



SISTEM KARDIOVASKULAR

DENNY AGUSTININGSIH

Tekanan Darah



- PARAMETER YANG MUDAH DIAMATI
- TERMASUK SALAH SATU TANDA VITAL

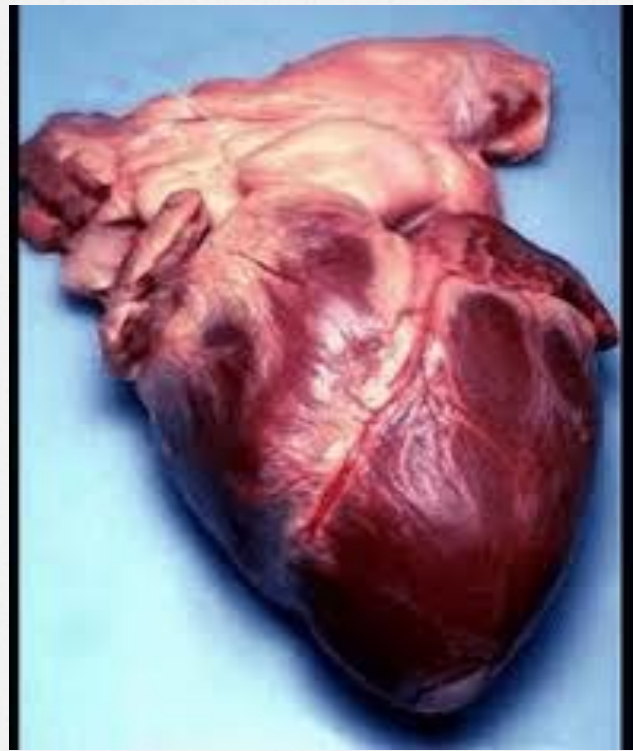
APA YANG DIMAKSUD DENGAN TEKANAN DARAH?

Desakan **darah** terhadap dinding **pembuluh darah** akibat pompa **jantung**

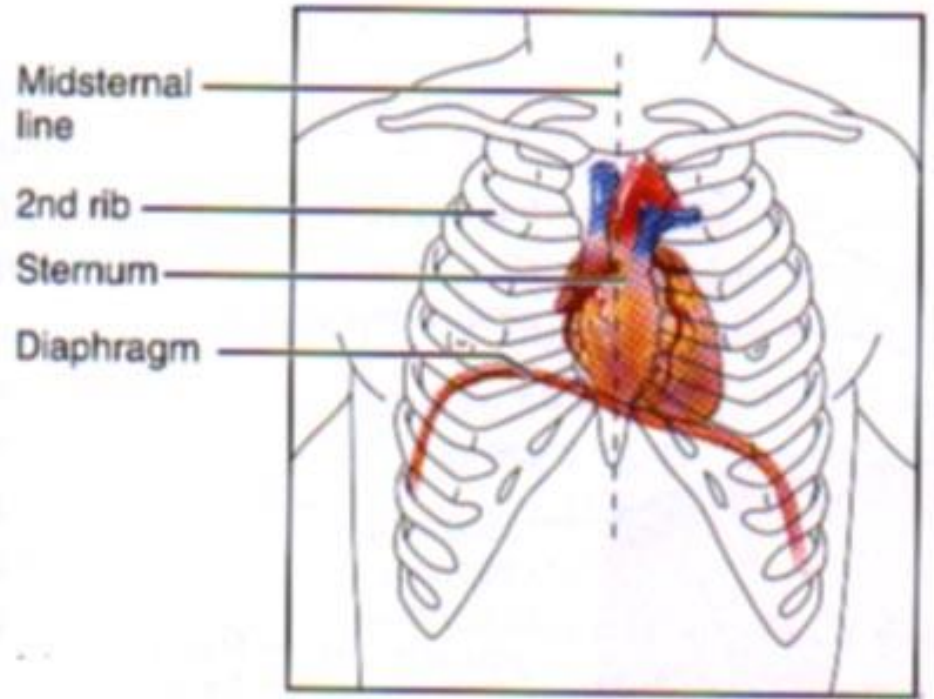
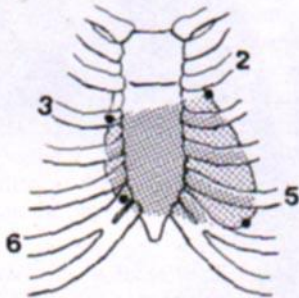
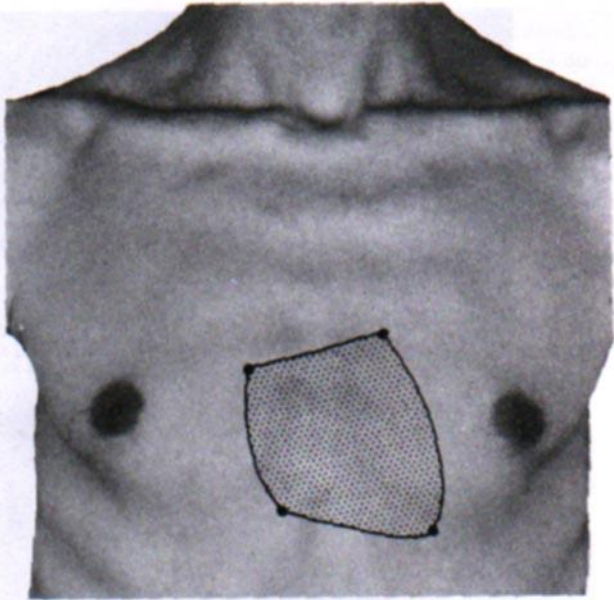
FAKTOR-FAKTOR YG MEMPENGARUHI TEKANAN DARAH

1. POMPA JANTUNG
2. KONDISI PEMBULUH DARAH ARTERI
 - o diameter
 - o kelenturan dinding
3. DARAH (cairan intravaskular)
 - o Volume
 - o viskositas

JANTUNG

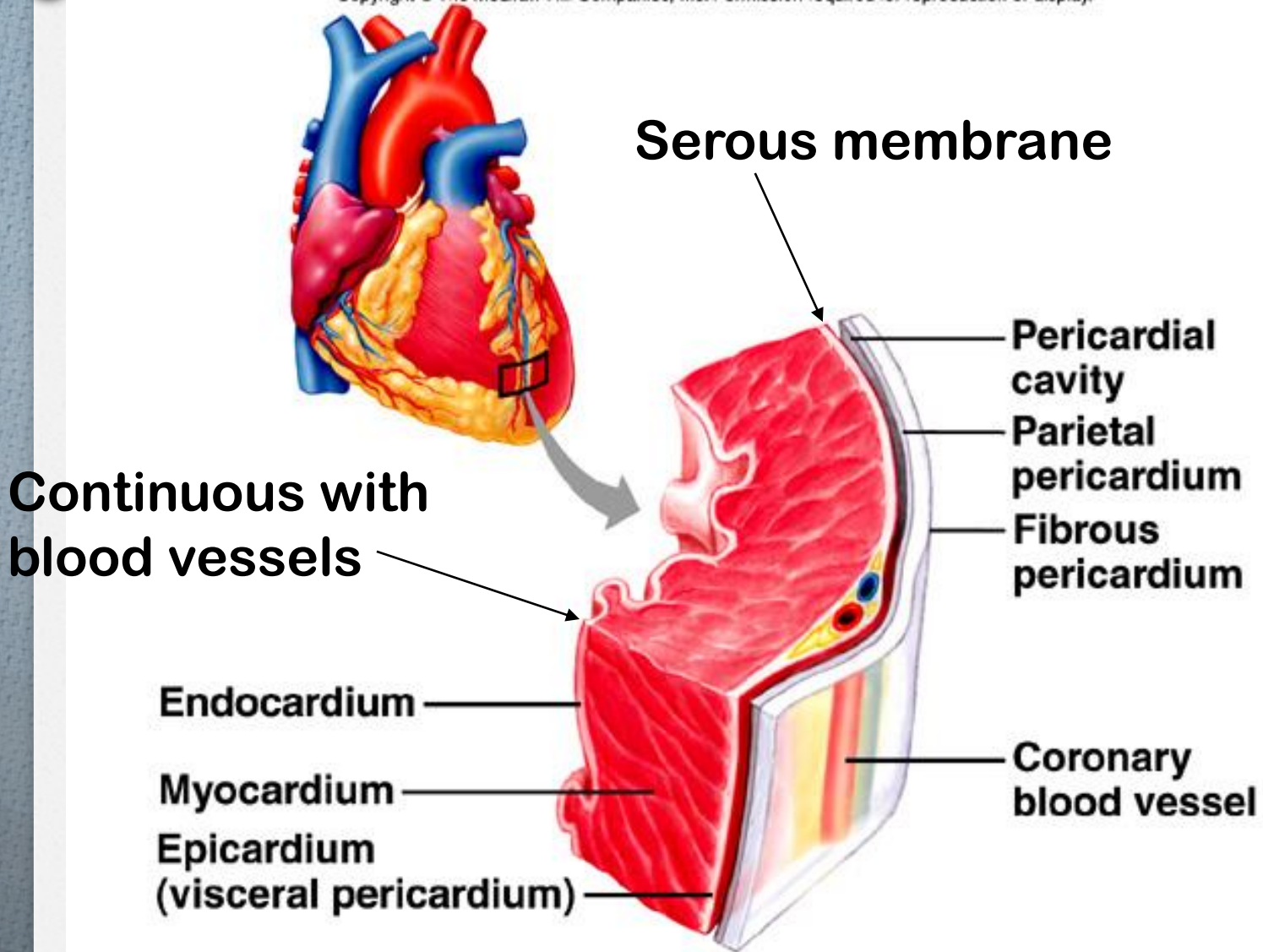


Location of Heart in Thorax



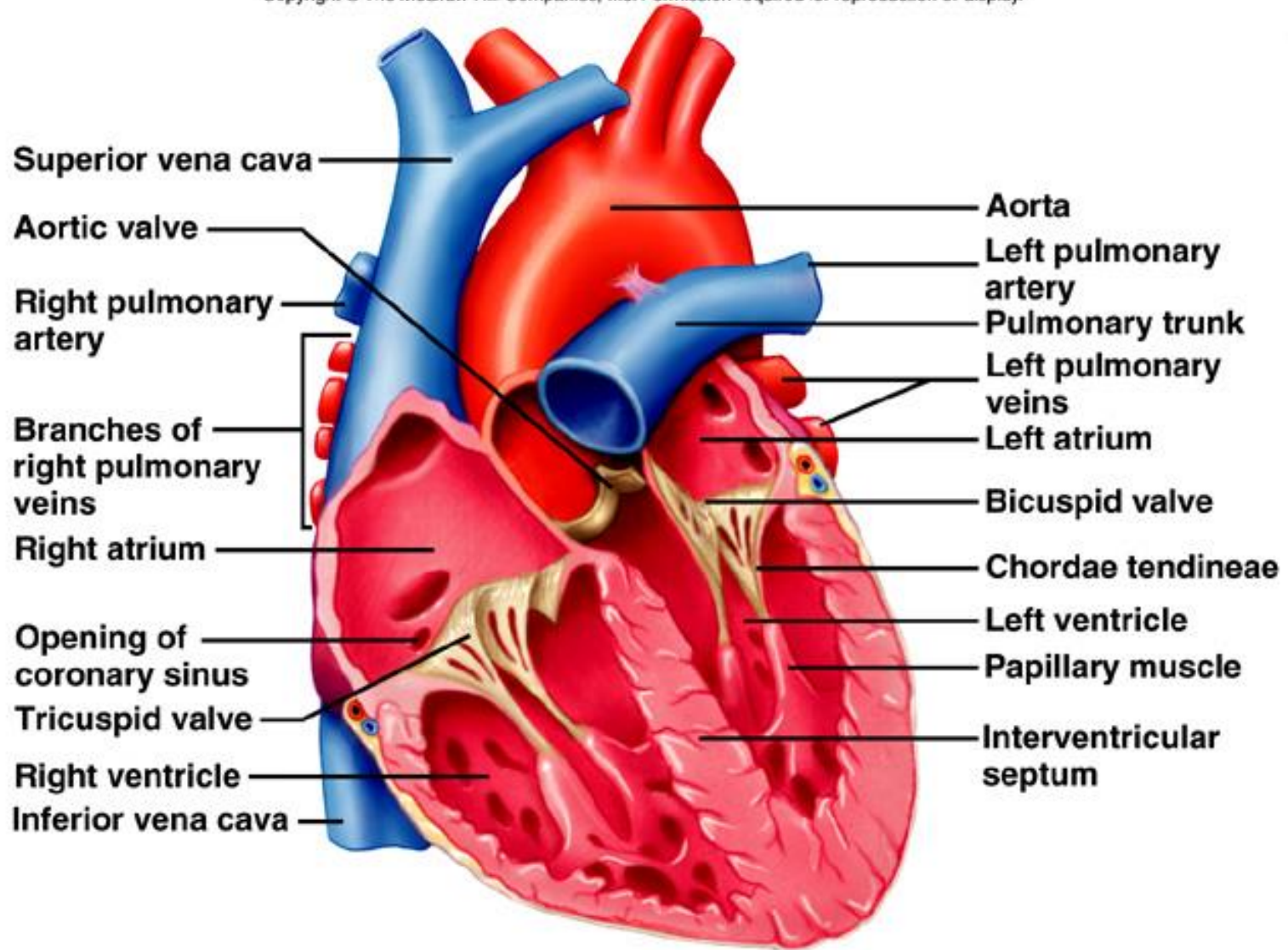
Lapisan-lapisan jantung

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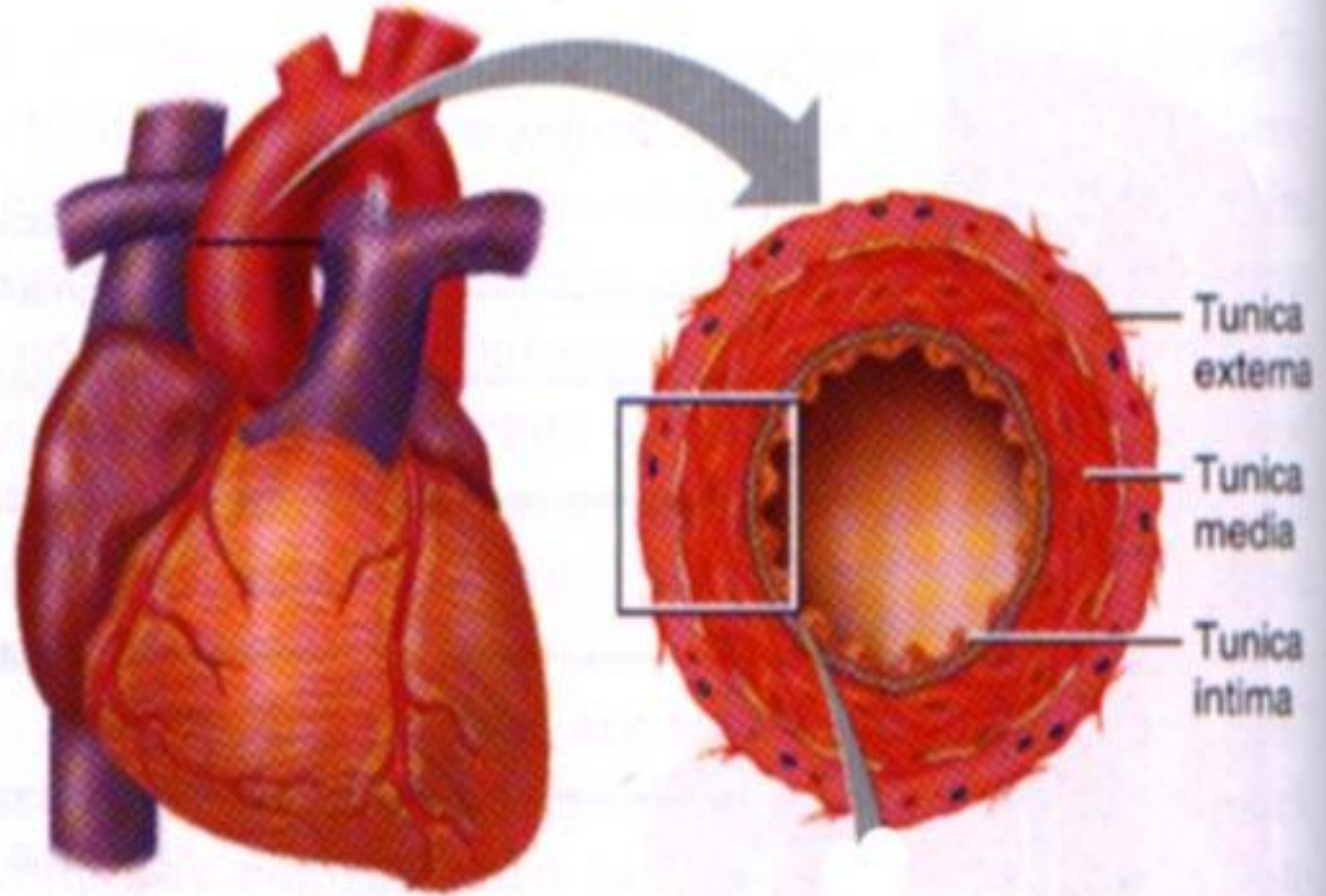


