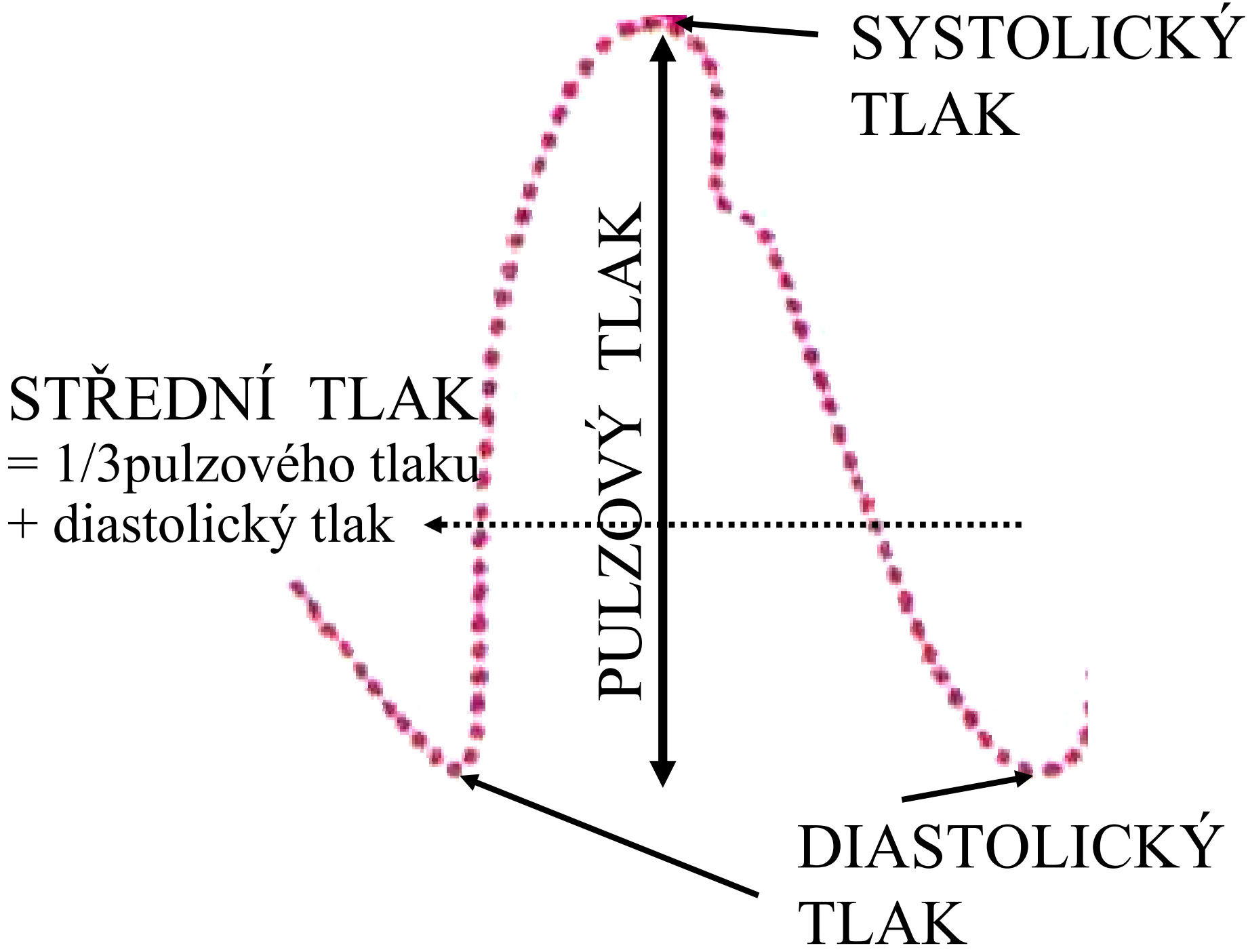
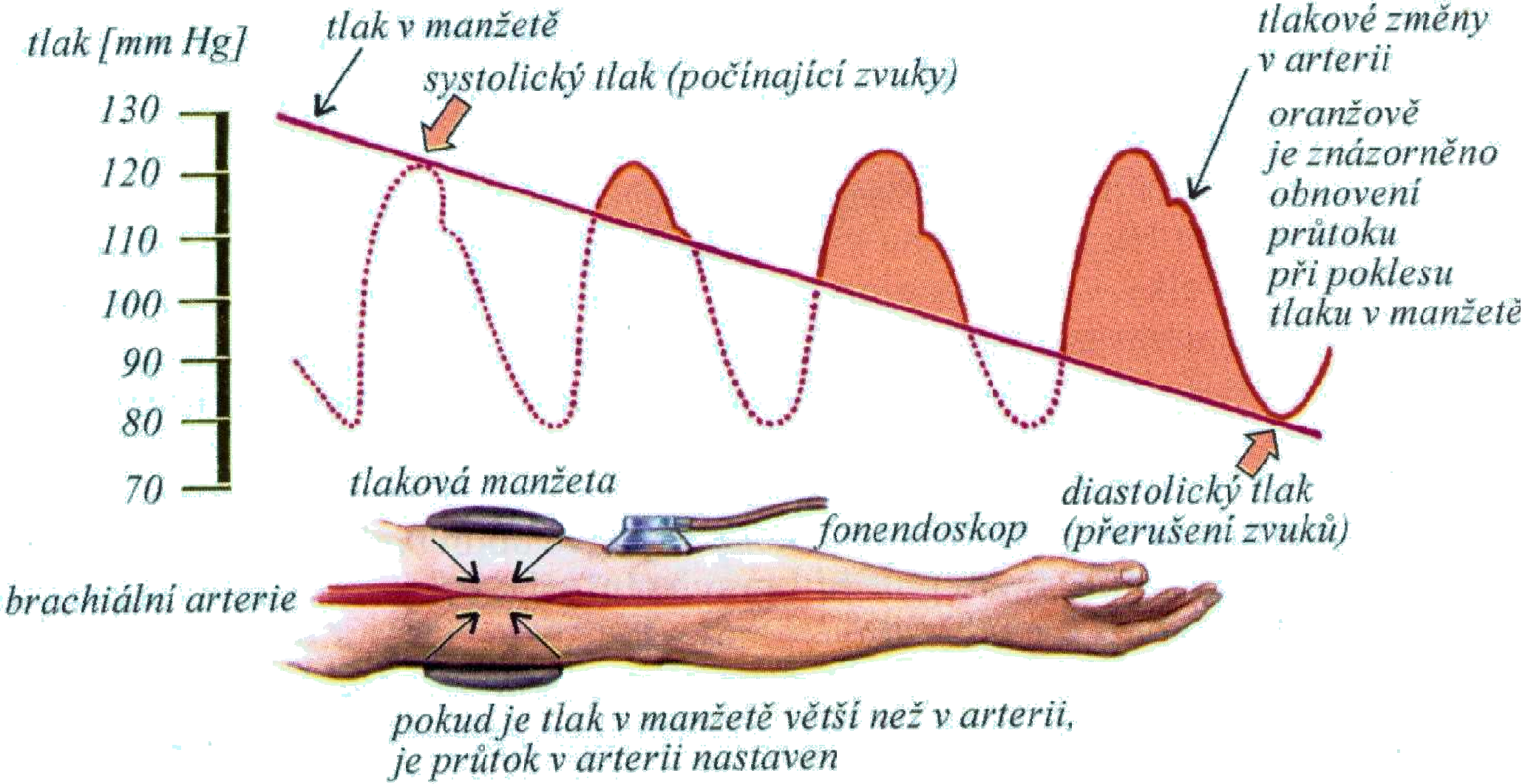
lymfatické cévy

