Cardiovascular Physiology II.

- Hemodynamics: the functional categorization of bloos vessels

- The function of the aorta and arteries

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41th LO: Hemodynamics: the functional categorization of blood vessels

Explain the concept of transmural pressure of blood vessels.
Explain the concept of vascular compliance, give the formula for its determination ($C=\Delta V/\Delta P$).
Which two main factors determine vascular compliance (starting volume and distensibility)?
Characterize the different vascular segments based on their compliance.
Explain the concept of critical closing pressure.
Describe the relationship among wall tension, transmural pressure, vessel radius and wall thickness using the equation of Laplace's law. Based on the relationship, in which vessel segment is the rupture of the vessel due to high wall tension most likely?
Give the definitions of hydraulic resistance and conductance. Explain the effects of adding resistance in series vs. in parallel on total resistance.
Characterize the contribution of arteries, arterioles, capillaries, venules, and veins to peripheral vascular resistance. Contrast blood pressure, cross sectional area, flow velocity, and blood volume in these vascular segments.

Blood vessels: elastic and branching tubes

- According to the Hagen-Poiseuille's law length and especially radius of vessels affect hydraulic resistance greatly. In contrast to the criteria of the law, the circulatory system consists of elastic, branching and not always cylindrical tubes.
- Blood flow at a given pressure gradient is determined by the resistance. Total peripheral resistance (TPR) is determined by vessels connected to each other in series or in parallel in a complex way.
- Because of vessel elasticity, incrases in distending blood pressure result also in increases in vessel radius (reducing resistance), cross sectional area and volume.
- Important concepts: transmural pressure, critical closing pressure, vascular compliance, wall tension (Laplace's law)

Ohm's law

$$Q = \Delta P / R$$

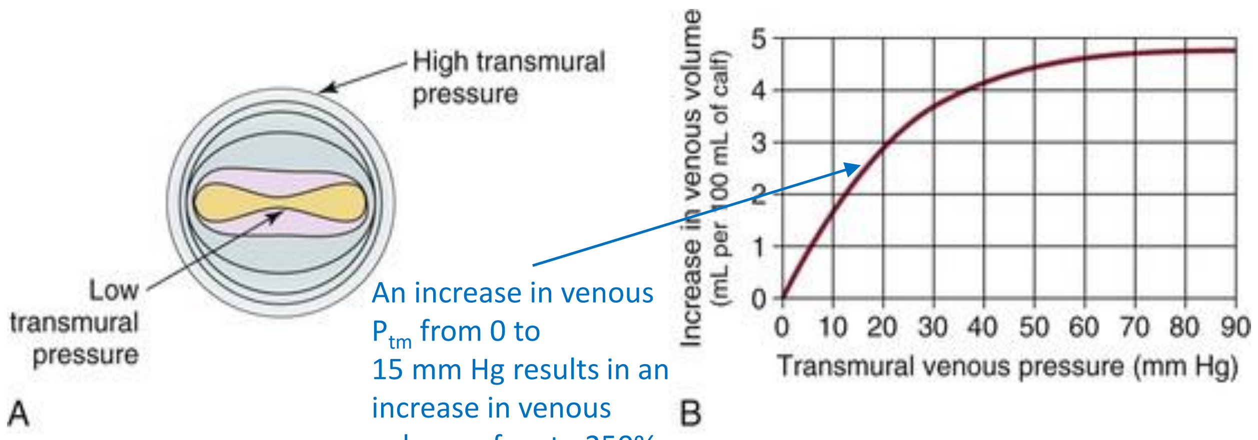
Hagen-Poiseuille's law

$$Q = \Delta P \frac{r^4 \pi}{L\eta 8} \qquad R = \frac{L\eta 8}{r^4 \pi}$$

Transmural pressure: the pressure distending the blood vessel

- „transmural”: across the wall
- The difference between the blood pressure and the pressure outside the vessel wall (interstitial pressure)
- The interstitial pressure normally doesn’ t play a significant role in determining transmural pressure in the systemic arteries (except in contracting muscle), but very important in the low pressure system (venous circulation)

$$P_{tm} = P_{blood} – P_{inst.}$$

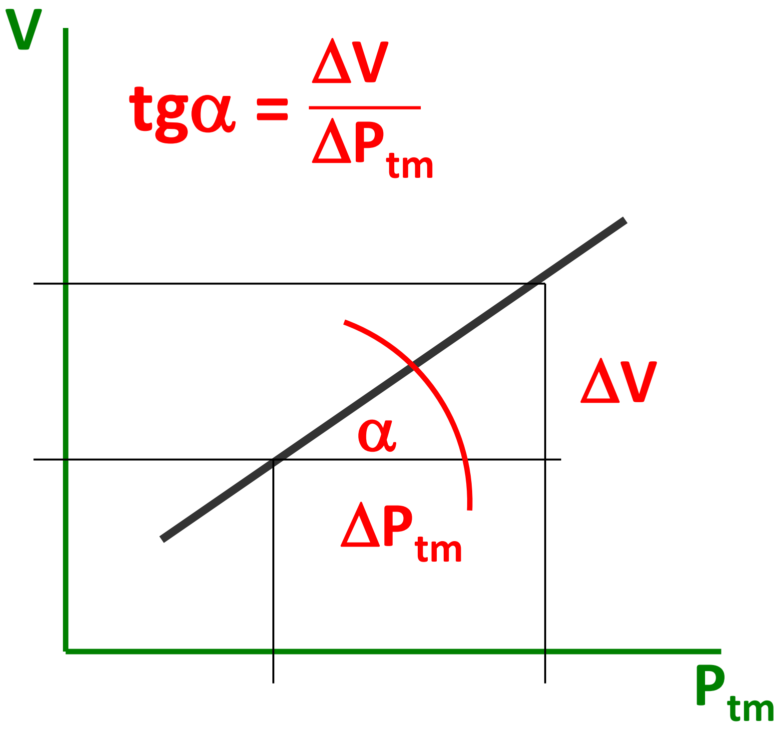


An increase in venous P_{tm} from 0 to 15 mm Hg results in an increase in venous volume of up to 250%. This corresponds to a change in shape from elliptical to circular.

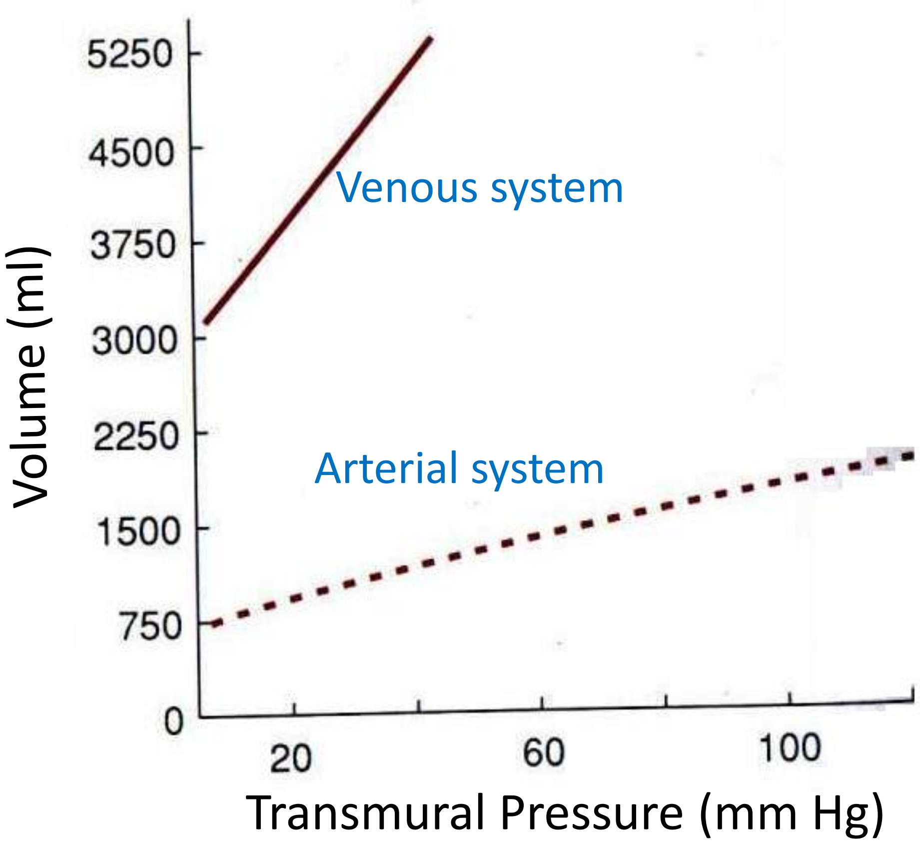
Once the vein is circular, much higher pressure is required to stretch the venous wall to add additional volume. At arterial pressures, veins become as stiff as arteries

Vascular Compliance: increase in vessel volume in response to unit increase in pressure

Compliance: the slope of the vessel volume and transmural pressure curve

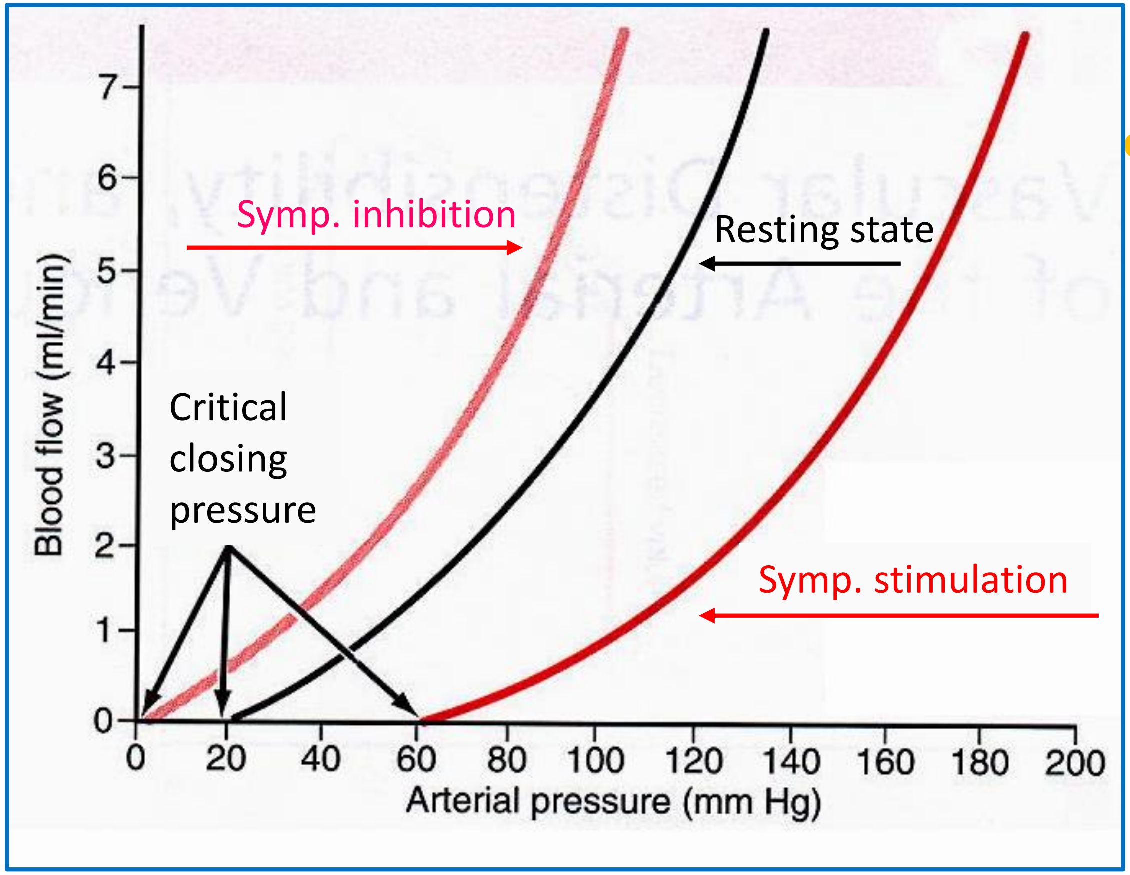


Depends on vessel distensibility and vessel volume



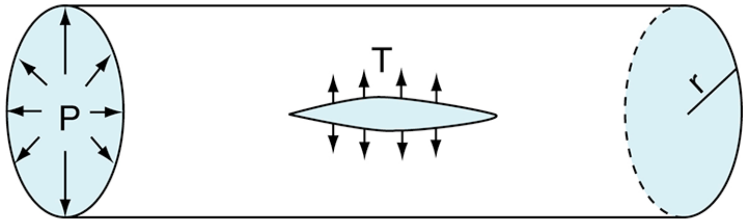
Venous Compliance is 20-24 times larger than arterial Compliance!

Blood vessels collapse, if the transmural pressure falls below the critical closing pressure value



Critical closing pressure in arteries is higher than the mean vascular filling pressure that develops after death (~7 mmHg). Therefore, arteries collapse after death, then fill up with air once the dissection begins. This misled scientists for a thousand years believing the arteries to transport air (reflected in their greek name, arteria means windpipe)

Laplace’s law



Wall Tension = Transmural Pressure x $\frac{\text{radius}}{\text{wall thickness}}$; $T = P_{tm} \times \frac{r}{h}$

Tension of vessels: the tangential force in response to the distending pressure. Wall tension is a force that would tear the vessel wall

Laplace’s law helps to identify which vessels are at risk of wall rupture

- veins – low (large radius, low blood pressure)
- capillaries – low (small radius, low blood pressure)
- arterioles – low (small radius, high pressure + thick wall)
- muscular arteries – low (moderate radius + thick wall, high pressure)
- aorta/ large elastic arteries – **high** (large radius + relatively thin wall, hihg pressure)

High blood pressure induced wall rupture is thus most likely in the aorta, wall weakening causes aneurysm formation that sets off a vicious circle.