Chambers of the heart; valves

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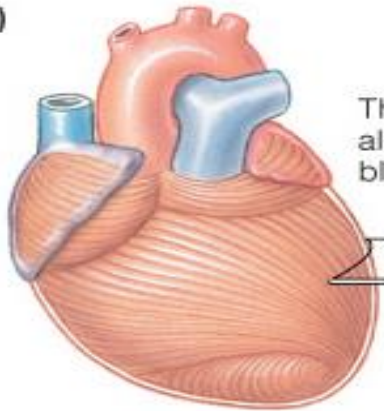


Pembuluh darah



Otot jantung

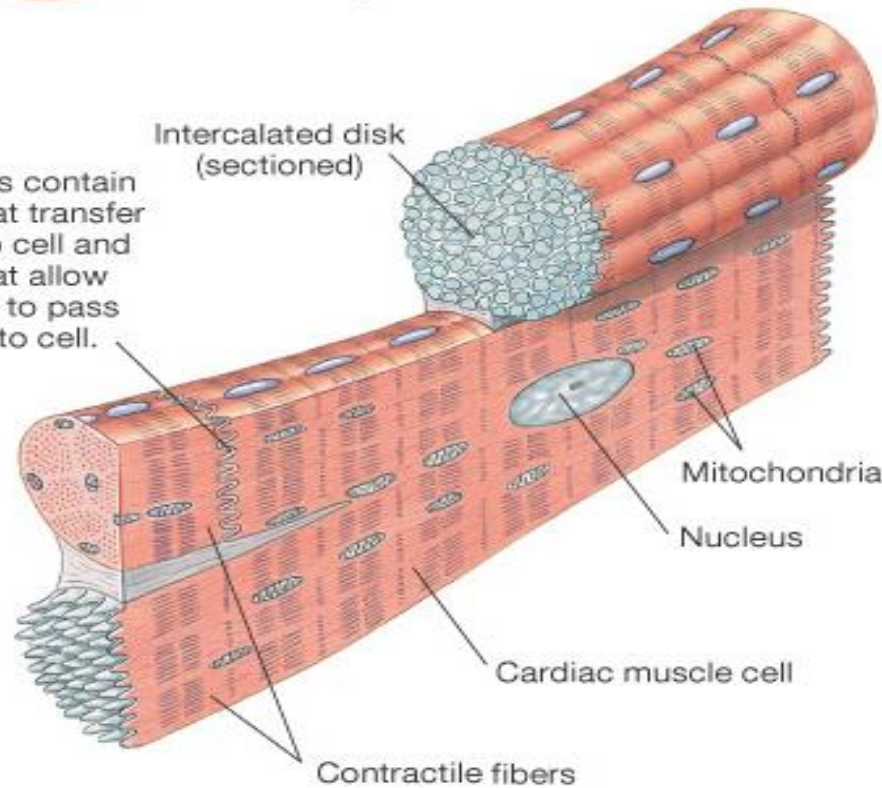
(a)



The spiral arrangement of ventricular muscle allows ventricular contraction to squeeze the blood upward from the apex of the heart.

(b)

Intercalated disks contain desmosomes that transfer force from cell to cell and gap junctions that allow electrical signals to pass rapidly from cell to cell.



Intercalated disk (sectioned)

Mitochondria

Nucleus

Cardiac muscle cell

Contractile fibers

BAGAIMANA JANTUNG DAPAT MEMOMPA DARAH ?

1. Faktor kelistrikan

2. Faktor Mekanik

DUA JENIS SEL OTOT JANTUNG

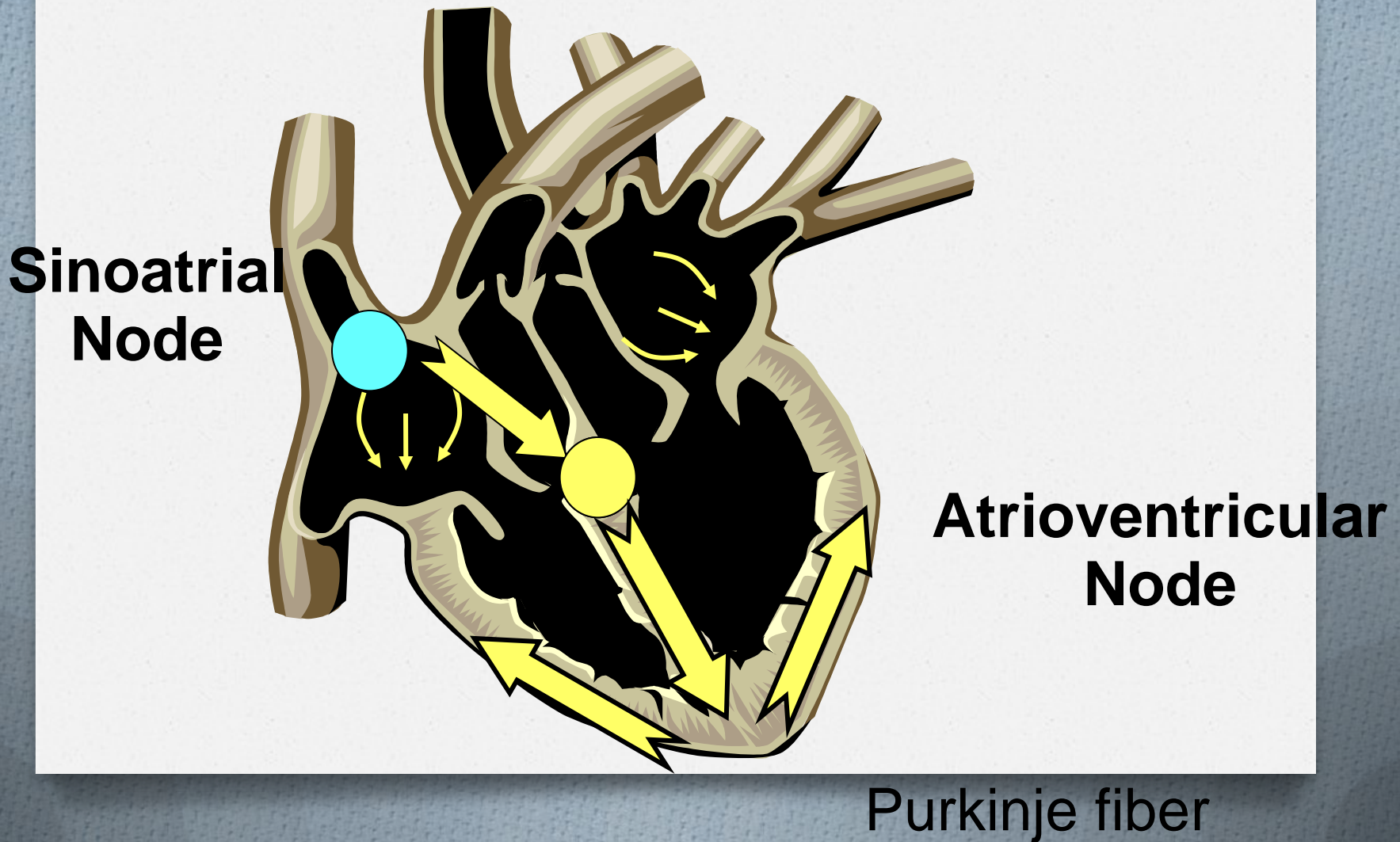
o SEL KONTRAKTIL: 95%- 99%

- Untuk kekuatan memompa darah
- Tidak mudah lelah

o SEL AUTORITMIK/AUTOMATIK: 1%-5%

- Bertanggung jawab untuk memulai dan/atau menjalankan impuls listrik
- pacemaker potential
- Menentukan frekuensi dan irama denyut jantung

SISTEM KONDUKSI OTOT JANTUNG



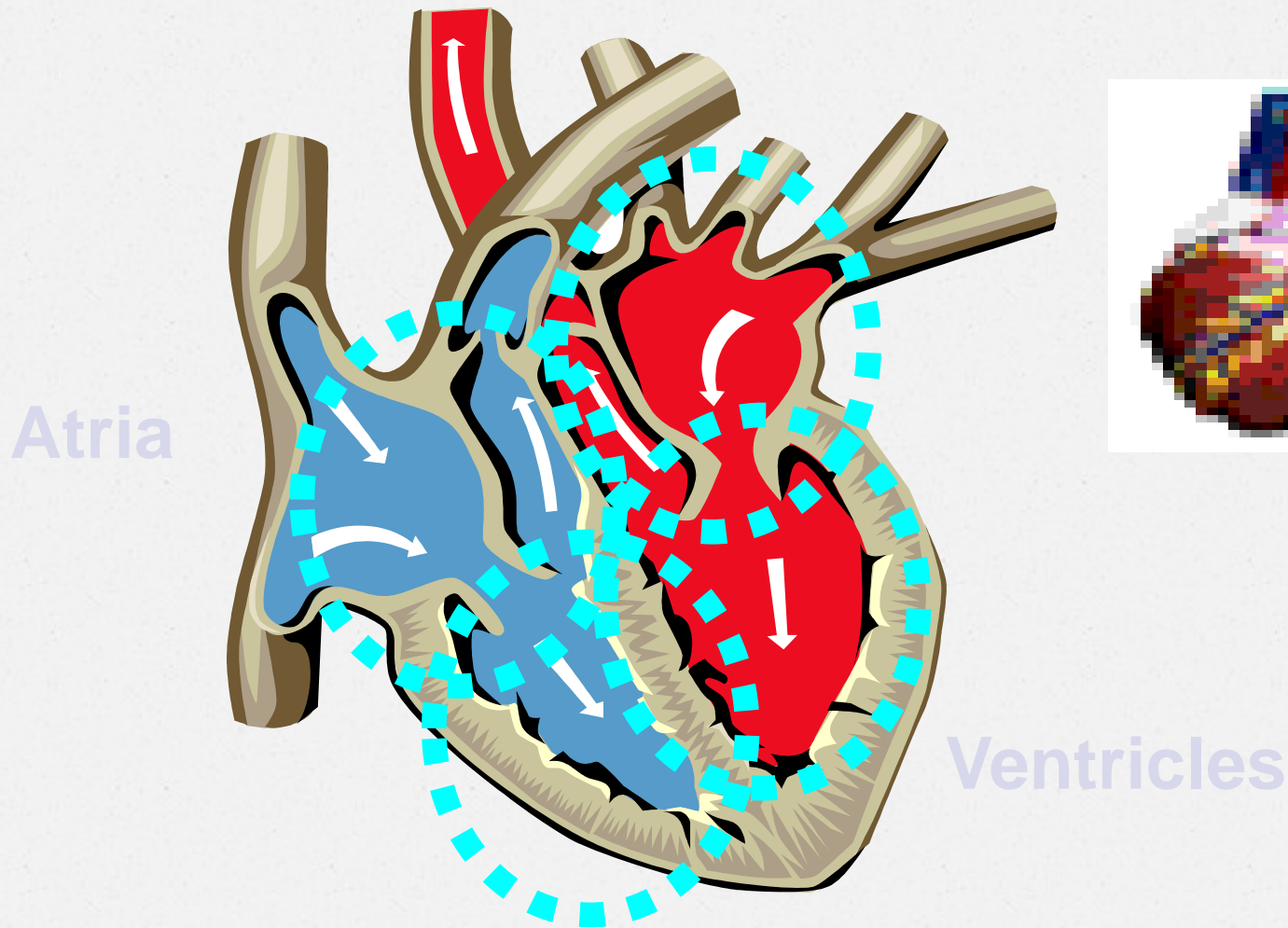
Kelistrikan jantung menentukan

1. Frekuensi denyut jantung
2. Irama denyut jantung
3. Terjadinya kontraksi otot jantung

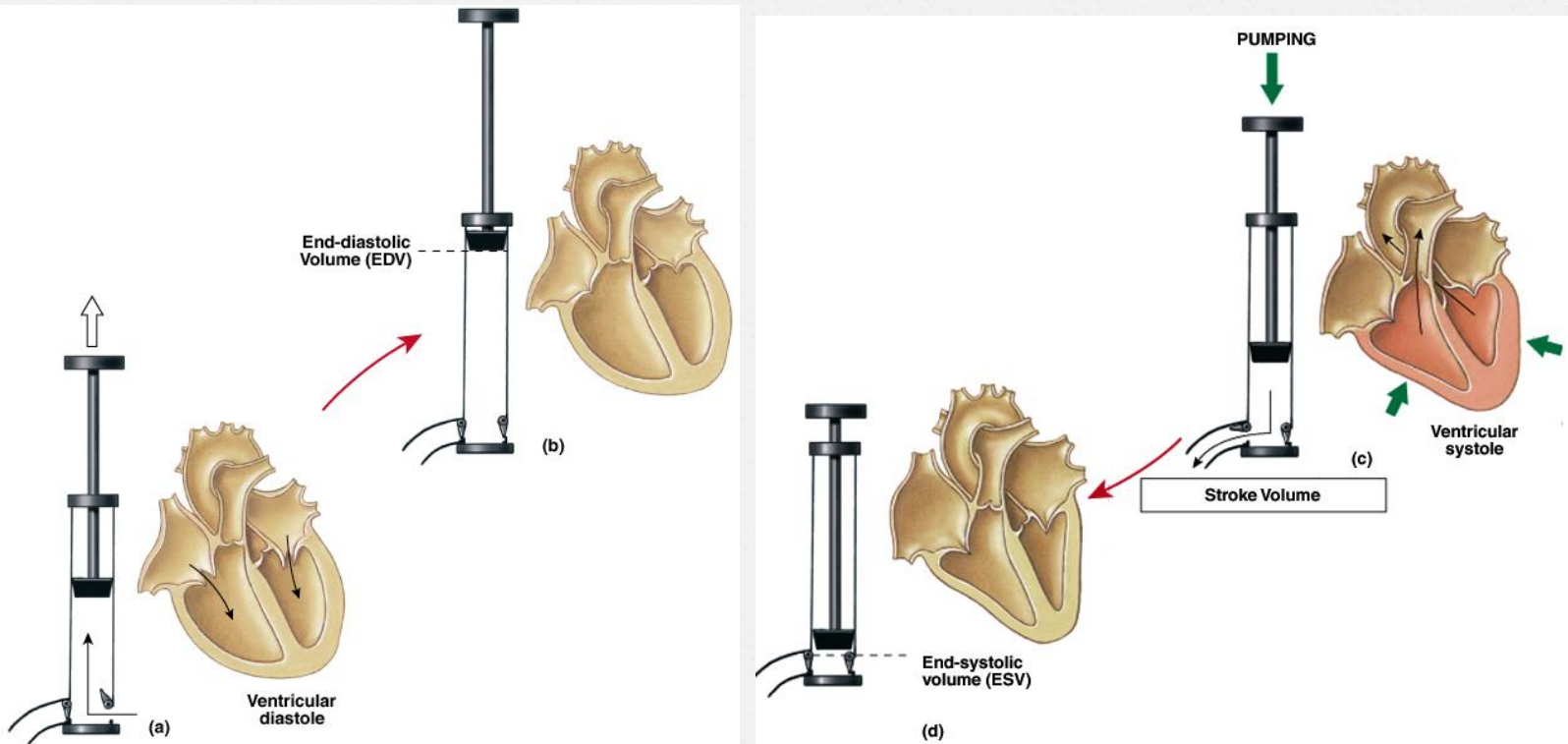
FAKTOR-FAKTOR YG MEMPENGARUHI FREKUENSI DENYUT JANTUNG

1. saraf simpatis dan parasimpatis
2. Hormon adrenalin, asetilkolin, tiroid, estrogen, testosteron
3. Ion Na, Ca, K
4. Suhu badan