TEPNY A ŽÍLY

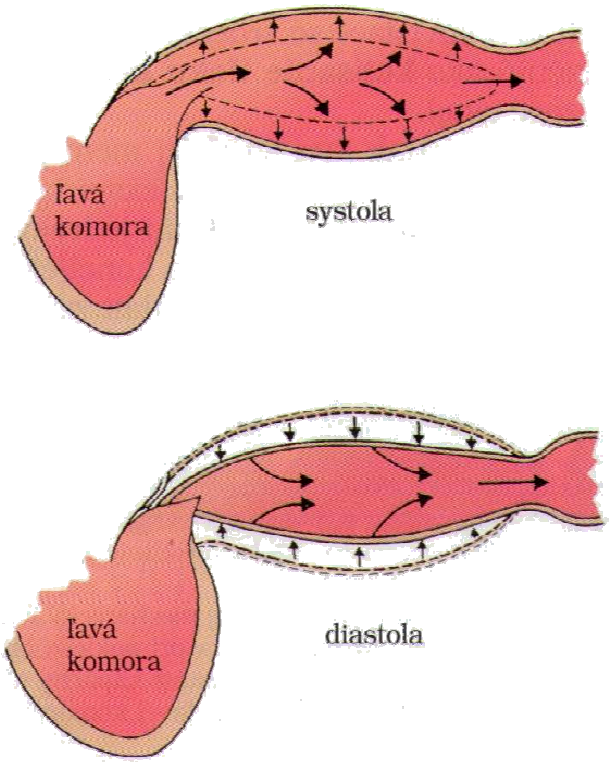

Žíla
Otevřená chlopeň
Zavřená chlopeň


Lumen – průsvit cévy
Epitel
Intima
Medie
Adventicie
Pojivová tkáň

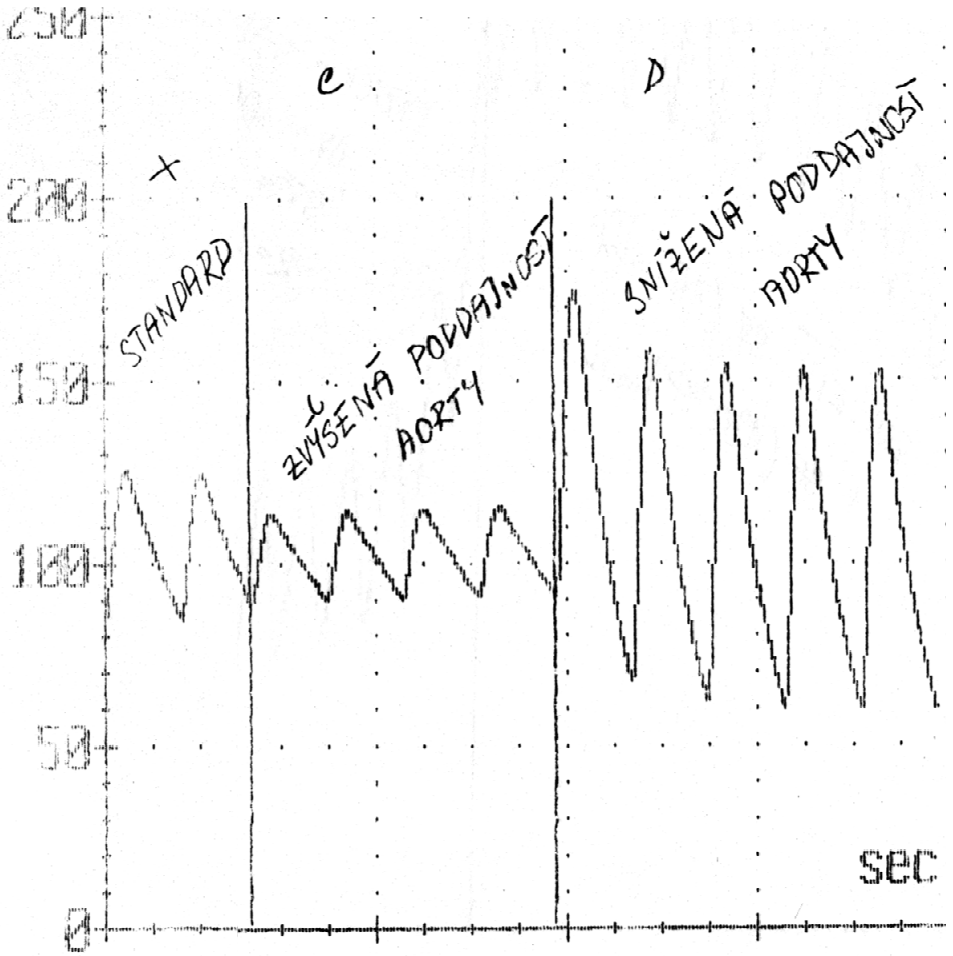

SYSTOLICKÝ
TLAK
PULZOVÝ TLAK
STŘEDNÍ TLAK
= 1/3pulzového tlaku
+ diastolický tlak
DIASTOLICKÝ
TLAK


tlak [mm Hg]
130
120
110
100
90
80
70
tlak v manžetě
systolický tlak (počínající zvuky)
tlakové změny v arterii
oranžově je znázorněno obnovení průtoku při poklesu tlaku v manžetě
tlaková manžeta
fonendoskop
diastolický tlak (přerušení zvuků)
brachiální arterie
pokud je tlak v manžetě větší než v arterii, je průtok v arterii nastaven

TEPNY – pružníkové cévy



Cévy se ↑ obsahem elastických vláken



Poddajnost (compliance)