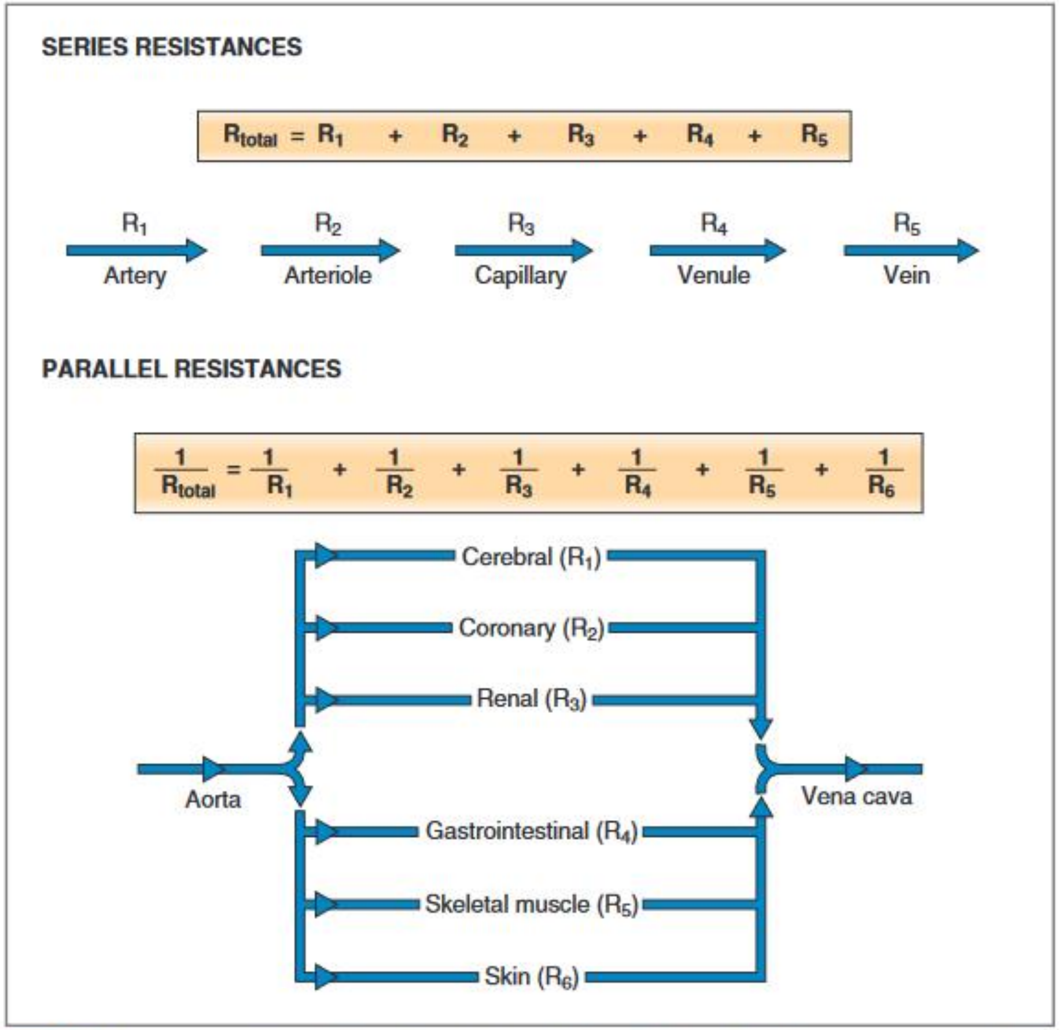


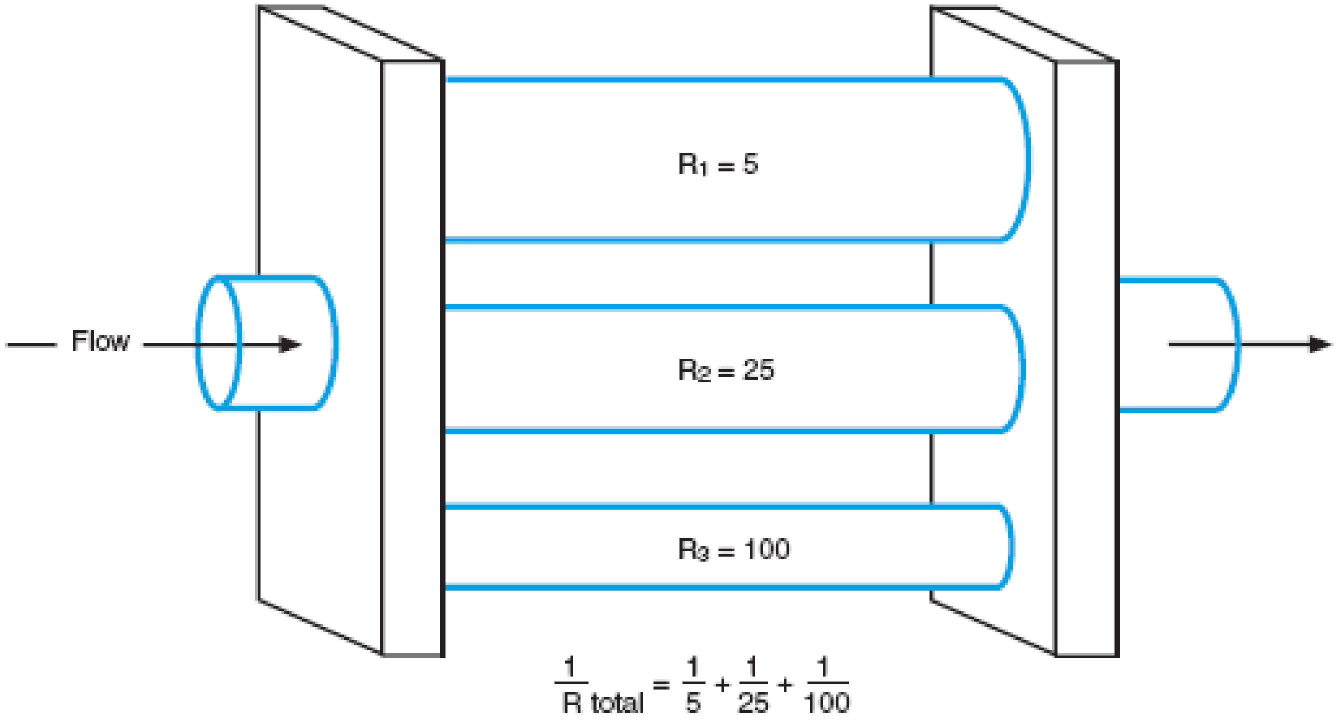
Figure 4–5 Arrangements of blood vessels in series and in parallel. The *arrows* show the direction of blood flow. R, Resistance (subscripts refer to individual resistances).

How can the vascular resistance be described in the branching vasculature?

- Vessels are connected to each other either in series or in parallel
- The different organs in the systemic circulation are connected in parallel
- The vessels of the SAME class are connected in parallel (arteries, arterioles, capillaries, veins etc.)
- Vessel classes are connected to each other in series (arteries to arterioles, arterioles to capillaries, capillaries to venules etc.)

Compound resistance in parallel connected tubes

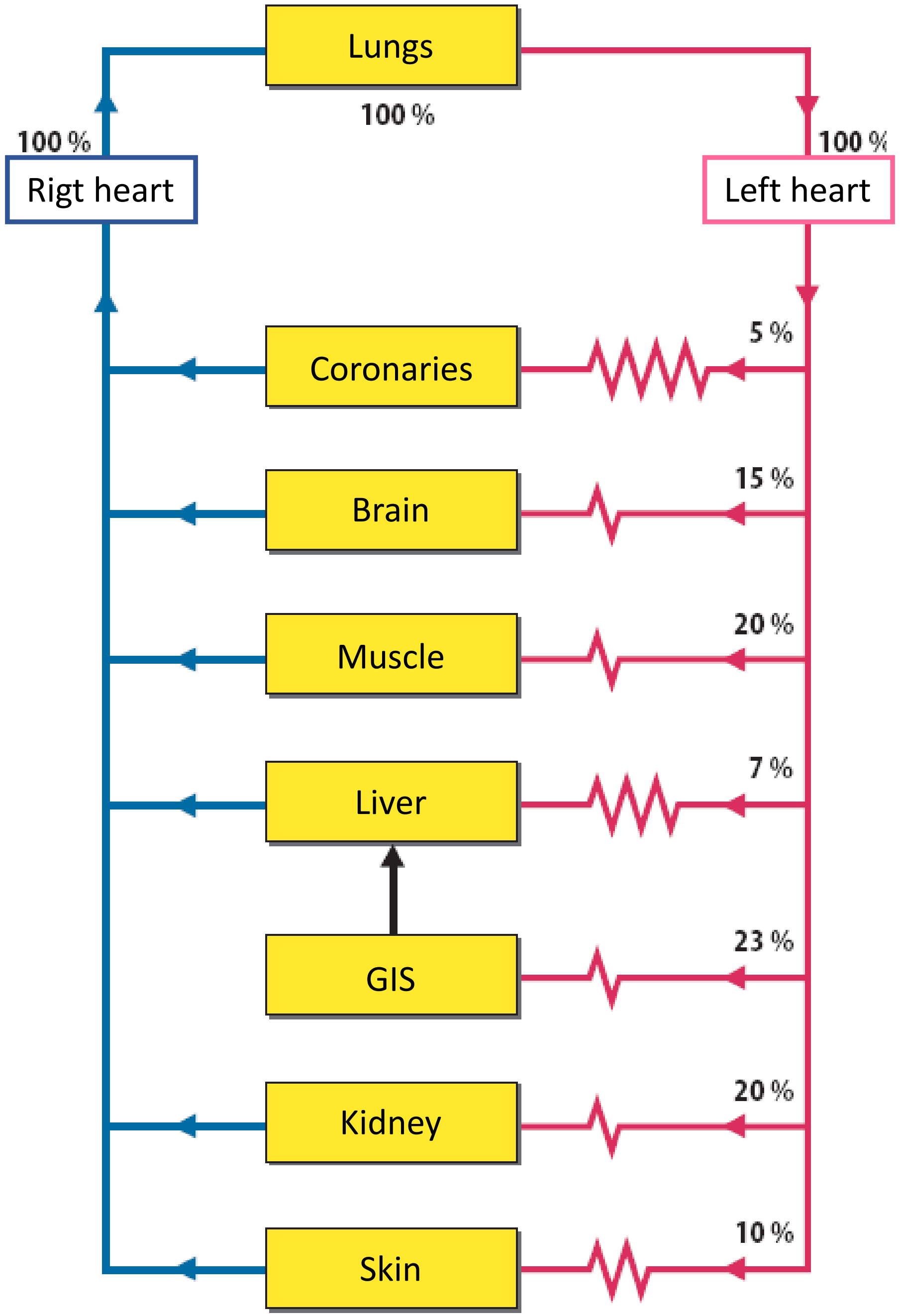
Note that total resistance is always smaller than the smallest individual resistance!



$$\frac{1}{R_t} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3}$$

$$\frac{1}{R_t} = \frac{1}{5} + \frac{1}{25} + \frac{1}{100} = \frac{25}{100} = \frac{1}{4}$$

$$R_t = 4\ \text{RU}$$



The organs of the systemic circulation are connected in parallel

- To describe these systems it is easier to use conductance instead of resistance
- $C = 1/R$, $C_{total} = 1/TPR$
- $C_{total} = C_{coronaries} + C_{brain} + C_{muscle} + \ldots$
- The figure shows the % of total peripheral conductance values in the systemic circulation

For instance, coronary circulation has 5% of total conductance that means that its resistance

$$R_{coronaries} = 1/C_{coronaries} = 1 / 0.05\ C_{total} = 20\ TPR$$

The TPR is smaller than any of the organ resistances in the systemic circulation

Compound resistance of parallel connected vascular segments

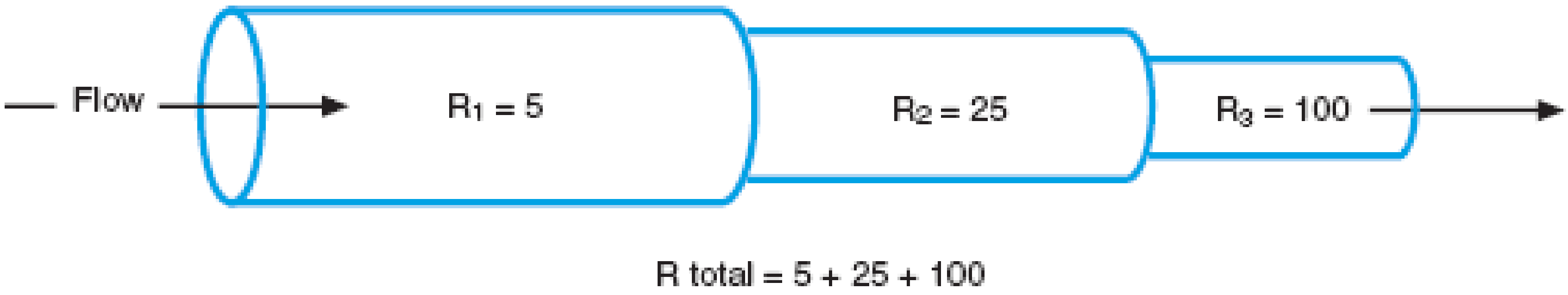
$$R_{segment} = \frac{1}{\left(\frac{1}{R1} + \frac{1}{R2} + \frac{1}{R3} + \quad \dots \quad + \frac{1}{Rn}\right)}$$

or

R = 1/C !!!

$$C_{segment} = C1 + C2 + C3 + C4 + \dots . \; Cn$$

Compound vascular resistance in each segment is increasing by larger R (individual resistance), and by smaller n (the number of parallel connected vessels). Perhaps it is easier to see that segmental **conductance** is smaller if individual conductances are smaller and the number of parallel connected vessels is smaller. Based on these principles the class of arterioles possess the greatest resistance (large individual R, relatively small n values)

