

SPECIAL PATHOPHYSIOLOGY

HYPERTENSION

1. Systolic blood pressure is elevated when it is over:
 1. 120mmHg.
 2. 130mmHg.
 3. 160mmHg.
 4. 140mmHg.
 5. 150mmHg.

2. Diastolic blood pressure is elevated when it is over:
 1. 80mmHg.
 2. 90mmHg.
 3. 95mmHg.
 4. 100mmHg.
 5. 110mmHg.

3. Which factors are able to increase systemic blood pressure?:
 1. Stroke volume of the heart.
 2. Cardiac output.
 3. Total peripheral vascular resistance (TPR).
 4. 2, 3.
 5. 1, 2, 3.

4. Blood pressure is increased when there is:
 1. Prevalence of pressor mechanisms.
 2. Activation of pressor and depressor factors.
 3. Insufficient depressor factors.
 4. Pressor-depressor dissociation.
 5. 1, 3.
 6. 1, 2, 3, 4.

5. Arterial hypertension is a condition of:
 1. Elevated blood pressure adequate to metabolic needs.
 2. Form of adaptation to generalized hyperperfusion.
 3. Functionally inadequate elevated blood pressure.
 4. Form of maintaining blood flow in vital organs.
 5. Circulatory overcompensation.

6. What is the pathogenetic classification of arterial hypertension?
 1. Congenital and acquired.
 2. Uncompensated and overcompensated.
 3. Infectious and non-infectious.
 4. Essential and symptomatic.

5. Localized and generalized.
 6. According to age and gender.
7. Which factors play an important role in the genesis of essential hypertension?
 1. Acute infections.
 2. Psychological traumas.
 3. Genetic predisposition.
 4. Kidney diseases.
 5. 2, 3.
 6. 1, 2, 3, 4.
 8. Which pathogenic units are involved in the pathogenesis of essential hypertension?
 1. Vessels.
 2. Renal.
 3. Autoimmune.
 4. Metabolic-perfusion.
 5. 1, 2.
 6. 1, 2, 3, 4.
 9. Retention of sodium and water in patients with essential hypertension is associated with:
 1. Decreased excretion of sodium and water.
 2. Lost potential of sodium and water excretion.
 3. Neuro-dependent retention of sodium and water.
 4. Renoprival retention of sodium and water.
 5. Vasopressin-dependent sodium and water retention.
 10. Renin is:
 1. Hormone produced by the kidneys
 2. Hormone that stimulates the function of the adrenal glands
 3. Enzyme produced by the kidneys
 4. Enzyme that catalyzes ATI-->ATII conversion
 5. 1,2
 6. 3,4
 11. What is the classification of essential hypertension according to the dominant pathogenetic mechanism?
 1. Hyper-, normo- and hypoperfusion.
 2. Hyperkinetic, volume and vasoconstrictory.
 3. High, normal and low-flow.
 4. Hyper-, normo- and hyporeactive.
 5. Stretch- and chemoreceptor dependent.
 12. Vasoconstriction elevates blood pressure by:
 1. Increasing heart rate.
 2. Increasing cardiac output.

3. Increased afterload.
 4. Increased total peripheral resistance.
 5. Generalized tissue hypoperfusion.
13. How the hyperkinetic mechanism increases blood pressure?
1. Venoconstriction increases central blood flow.
 2. Primary hypervolemia.
 3. Positive chronotropic and inotropic effects.
 4. Generalized arterio-venous shunting of blood.
 5. 1, 3.
 6. 1, 2, 4.
14. The main pathogenetic unit of volume-dependent arterial hypertension is:
1. Decreased renal excretion of sodium and water.
 2. Increased sympathetic tone.
 3. Lost baroreceptor depressor mechanism.
 4. Genetically, higher extracellular volume.
 5. Ineffective Darrow-Yannet mechanism.
15. Mandatory factor for stabilization of high blood pressure is:
1. Sustainable increase in extracellular fluid volume.
 2. Increased peripheral vascular resistance inadequate to the actual blood volume.
 3. Inadequately elevated hematocrit.
 4. Shortened circulating time.
 5. Increased cardiac output.
16. Which renal prostaglandin has depressor activity?
1. $\text{Pgf}_{2\alpha}$.
 2. Pge_2 and A_2 .
 3. Pgd and H .
 4. Pga_2 and B_2 .
 5. Endoperoxide.
17. Which are the pathogenic forms of renal hypertension?
1. Acute and chronic.
 2. Latent and manifested.
 3. Renal-parenchymal and renal-vascular.
 4. Compensated and decompensated.
 5. 1, 2.
18. Renal-parenchymal hypertension develops in cases of:
1. Glomerulonephritis, chronic pyelonephritis.
 2. Surgical removal of the kidney.
 3. Fibromuscular hyperplasia of a. renalis.

4. Embolism of a. renalis.
5. Presence of an aberrant vessel.

19. The main pathogenetic mechanism in renal-parenchymal hypertension is:

1. Relatively increased renin activity.
2. Decreased ability of the kidneys to excrete sodium and water.
3. Increased level of renal prostaglandins and kinins.
4. 1, 3.
5. 1, 2.

20. Renal-vascular hypertension develops in:

1. Reduction of the renal parenchyma.
2. Stenosis of a. renalis.
3. Bilateral nephrectomy.
4. Polycystic kidney disease.
5. Acute pyelonephritis.

21. Which is the main pathogenetic mechanism for development of renal-vascular hypertension?

1. Decreased ability of the kidneys to excrete Na^+ and water.
2. Increased concentration of Na^+ in the blood vessel wall.
3. Activation of the renin-angiotensin-aldosterone system.
4. Stimulation of the depressor part of the vasomotor center.
5. Reduced sensitivity of the renal receptors for ADH.

22. Arterial hypertension in atherosclerosis is a result of:

1. Increased Na^+ concentration in the blood.
2. Increased release of pressor factors.
3. Reduced elasticity of the large arterial vessels.
4. Increased pressor effect of the vasomotor center.
5. 1, 4.

23. Arterial hypertension in thyrotoxicosis is:

1. Diastolic type.
2. Systolic /hyperkinetic/ type.
3. Volume type.
4. Blood pressure is not affected in thyrotoxicosis.
5. Systolic-diastolic type.

24. The main hypertensive mechanism in hyperaldosteronism is:

1. Spasm of the peripheral blood vessels.
2. Increased Na^+ and water retention in the body.
3. Sensitization of the vascular wall to pressor factors.

4. Hypertrophy of the vascular wall.
5. Stimulation of the renin-angiotensin system.

25. The main hypertensive mechanism in hyperglucocorticism is:

1. Peripheral blood vessels spasm.
2. Increased Na^+ and water retention in the body.
3. Sensitization of the vascular wall to pressor factors.
4. Hypertrophy of the vascular wall.
5. Altered sensitivity of stretch- and chemoreceptors.

26. Endocrine hypertension in pheochromocytoma is determined by:

1. Increased peripheral vascular resistance.
2. Tachycardia with increased cardiac output.
3. Increased Na^+ and water retention in the body.
4. 1, 2, 3.
5. 1, 2.