$$C = \frac{\Delta V}{\Delta P}$$

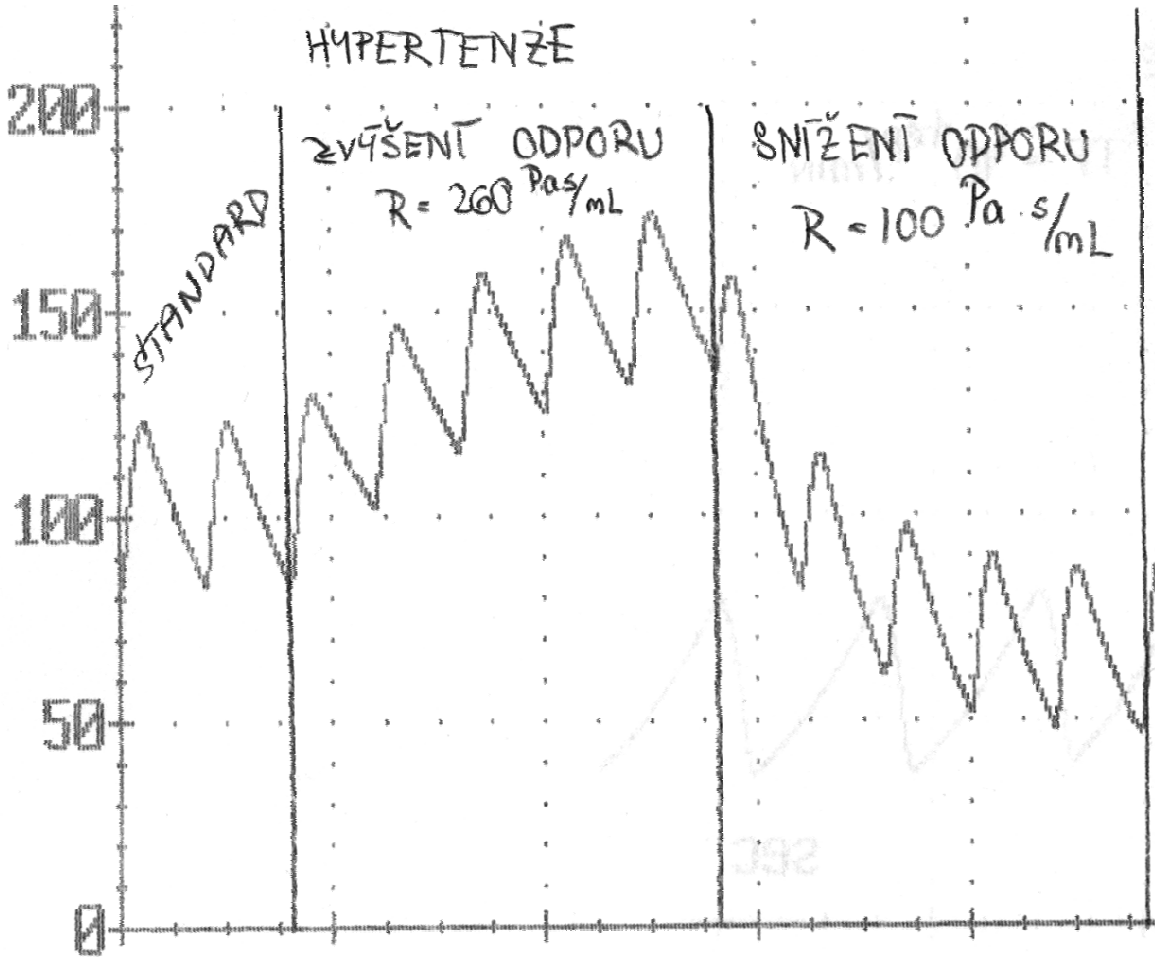
TEPÉNKY – odporové cévy

Cévy se ↑ obsahem svalových buněk ve stěně

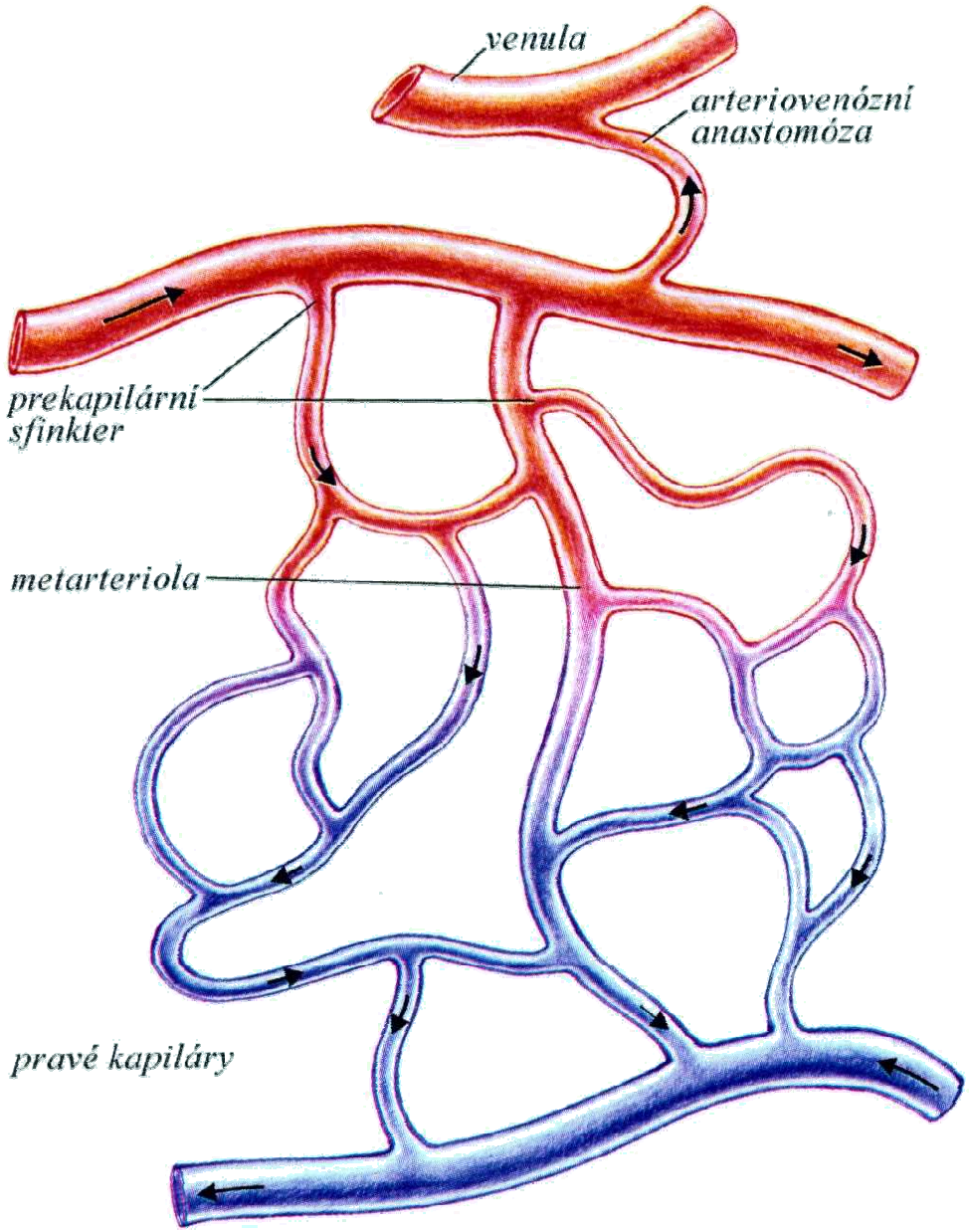
$$\mathbf{R = \frac{\Delta P}{Q}}$$

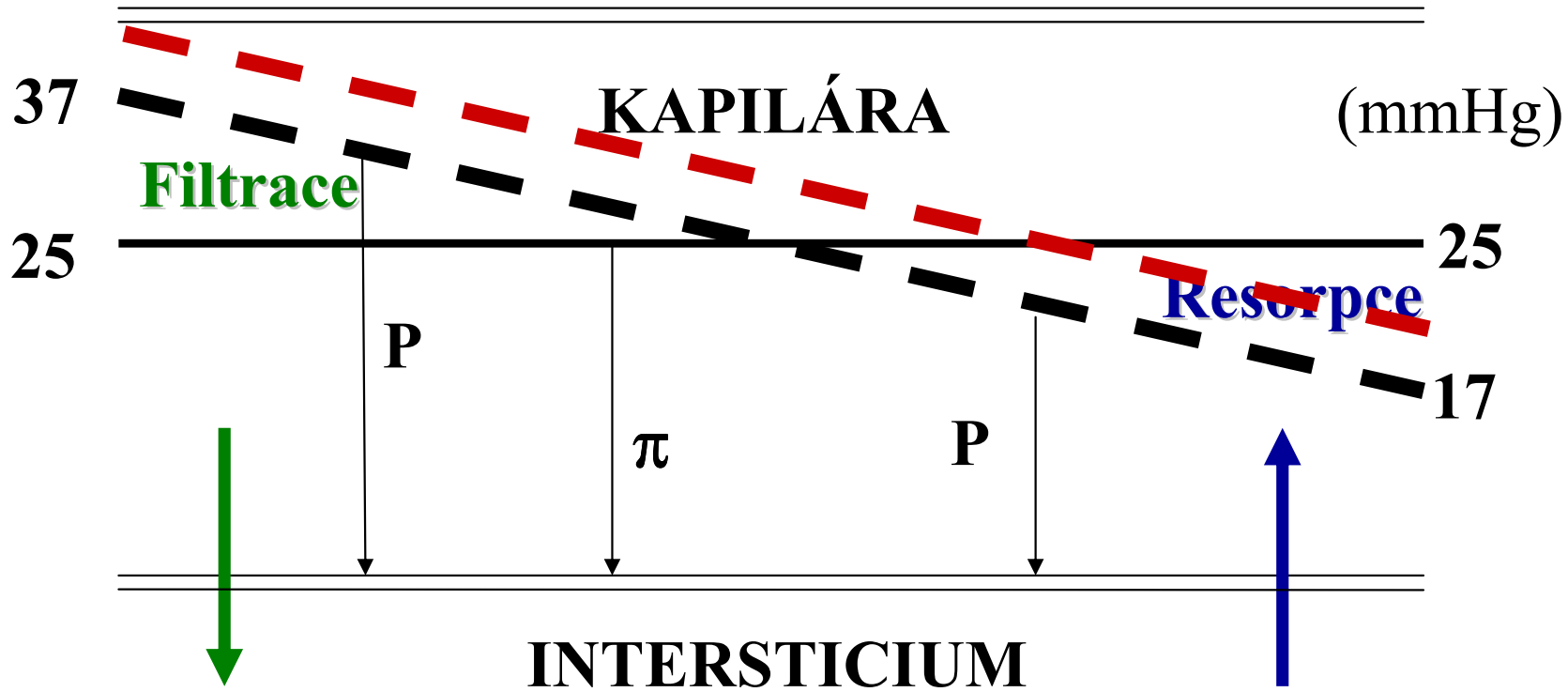
$$\mathbf{R = \frac{8 * \eta * l}{\pi * r^4}}$$

$$R = \frac{8 * \eta * l * \pi}{S^2}$$



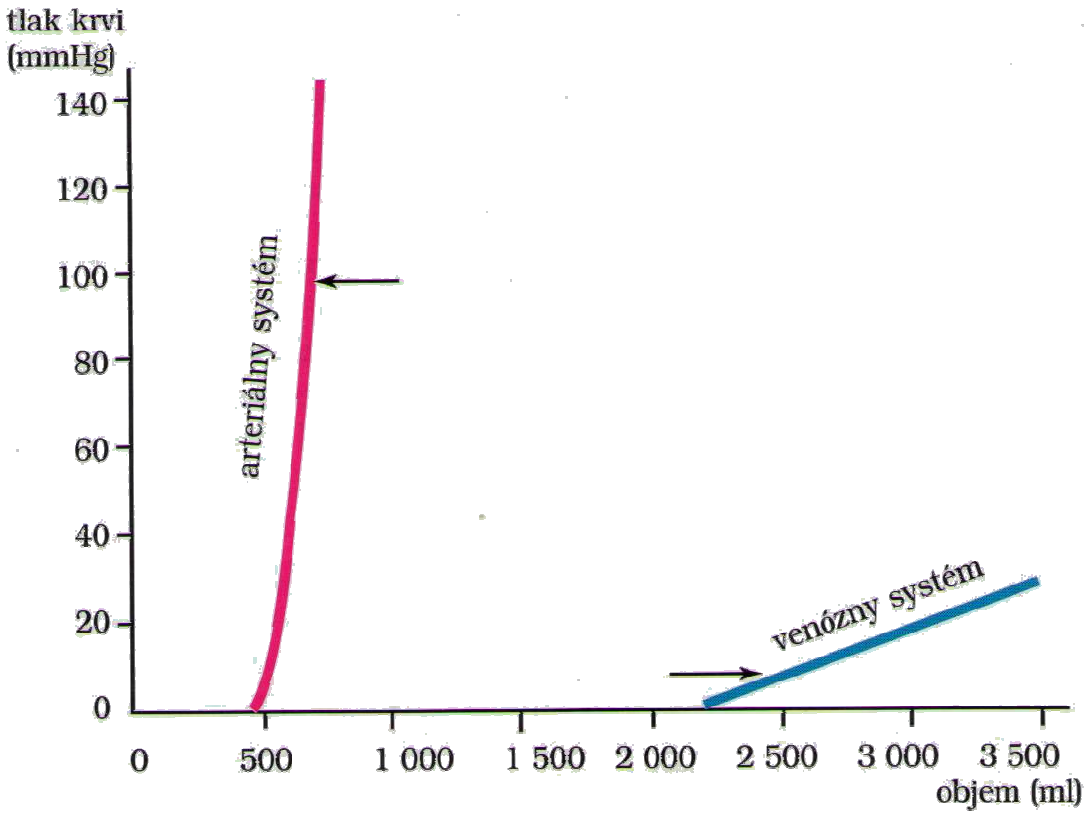
KAPILÁRY – výměnné cévy





P… hydrostatický tlak
π…onkotický tlak bílkovin
P> π - filtrace (prostup tekutiny z cév do tkání)
P< π - resorpce (prostup tekutiny z tkání do cév)