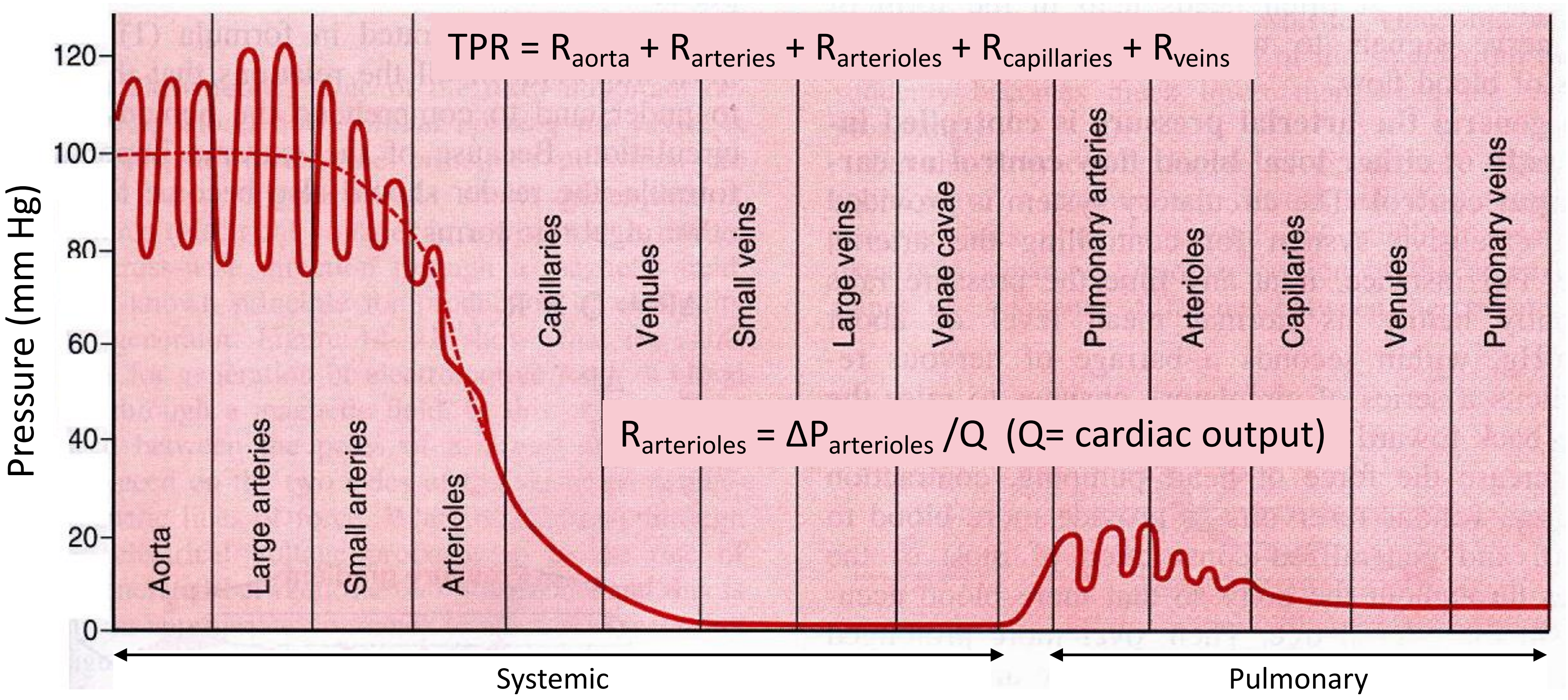


Combined resistance in series connection

- $R_{total} = R_1 + R_2 + R_3 + \ldots R_n$
- $TPR = R_{aorta} + R_{arteries} + R_{arterioles} + R_{capillaries} + R_{veins}$

Note that the total resistance is always greater than the largest resistance!

Blood pressure/ segmental resistance in the circulation

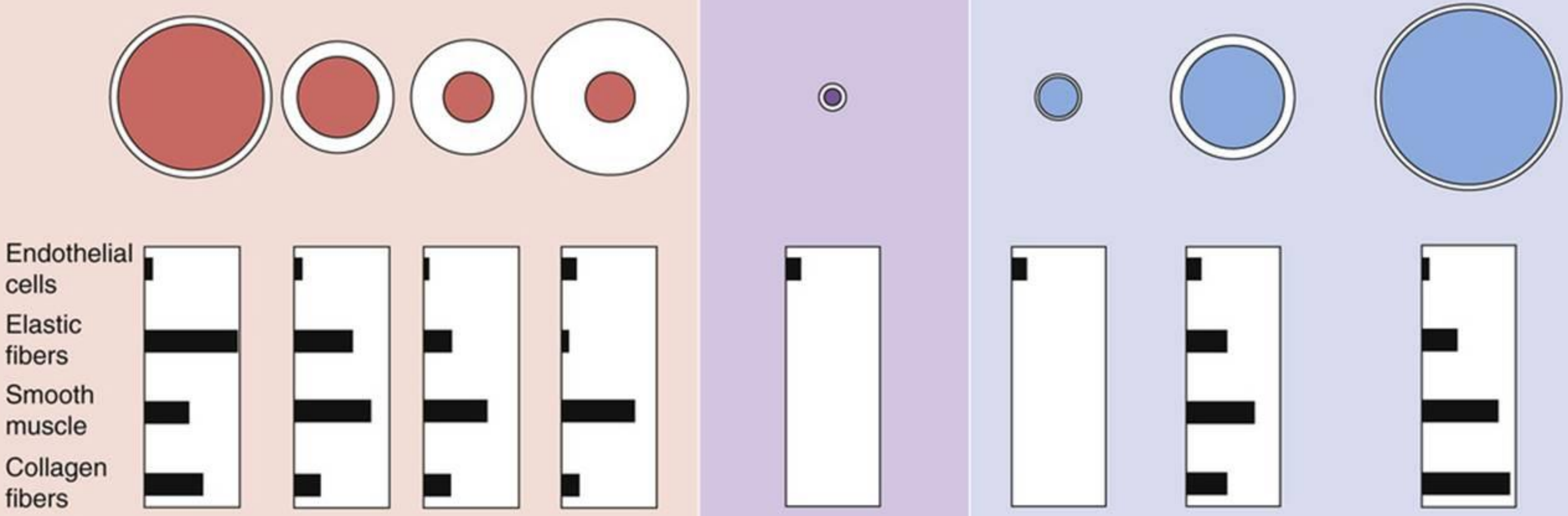


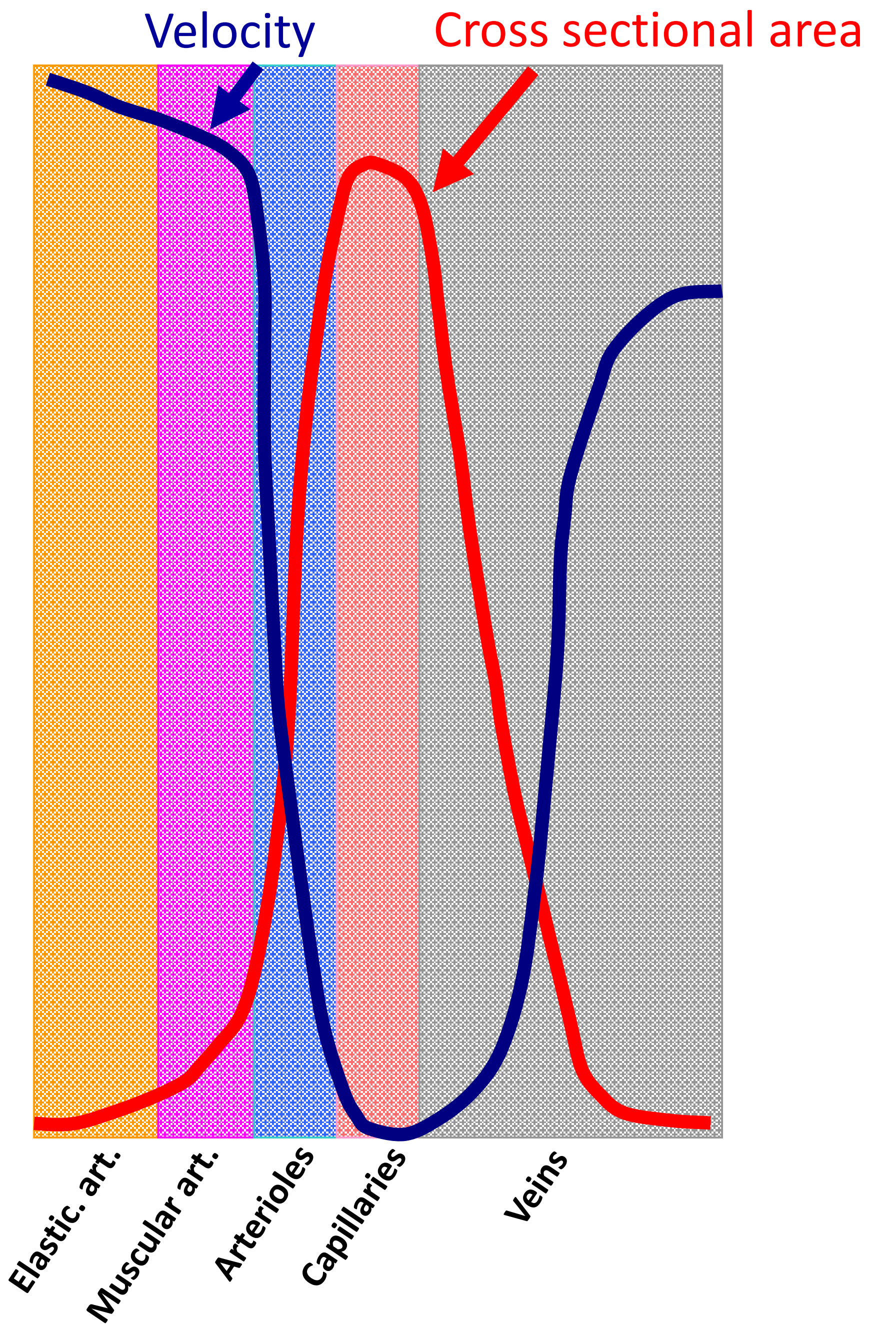
The total flow through each level of the system is the same (Q = constant)

- $R_{arterioles} >> R_{capillaries} > R_{veins} > R_{arteries} > R_{aorta}$
- Blood pressure drop is greatest in the arterioles because this segment has the largest vascular resistance, essentially determining the total peripheral resistance (TPR)
- Control of arterial blood pressure and local blood flow is taking place in the arterioles

Classes of vessels: structure and function

	Aorta	Medium artery	Arteriole	Precapillary sphincter	True capillary	Venule	Vein	Vena cava
Internal radius:	12 mm	2 mm	15 μm	15 μm	3 μm	10 μm	2.5 mm	15 mm
Wall thickness:	2 mm	1 mm	20 μm	30 μm	1 μm	2 μm	0.5 mm	1.5 mm
Function	Windkessel function (continuous flow)	Distribution	Resistance, blood pressure and local flow control		Exchange	Protein and cell trafic	Venous return control, cadiac output determination	

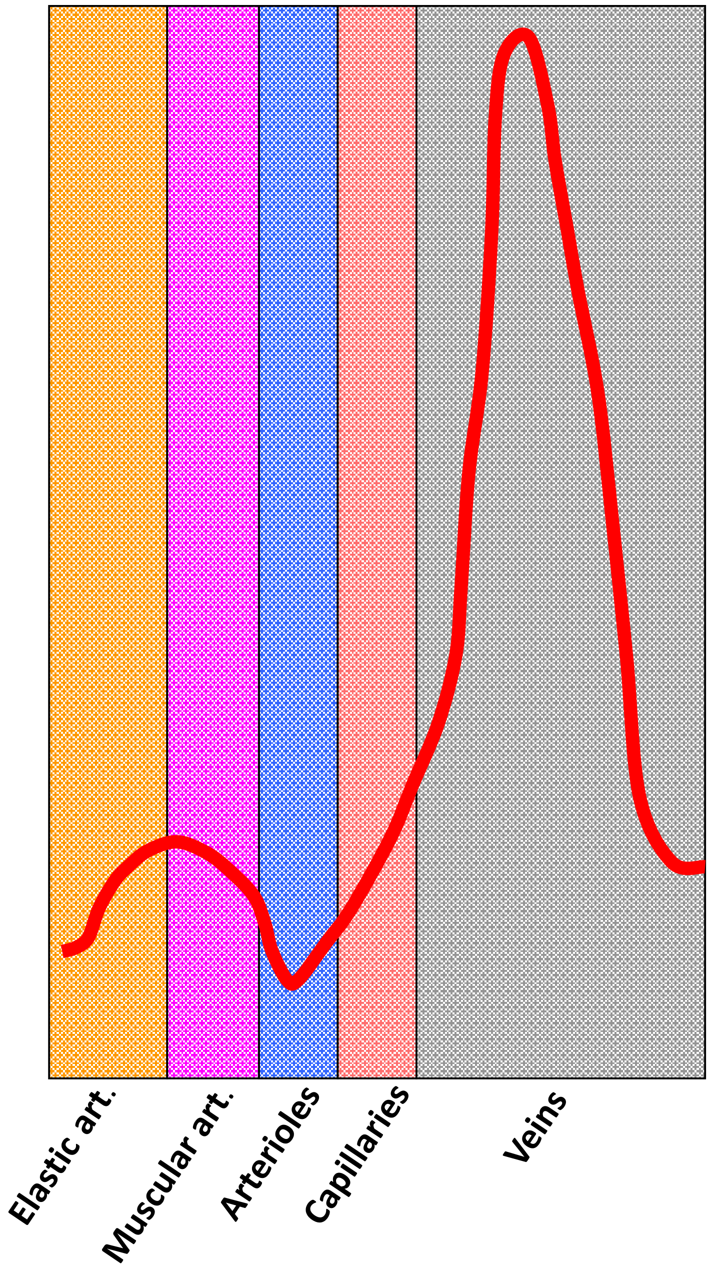




Cross sectional area and velocity of blood flow

Q = A x v

	cm²	cm/s
Aorta	4	22.5
Arteries	20	
Arterioles	40	
Capillaries	3000	0.03
Venules	250	
Veins	80	
Vena cavae	8	11



Distribution of blood volume

Depends on pressure and compliance, 2/3 of the total blood volume is in the venous system

	%
Heart	7
Aorta	6
Arteries	6
Arterioles	2
Capillaries	6
Veins	64
Pulmonary circ.	9

42nd LO: The function of the aorta and arteries

Describe the invasive and non-invasive methods of arterial blood pressure determinations (catheter + pressure transducer, and sphygmomanometry).

