Arterial Hypertension

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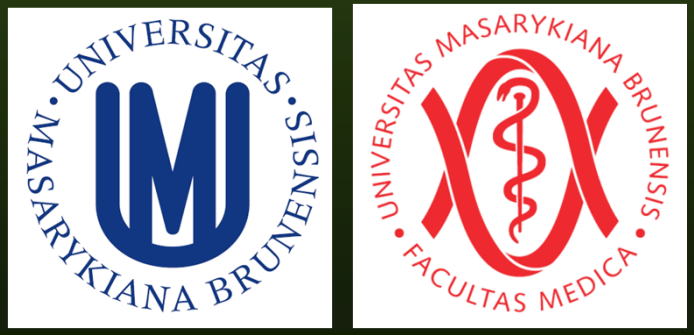

This presentation includes only the most important terms and facts. Its content by itself is not a sufficient source of information required to pass the Physiology exam.

Definition and Consequences

Arterial hypertension - chronic increase of the systemic blood pressure.

Symptoms indistinctive and nonspecific in the first stages of hypertension → almost 50% of the hypertensive patients do not know about their hypertension!

If not diagnosed in time and adequately treated, arterial hypertension results in:

- overload of the left ventricle (hypertrophy, heart failure)
- arteriosclerosis
  - increased risk of the myocardial infarction
  - increased risk of the stroke
  - the renal failure, etc.

Hypertension significantly shortens the life span.

Definition and Consequences

Arterial hypertension - chronic increase of the systemic blood pressure.

Table 1 Definitions and classification of blood pressure (BP) levels (mmHg)

Category	Systolic		Diastolic
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension	≥140	and	<90

Guidelines for the management of arterial hypertension. Eur Heart J 2007;28:1462-1536.

Methods of blood pressure measurement

Proper way of blood pressure measurement

Definition and Consequences

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Definition and Consequences

Stratification of cardiovascular risk

Blood pressure (mmHg)					
Other risk factors, OD or Disease	Normal SBP 120–129 or DBP 80–84	High normal SBP 130–139 or DBP 85–89	Grade 1 HT SBP 140–159 or DBP 90–99	Grade 2 HT SBP 160–179 or DBP 100–109	Grade 3 HT SBP ≥ 180 or DBP ≥ 110
No other risk factors	Average risk	Average risk	Low added risk	Moderate added risk	High added risk
1–2 risk factors	Low added risk	Low added risk	Moderate added risk	Moderate added risk	Very high added risk
3 or more risk factors, MS, OD or Diabetes	Moderate added risk	High added risk	High added risk	High added risk	Very high added risk
Established CV or renal disease	Very high added risk	Very high added risk	Very high added risk	Very high added risk	Very high added risk

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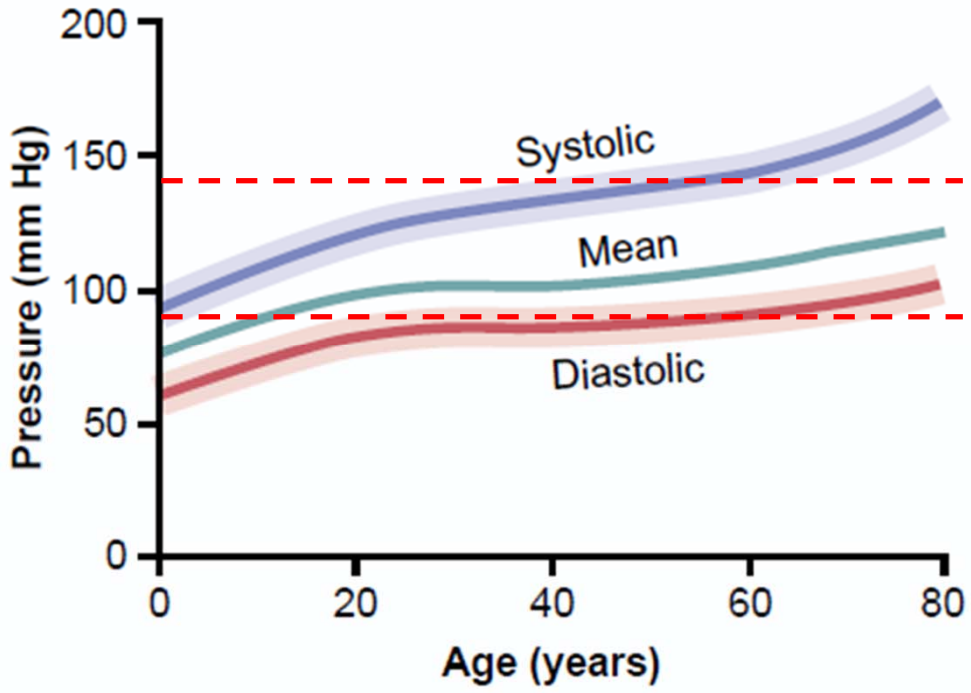
Definition and Consequences