ŽÍLY – kapacitní cévy



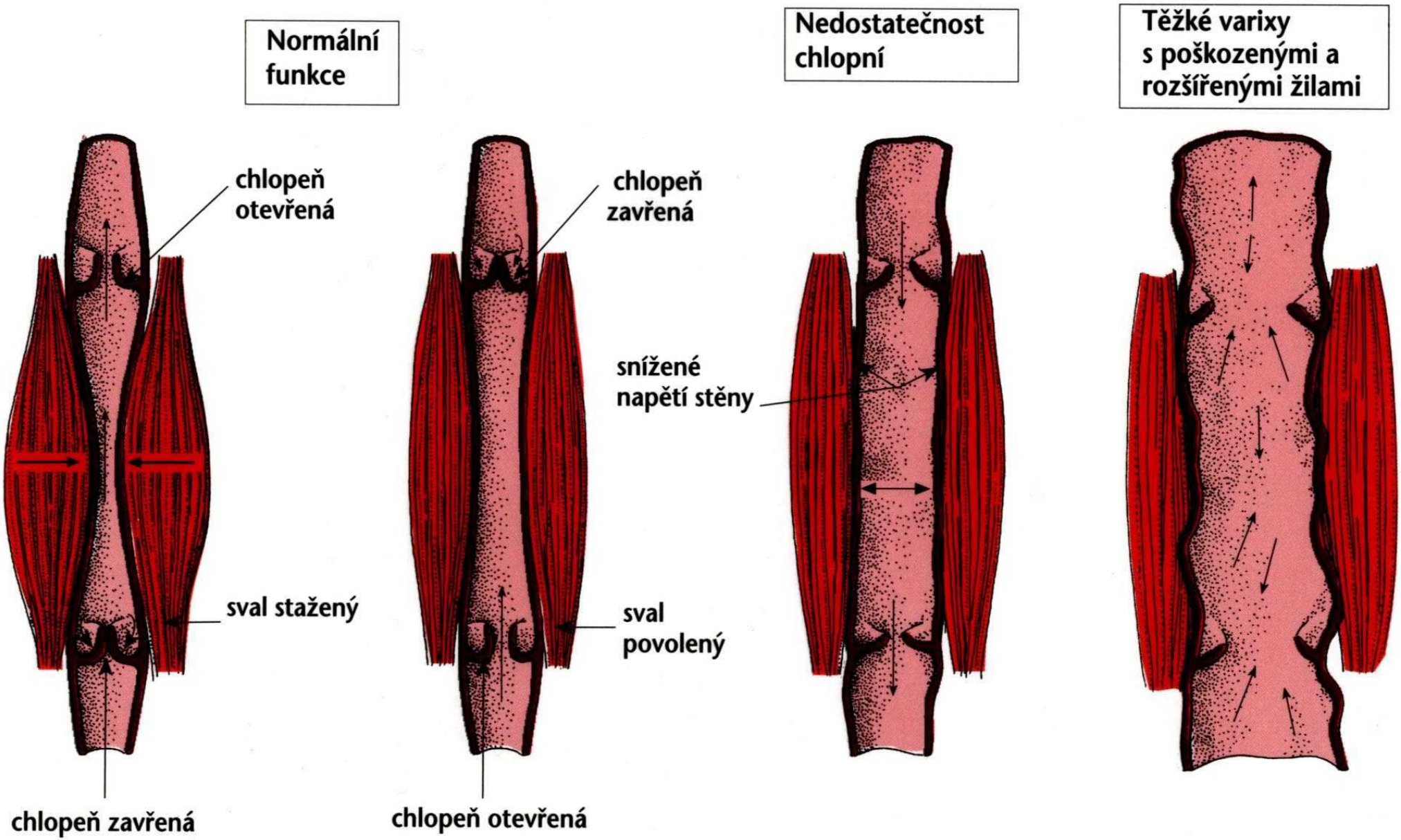
MECHANISMY ŽILNÍHO NÁVRATU

síla ze zadu - tepenný tlak produkovaný systolickou prací levé komory

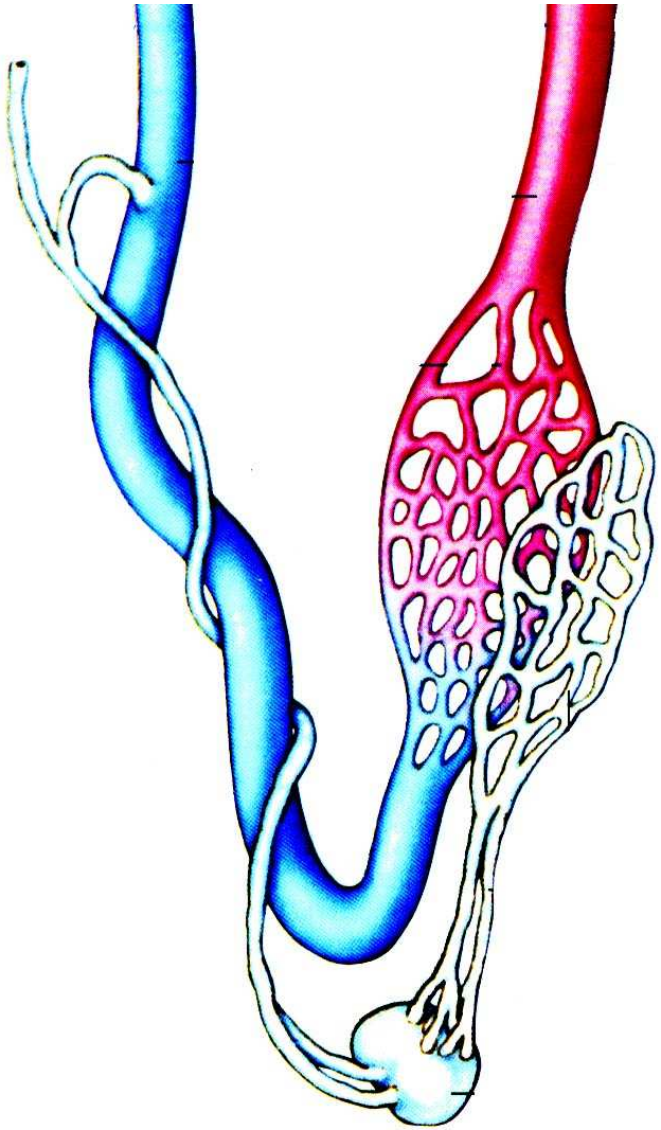
sací síla srdce - nasátí krve do síní během systoly komor

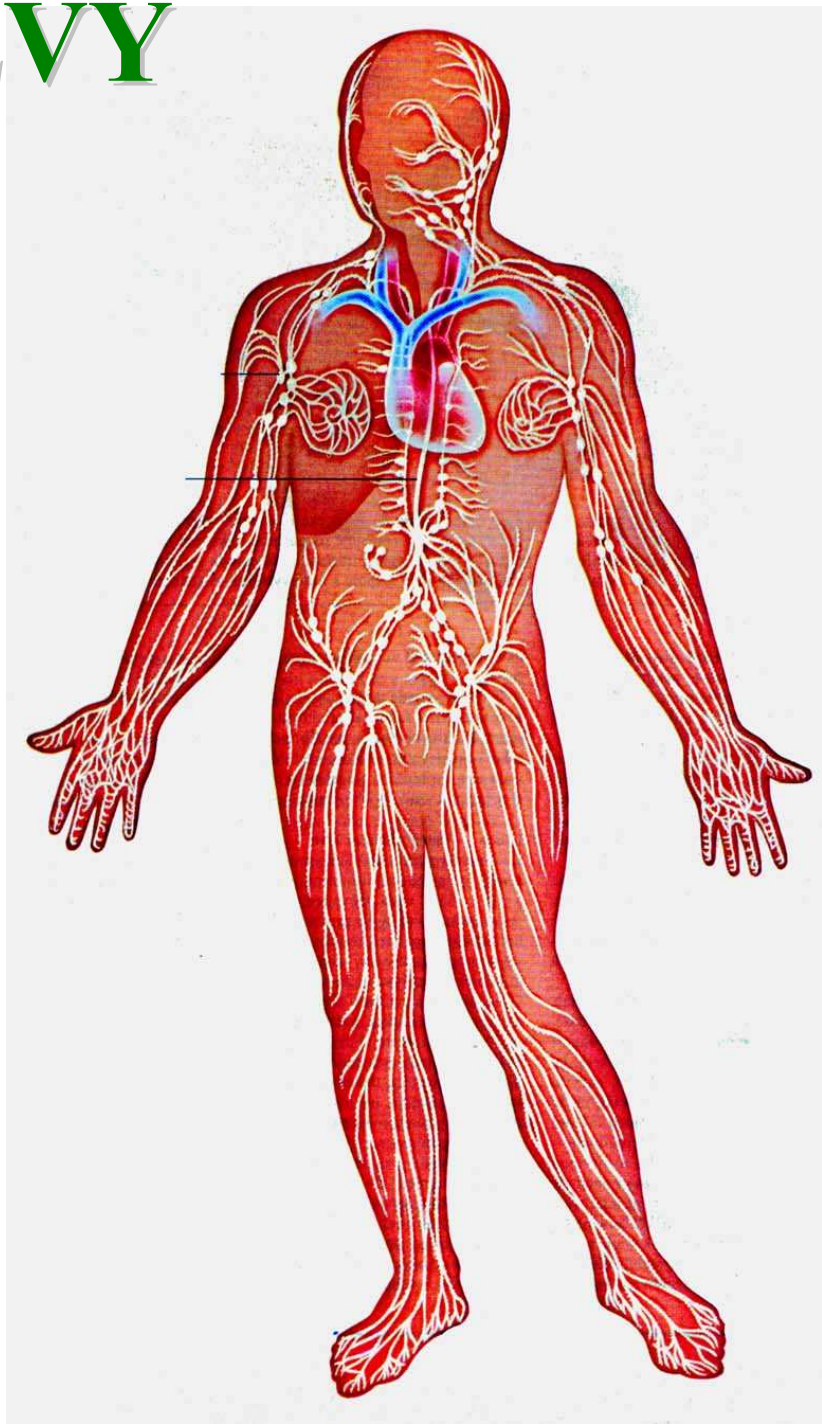
nitrohrudní podtlak - urychlení proudu krve při průchodu duté žíly bránici

svalová pumpa a chlopně

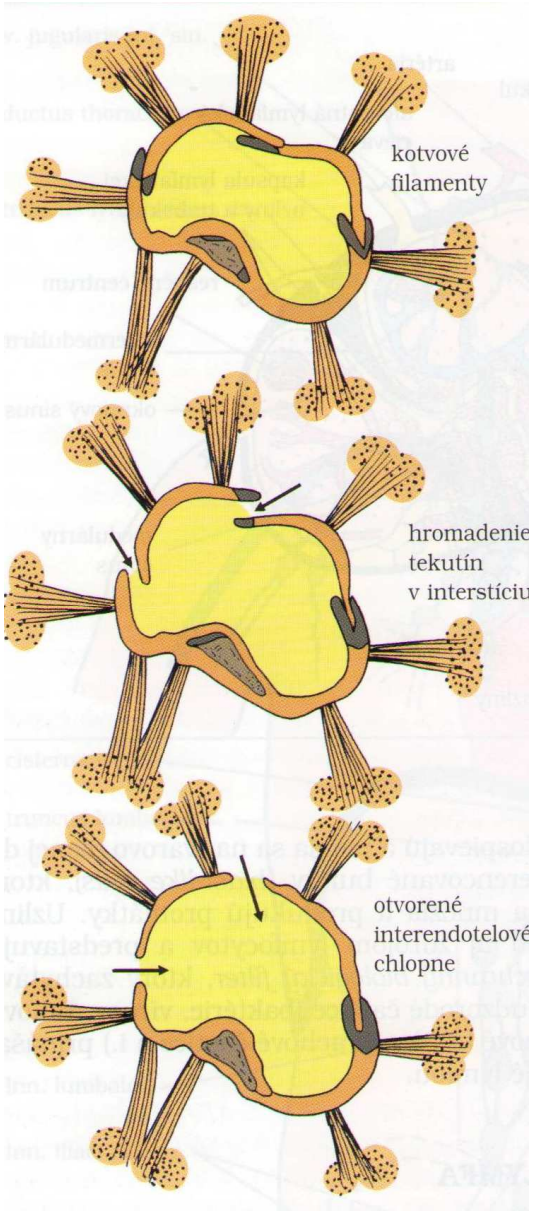

Normální funkce
chlopeň otevřená
sval stažený
chlopeň zavřená
chlopeň zavřená
snížené napětí stěny
sval povolený
chlopeň otevřená
Nedostatečnost chlopní
Těžké varixy s poškozenými a rozšířenými žilami