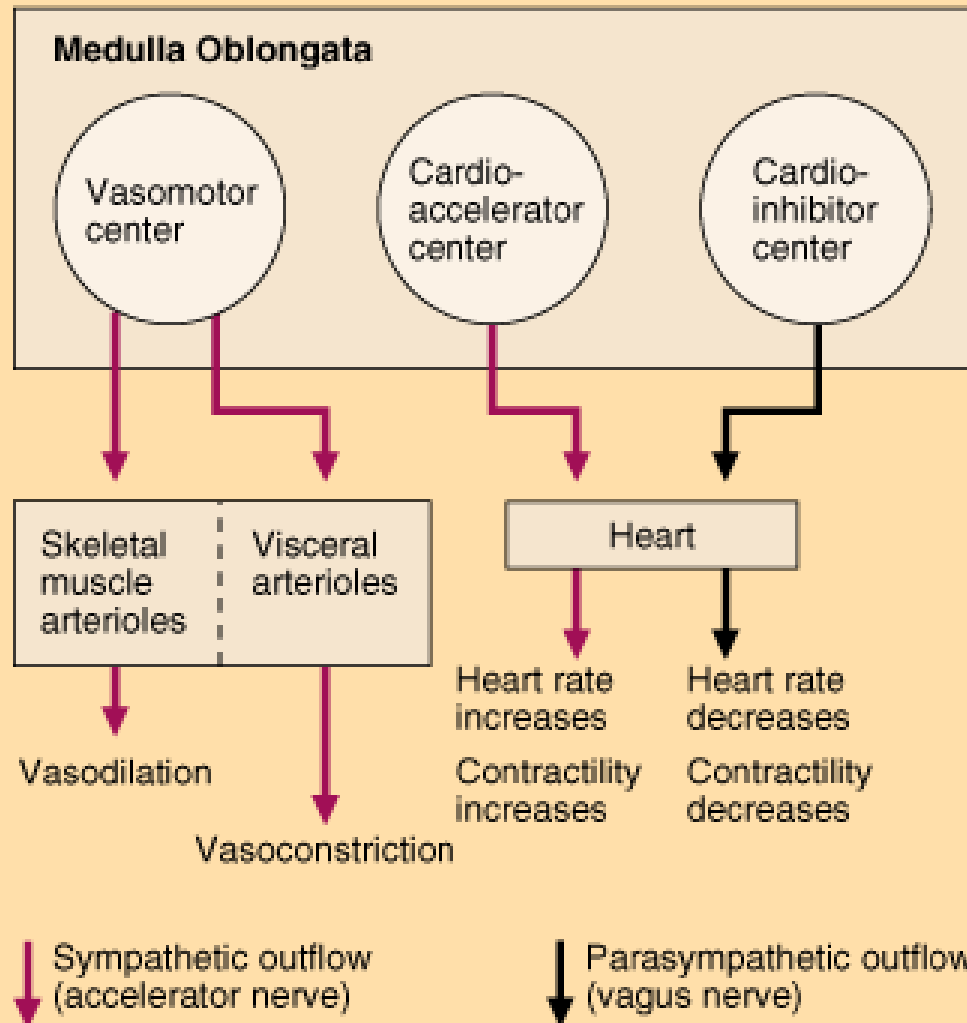
TWO SEPARATE PUMPS



POMPA JANTUNG

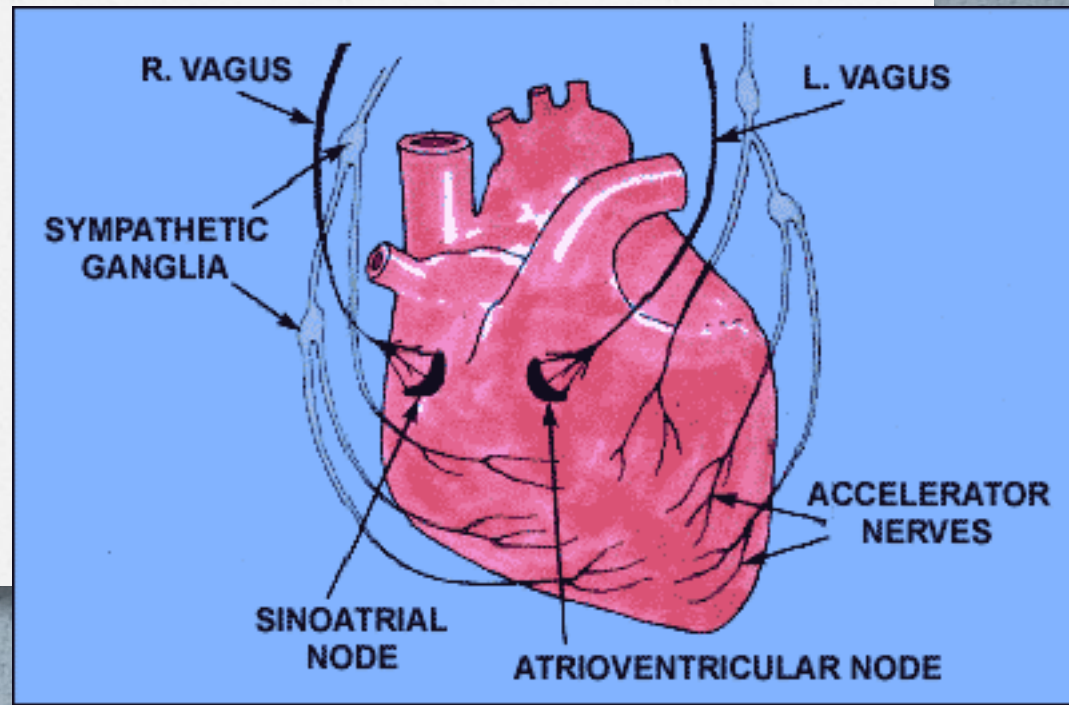
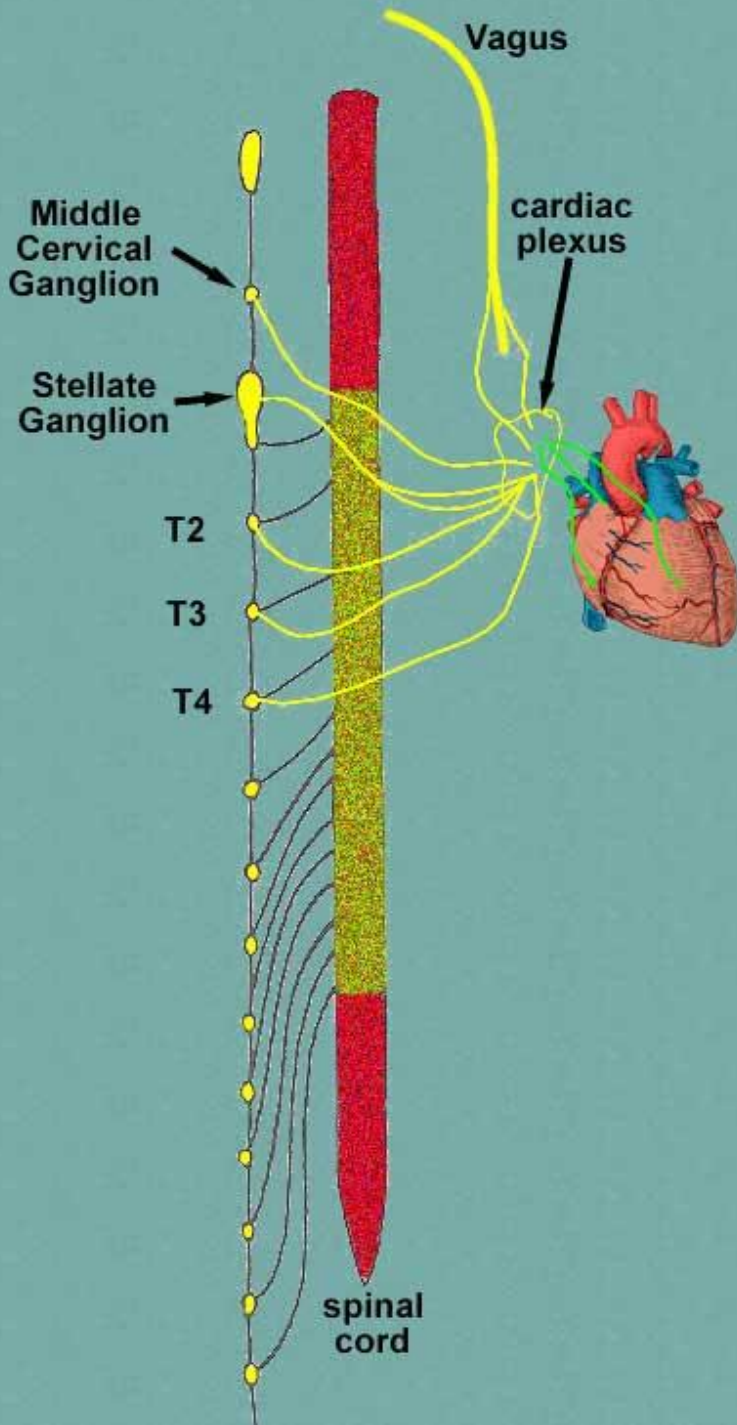


► Neural Control of Cardiovascular Function

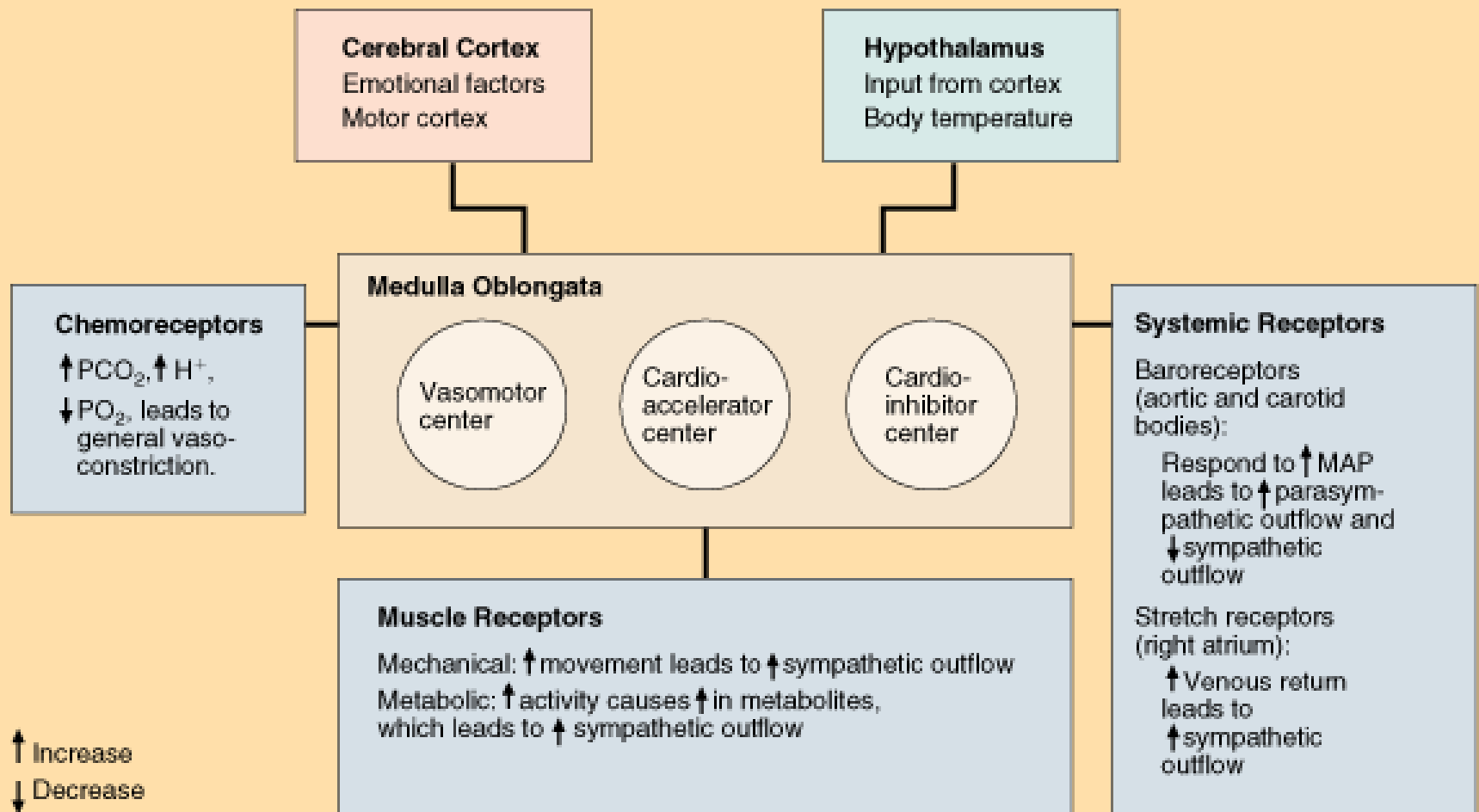


Heart Innervation

- Heart receives visceral motor innervation
 - Sympathetic (speeds up)
 - Parasympathetic (slows down)