Guyton and Hall.
Textbook of Medical Physiology, 11th edition

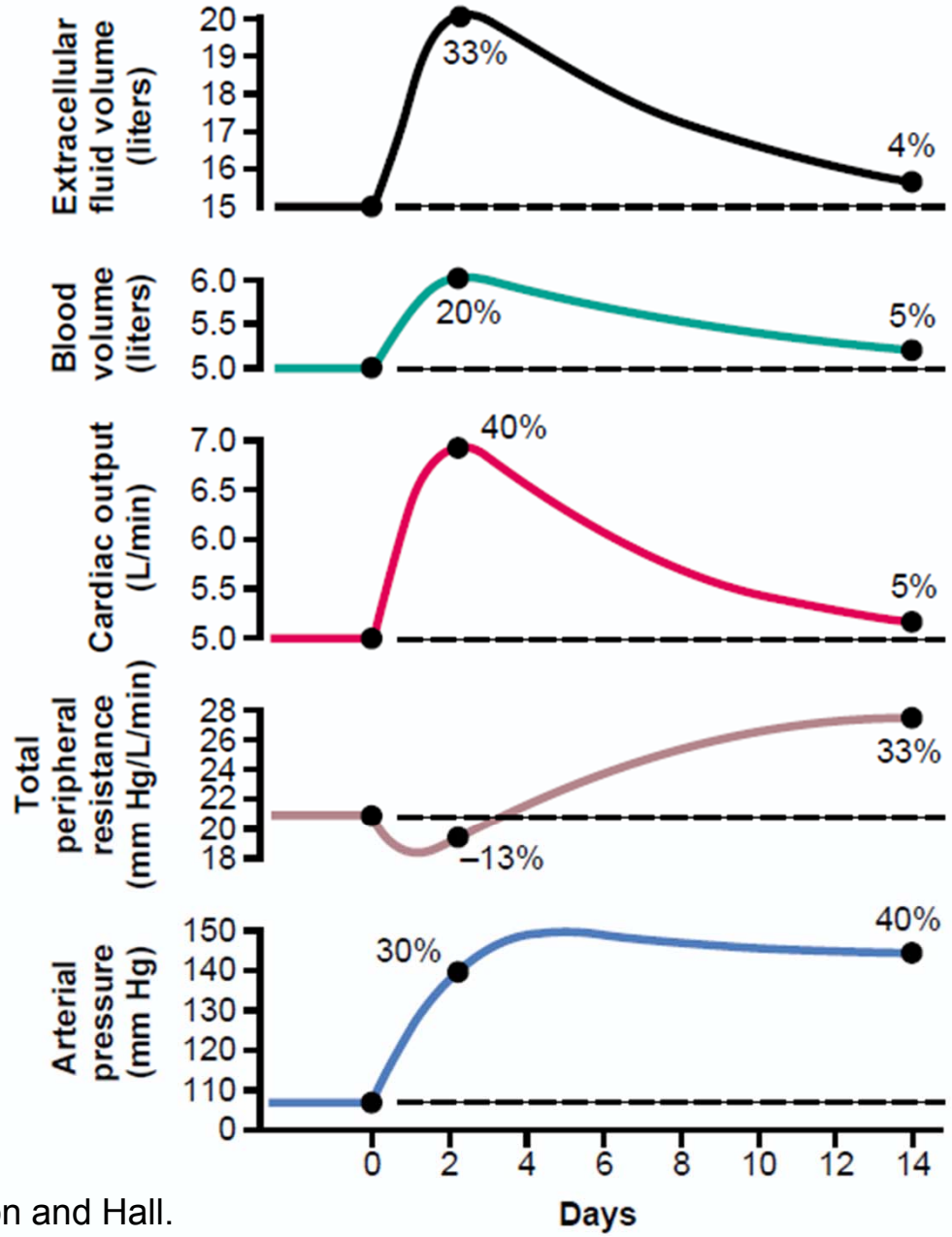
in children and adolescents – special percentile tables

Factors Determining Blood Pressure

Ohm´s law

$U = I \cdot R \longrightarrow P = CO \cdot TPR$

- ↑ cardiac output ↑
  - → volume-loading (hyperdynamic, volume dependent) h.
- ↑ total peripheral resistance
  - → resistance (non-volume dependent) h.



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$C = \Delta V / \Delta P$

- ↓ compliance → isolated systolic hypertension

heart

CO = SV . HR

HR is guided by sympathetic and parasympathetic system

SV depends on:

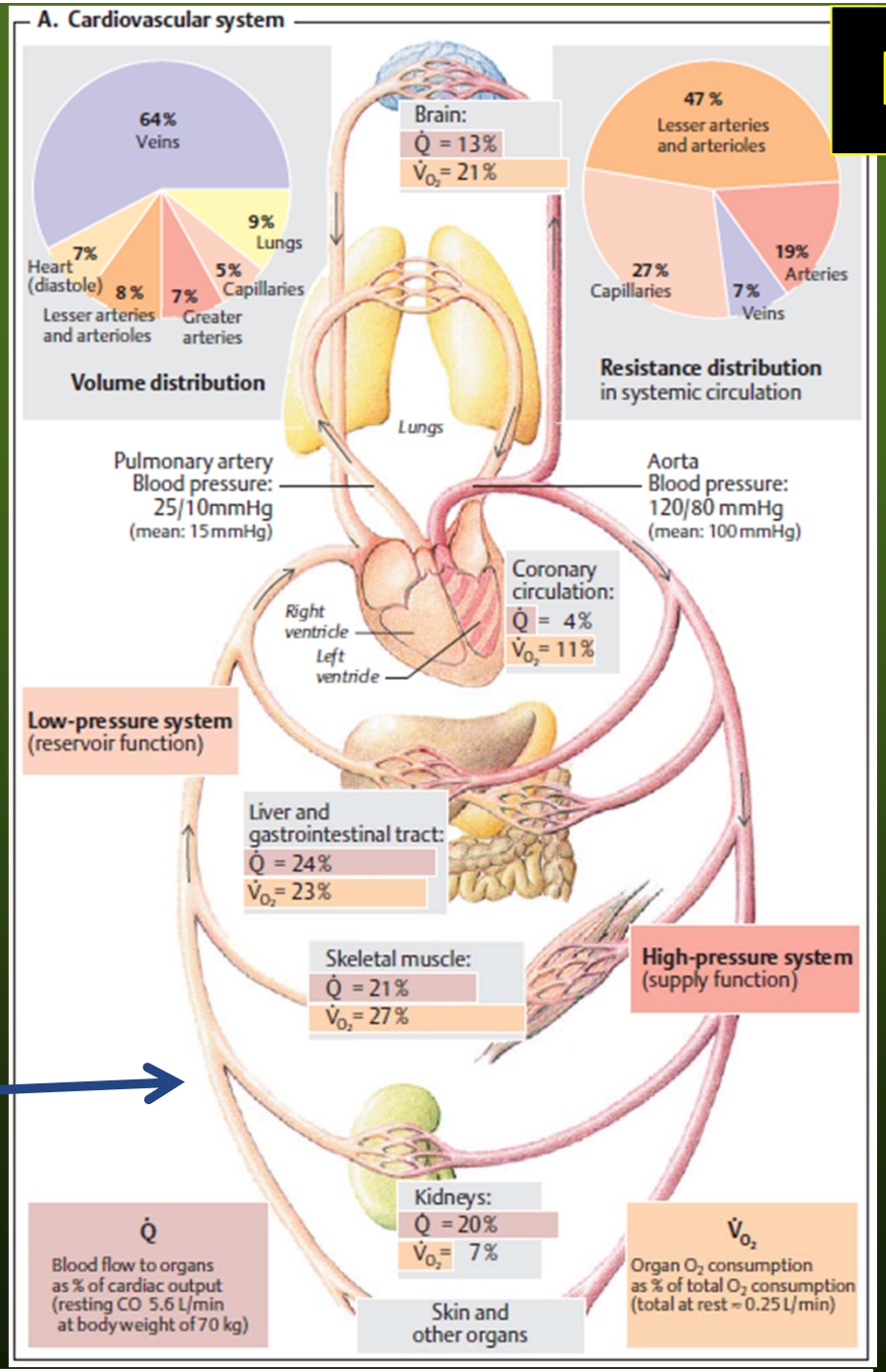
1.<u>venous return</u> (blood volume, tonus of veins)

2.<u>contractility</u>

3.<u>peripheral pressure</u>

veins

blood reservoire



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P = CO . TPR

kidneys

regulation of blood volume

heart

CO = SV . HR

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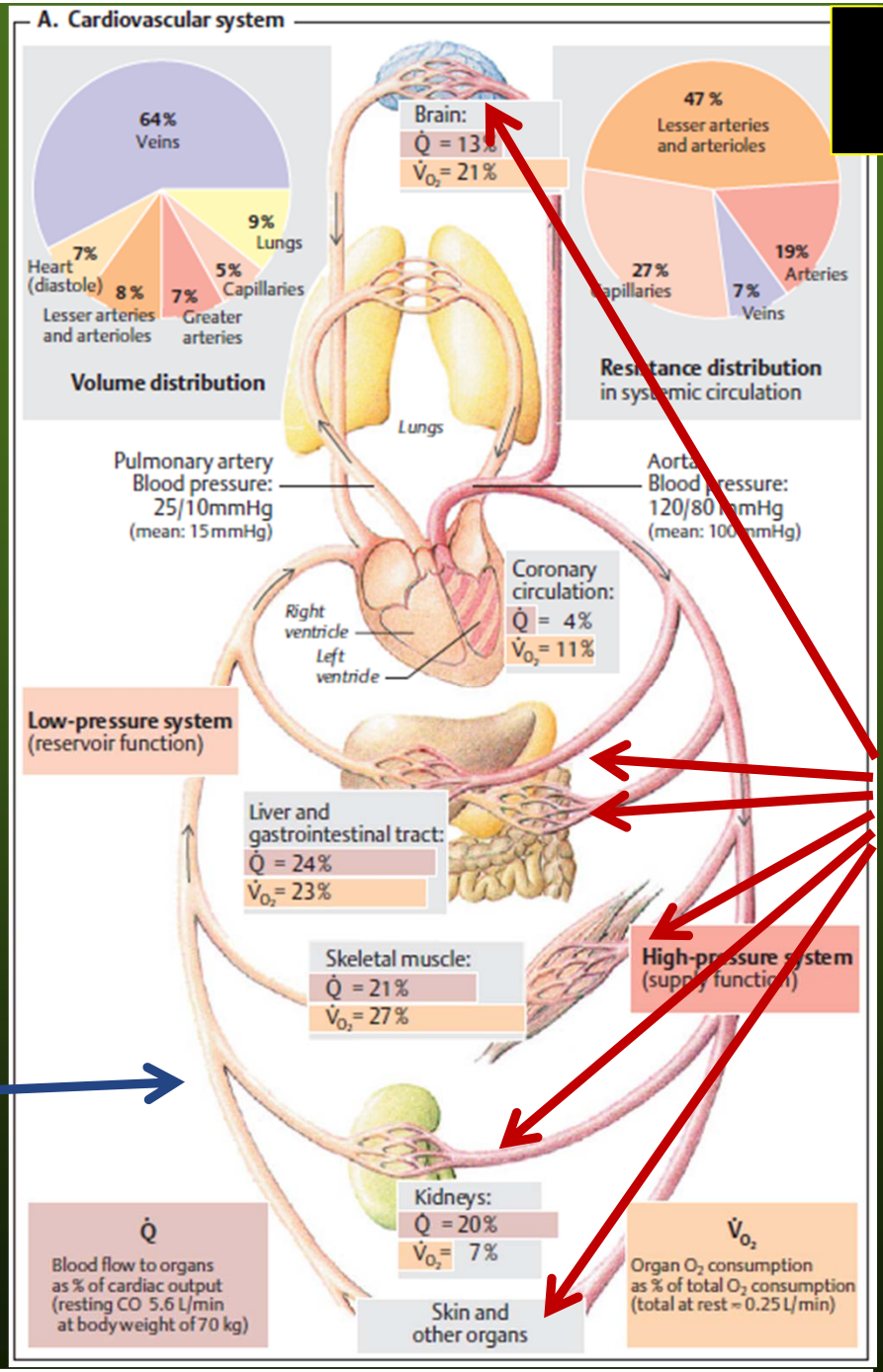