LYMFATICKÉ CÉVY

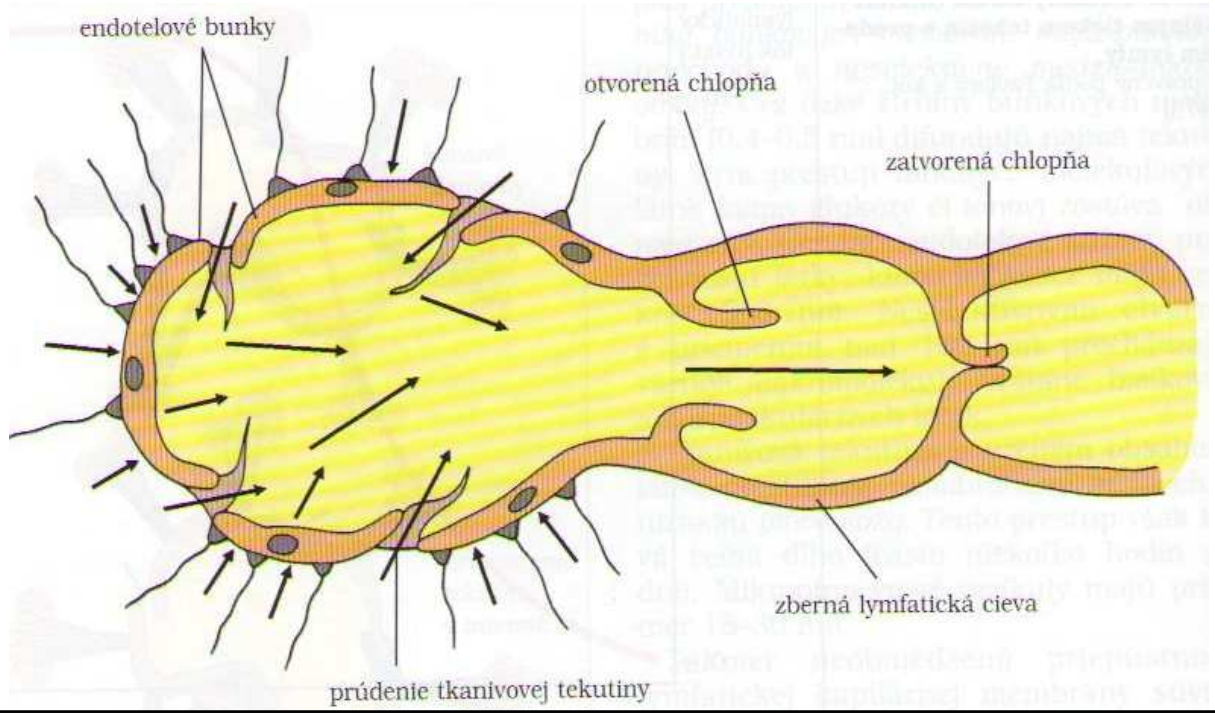


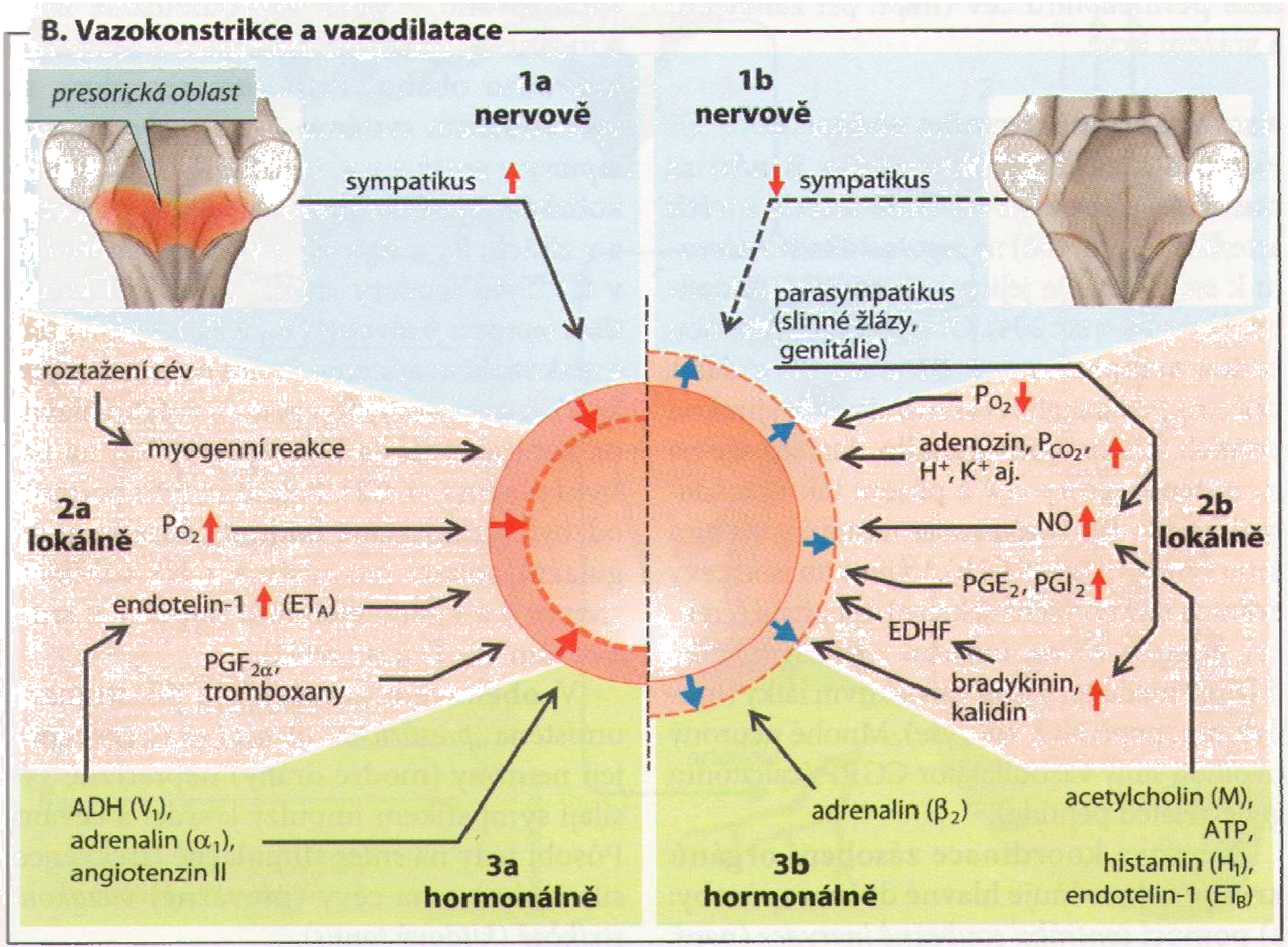


LYMFA

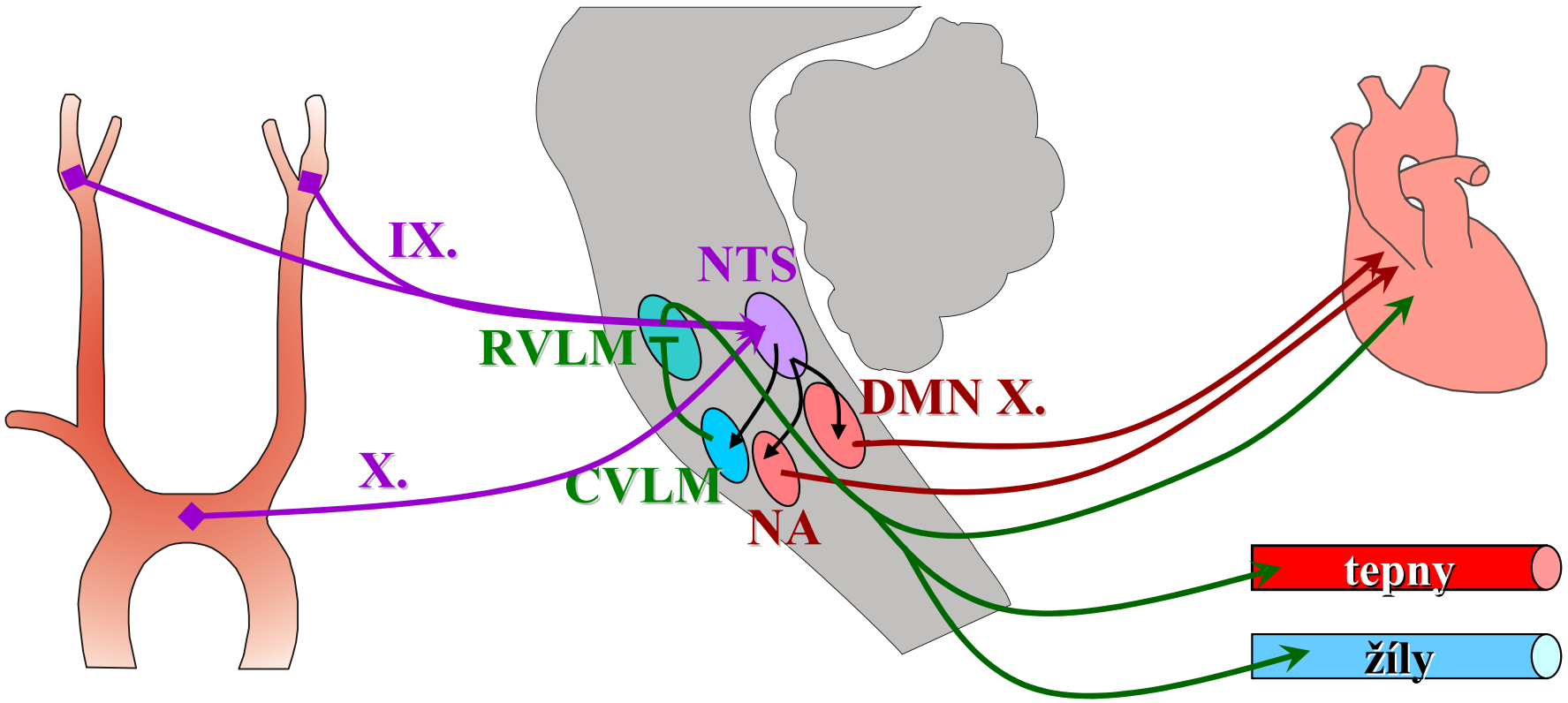


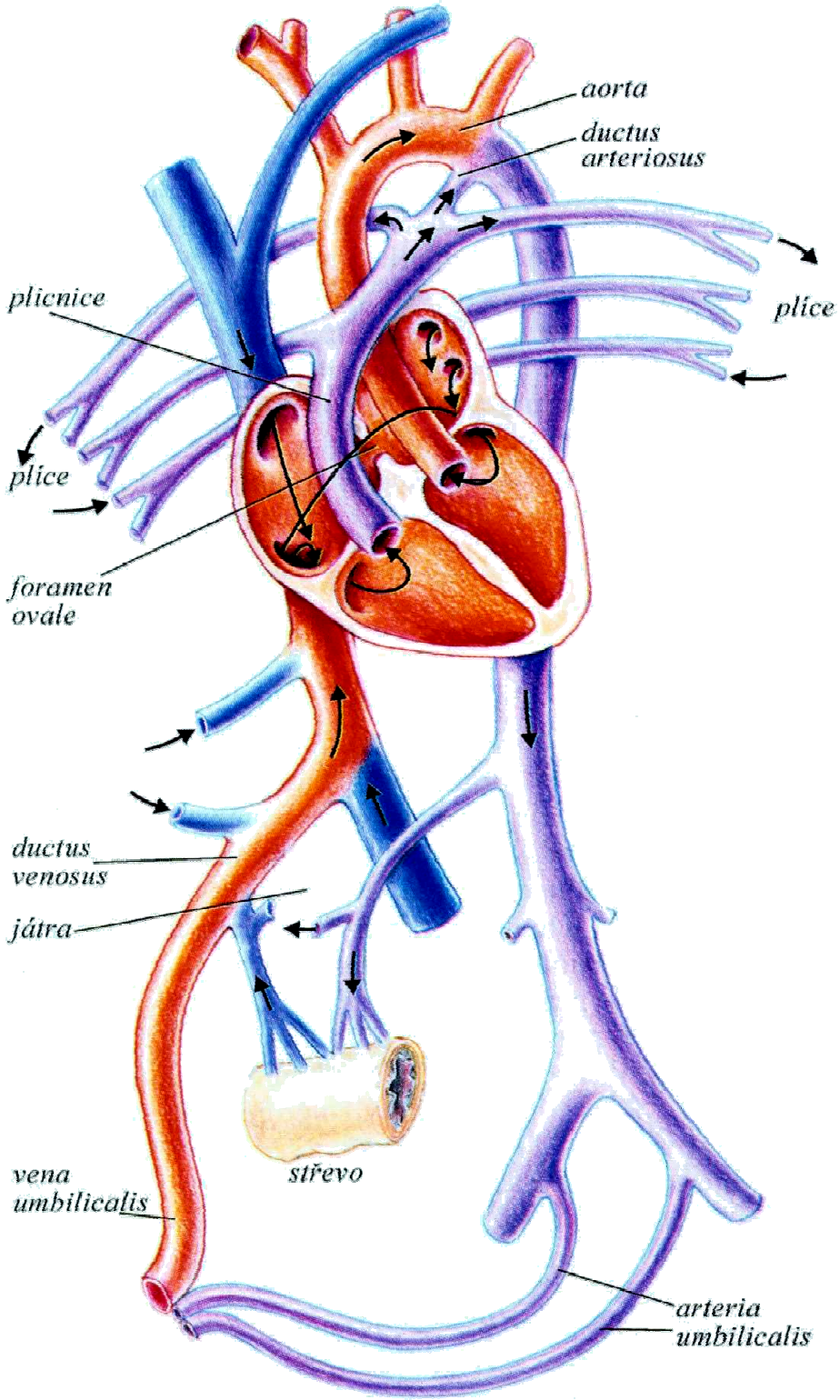
- odvádí 2 l intersticiální tekutiny za den
- podobné složení jako intersticiální tekutina nebo plazma
- ↓ množství bílkovin
- 2/3 tuků je přijato střevními lymfatickými cévami → *mléčné zabarvení lymfy*
- neobsahuje červené krvinky a destičky
- v lymfě před uzlíky málo lymfocytů
- v lymfě za uzlíkem 10x více lymfocytů než v plazmě