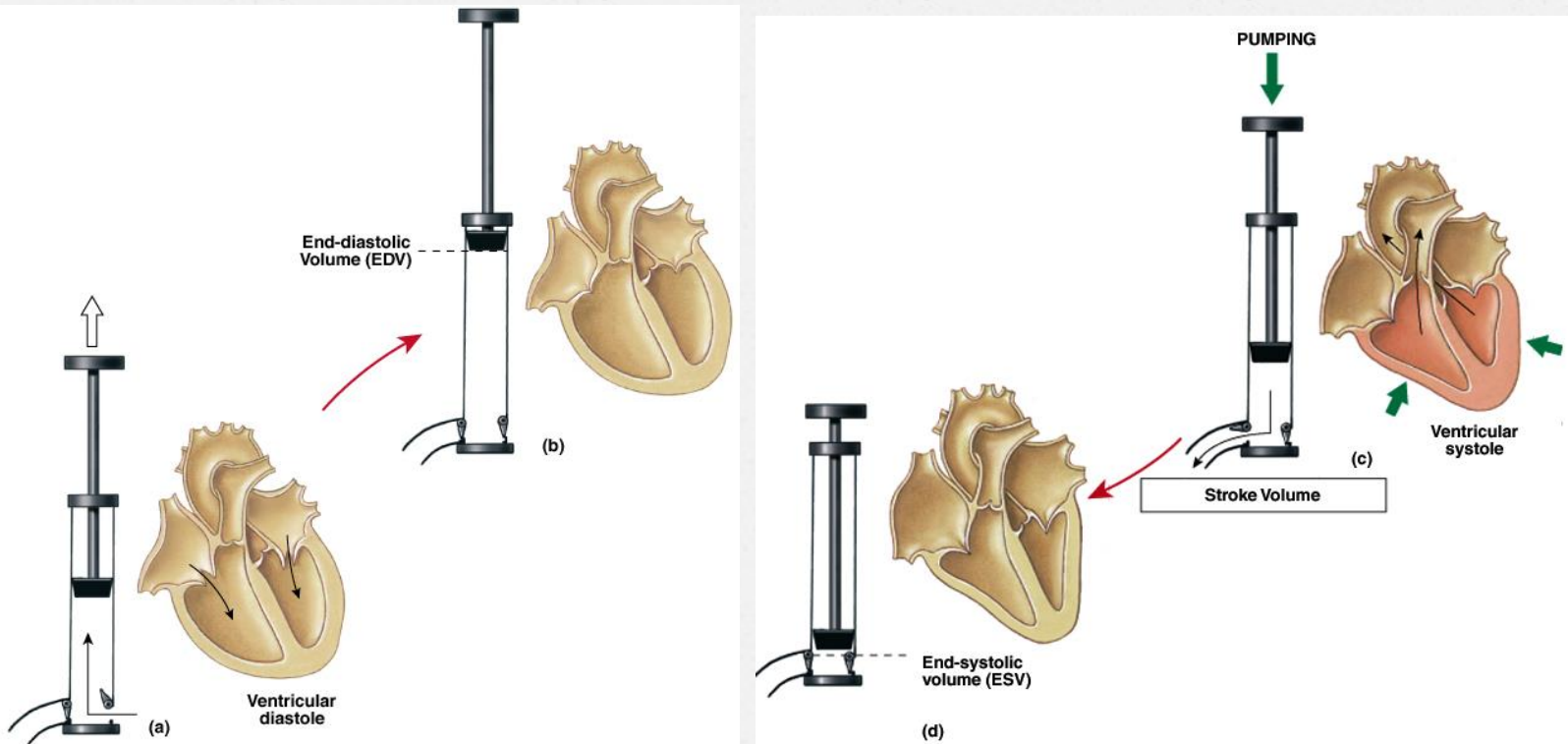


► Factors Affecting Neural Control of Cardiovascular Function



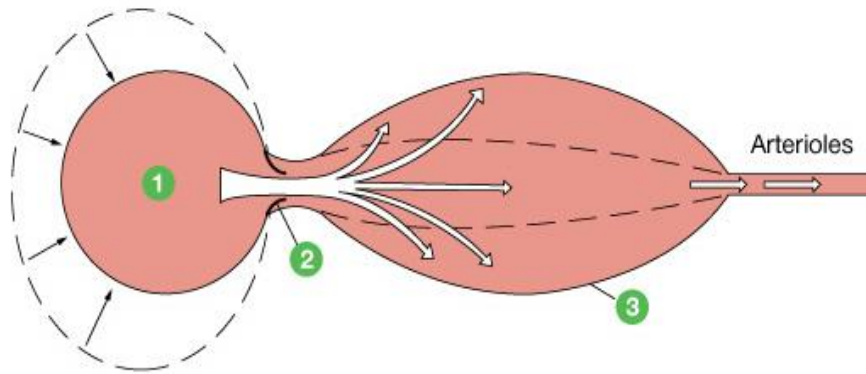
PEMBULUH DARAH

POMPA JANTUNG



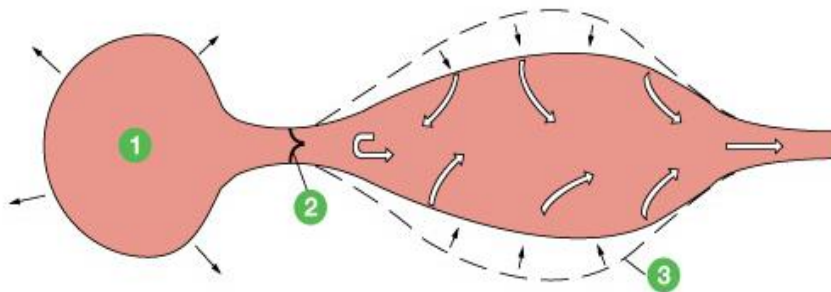
TEKANAN DARAH

(a) Ventricular contraction



- 1 Ventricle contracts.
- 2 Semilunar valve opens.
- 3 Aorta and arteries expand and store pressure in elastic walls.

(b) Ventricular relaxation



- 1 Isovolumic ventricular relaxation
- 2 Semilunar valve shuts.
- 3 Elastic recoil of arteries sends blood forward into rest of circulatory system.

Figure 15-4: Elastic recoil in the arteries

- o Arteri dapat dibayangkan seperti balon panjang tanpa lubang terbuka di ujungnya



- o Selama masih ada aliran udara, maka balon tetap mengembang

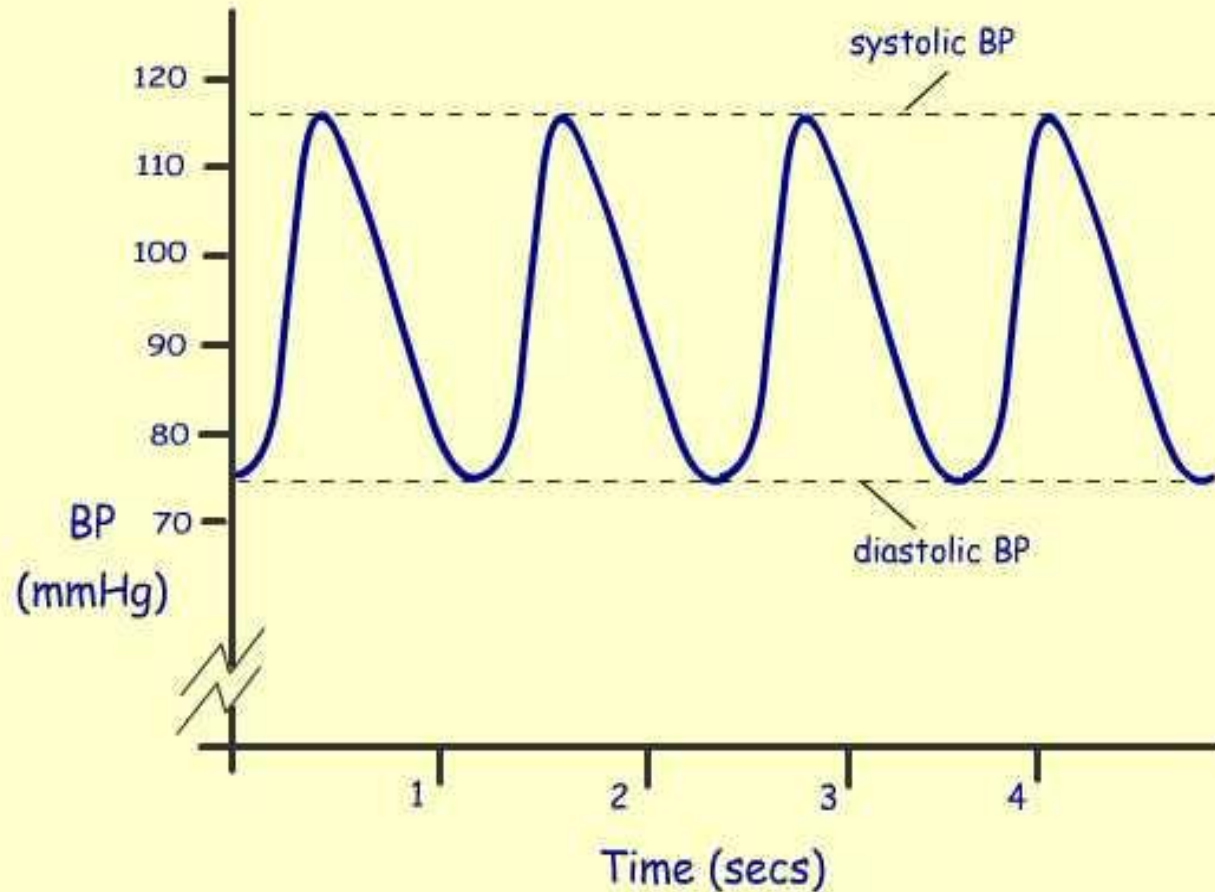
Systole and Diastole

- o Kondisi arteri berfluktuasi diantara systole dan diastole
- o Saat systole, tekanan di arteri meningkat akibat darah mengalir dari jantung
 - o dinding arteri meregang
 - o Teraba tegangan di arteri perifer sebagai denyut nadi

Systole and Diastole

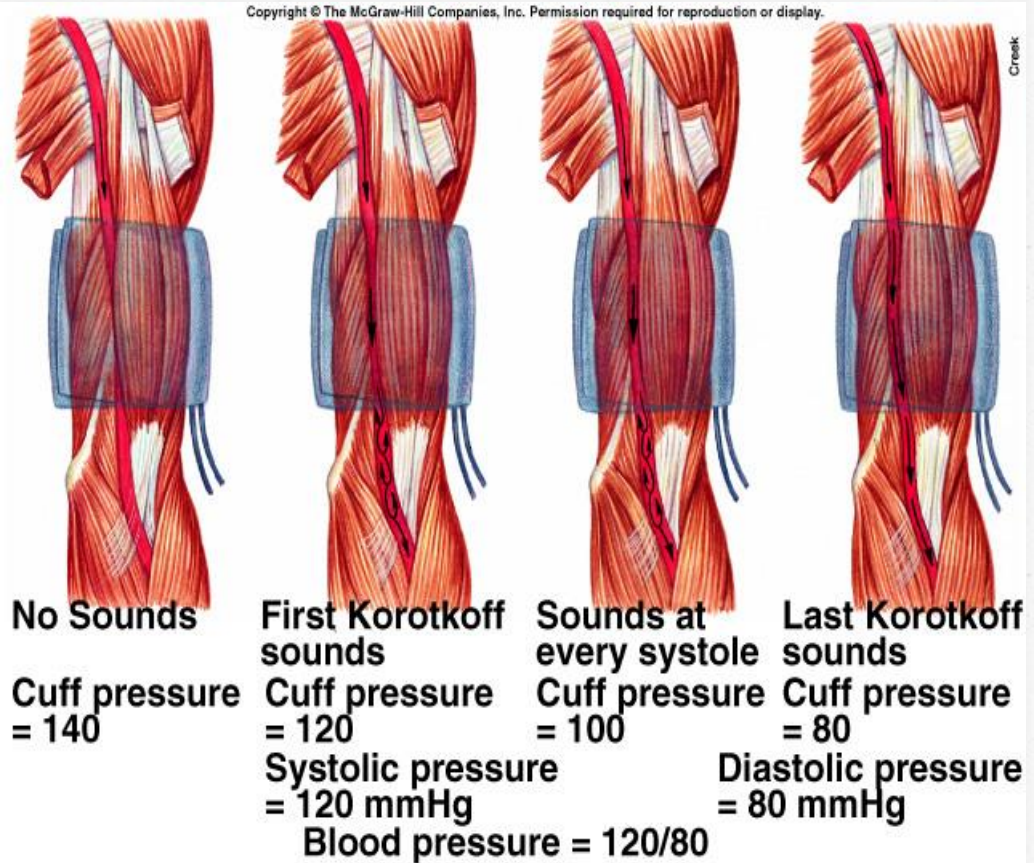
- o Saat diastole, elastic recoil arteri mendorong darah masuk kapiler
 - o Tekanan di arteri menurun karena darah berpindah
 - o Tidak pernah sampai 0 mmHg

Here is a graph of changes in arterial BP

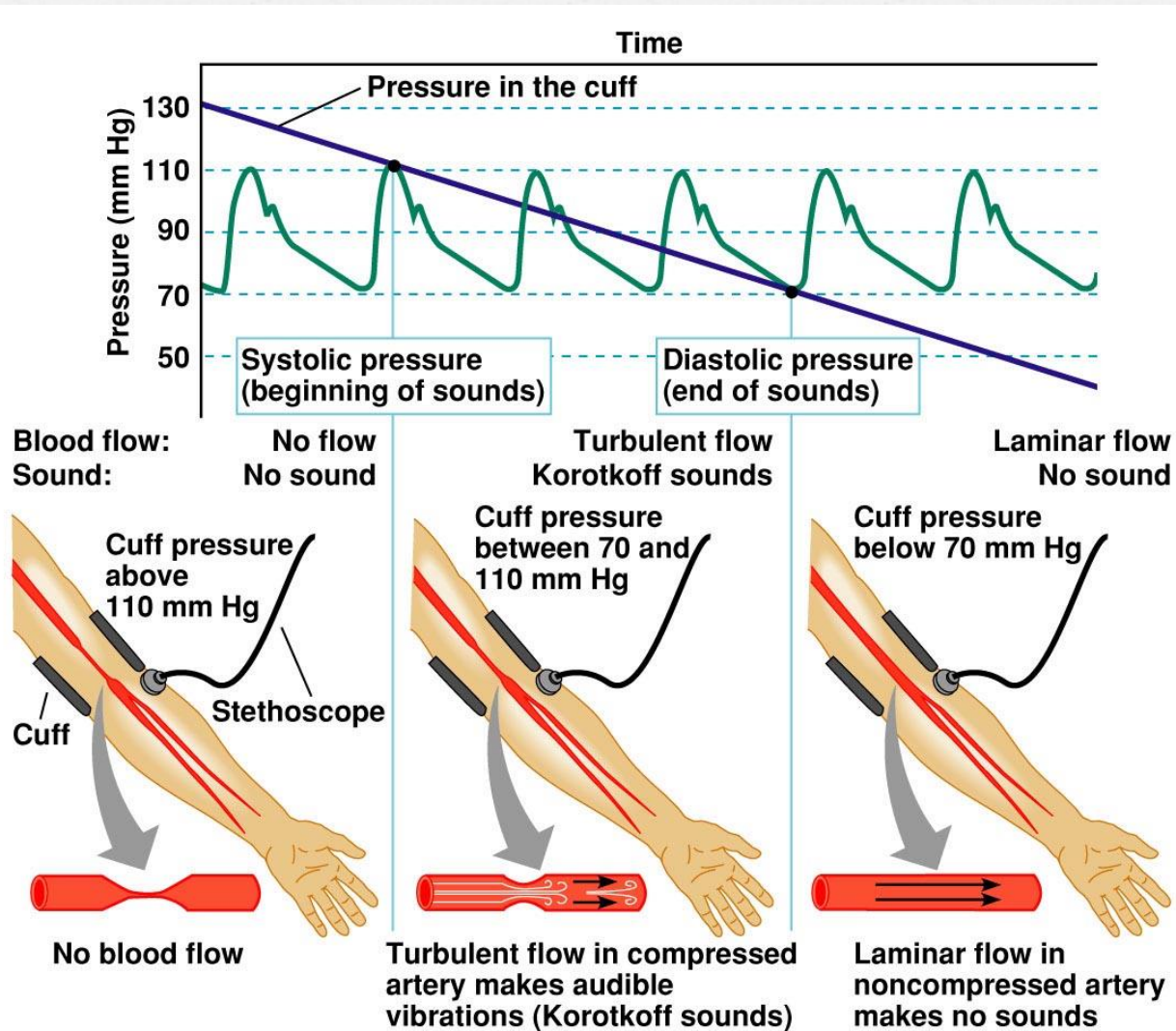


Measurement of Blood Pressure

- Blood pressure cuff is inflated above systolic pressure, occluding the artery.
- As cuff pressure is lowered, the blood will flow only when systolic pressure is above cuff pressure, producing the sounds of Korotkoff.
- Korotkoff sounds will be heard until cuff pressure equals diastolic pressure, causing the sounds to disappear.