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P = CO . TPR

arterioles

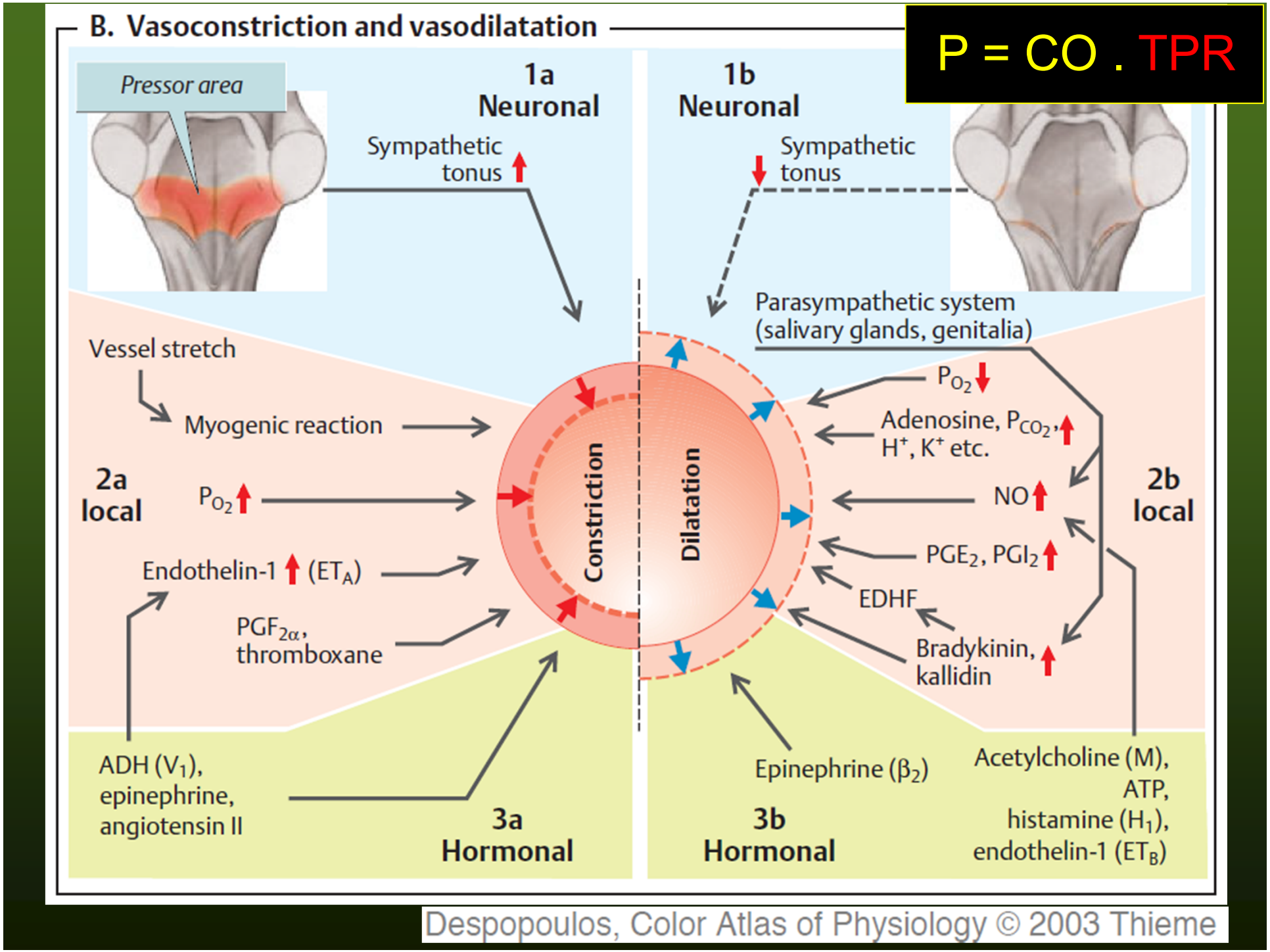
regulation of TPR

kidneys

regulation of blood volume

also TPR (RAS)