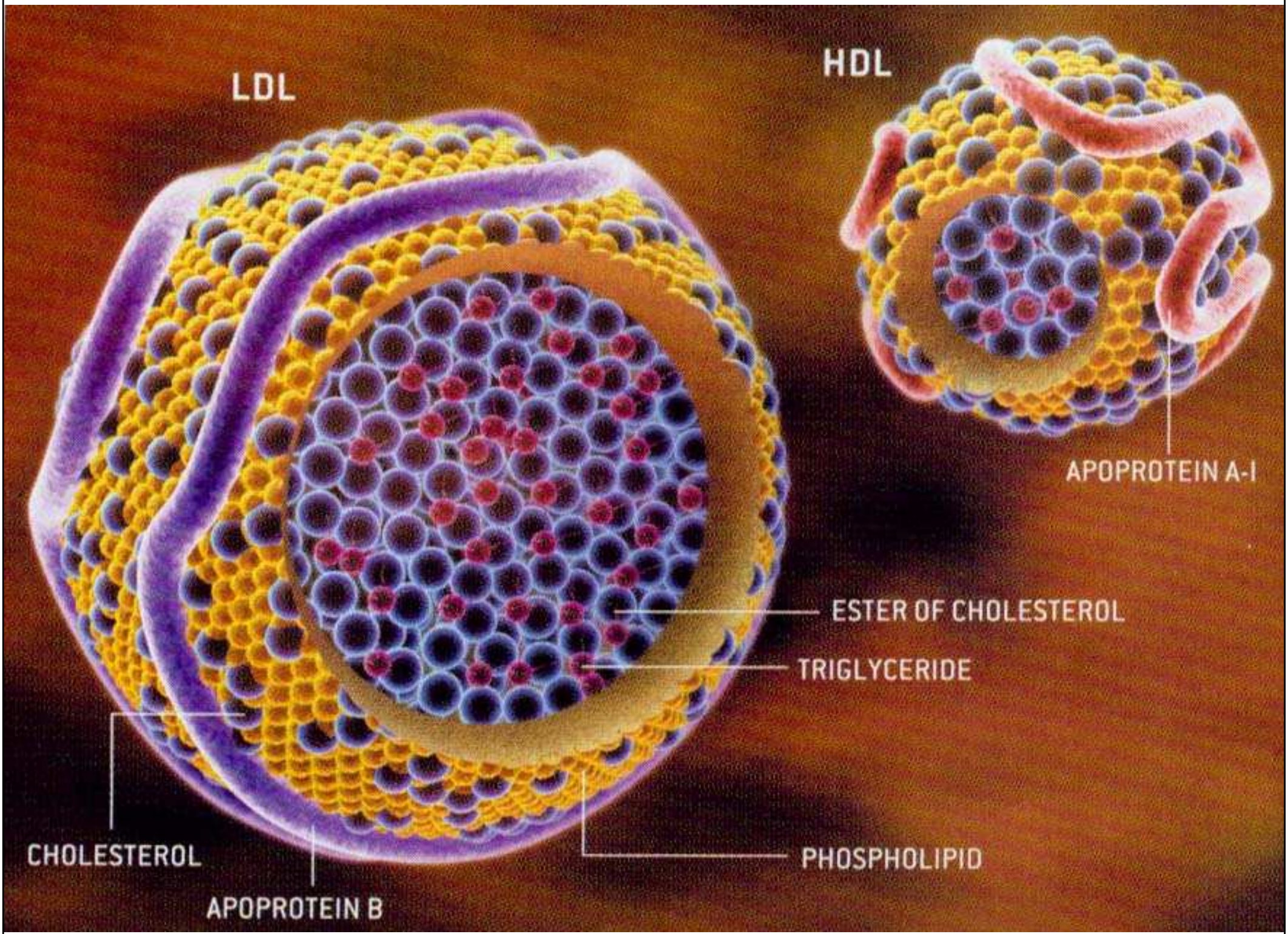



B. Vazokonstrikce a vazodilatace
presorická oblast
1a nervově
sympatikus
1b nervově
sympatikus
parasympatikus (slinné žlázy, genitálie)
roztažení cév
myogenní reakce
2a lokálně
P_{O_2}
endotelin-1 (ET_A)
$PGF_{2\alpha}$, tromboxany
ADH (V_1), adrenalin (α_1), angiotenzin II
3a hormonálně
P_{O_2}
adenozin, P_{CO_2}, H^+, K^+ aj.
NO
PGE_2, PGI_2
EDHF
bradykinin, kalidin
2b lokálně
adrenalin (β_2)
3b hormonálně
acetylcholin (M), ATP, histamin (H_1), endotelin-1 (ET_B)