Draw the blood pressure curve of the aorta. Give the definitions and reference values of arterial systolic, diastolic, mean, and pulse pressures.

Describe the Windkessel function of the aorta.

Describe the effects of changes in a) stroke volume, b) arterial compliance, and c) total peripheral resistance, on arterial systolic, diastolic, mean, and pulse pressure values.

Describe the propagation of the pressure pulse, the changes in the shape of the pressure waveform from the aorta to the peripheral arteries. Contrast the pressure pulse with the flow pulse.

Describe the arterial pulse qualities assessed with palpation..


EXERCITATIO
ANATOMICA DE
MOTV CORDIS ET SAN-
GVINIS IN ANIMALI-
BVS,
GVILIELMI HARVEI ANGLI,
Medici Regii, & Professoris Anatomiæ in Col-
legio Medicorum Londinensi.

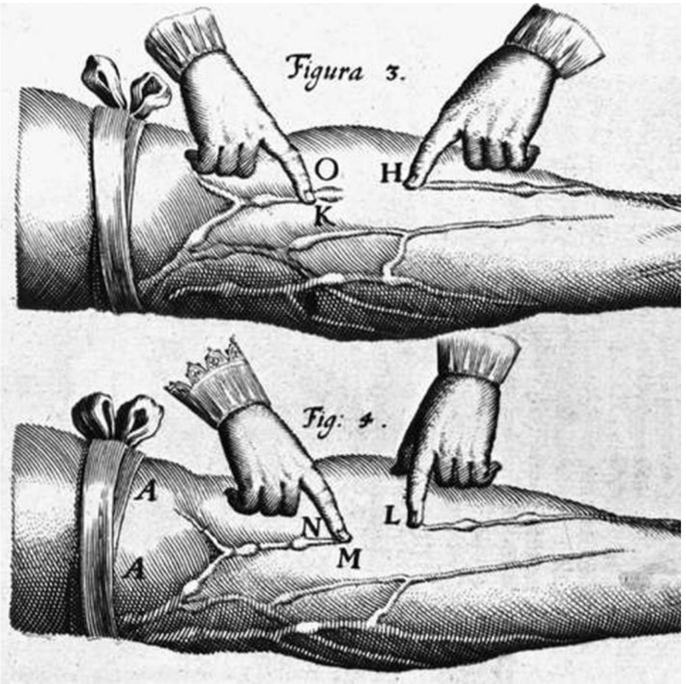
FRANCOFVRTI,
Sumptibus GVILIELMI FITZERI.
ANNO M. DC. XXVIII.

Harvey's book on the circulation is the beginning of modern medicine

Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus (Frankfurt, 1628)

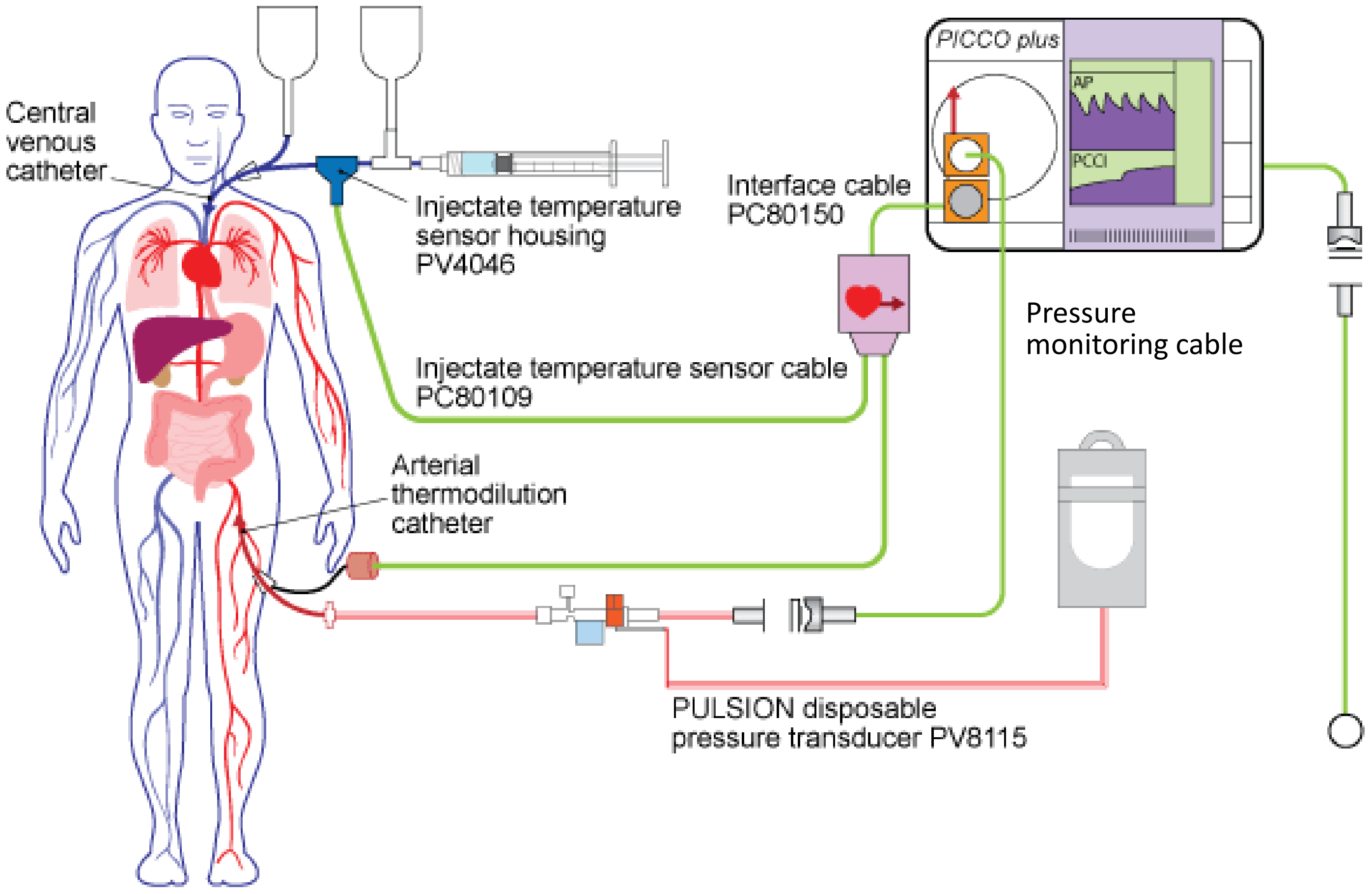
„Primum sese contrahit auricula, et in illa contractione sanguinem contentum in ventriculum cordis conjicit, quo repleto cor sese erigit, continuo omnes nervos tendit, contrahit ventriculos, et pulsum facit, quo pulsu immissum ab auricula sanguinem continenter protrudit in arterias: dexter ventriculus in pulmones, sinister ventriculus in aortam, et per arterias in universum corpus."

„The atrium contracts first, and with that contraction it pushes the contained blood into the heart's ventricle. When the ventricle is filled, the heart bulges, immediately flexes all its muscles, contracts the ventricles and make a pulse, by which pulse it pushes the blood received from the atrium directly into the arteries: the right ventricle towards the lung, the left ventricle into the aorta, and through the arteries into the whole body."



William Harvey (1578 – 1657)

The invasive method of blood pressure determination

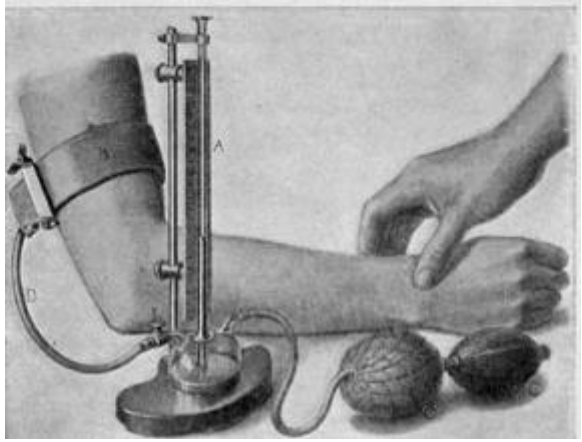


Blood pressure can be measured directly with a needle-tipped cannula inserted into the artery, which can be connected to a pressure gauge to continuously monitor the arterial pressure.

This method is used in intensive care and during surgeries.

The non-invasive method of blood pressure determination

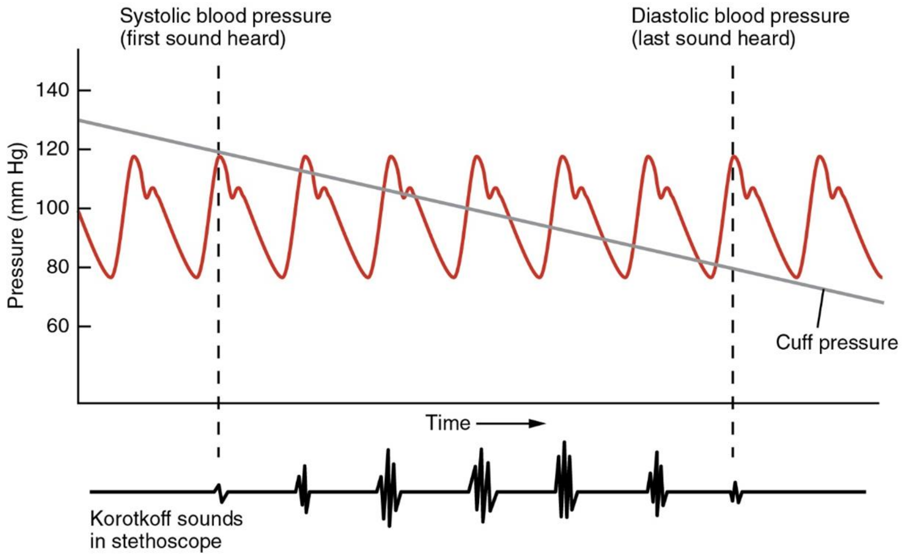

Scipione Riva-Rocci
(1863 -1937)

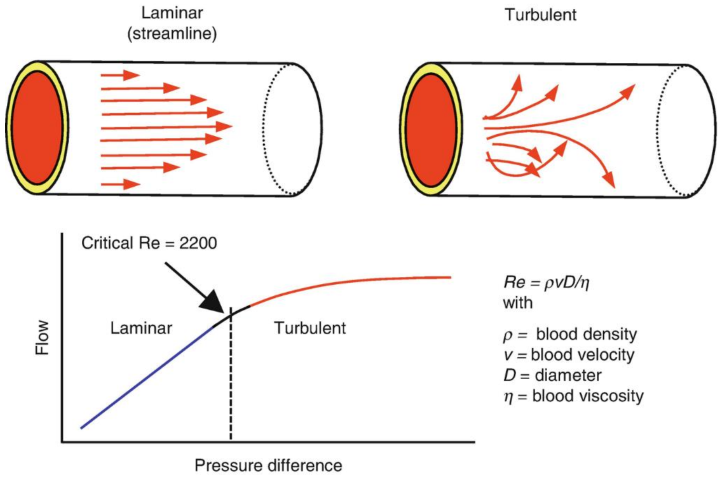

Riva-Rocci sphygmomanometer

- *Scipione Riva-Rocci* invented the mercury sphygmomanometer in 1896
- The inflatable cuff was only 5 cm wide
- He checked the return of the pulse by palpation

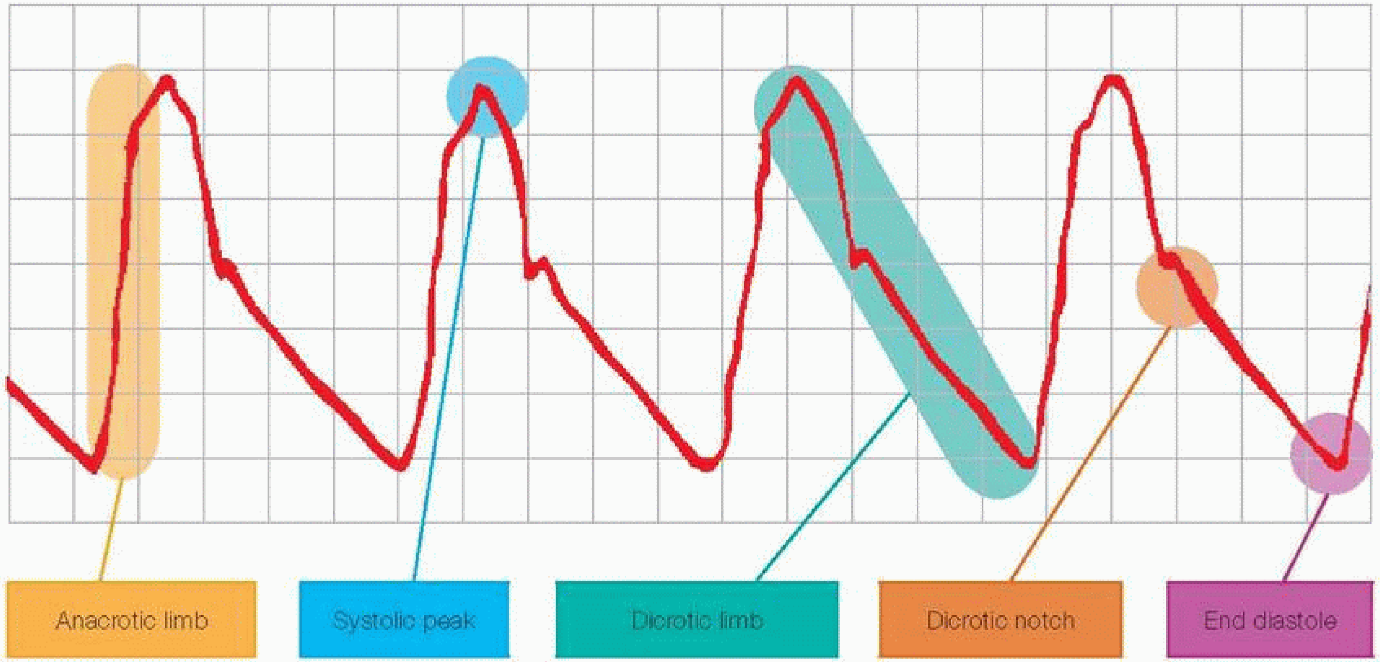
- *Nikolai Sergeyevich Korotkov* improved Riva-Rocci's method in1905
- Wider cuff
- He checked the return of the pulse by auscultation (Korotkov sounds)
- Estimation of systolic and diastolic pressure


Nikolai Sergeyevich Korotkov
(1874 -1920)





Normal arterial waveform



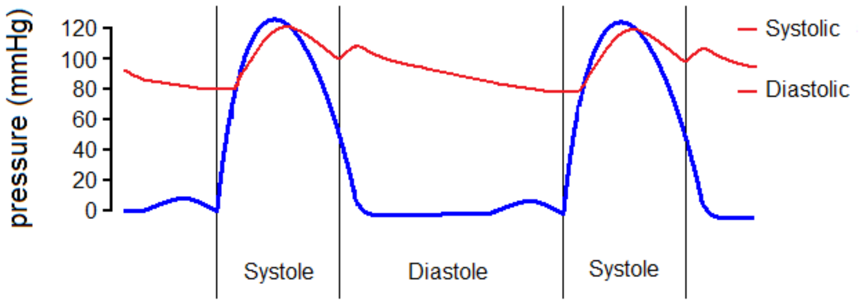
The *anacrotic limb* marks the waveform's initial upstroke, which occurs as blood is rapidly ejected from the ventricle through the open aortic valve into the aorta.