P = CO . TPR



heart

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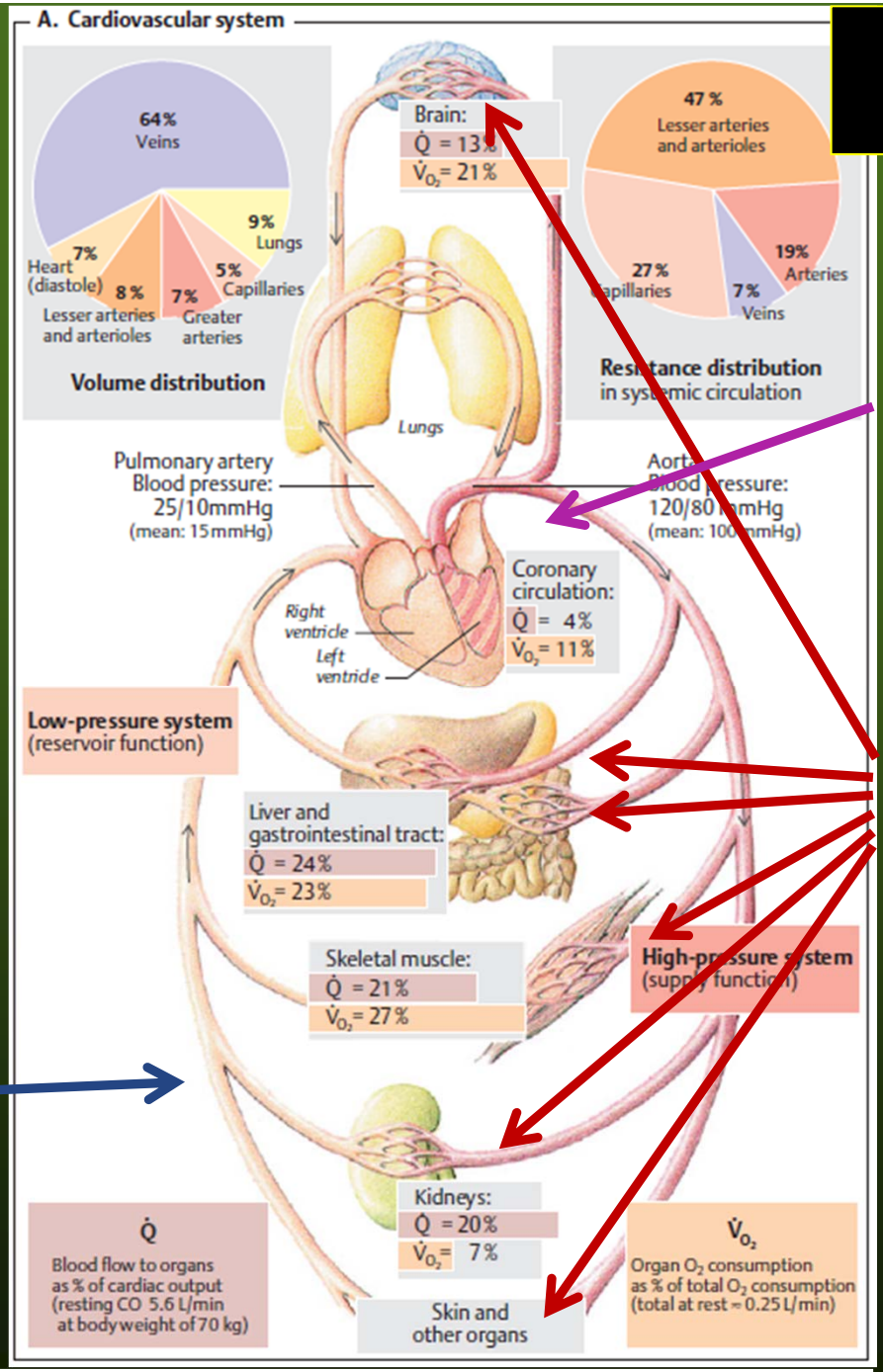

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P = CO . TPR

aorta and big elastic arteries

compliance

arterioles

regulation of TPR

kidneys

regulation of blood volume, also TPR (RAS)

heart

CO = SV . HR

HR is guided by sympathetic and parasympathetic system

SV depends on:

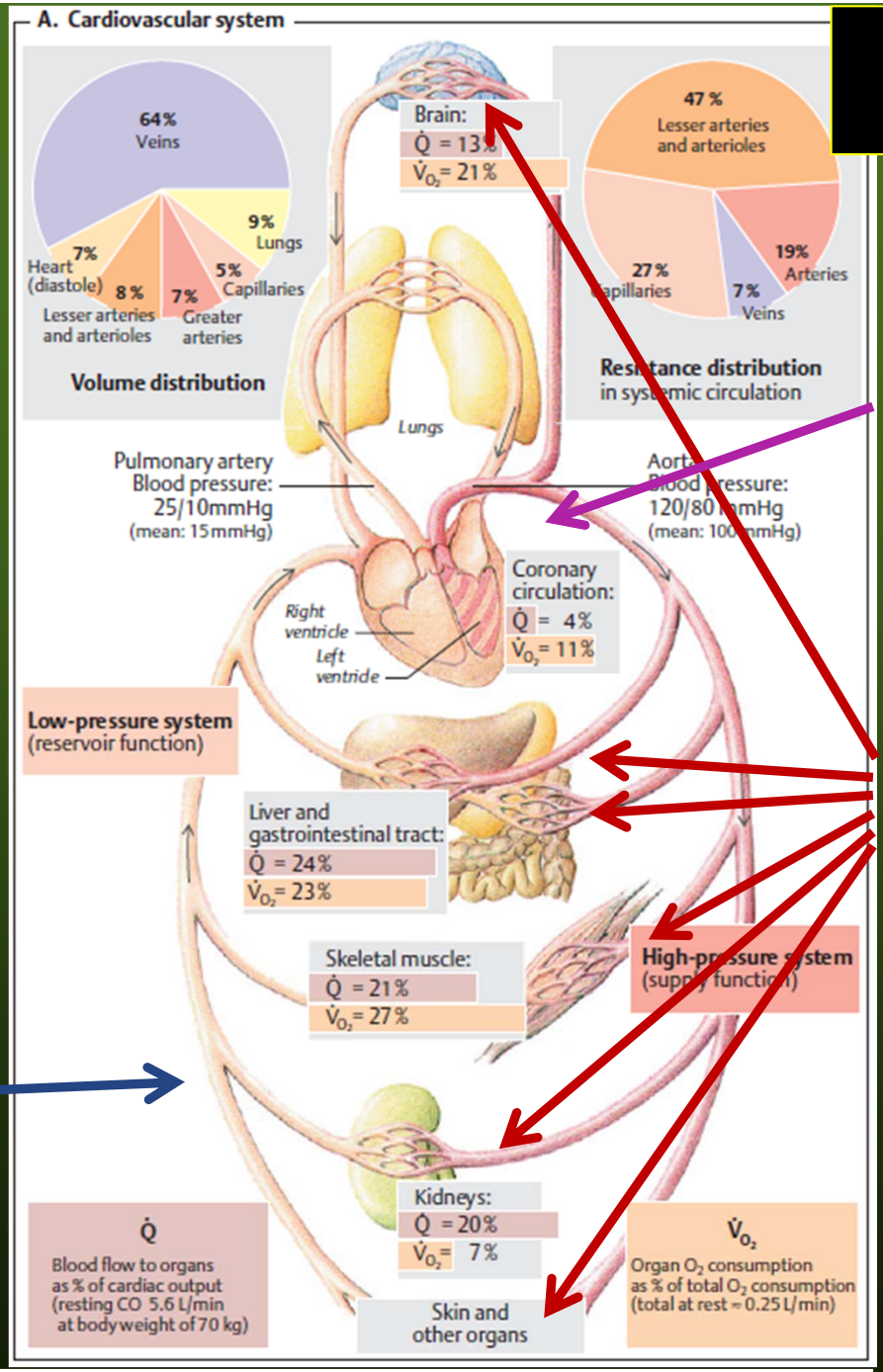
1.<u>venous return</u> (blood volume, tonus of veins)