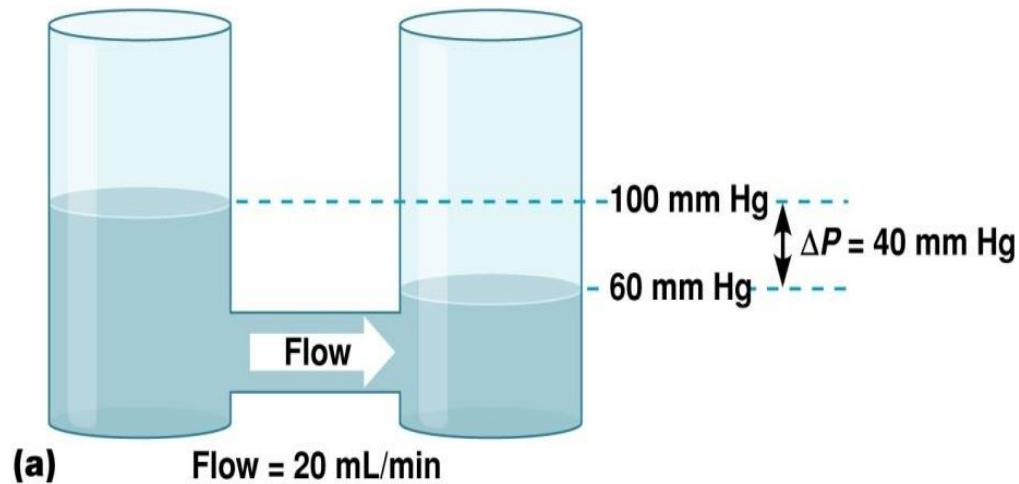
Arterial blood pressure

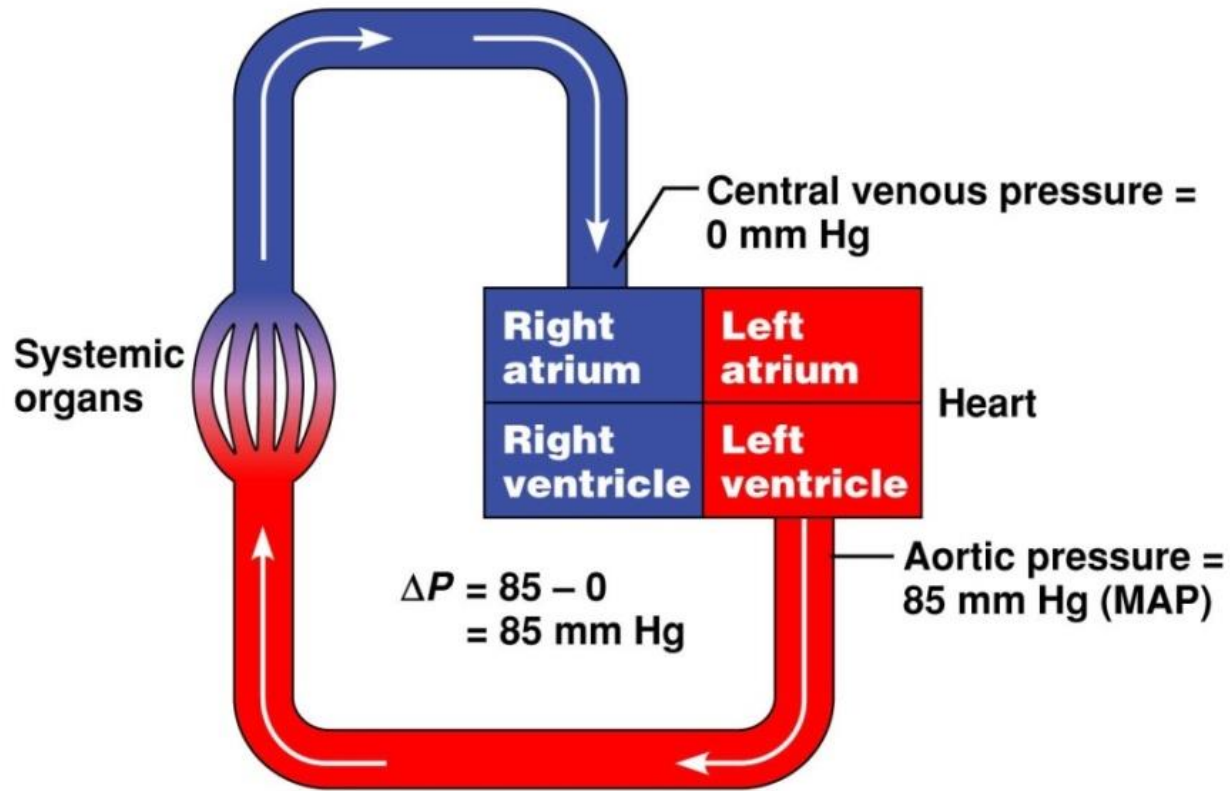


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Physical laws governing blood flow and blood pressure

Flow of blood through out body =
pressure gradient within vessels X resistance to flow

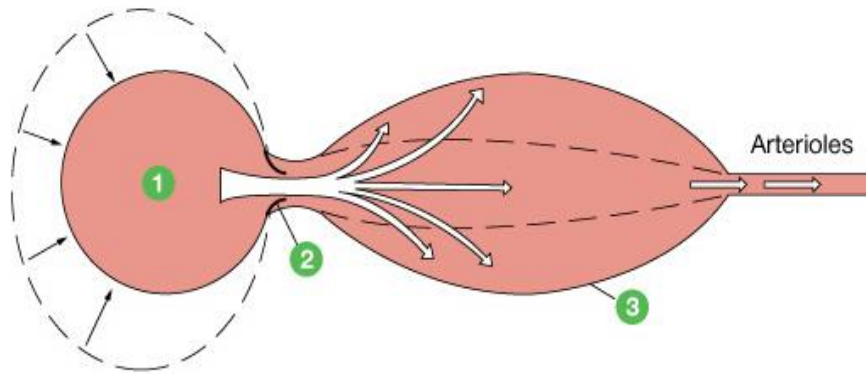




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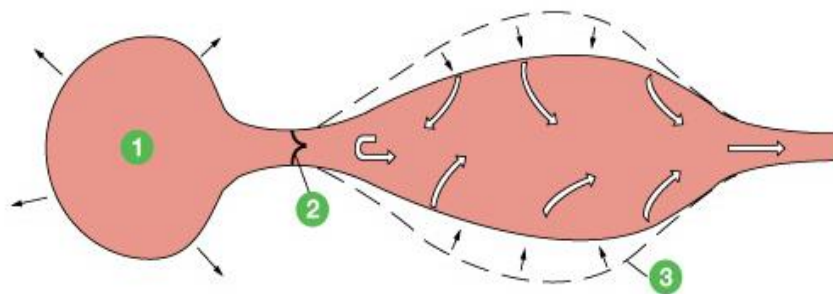
Pressure gradient:
aortic pressure – central venous pressure

(a) Ventricular contraction



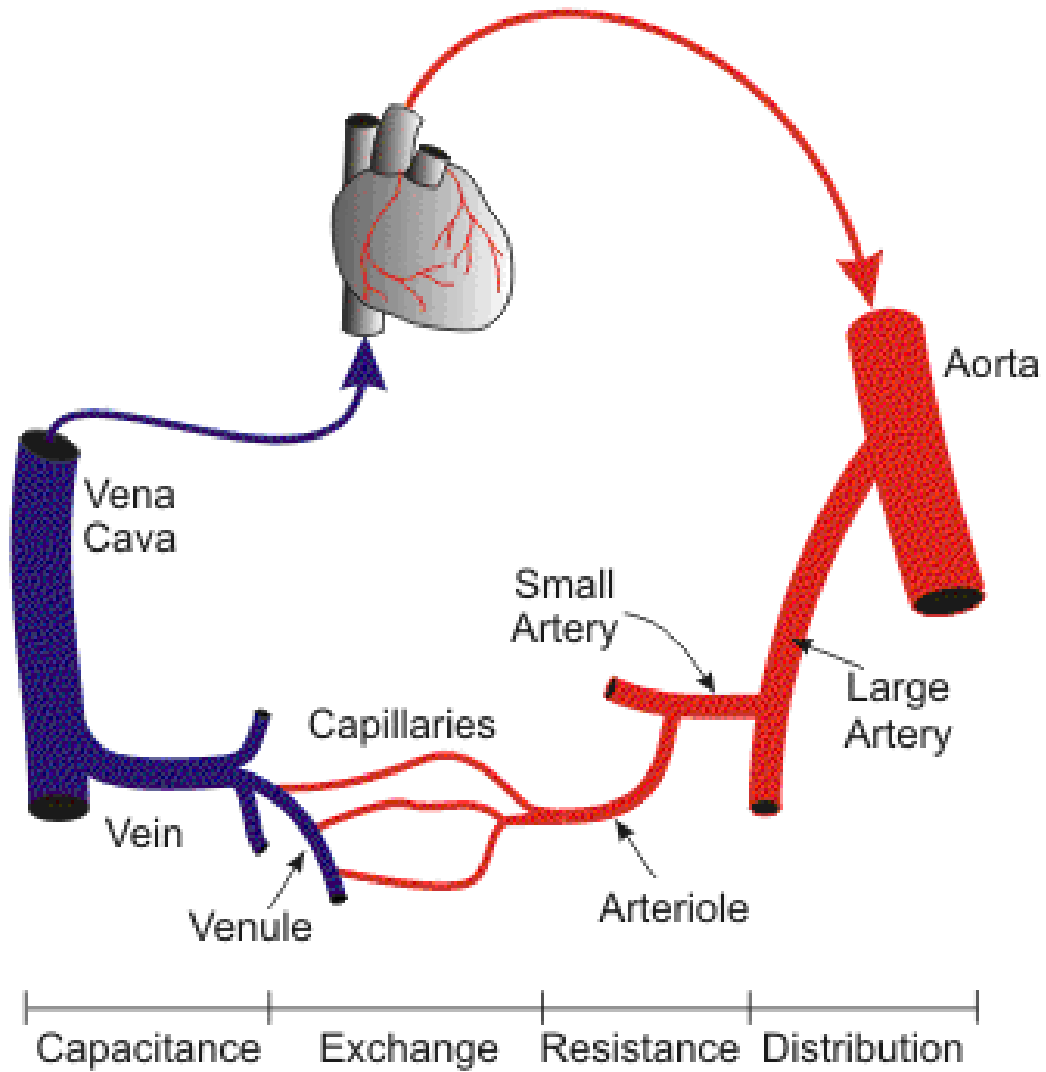
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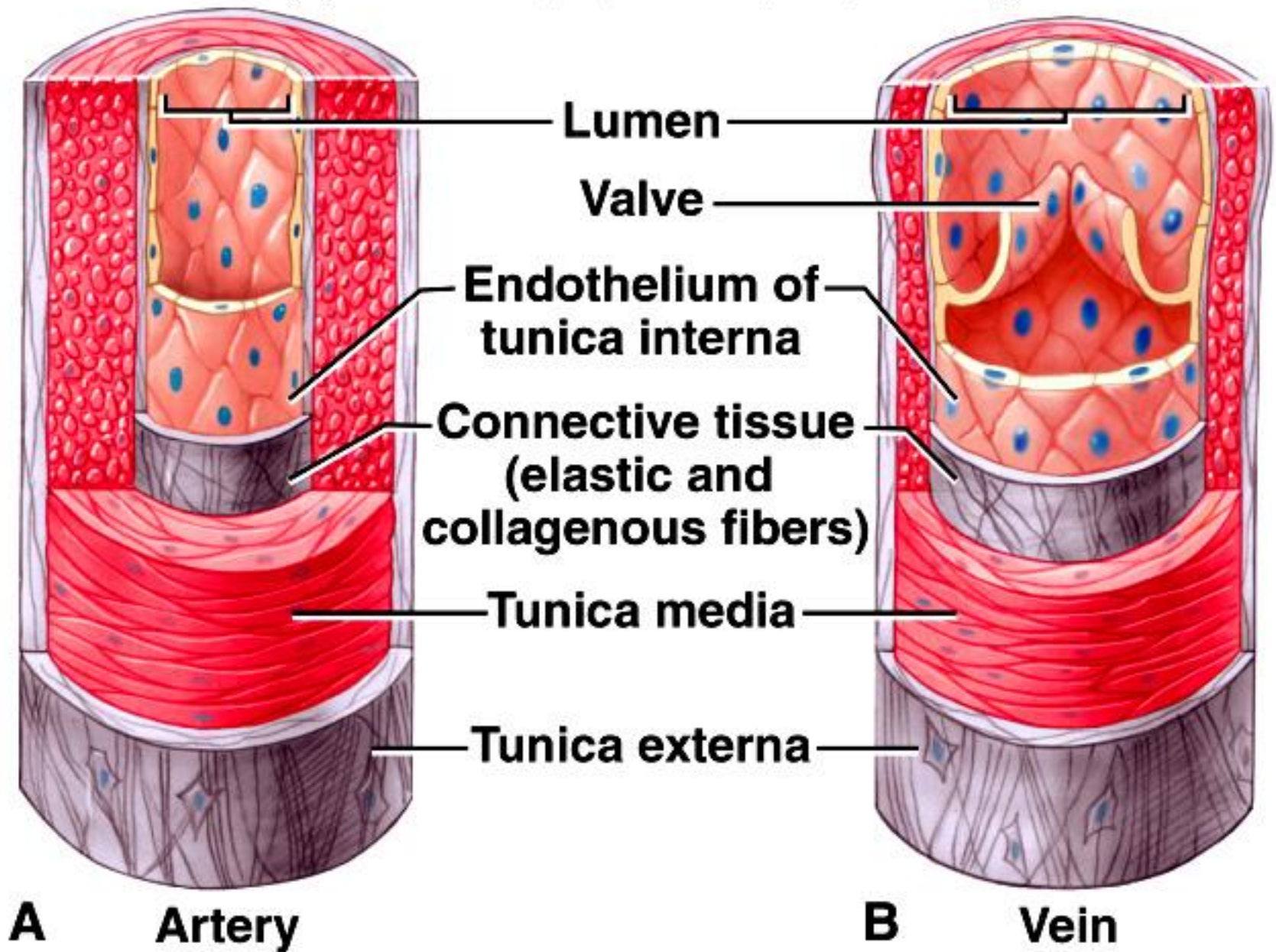
(b) Ventricular relaxation



- 1 Isovolumic ventricular relaxation
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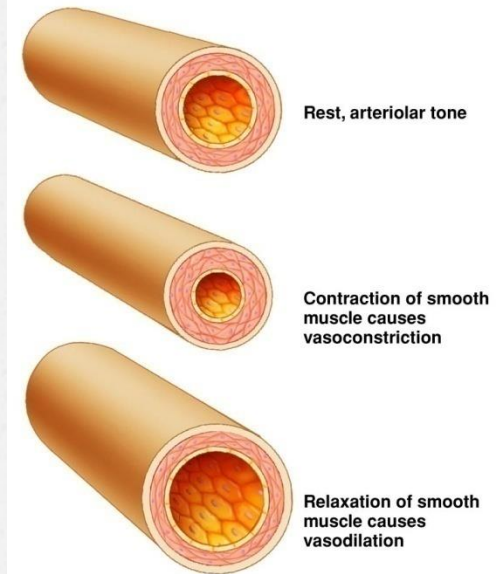
Figure 15-4: Elastic recoil in the arteries





Regulation of blood flow in arteries

- o It is important to adjust blood flow to organ needs → Flow of blood to particular organ can be regulated by varying resistance to flow (or blood vessel diameter)
- o **Vasoconstriction** of blood vessel smooth muscle is controlled both by the ANS and at the local level.
- o **Four factors** control arterial flow at the organ level:
 - change in metabolic activity
 - changes in blood flow
 - stretch of arterial smooth muscle
 - local chemical messengers



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Intrinsic control of local arterial blood flow

o Change in metabolic activity

- o Usually linked to CO_2 and O_2 levels ($\uparrow \text{CO}_2 \rightarrow$ vasodilation $\rightarrow \uparrow$ blood flow) intrinsic control

o Changes in blood flow

- decreased blood flow \rightarrow increased metabolic wastes \rightarrow vasodilation

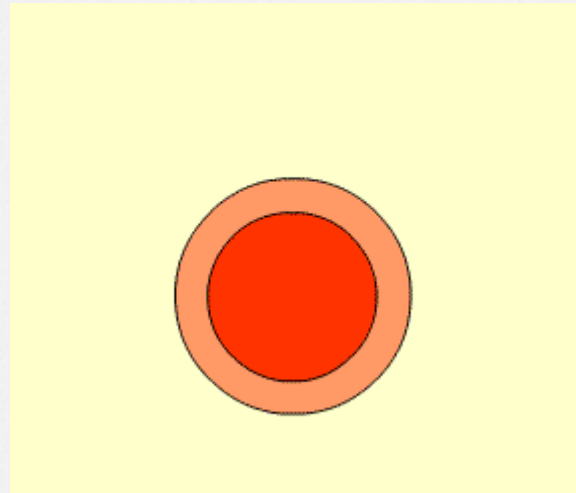
o Stretch of arterial wall = myogenic response