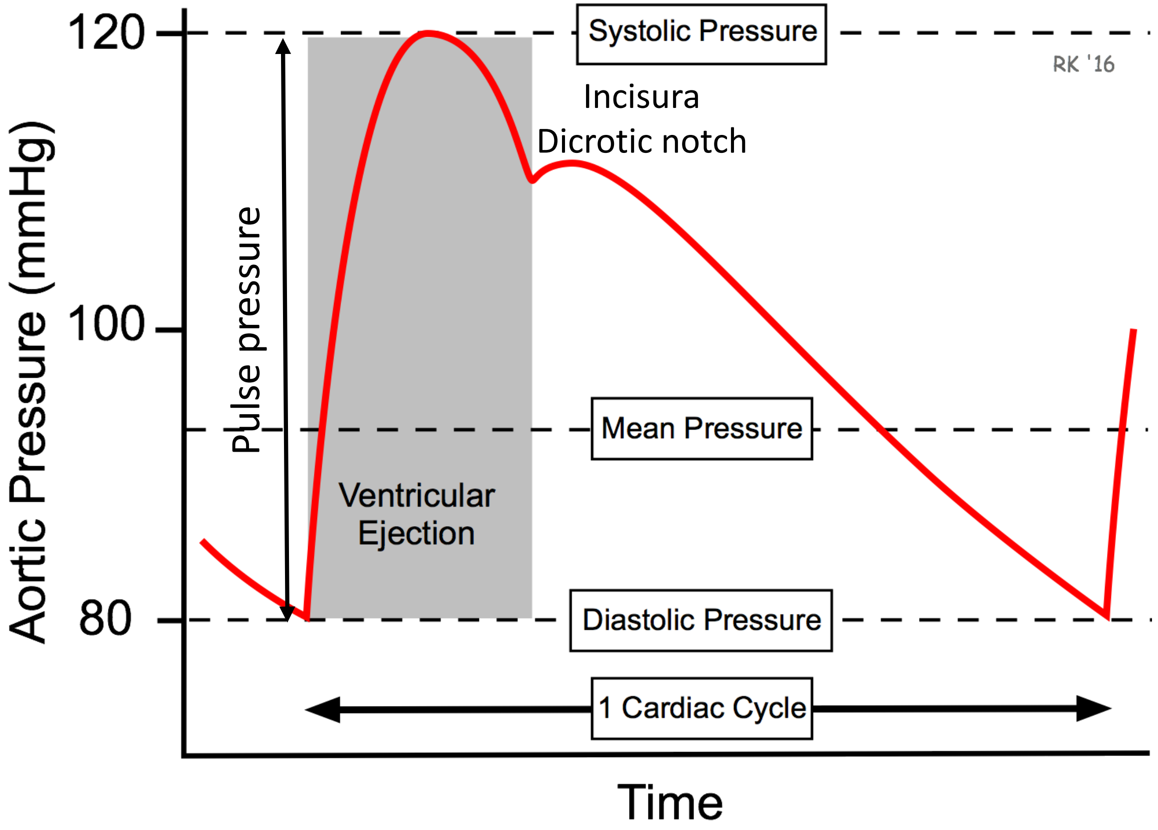
Arterial pressure then rises sharply, resulting in the *systolic peak*—the waveform's highest point.

As blood continues into the peripheral vessels, arterial pressure falls and the waveform begins a downward trend, called the *dicrotic limb*. Arterial pressure usually keeps falling until pressure in the ventricle is less than pressure in the aortic root.

When ventricular pressure is lower than aortic root pressure, the aortic valve closes. This event appears as a small notch on the waveform's downside, called the *dicrotic notch*.

When the aortic valve closes, diastole begins, progressing until aortic root pressure gradually falls to its lowest point. On the waveform, this is known as *end diastole*.





Arterial blood pressure

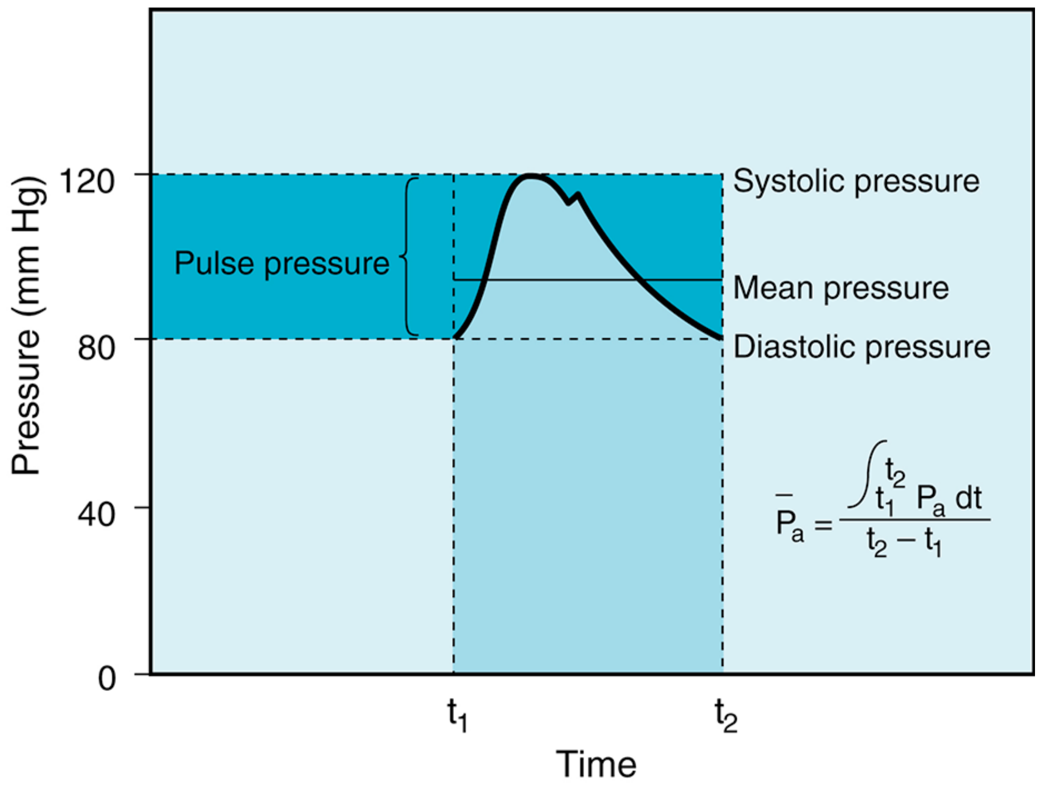
systolic/diastolic/pulse pressure ($P_s/P_d/P_p$)

mean arterial blood pressure (MABP) (the determinant of the perfusion pressure)

MABP ~ $P_d + 1/3(P_s - P_d)$

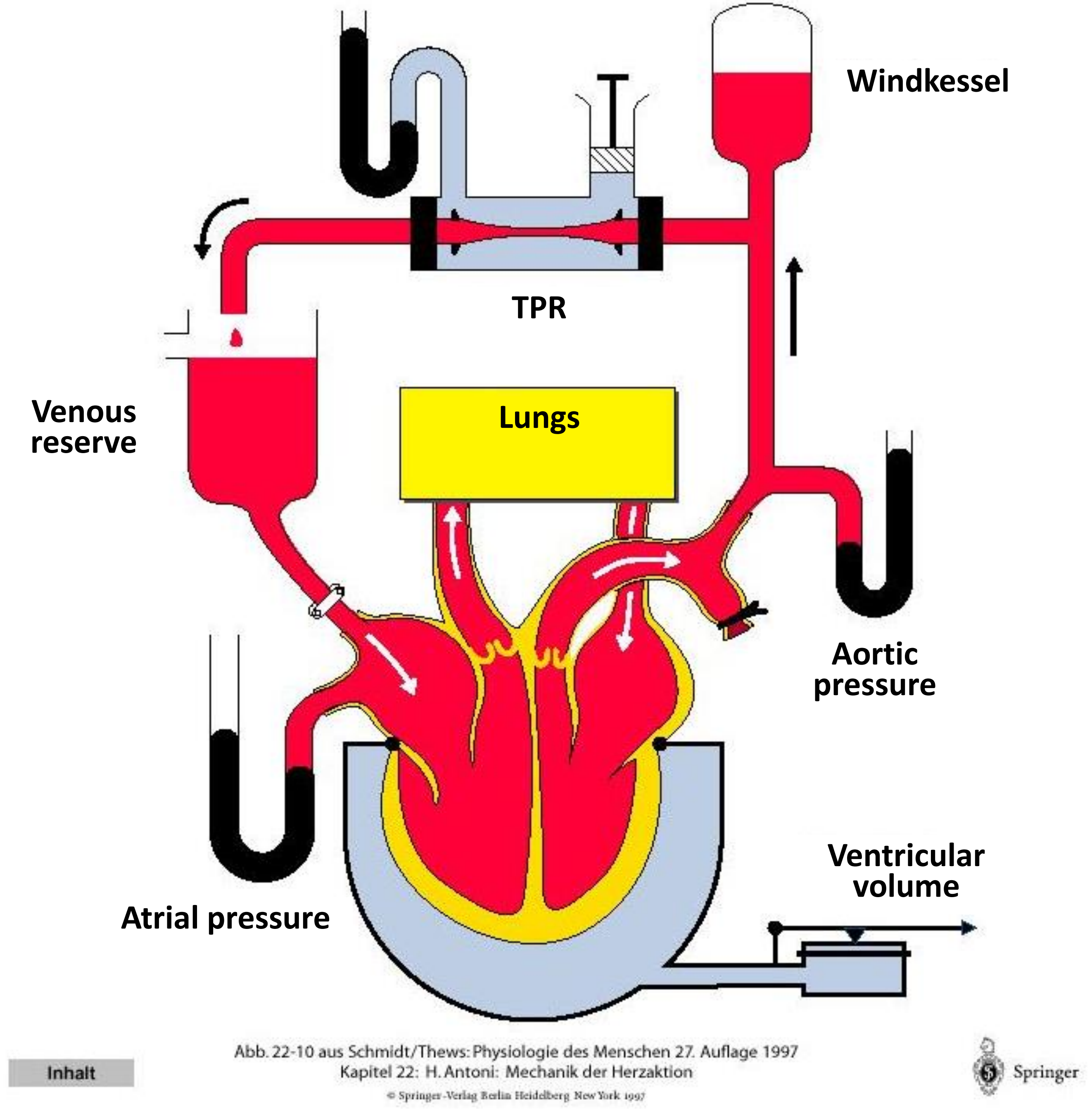
However…

Patient monitors actually integrate the pressure signal to determine MABP precisely