2.<u>contractility</u>

3.<u>peripheral pressure</u>

veins

blood reservoire



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P = CO . TPR

aorta and big elastic arteries

compliance

arterioles

regulation of TPR

regulation of blood volume:

- kidneys
- thirst
- ADH

heart

CO = SV . HR

HR is guided by sympathetic and parasympathetic system

SV (

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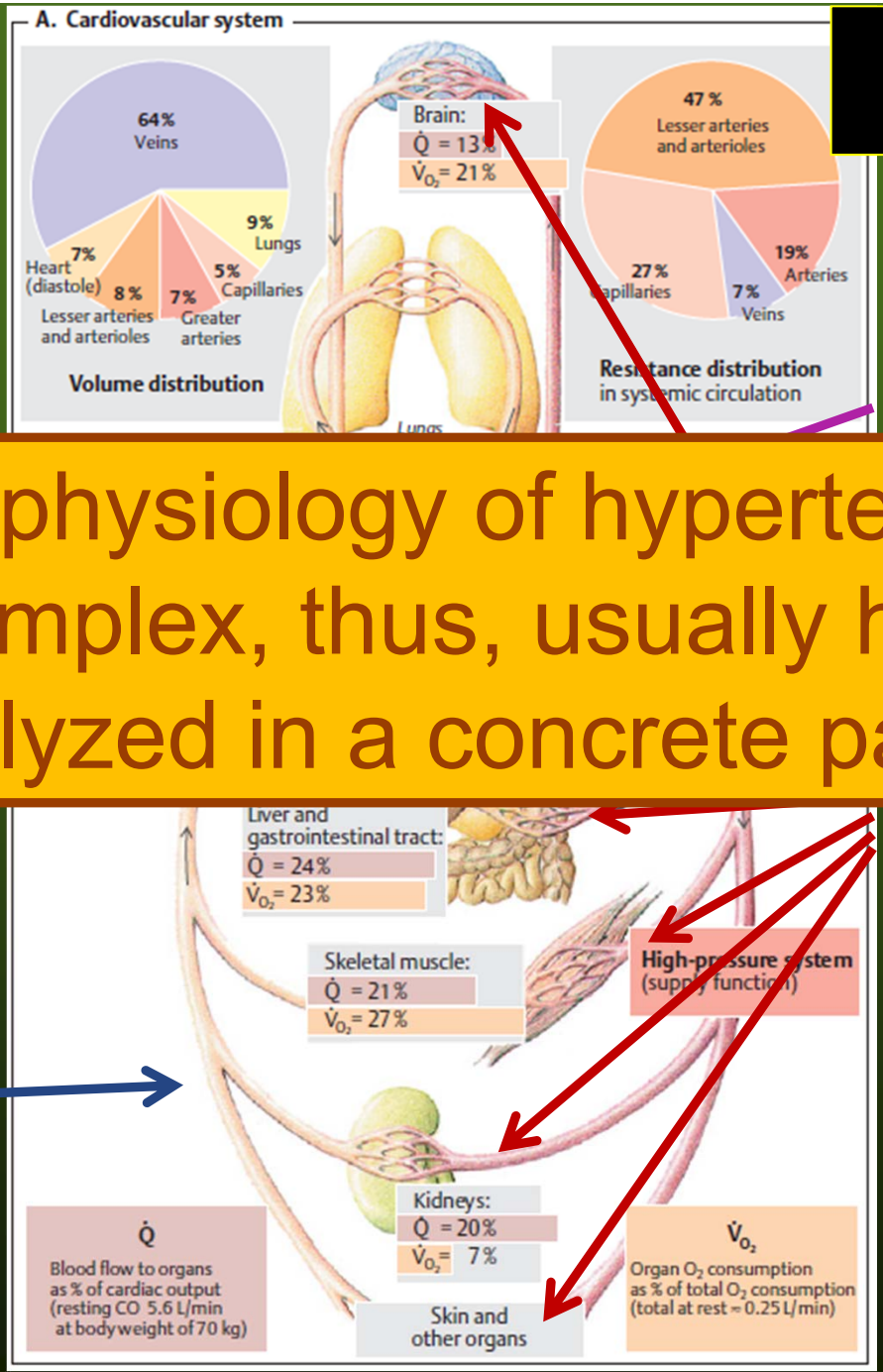
to