- Stretch of arterial wall due to increased pressure \rightarrow reflex constriction

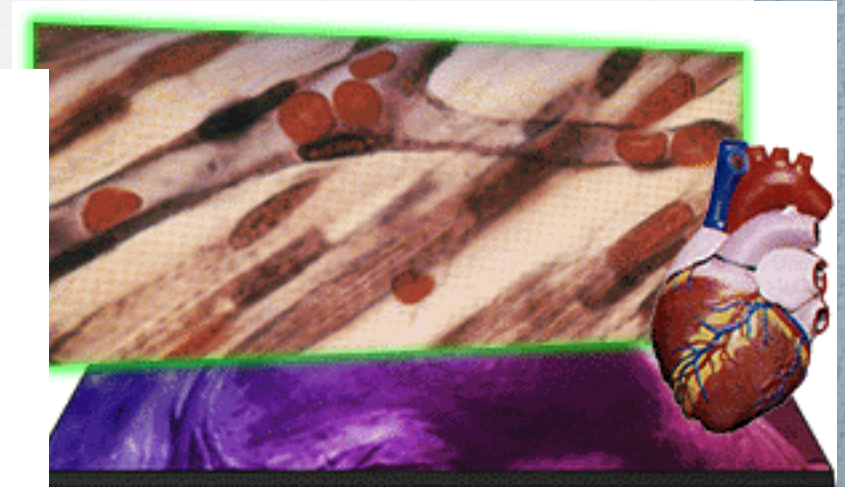
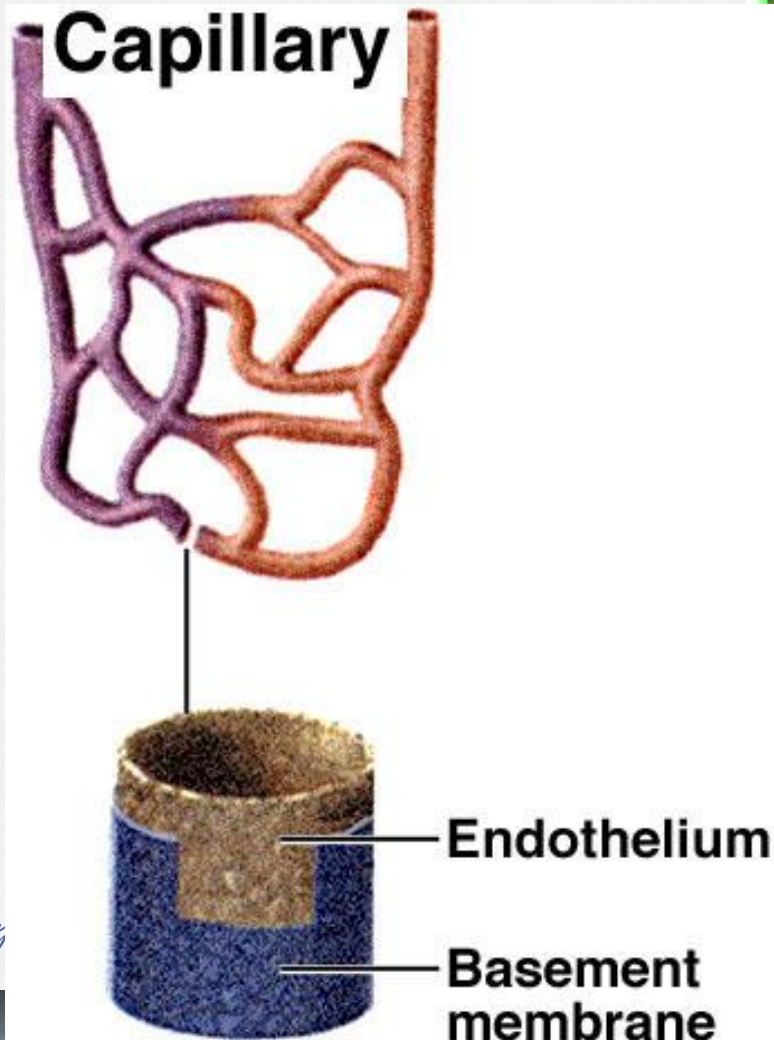
o Locally secreted chemicals can promote vasoconstriction or most commonly vasodilation

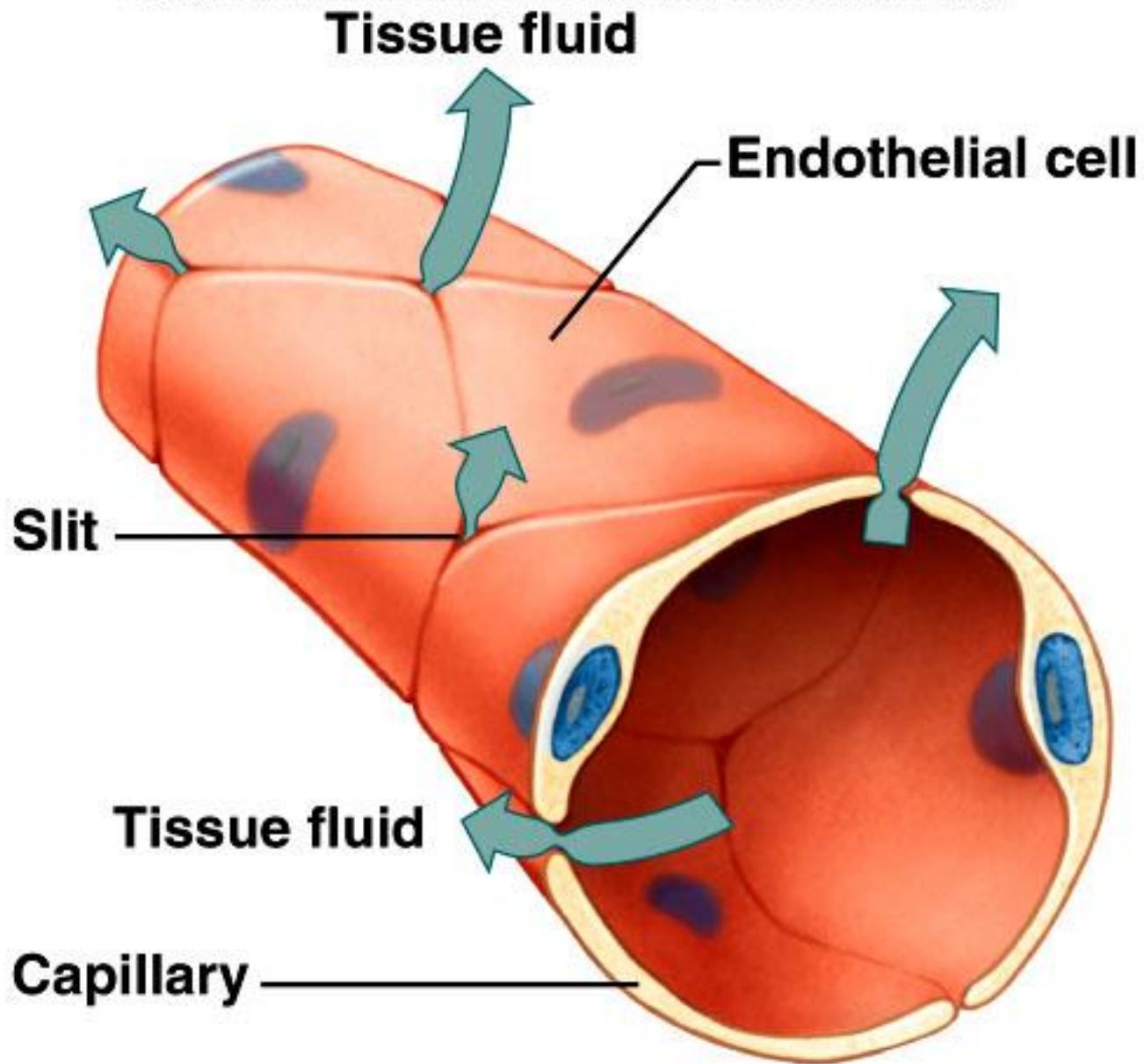
- inflammatory chemicals, (nitric oxide, CO_2)

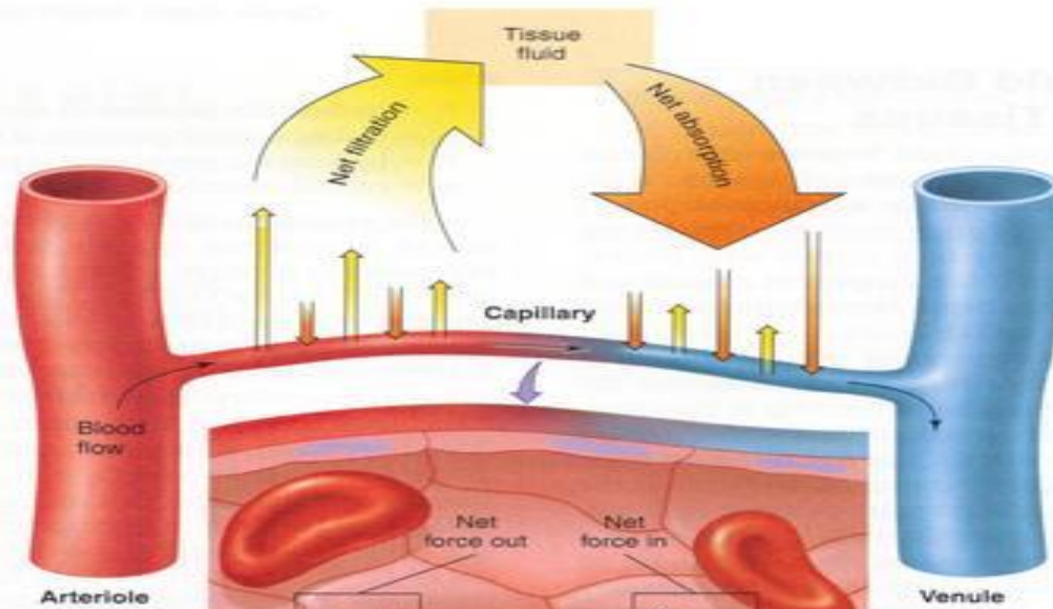
Artery in systole and diastole



Kapiler







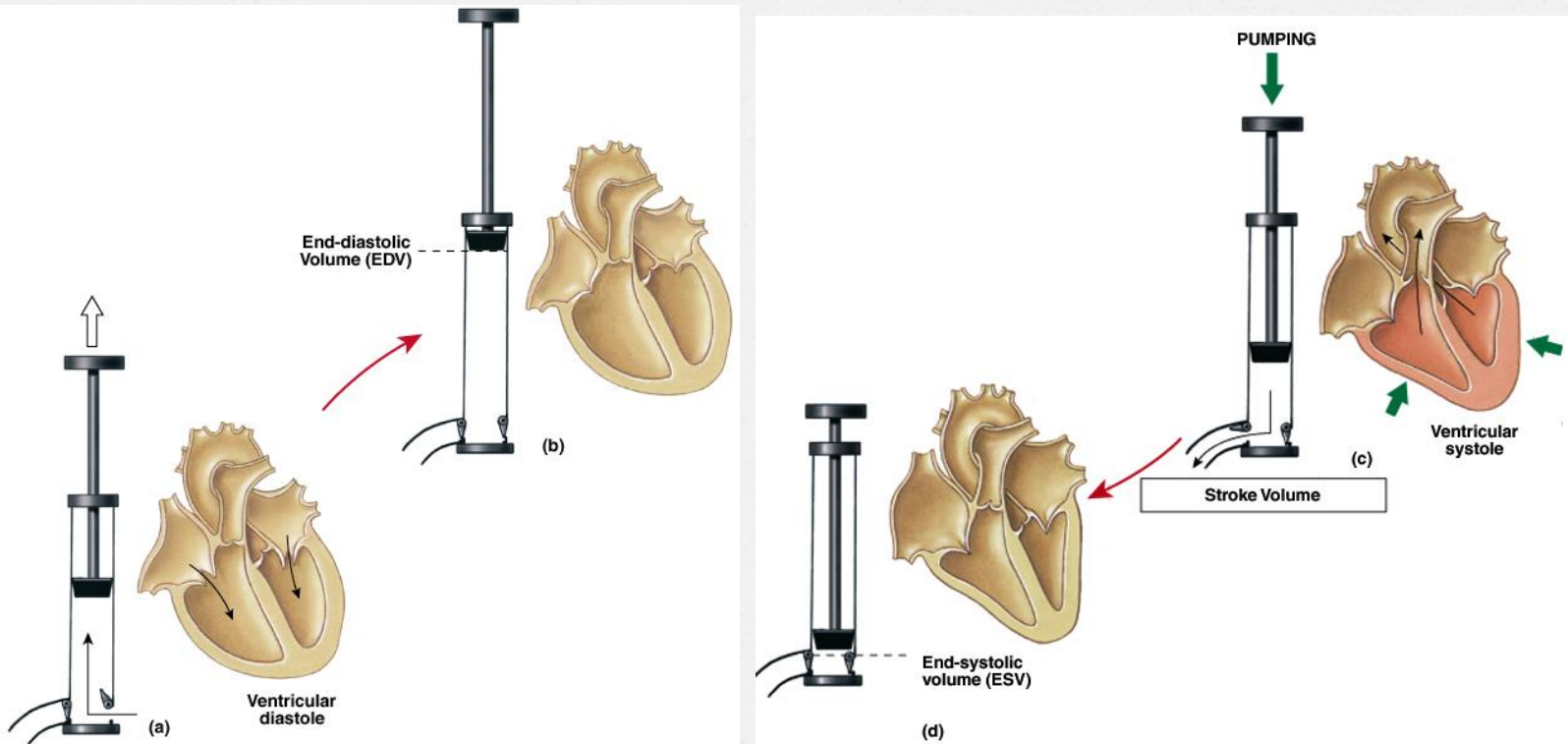
Arterial end of capillary	Venous end of capillary
$(P_c + \pi_i) - (P_i + \pi_p)$ (Fluid out) (Fluid in)	$(P_c + \pi_i) - (P_i + \pi_p)$ (Fluid out) (Fluid in)
$(37 + 0) - (1 + 25)$ $= 11 \text{ mmHg}$ Net filtration	$(17 + 0) - (1 + 25)$ $= -9 \text{ mmHg}$ Net absorption

Where P_c = hydrostatic pressure in the capillary
 π_i = colloid osmotic pressure of interstitial fluid
 P_i = hydrostatic pressure of interstitial fluid
 π_p = colloid osmotic pressure of blood plasma