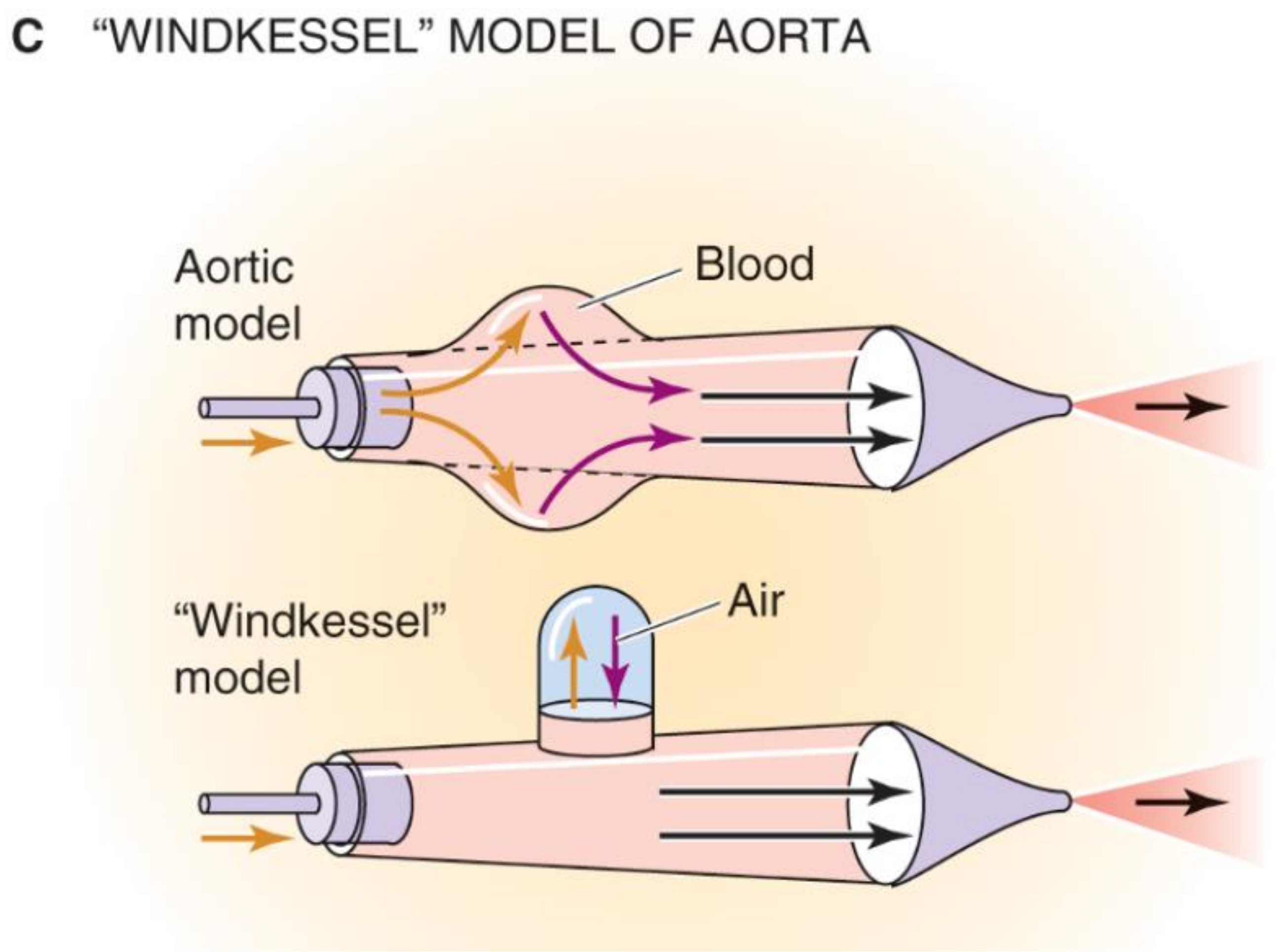


The formula shows that length of the cardiac cycle (pulse rate) affects MABP!

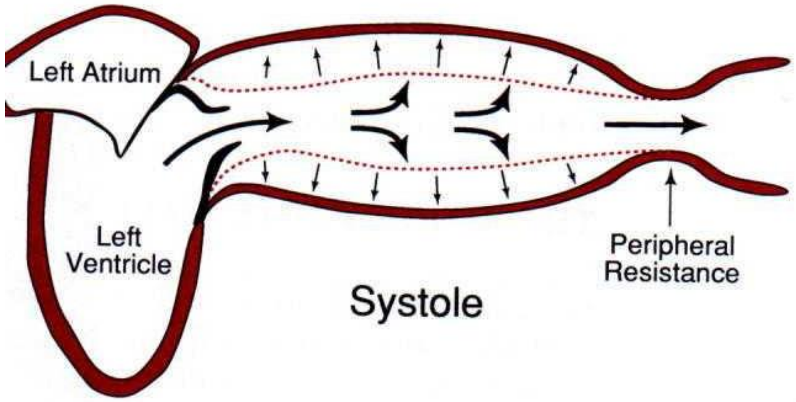
Starling's heart-lung preparation



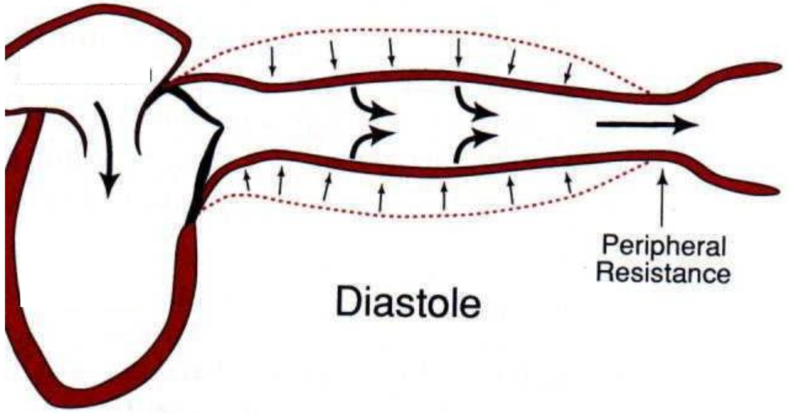
Windkessel = air tank, transient energy storage of the pump’s energy



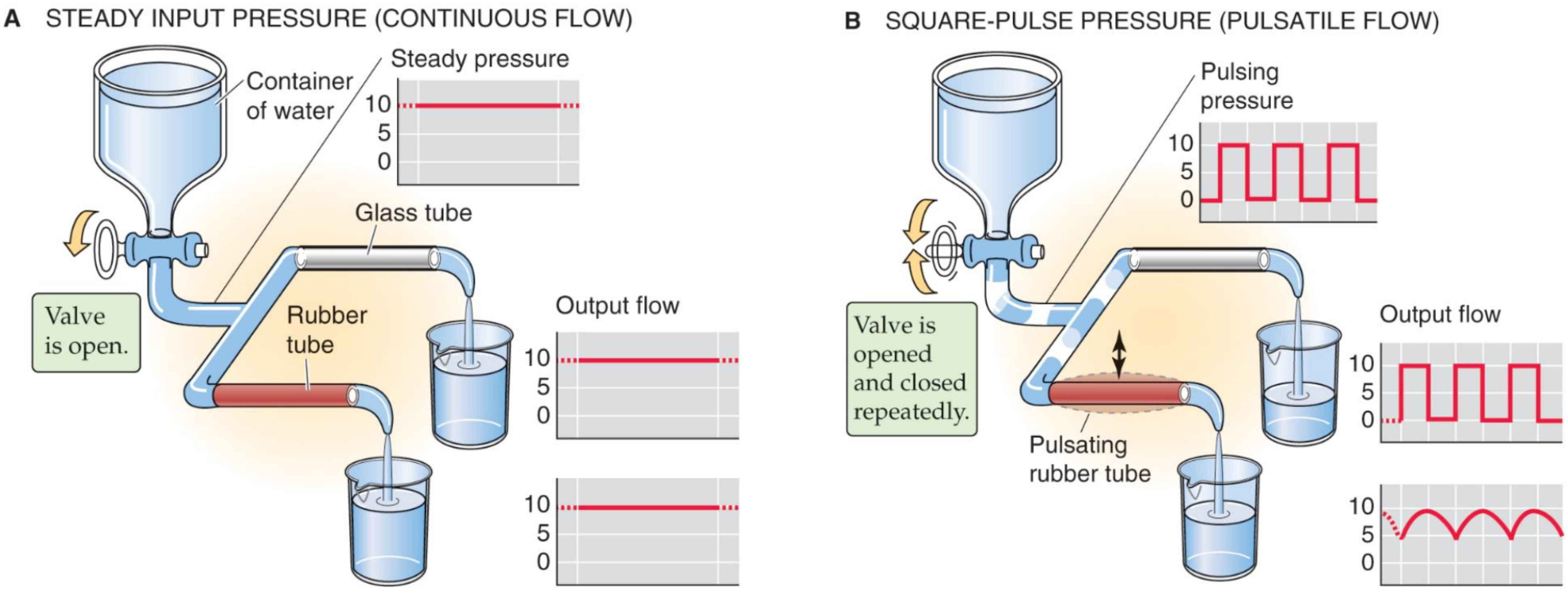
Windkessel function of elastic arteries



During ventricular ejection, the distended aorta store blood and energy of the contraction.

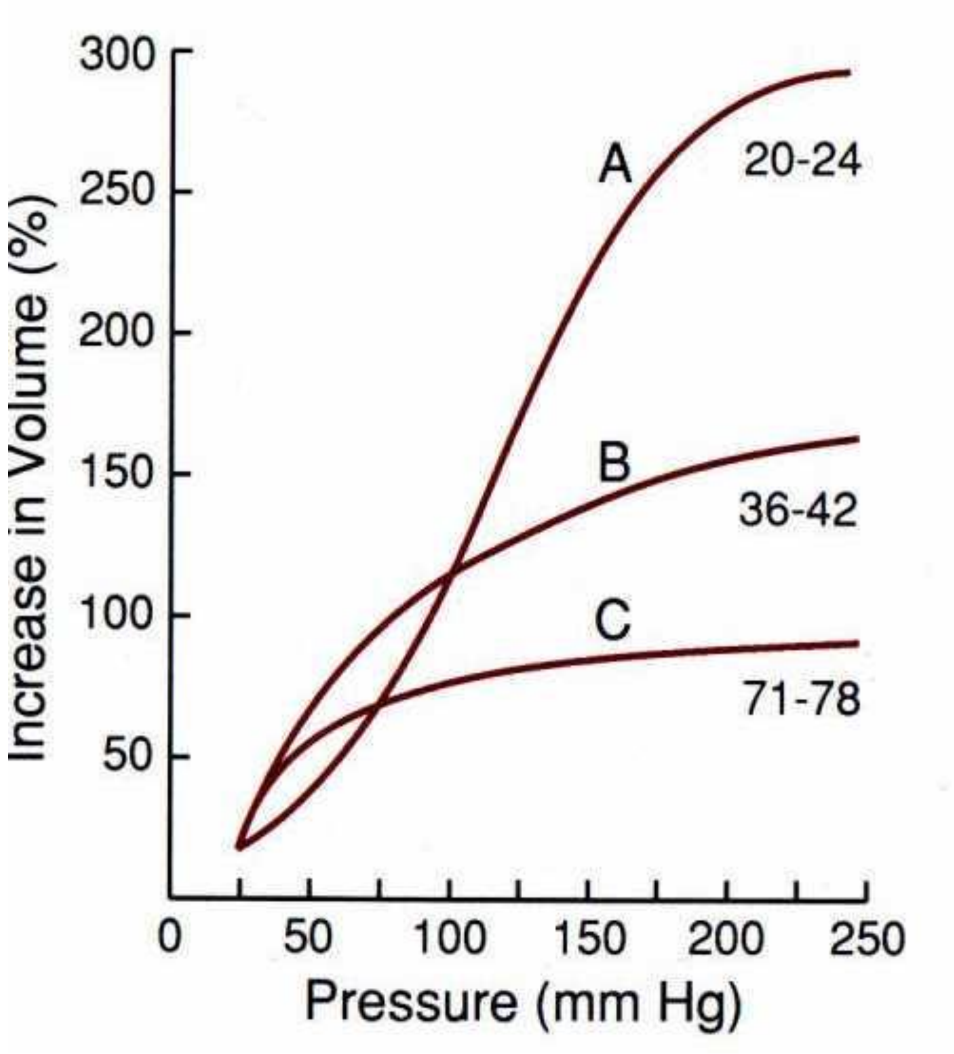


During diastole, the aorta passively contracts and pushes blood forward toward the periphery.