Baroreflex

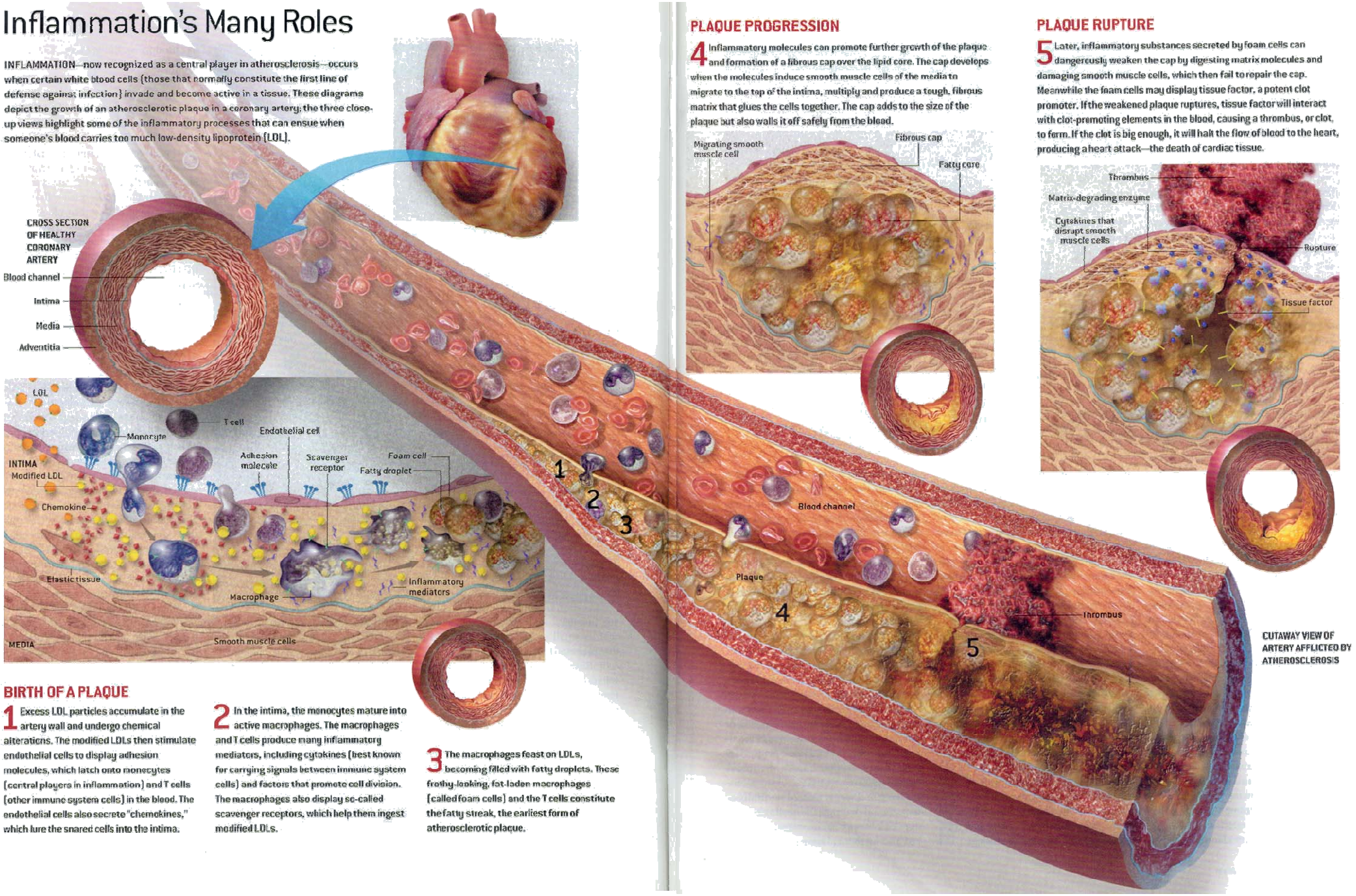



aorta
ductus arteriosus
plicnice
plíce
plíce
foramen ovale
ductus venosus
játra
střevo
vena umbilicalis
arteria umbilicalis


LDL
HDL
APOPROTEIN A-I
ESTER OF CHOLESTEROL
TRIGLYCERIDE
CHOLESTEROL
PHOSPHOLIPID
APOPROTEIN B

Inflammation's Many Roles

INFLAMMATION—now recognized as a central player in atherosclerosis—occurs when certain white blood cells (those that normally constitute the first line of defense against infection) invade and become active in a tissue. These diagrams depict the growth of an atherosclerotic plaque in a coronary artery; the three close-up views highlight some of the inflammatory processes that can ensue when someone's blood carries too much low-density lipoprotein (LDL).



CUTAWAY VIEW OF ARTERY AFFLICTED BY ATHEROSCLEROSIS

BIRTH OF A PLAQUE

1 Excess LDL particles accumulate in the artery wall and undergo chemical alterations. The modified LDLs then stimulate endothelial cells to display adhesion molecules, which latch onto monocytes (central players in inflammation) and T cells (other immune system cells) in the blood. The endothelial cells also secrete "chemokines," which lure the snared cells into the intima.

2 In the intima, the monocytes mature into active macrophages. The macrophages and T cells produce many inflammatory mediators, including cytokines (best known for carrying signals between immune system cells) and factors that promote cell division. The macrophages also display so-called scavenger receptors, which help them ingest modified LDLs.

3 The macrophages feast on LDLs, becoming filled with fatty droplets. These frothy-looking, fat-laden macrophages (called foam cells) and the T cells constitute the fatty streak, the earliest form of atherosclerotic plaque.

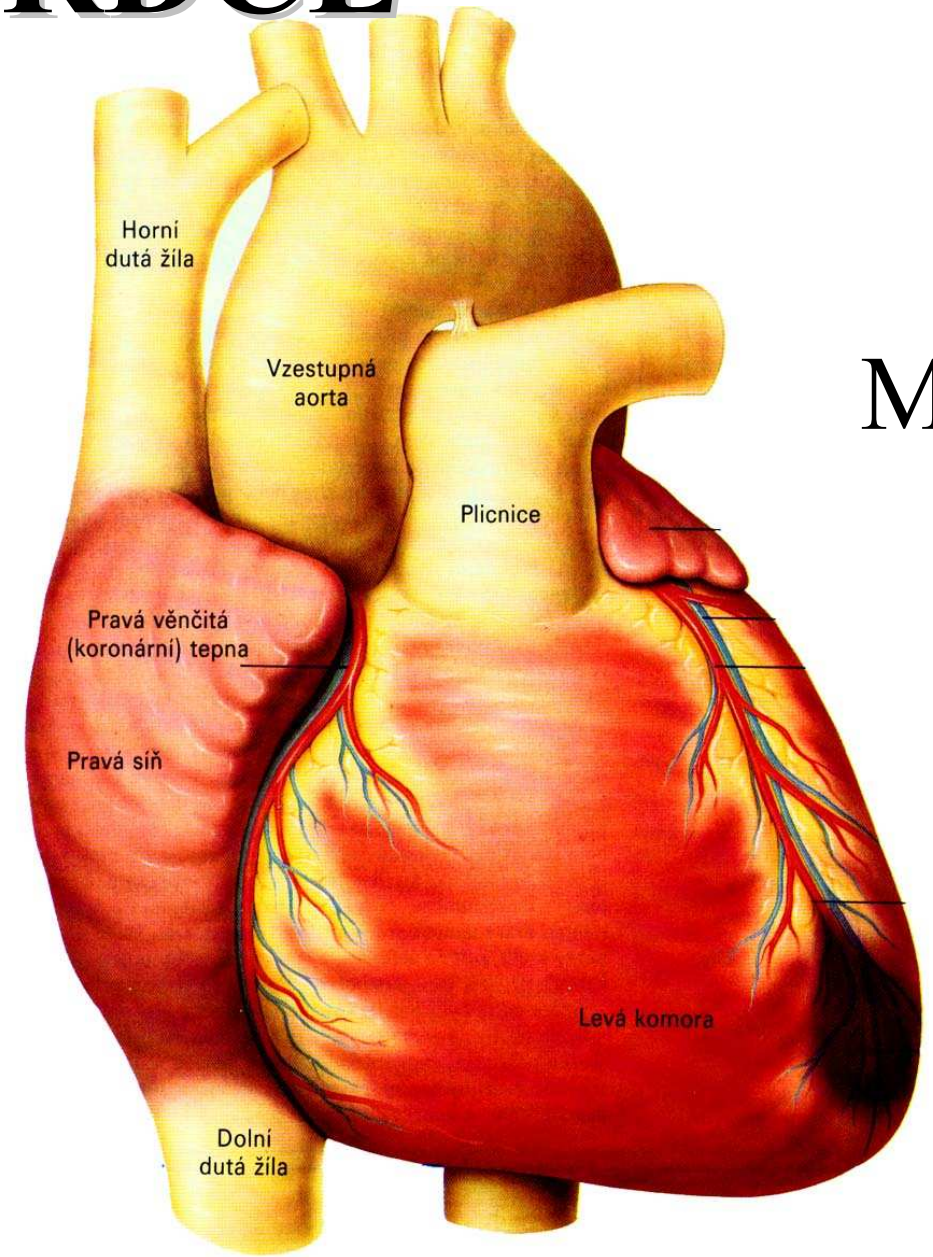
PLAQUE PROGRESSION

4 Inflammatory molecules can promote further growth of the plaque and formation of a fibrous cap over the lipid core. The cap develops when the molecules induce smooth muscle cells of the media to migrate to the top of the intima, multiply and produce a tough, fibrous matrix that glues the cells together. The cap adds to the size of the plaque but also walls it off safely from the blood.