VOLUME DARAH

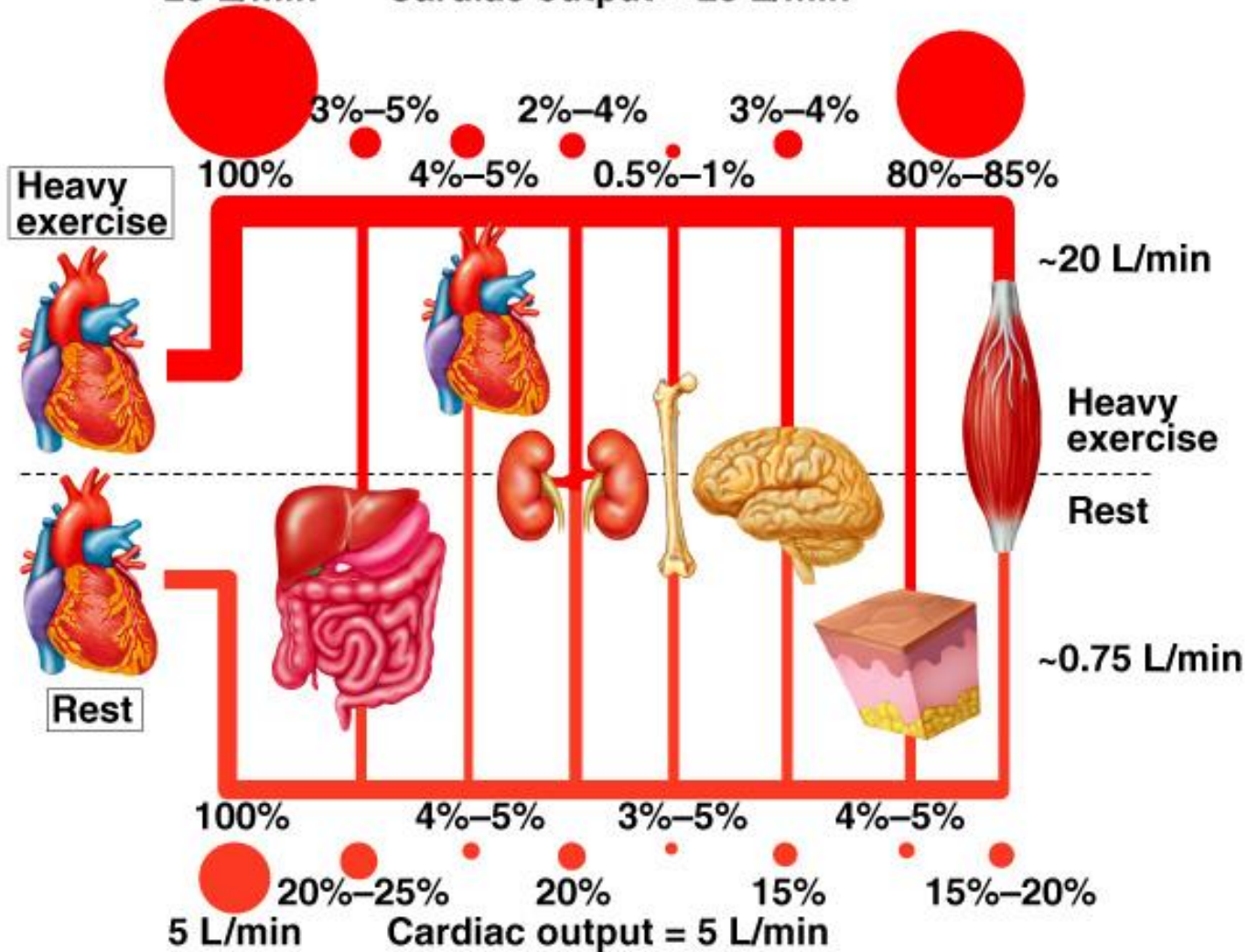
Stroke volume
End-diastolik volume
End-sistolik volume
Cardiac output
Ejection fraction

POMPA JANTUNG



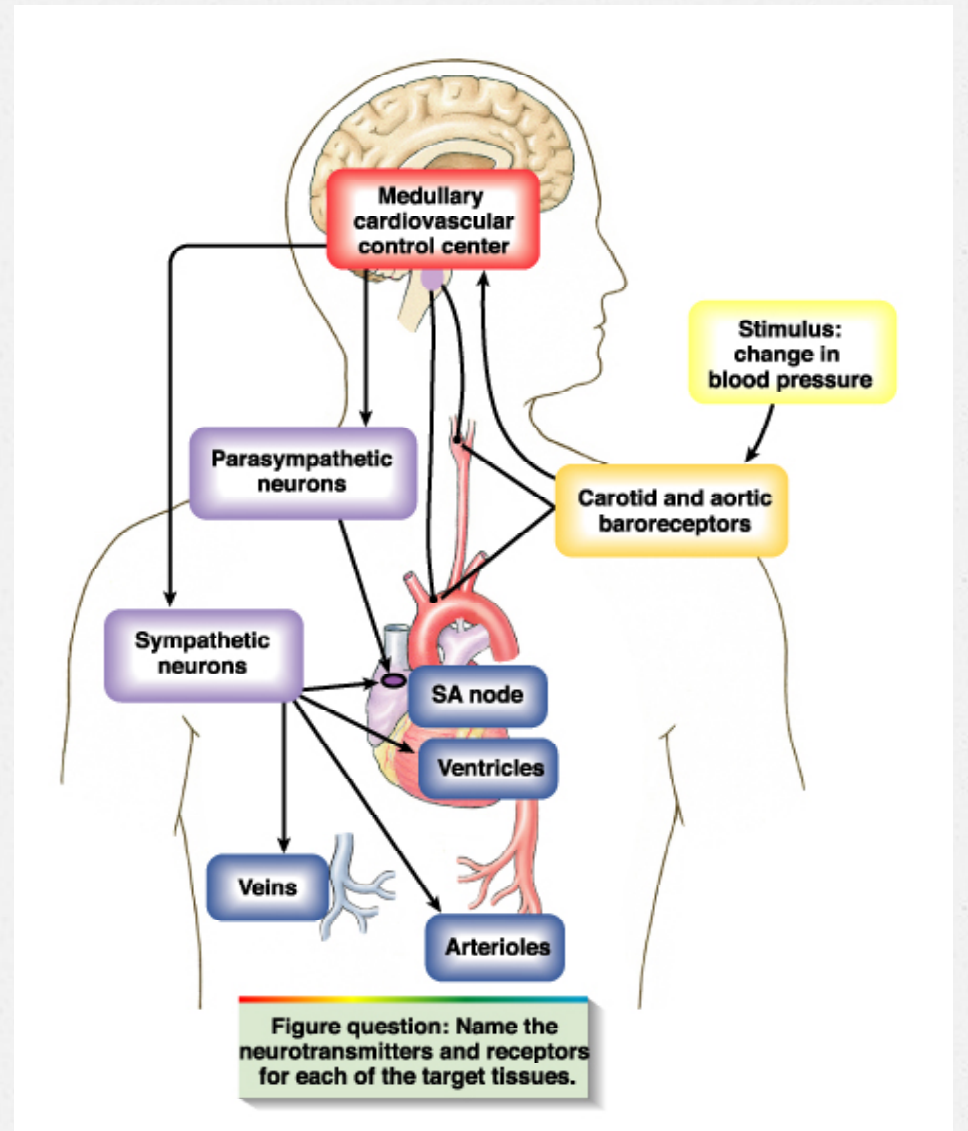
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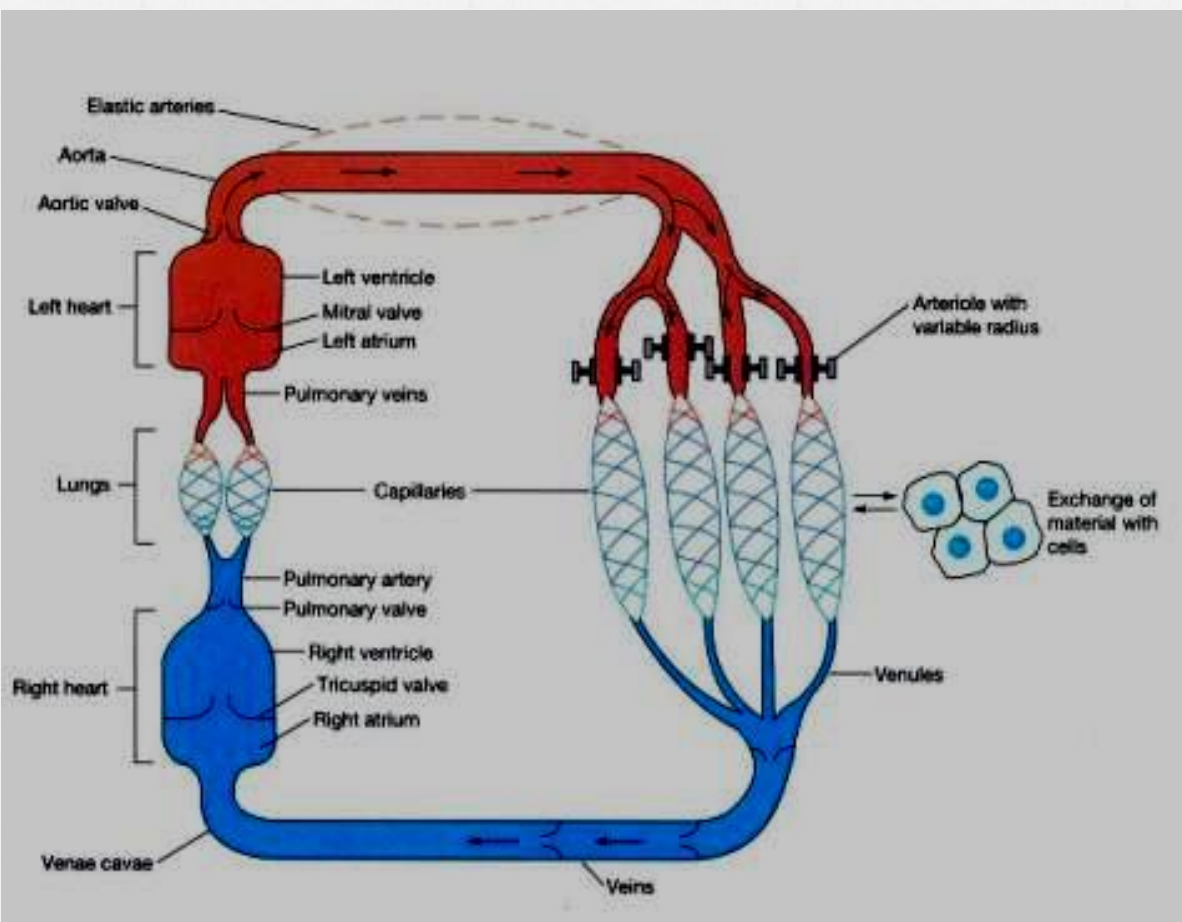
25 L/min Cardiac output = 25 L/min



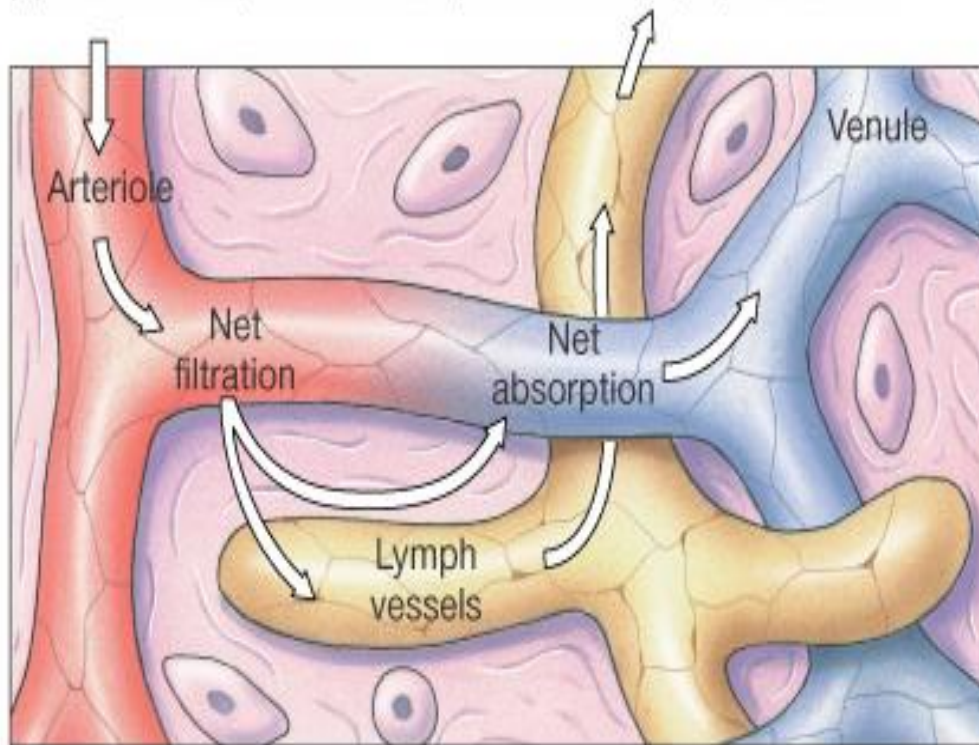
Regulation of Blood Pressure

- Main coordinating center is in the medulla oblongata of the brain; medullary cardiovascular control center
- Reflex control of blood pressure
 - Baroreceptor reflex





(b) Relationship between capillaries and lymph vessels



The excess water and solutes that filter out of the capillary are picked up by the lymph vessels and returned to the circulation.

HEMODINAMIKA

- Variabel-variabel fisik yang berhubungan dengan bagaimana darah mengalir
- Prinsip-prinsip dasar fluida cairan
- Komponen dinamika cairan dalam tabung
 - Energi :
 - tekanan atau beda tekanan antara 2 tempat
 - Gaya per satuan luas
 - Densitas cairan
 - Gravitasi
 - Panjang tabung

ALIRAN CAIRAN MELALUI TABUNG

1. ALIRAN LAMINAR

- o Sebagai arus yang paralel
- o Arus yg tepat berdekatan dgn dinding pembuluh stasioner, yg mendekati pusat kecepatan lbh tinggi, maksimum di pusat tabung
 - o Cairan **Newtonian** (mis. Air) berbentuk parabola dg ujung tajam,
 - o **Non-Newtonian** (mis. Darah) ujung lbh tumpul
 - o Sel-sel darah mengalir sejajar dgn arah aliran & bergerak ke tengah
 - o Memudahkan masuknya darah ke arteriola

ALIRAN CAIRAN MELALUI TABUNG

2. ALIRAN TURBULEN

- o Ketika kecepatan mencapai nilai kritis (Reynolds)
 - o Angka Reynolds (Re) = $(vD\rho)/\eta$
 - o V-kecepatan, D-diameter, ρ -densitas, η -viskositas
 - o Re mencapai $> 200-400$ terjadi turbulen saat di percabangan arteri kmd hilang ketika sdh lurus
 - o Re kritis aliran turbulen pd pembuluh darah lurus 2000
 - o Aliran ini menyebabkan kehilangan energi sbg bunyi & panas

ALIRAN CAIRAN MELALUI TABUNG

- o Keadaan2 yg menyebabkan aliran turbulen
 - o Kec aliran yg tinggi (aorta proximal dan a. pulmonaris)
 - o Pulsasi arteri
 - o Perubahan diameter mendadak
 - o Pembuluh darah dg diameter besar

3. ALIRAN *SINGLE-FILE*

- o Aliran dgn perubahan btk SDM (aliran berbentuk bolus)

PENGUKURAN ALIRAN DARAH (FLOWMETER)

o ELECTROMAGNETIC FLOWMETER

- o Tdk invasif
- o Dpt merekam perubahan aliran $<1/100$ per detik
- o Dpt merekam aliran pulsatil maupun stabil
- o Tradisional:
 - o Menempelkan elektroda di pembuluh darah yg dikelilingi oleh medan magnet kuat
 - o Kec aliran darah proporsional dg arus listrik yg dihasilkan antara 2 elektroda dan direkam dg voltmeter
- o Modern:
 - o Menggunakan probe yg ditempelkan disekeliling pembuluh

PENGUKURAN ALIRAN DARAH (FLOWMETER)

- o ULTRASONIC DOPPLER FLOWMETER
 - o Keuntungan sama dg elektromagnetik
 - o Menempelkan kristal piezoelektrik kecil di dinding pembuluh darah
 - o Ketika diberi aliran listrik akan memancarkan ultrasound dg frekuensi bbrp ratus ribu siklus/detik ke arah darah yg mengalir
 - o Bunyi akan dipantulkan oleh SDM yg mengalir dg frekuensi yg lbh rendah
 - o Kecepatan merupakan selisih frekuensi antara yg dipancarkan dg yg dipantulkan kembali

Viskositas

- o Pada Newtonian fluid viskositas tdk tergantung dimensi tabung dan kecepatan aliran ok memenuhi aliran laminar.
- o Tahanan intrinsik thd aliran disebabkan krn adanya gesekan antara lapisan-lapisan yg berdekatan yg akan menentukan viskositasnya