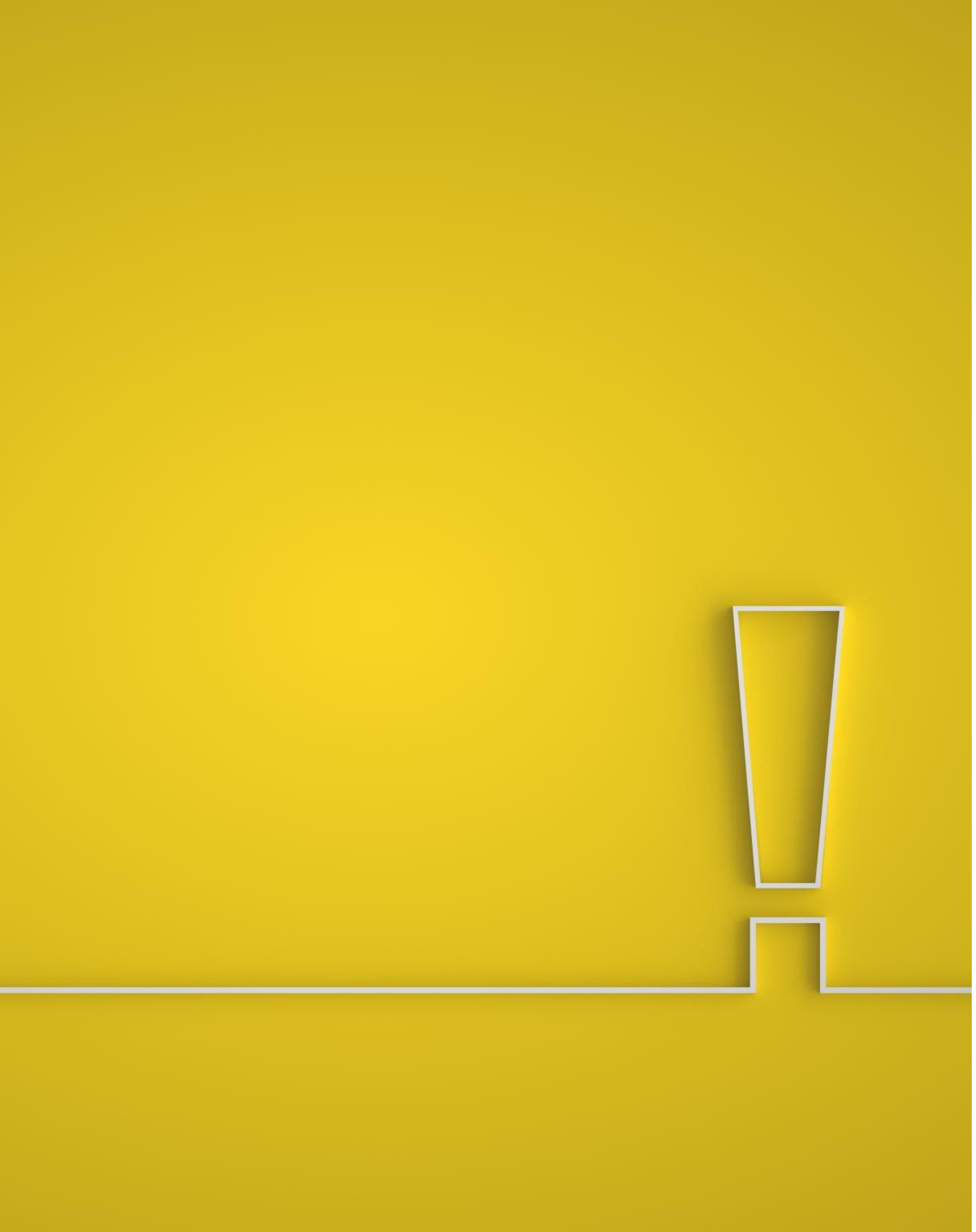


An elastic tube is better when flow is pulsatile

Aging decreases aortic elasticity



Compliance decreases in the higher blood pressure range



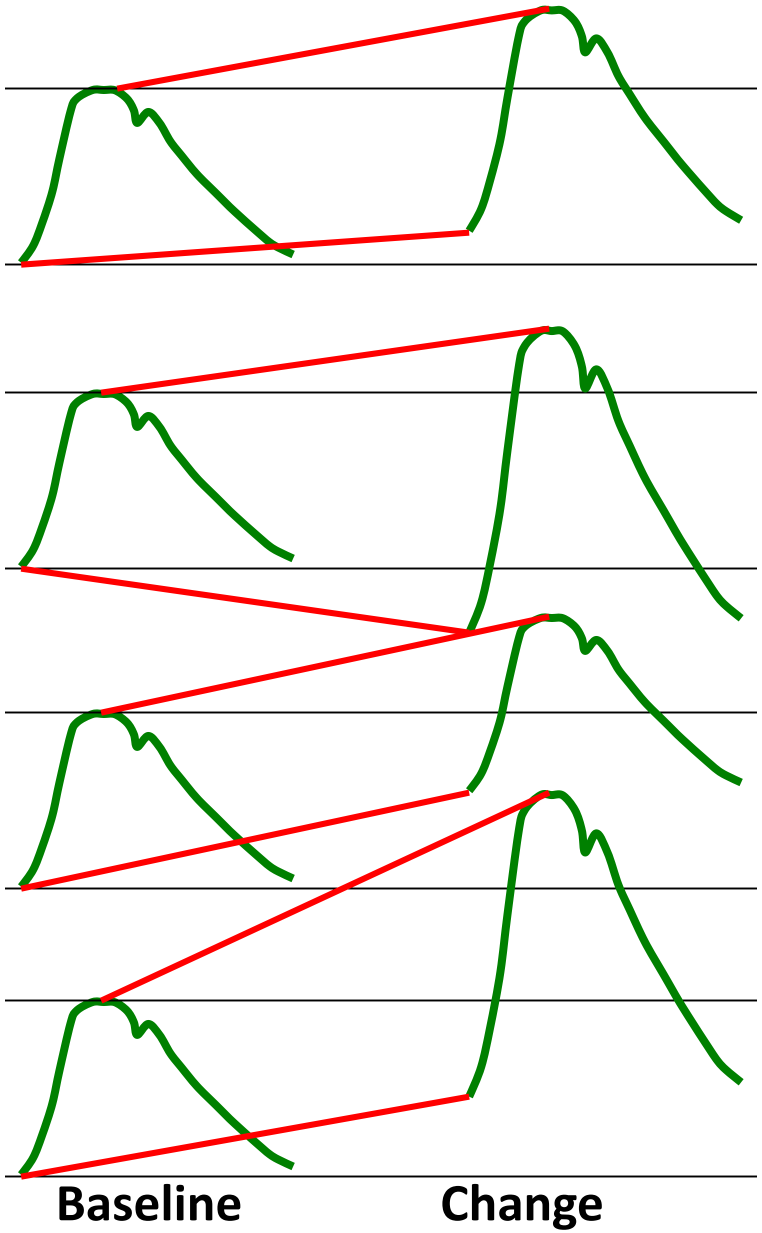
Factors affecting arterial blood pressure

Stroke volume (SV): its INCREASE will make the pulse larger, so systolic, mean, diastolic pressures all increase AND pulse pressure increases too.

Elasticity of the aorta (compliance): its REDUCTION by aging will increase pulsation: mean pressure is UNAFFECTED, systolic pressure is increased, diastolic is decreased and pulse pressure GREATLY increased

Total peripheral resistance (TPR): its INCREASE will INCREASE the MABP but has no effect on pulsation, so systolic, mean, diastolic pressures all increase BUT the pulse pressure does not change

Summary of arterial blood pressure changes



Baseline

$\frac{120}{80}$

Increased stroke volume

$\frac{140}{90}$ Hgmm

$\frac{120}{80}$

Decreased compliance (elasticity)

$\frac{140}{60}$ Hgmm

$\frac{120}{80}$

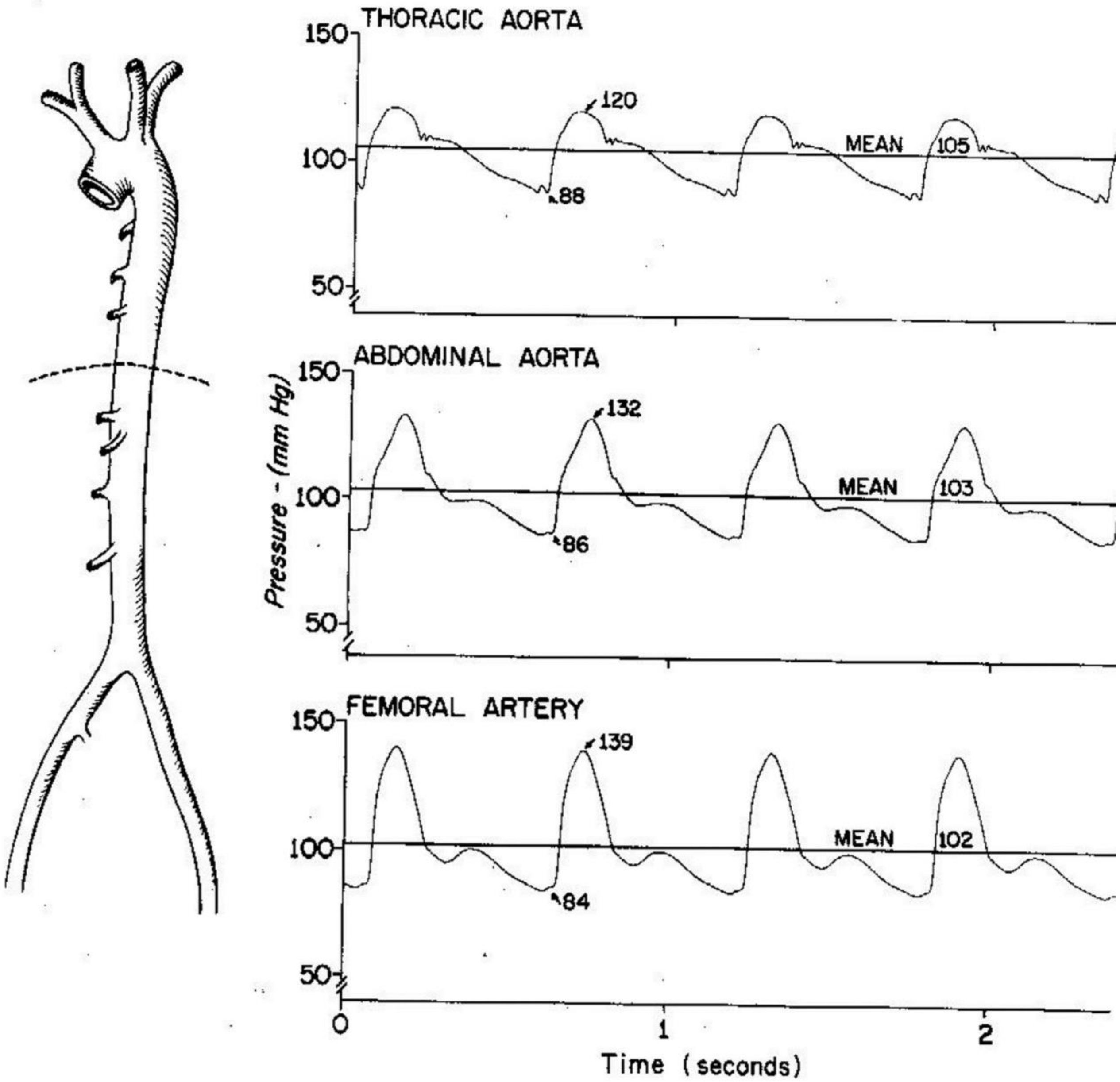
Increased TPR

$\frac{150}{110}$ Hgmm

$\frac{120}{80}$

Increased TPR + decreased compliance

$\frac{180}{110}$ Hgmm



Change in the pressure pulse shape during propagation

Towards the periphery

- systolic peak increases
- incisura disappears
- diastolic peak appears
- time lag

Propagation of the pressure pulse

Pulse wave velocity (PWV)

- in the aorta: 3-5 m/sec;
- in the little arteries: 15-30 m/sec.

Mean flow velocity is about 30 cm/sec in the aorta

- and decreases toward the periphery.

v = Q/A (distance traveled by the particles)

(blood) flow ≠ flow velocity!

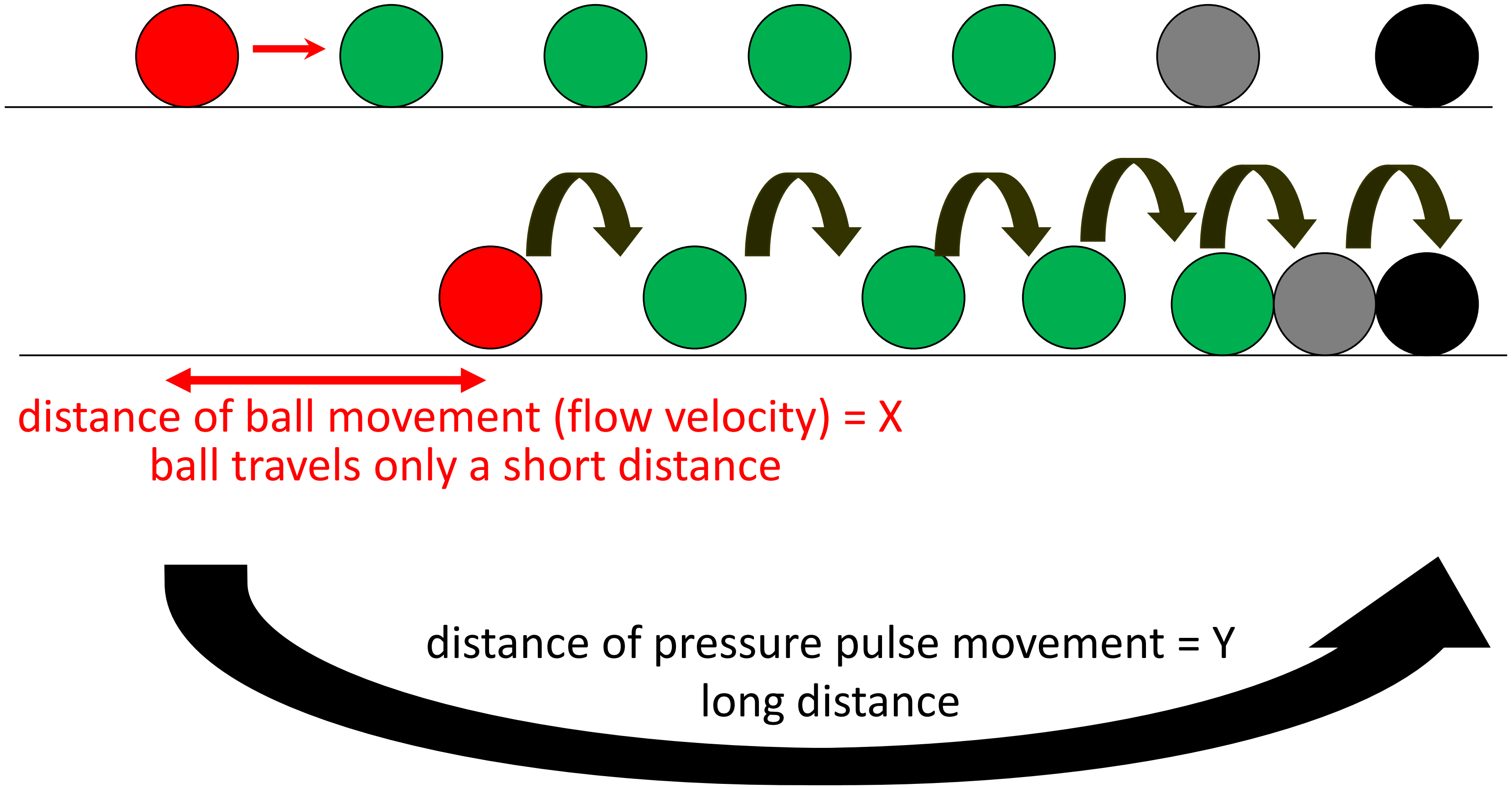
PWV is increased by

- decreasing wall elasticity (stiffness, E)
- increasing wall thickness (h)
- decreasing vascular radius (ri)