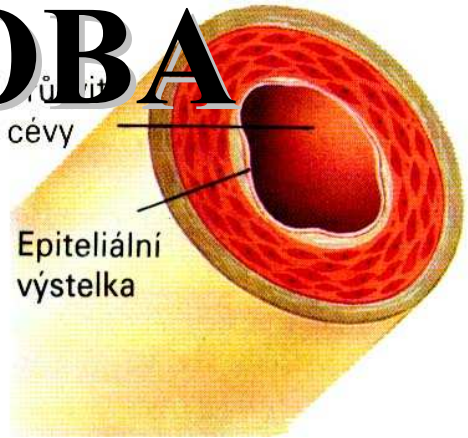
PLAQUE RUPTURE

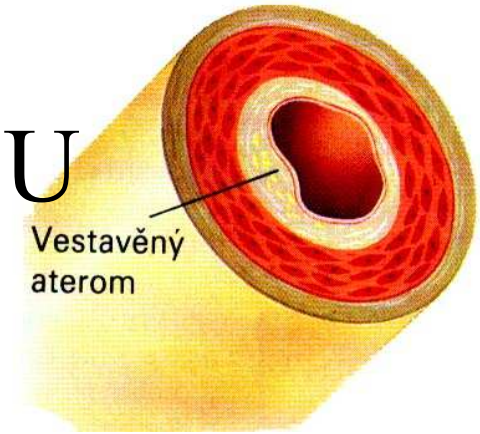
5 Later, inflammatory substances secreted by foam cells can dangerously weaken the cap by digesting matrix molecules and damaging smooth muscle cells, which then fail to repair the cap. Meanwhile the foam cells may display tissue factor, a potent clot promoter. If the weakened plaque ruptures, tissue factor will interact with clot-promoting elements in the blood, causing a thrombus, or clot, to form. If the clot is big enough, it will halt the flow of blood to the heart, producing a heart attack—the death of cardiac tissue.

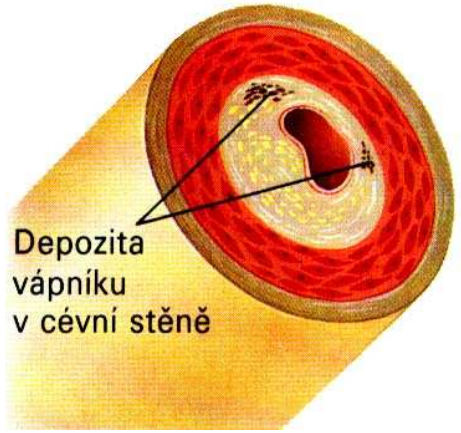
ISCHEMICKÁ CHOROBA SRDCE

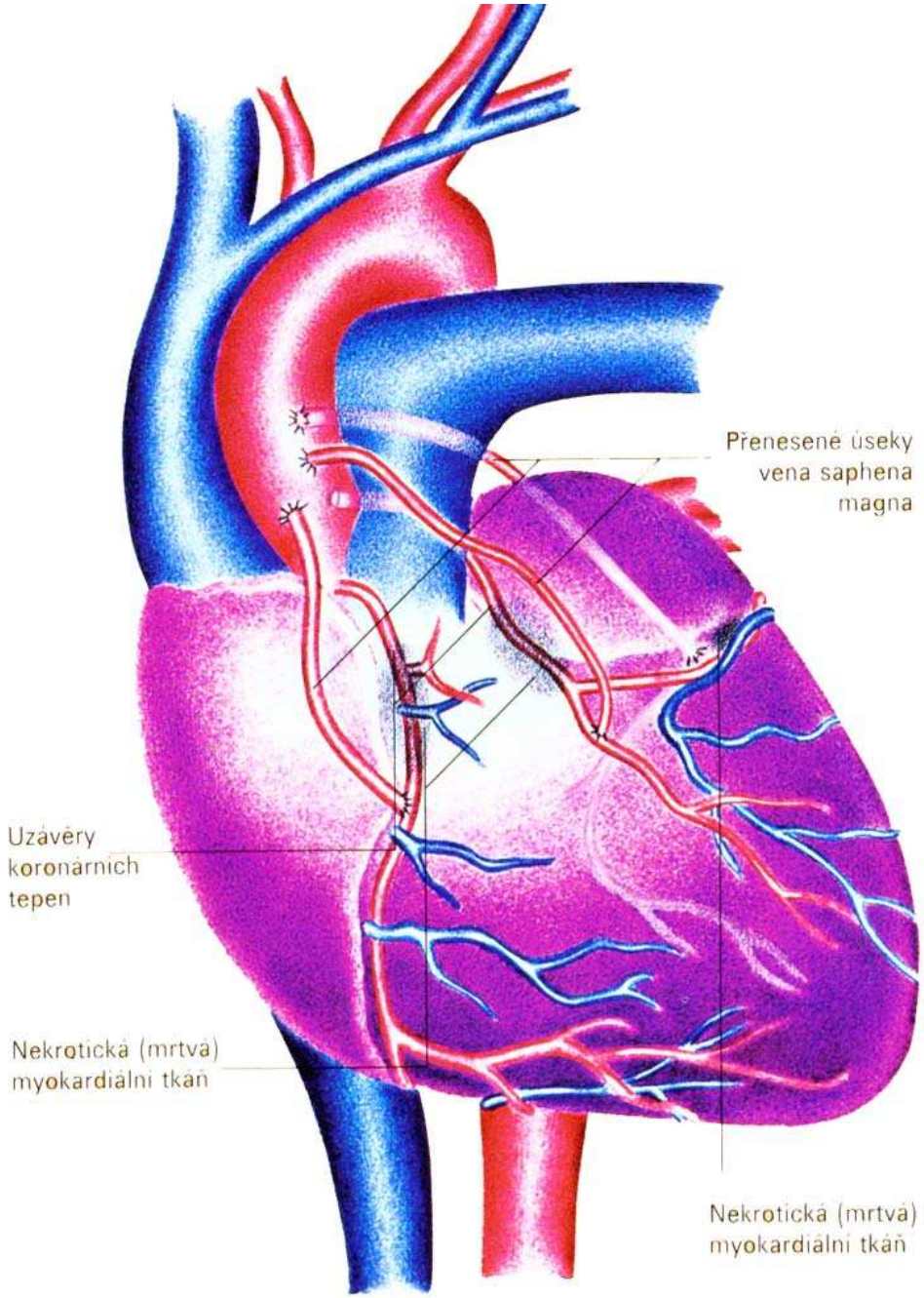


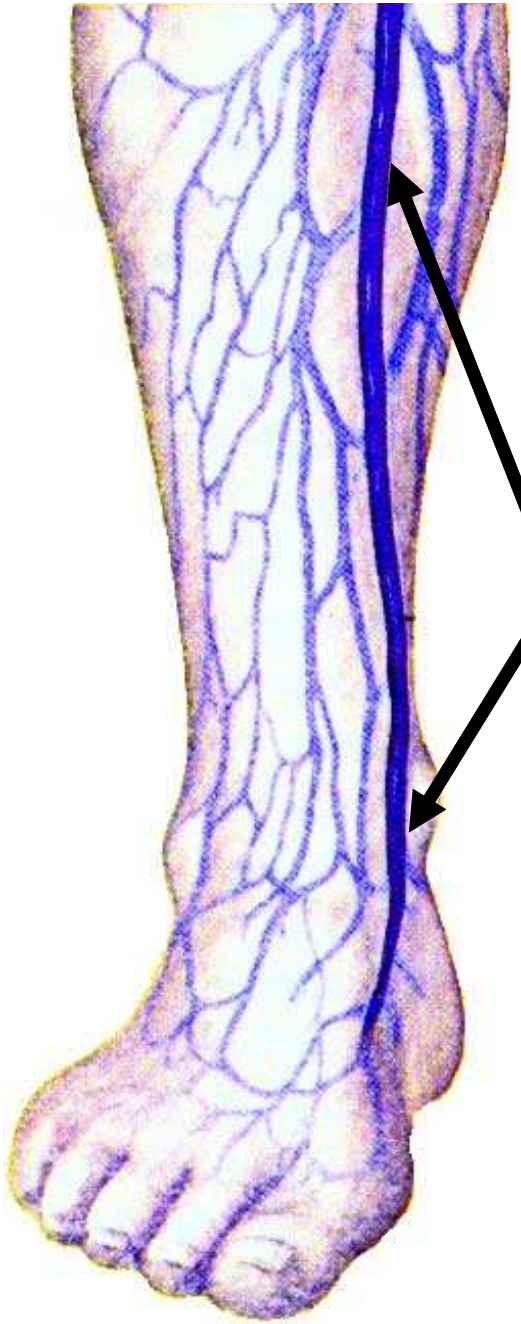
INFARKT MYOKARDU (záhať)











vaena saphena magna