3.<u>peripheral pressure</u>

veins

blood reservoire

P = CO . TPR



aorta and big elastic arteries

Pathophysiology of hypertension is very complex, thus, usually hard to be analyzed in a concrete patient!

regulation of TPR

regulation of blood volume:

- kidneys
- thirst
- ADH

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Classification

A. Essential (primary) hypertension

- „hypertension of an unknown origin“
- 90 – 95%

B. Secondary (symptomatic) hypertension

- symptom of another primary disease with identifiable cause

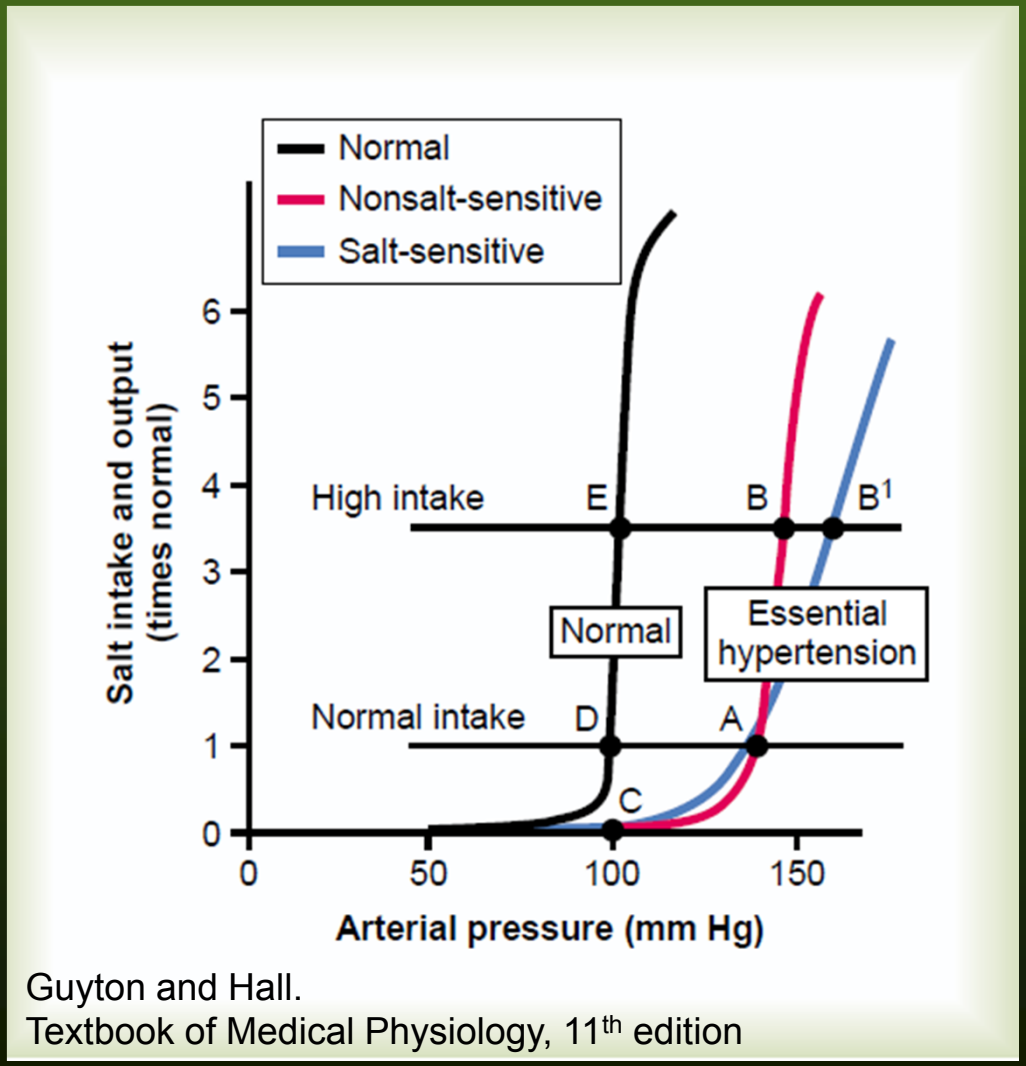
Essential Hypertension

- strong hereditary tendency in some patients
  (polygenic ground)
- provoking factors:
  - excess weight gain, obesity – account for about 65-70% of the risk for developing of essential hypertension
  - sedentary lifestyle

  New clinical guidelines recommend increased physical activity and weight loss as the first step in treating most patients with the essential hypertension.
  - stress (namely mental)
  - excessive sodium intake (interpopulation studies – Eskimos *vs.* people living in the North Japan)

Essential Hypertension

Sodium-loading renal function curves



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Increased salt intake

↓

Increased extracellular volume

↓

Increased arterial pressure

↓

Decreased renin and angiotensin

↓

Decreased renal retention of salt and water

↓

Return of extracellular volume almost to normal

↓

Return of arterial pressure almost to normal

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Essential Hypertension

Isolated Systolic Hypertension

- ↑ systolic and pulse pressure
- in the elderly
- due to:
  - age-dependent remodelling of the wall of elastic arteries (less elastic and more collagen fibres)