Moens-Korteweg equation

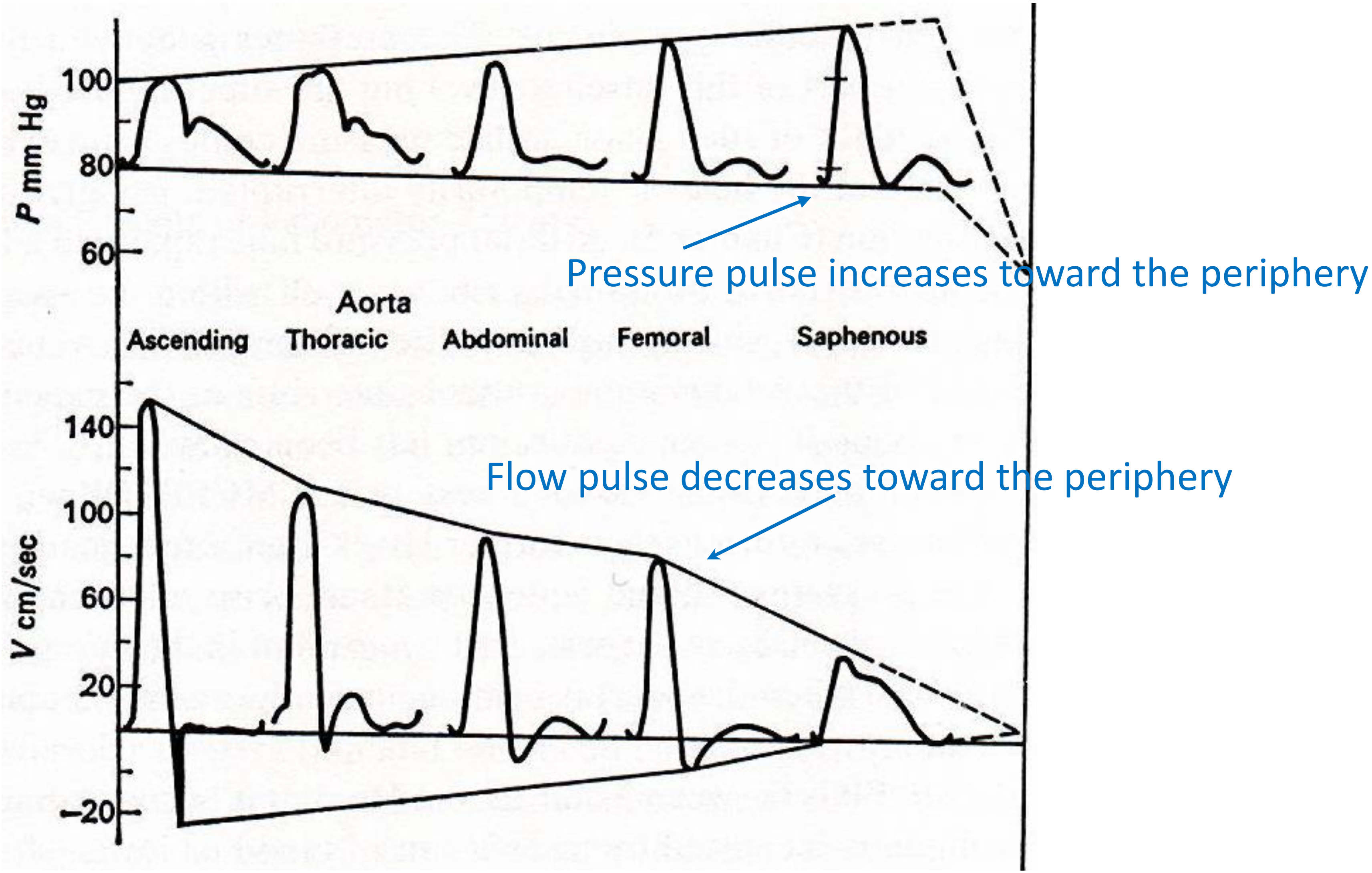
$$PWV = (Eh/2\rho r_i)^{1/2}$$

Flow velocity and propagation of pressure pulse



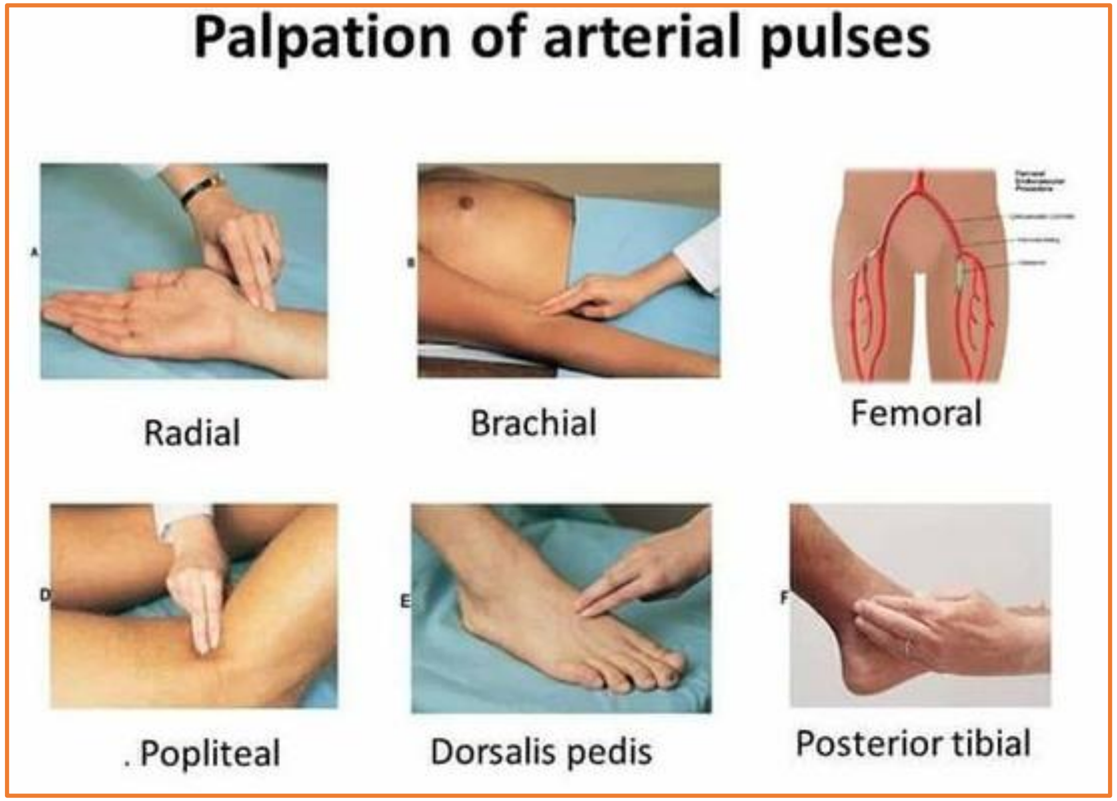
The propagation velocity of the pressure pulse (Y/t) is much faster, than the velocity of blood flow (X/t)

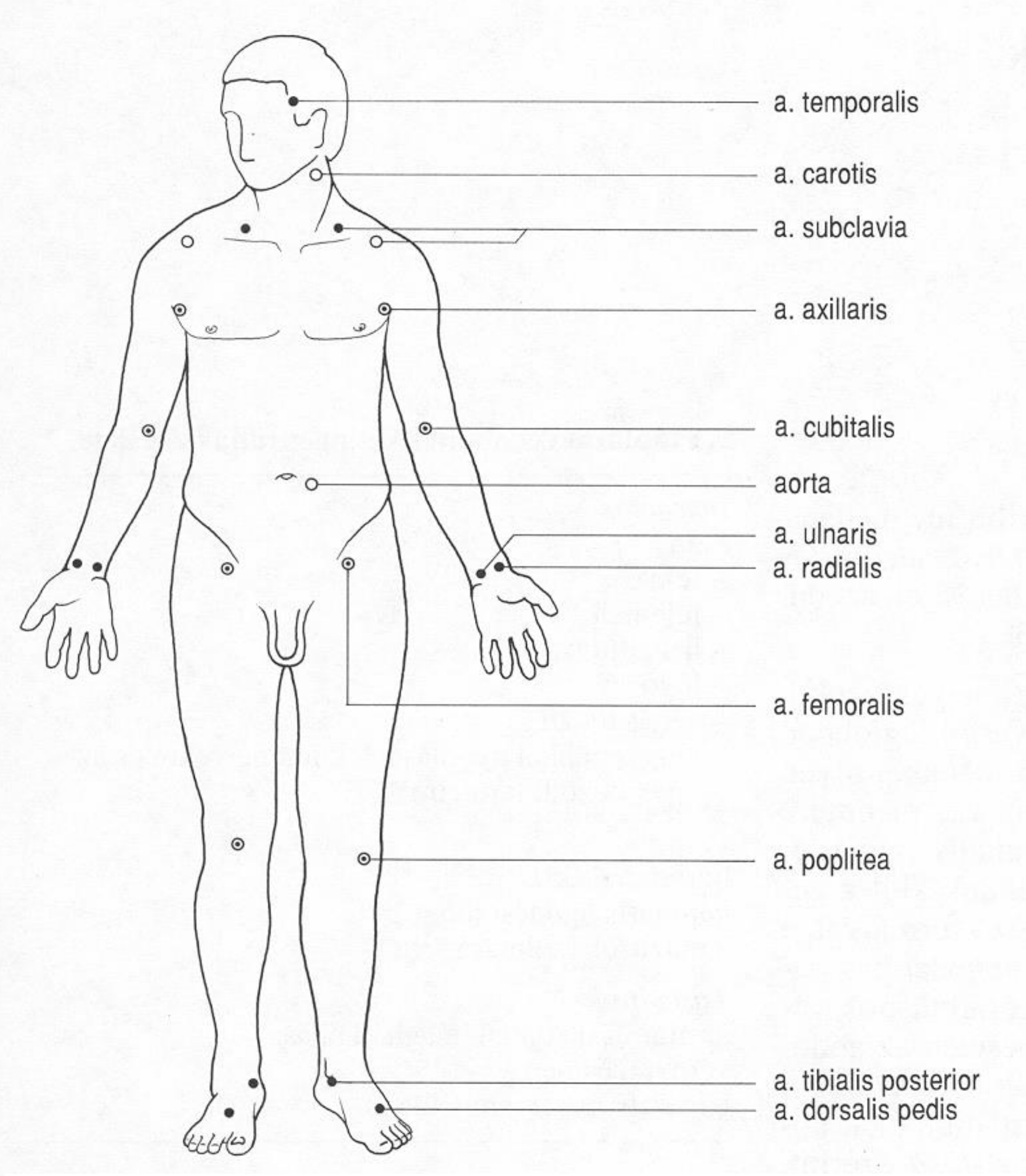
Pressure puse and flow pulse



The arterial pulse

- The pressure changes in the aorta during the cardiac cycle are transmitted along the arteries as a pressure pulse - creating a volume pulse that can be palpated by pressing the arteries against a flat hard surface
- the pulse depends on the function of BOTH the heart and the transmitting arteries!





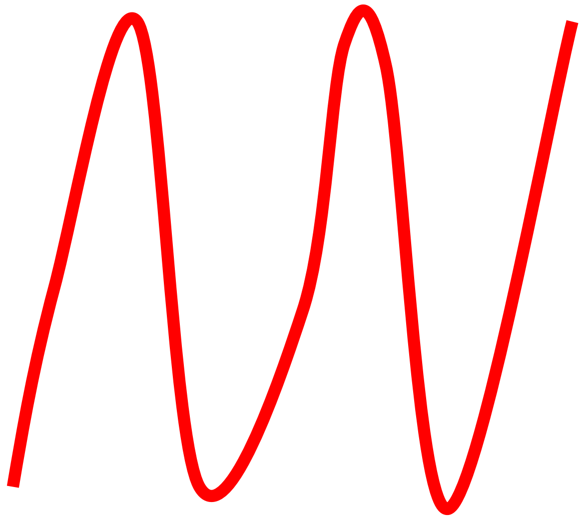
Palpation sites

Arterial pulse qualities (in latinul)

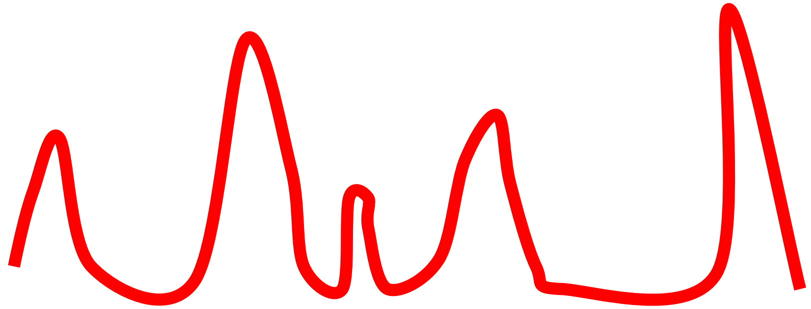
1.pulsus frequens	*pulsus rarus*	(frequency; high-low)
2.pulsus regularis	*pulsus irregularis*	(rhytmicity; regular- irregular)
3.pulsus altus	*pulsus parvus*	(amplitude of pulse wave= arterial wall deflection; high-low)
4.pulsus celer	*pulsus tardus*	(steepness of pulse wave= arterial wall deflection speed; rapid-slow rise)
5.pulsus durus	*pulsus mollis*	(pulse suppression; hard-soft)
6.pulsus aequalis	*pulsus inaequalis*	(similarity; similar-different)

1. Physiologically, the frequency of the heart reflects the pulse frequency; tachycardia-bradycardia
2. Physiological respiratory arrhythmia: the pulse is more frequent during inspiration; Bainbridge reflex (increase in atrial pressure increases heart rate)
3. Depends on the volume of stroke volume and the elasticity of the arterial wall
4. Depends on the velocity of the pressure change
5. Determined by the MABP
6. When fullness, height, speed and hardness of the pulse waves are equal, this is called *pulsus aequalis*

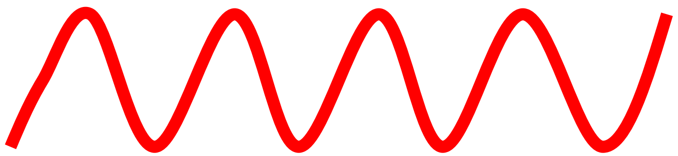
Arterial pulses: examples



Pulsus celer et altus
Aortic valve disfunction, when the ventricule is filling from 2 directions



Pulsus irregularis et inaequalis = arrhythmia absoluta
The time intervals between beats are different and the fullness of the pulses are also different



Pulsus frequens, parvus et mollis = pulsus filiformis
In hypovolaemia (collapsus, shock) the frequency is high, the amplitude is low and the pulse is easy to suppress: filiform pulse