Viskositas

- Non-Newtonian Fluid (DARAH)
- Punya sel-sel dlm cairan, shg viskositas dan tahanan aliran tergantung :
 - Kecepatan aliran, hematokrit, diameter pembuluh darah kecil, sumbu SDM dan kemampuan SDM utk berubah bentuk
- Pengukuran dgn Viskometer Rotasional yg dpt menganalisis sifat-sifat rheologis darah

Viskositas

- Viskositas air pada 20 °C adalah 1mPascal.s & hanya ~0.69mPa.s pada 37 °C
- Viskositas plasma ~1.2mPa.s pada 37 °C (krn albumin & globulin)
- Viskositas darah keseluruhan didominasi oleh hematokrit (jika 47% adalah ~2.8mPa.s)
 - Jk hematokrit naik sampai 60 atau 70 (polisitemia) mk viskositas akan naik 10x lipat
- Saling bertabrakan antara SDM menyebabkan viskositas mkn tinggi.
 - Tergantung fleksibilitas SDM

Efek Fahraeus - Lindqvist

- o Jika darah dialirkan melalui tabung kaca, maka viskositas nyata akan konstan pada diameter tabung > 1 mm, jika diameter diturunkan hingga < 1 mm maka viskositas akan menurun
 - o Diduga krn perubahan orientasi SDM saat melewati diameter kecil tsb
 - o SDM bergerak dg axis panjang paralel thd arah aliran, terjadi migrasi axis, perubahan bentuk dan rotasi membran yg akan mengubah darah menjadi emulsi dan menurunkan viskositas

Formasi Rouleaux

- Jika kecepatan aliran rendah maka shear rate rendah akan menimbulkan kecenderungan SDm membentuk agregasi (rouleaux) shg kecepatan akan meningkat.
- Pembentukan rouleaux ini tergantung pd konsentrasi protein molekul besar di plasma khususnya fibrinogen

Deformabilitas SDM

- Viskositas darah berbanding terbalik dgn deformabilitas SDM
- Btk SDM adalah bikonkaf dgn diameter 7 mikron
- Jika masuk kapiler yg berdiameter lebih kecil (3-7 mikron) berubah menjadi seperti peluru / parasut
- Perubahan btk tergantung pd konsentrasi Ca^{2+} dlm plasma
 - Jk O_2 rendah, Ca^{2+} banyak masuk SDM, mejadi kaku shg viskositas naik

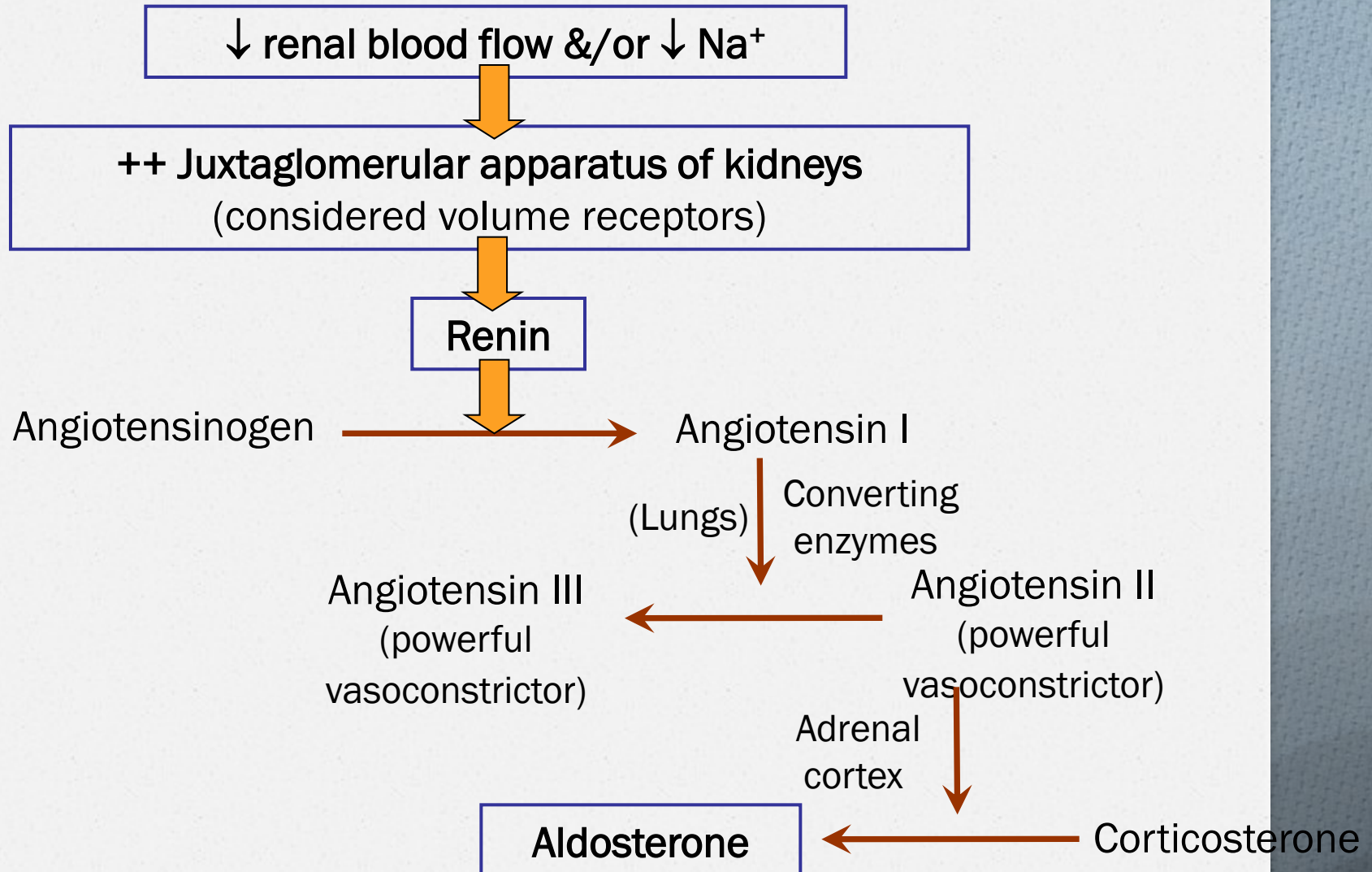
Regulation of Blood Volume:

- A long-term regulatory mechanism.
- Mainly renal:
 1. Renin-Angiotensin System.
 2. Anti-diuretic hormone (ADH), or vasopressin.
 3. Low-pressure volume receptors.

1. Renin-Angiotensin System:

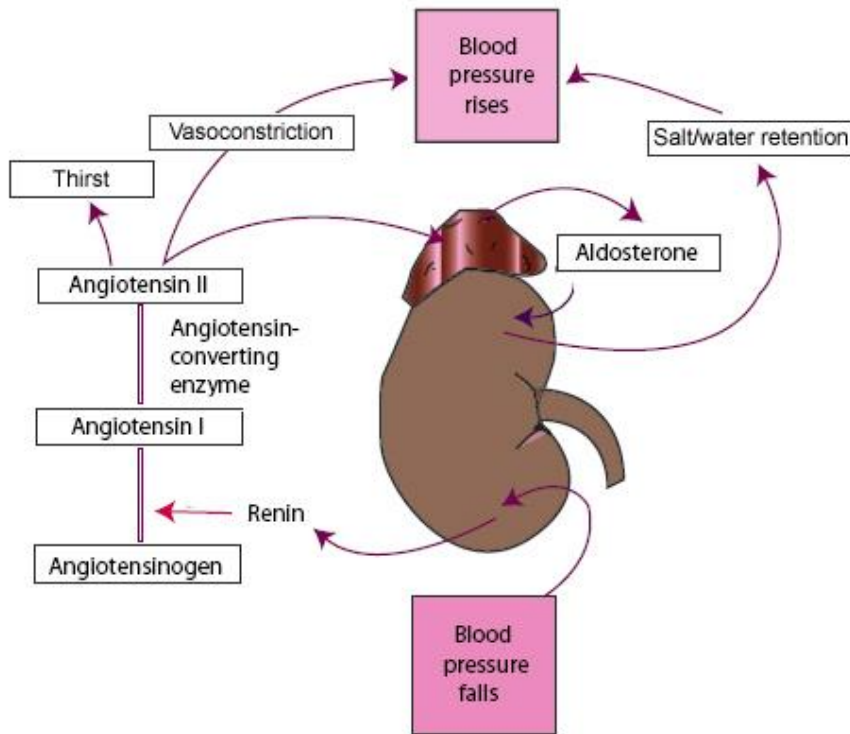
- Most important mechanism for Na^+ retention in order to maintain the blood volume.
- Any drop of renal blood flow &/or $\downarrow \text{Na}^+$, will stimulate volume receptors found in juxtaglomerular apparatus of the kidneys to secrete **Renin** which will act on the **Angiotensin System** leading to production of **aldosterone**.

• Renin-Angiotensin System:



□ N.B. Aldosterone is the main regulator of Na^+ retention.

Control of blood volume



Aldosterone:

Secretion by the adrenal cortex triggered by angiotensin II

Promotes sodium reabsorption by the kidney tubules (Na^+ moves back into the blood)

H_2O follows by osmosis

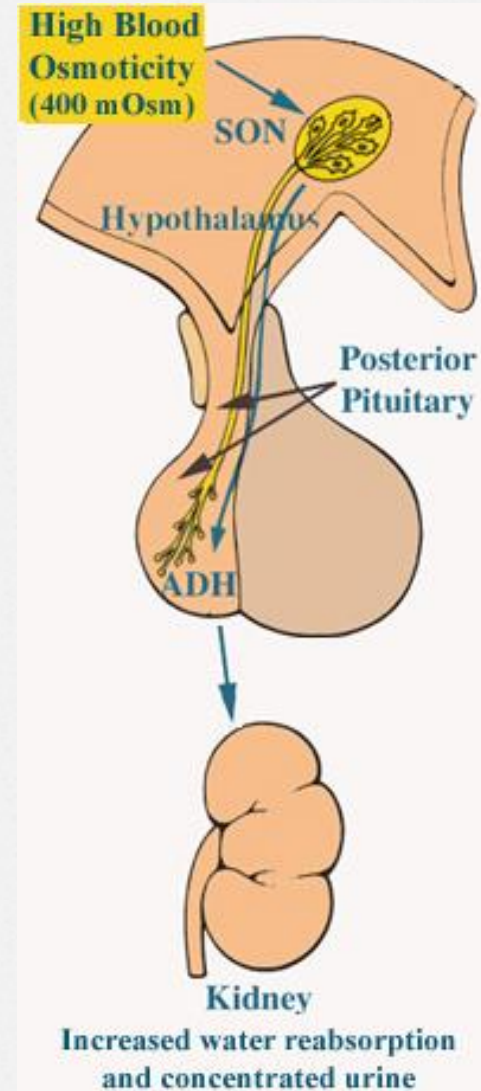
Whereas ADH promotes H_2O reabsorption only (in response to dehydration), aldosterone promotes reabsorption of both H_2O and salt (in response to \downarrow BP)

2. Anti-diuretic hormone (ADH), or vasopressin:

- Hypovolemia & dehydration will stimulate the osmoreceptors in the hypothalamus, which will lead to release of ADH from posterior pituitary gland.
- ADH will cause water reabsorption at kidney tubules.

Control of blood volume

- Anti-diuretic hormone = ADH
- Secreted by the posterior pituitary in response to \uparrow blood osmolarity (often due to dehydration)
- Promote water reabsorption by the kidney tubules \rightarrow H₂O moves back into the blood \rightarrow less urine formed

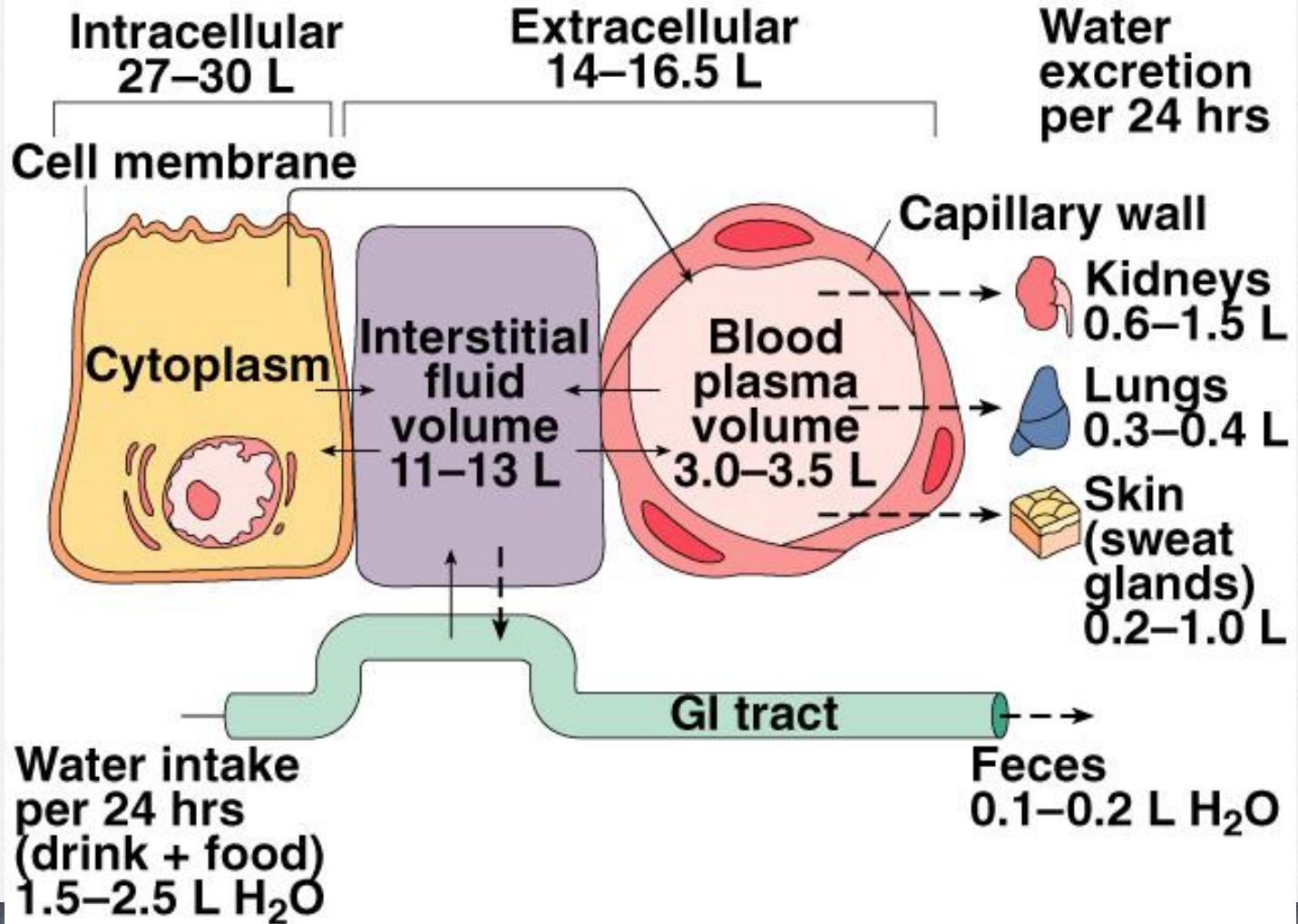


3. Low-pressure volume receptors:

- Atrial natriuretic peptide (ANP) hormone, is secreted from the wall of right atrium to regulate Na^+ excretion in order to maintain blood volume.

Blood Volume (continued)

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MAP

TPR

CO

HR

SV

ANS

Hormones

Chemicals

Viscosity

Blood vessel length

Blood vessel diameter

Local factors

ANS

Hormones

Lytes

Body temp

Brain

ED

V

Venous Return

Kidney

Angiotensin

Aldosterone

ADH

Respiratory pump

Skeletal muscle pump

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