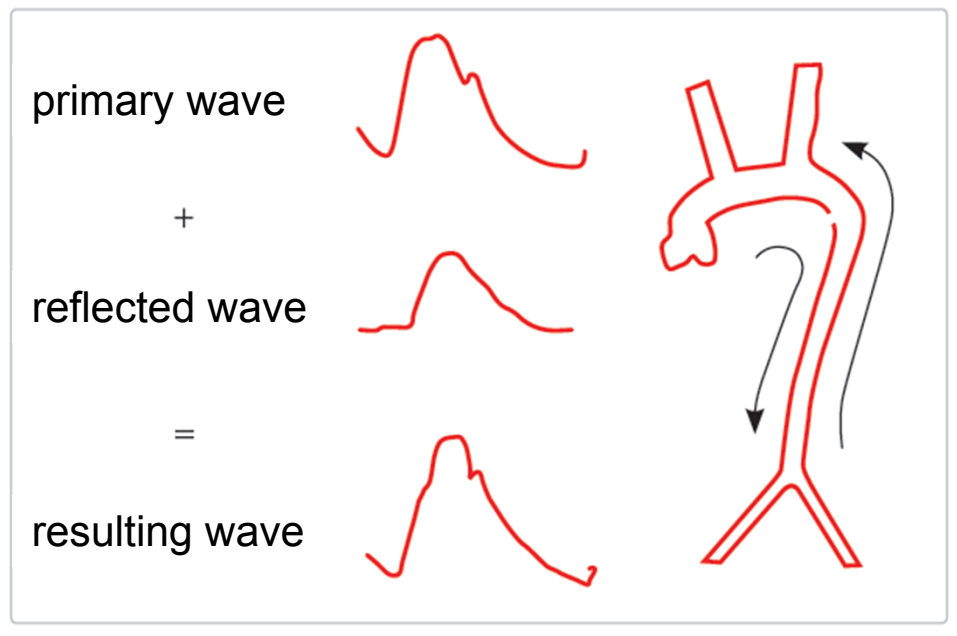
    → ↑ stiffness, ~↓ compliance:

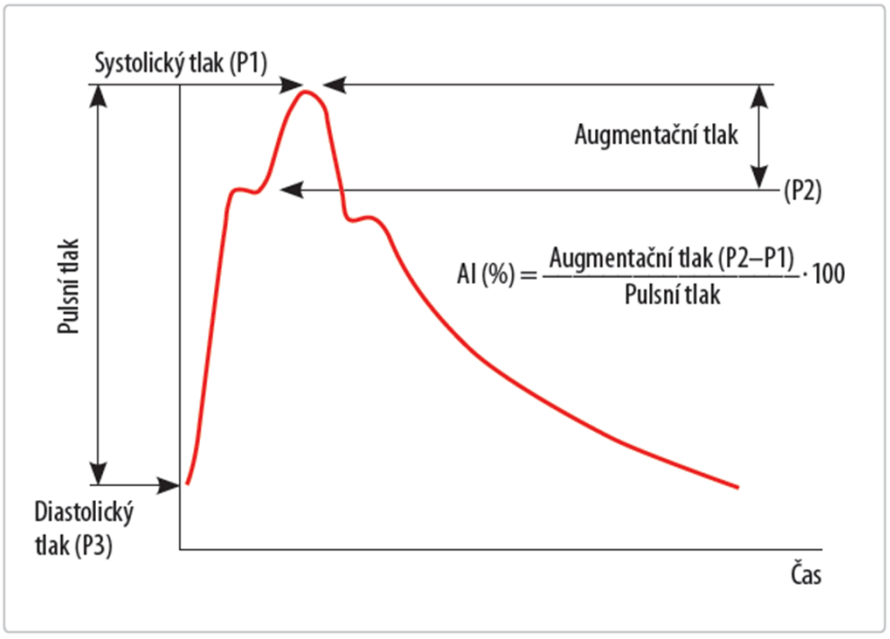
    1. → ↓ distension of elastic arteries during the systole → steeply ↑ arterial systolic pressure + ↓ blood volume (and also pressure) in arteries during the diastole
    2. → ↑ pulse wave velocity
  - endothelial dysfunction

Essential Hypertension

Isolated Systolic Hypertension

↑ pulse wave velocity → the secondary, reflected pulse wave comes back to the aorta and elastic arteries sooner and, thus, superimposes on the primary pulse wave still during the systolic phase → ↑ systolic pressure and may even ↓ diastolic pressure





Rosa J, Widimský J jr. **Charakteristika velkých tepen u arteriální hypertenze.** *Cor Vasa 2011;53:418–422.*

Essential Hypertension

Treatment

New clinical guidelines recommend increased physical activity and weight loss as the first step in treating most patients with EH.

Decrease of sodium and increase of potassium intake, relaxation ...

$P = CO \cdot TPR$

- ❖ vasodilatory drugs
  - ↓ TPR, some of them ↑ renal blood flow as well (ACEI)
  - a. by inhibiting sympathetic nervous system (sympatolytics)
  - b. by directly paralyzing the smooth muscle of the renal vasculature (vasodilatory agents or calcium channel blockers)
  - c. by blocking action of the renin-angiotensin system on the renal blood vessels or tubules (inhibitors of angiotensin I-converting enzyme, ACEI)
- ❖ natriuretic (diuretic) drugs
  - ↓ renal tubular reabsorption of salt and water → ↓ CO (by blocking the active transport of sodium through the tubular wall)

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Secondary Hypertension

1. Renal hypertension
   - Prerenal causes - Renovascular hypertension
   - Acute and chronic diseases of the renal parenchyma
   - Postrenal causes (renal vein trombosis, urinary tract obstruction)
   - Renin-producing renal tumor
2. Endocrine hypertension
   - Adrenocortical hyperfunction (Cushing´s, Conn´s, adrenogenital sy)
   - Sympatoadrenal hyperfunction (pheochromocytoma)
   - Exogenic hormones (gluko-, mineralocorticoids, sympatomimetics)
   - Hyperthyroidism
   - Acromegaly
3. Coarctation of the aorta
4. Hypertension in preeklampsia
5. Neurogenic hypertension