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БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ФАРМАКОЛОГИИ

ФАРМАКОЛОГИЯ

PHARMACOLOGY

Тесты
для специальности «Лечебное дело»

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PHARMACOKINETICS. BASIC CONCEPTS

1. The increase in ionization of weak electrolytes causes GIT absorption to:

a) Increase; b) Decrease; c) Stay the same.

2. Intramuscular injections provide high rates of absorption for:

a) Non-polar lipophilic drugs only;
b) Polar hydrophilic drugs only;
c) Both lipophilic and hydrophilic drugs.

3. Elimination half-life period:

a) Time equal to one-half of a full elimination period;

b) Time needed to decrease plasma concentration of a drug by 2 on the exponential part of a pharmacokinetic curve.

4. To accelerate the excretion of weak bases by the kidneys it's necessary to:

a) Alkalinize the urine; ёё
b) Acidify the urine;
c) Maintain neutral pH.

5. Extent of oral drug absorption determines:

a) Clearance;
b) Bioavailability;
c) Ionization constant;
d) Elimination half-life;
e) Elimination rate constant;
f) Volume of distribution.

6. Volume of distribution indicates:

a) The volume of body fluids in which drugs are distributed uniformly;

b) The volume of fluid in which a drug distributes uniformly in a concentration equal to that of blood plasma;

c) The volume of fluid in which a drug distributes uniformly in a concentration equal to that of tissue fluids;

d) The volume of fluid in which a drug distributes uniformly in a therapeutic concentration.

7. Total clearance is characteristic of:

a) Drug absorption;
b) Drug distribution;
c) Drug elimination;
d) Drug deposition.

8. Principal mechanism of drug absorption from the GIT:

a) Active transport;
b) Passive diffusion through a lipid barrier;
c) Diffusion through aqueous pores and intercellular spaces;
d) Microvesicular transport.

9. Determinants of renal clearance:

a) Metabolic transformation;
b) Glomerular filtration;
c) Tubular reabsorption;
d) Tubular secretion;
e) Conjugation.

10. Which of these enteral routes of administration provide absorption into the systemic circulation bypassing or partially bypassing the liver?

a) Oral (swallow);
b) Sublingual;
c) Transbuccal;
d) Into the duodenum;
e) Rectal.

11. Indicate the determinants of hepatic clearance:

a) Rate of biotransformation reactions in the liver;
b) Liver blood flow;
c) Unbound fraction of a drug;
d) Bioavailability;
e) Volume of distribution.

12. The direction of biotransformation reactions in the liver is:

a) A decrease of hydrophilicity;
b) Increase of hydrophilicity;
c) Increase of activity;
d) Decrease of activity;
e) Increase of polarity;
f) Decrease of polarity.

13. Biotransformation of drugs gives metabolites:

a) Which are poorly reabsorbed across the renal tubule;
b) Which are highly reabsorbed across the renal tubule;
c) Which are poorly absorbed from the intestines;
d) Which are highly absorbed from the intestines;
e) Rapidly leave the organism;
f) Slowly leave the organism.

14. Oral bioavailability is determined by:

a) Extent of gastrointestinal absorption;
b) Plasma protein binding;
c) First pass liver metabolism;
d) Rate of distribution throughout the body;
e) Quality of pharmaceutical drug formulation.

15. Liver cirrhosis may alter the pharmacokinetics of drugs:

a) Decreases presystemic elimination;
b) Increases the free fraction of drugs in plasma;
c) Decreases drug clearance;
d) Increases elimination half-life ($T_{1/2}$);
e) Increases bioavailability;
f) Decreases the volume of distribution.

16. Features of rectal route of administration:

a) Is used only in clinics;
b) Influence of digestive enzymes is presence;
c) May be used in unconscious patients;
d) Drugs destroying in the GIT can be applied;
e) Some fraction of a drug bypasses the liver;
f) Needs the trained medical personnel.

17. Features of intravenous route:

a) Maximum accuracy of dosing;
b) Provides highest possible bioavailability;
c) Fast onset of action;
d) Need to sterilize drugs and adhere to aseptic techniques;
e) Plasma steady state concentration of a drug is achieved in 2 half-lives.

18. Which dose of Drug M should be injected to a patient weighting 50 kg to rapidly achieve a plasma concentration of 30 mg/l (Vd = 0,1 l/kg)?

a) 150,0 mg; c) 450,0 mg; e) 900,0 mg;
b) 300,0 mg; d) 750,0 mg; f) 1500,0 mg.

19. Arrange the drugs in ascending order by intestinal absorption rate (pH = 7,2)

a) Weak acid A (pK = 3,5); c) Weak base C (pK = 8,2);
b) Weak acid B (pK = 5,2); d) Weak base D (pK = 7,2).

20. Arrange the drugs with different distribution patterns in ascending order by the loading doses needed to achieve plasma Css = 1 mg/ml (intravenous administration):

a) B (Vd = 2,0 l/kg); c) E (Vd = 4,0 l/kg); e) D (Vd = 1,5 l/kg).
b) C (Vd = 0,5 l/kg); d) A (Vd = 0,2 l/kg);

PHARMACODYNAMIC

1. Intrinsic activity is:

a) Ability to bind to specific receptors;
b) Ability to stimulate specific receptors and cause an effect upon binding;
c) Ability to block specific receptors and cause an effect upon binding;
d) Ability to compete with endogenous ligands for specific receptors.

2. Drugs with low intrinsic activity are called:

a) Agonists-antagonists; c) Antagonists;
b) Partial agonists; d) Full agonists.

3. Drugs with high intrinsic activity are called:

a) Agonists-antagonists; c) Antagonists;
b) Partial agonists; d) Full agonists.

4. Drugs stimulating one receptor subtype and blocking another one are called:

a) Agonists-antagonists; c) Antagonists;
b) Partial agonists; d) Full agonists.

5. Drugs with no intrinsic activity are called:

a) Agonists-antagonists; c) Antagonists;
b) Partial agonists; d) Full agonists.

6. The measure of efficacy:

a) Maximal effective dose;
b) Maximal effect (Emax);
c) The dose that causes maximal effect;
d) Therapeutic range;
e) Therapeutic index.

7. Synergism is:

a) Enhanced effect of a drug combination;
b) Decreased drug effect following by repeated administration;
c) Decreased effect of a drug combination;
d) Decreased effect of a drug following by its prolonged application.

8. Potentiation is:

a) The sum of drug effects;
b) The enhancement of action of one drug by another drug that is inactive;
c) Enhanced effect of a drug following by repeated administration;
d) Kind of drug-drug interaction resulting in an effect that is less than the sum of effects when the drugs are given individually.

9. Antagonism is:

a) Decreased effect following by repeated drug administration;
b) The combined effect of two or more drugs is less than the sum of the effects when the drugs are given individually;
c) The enhancement of action of one drug by another drug that is inactive;
d) Enhanced effect following by dose reduction.

10. Repeated use of drugs leads to the following negative consequences:

a) Cumulation;
b) Tachyphylaxis;
c) Drug dependence;
d) Tolerance;
e) Idiosyncrasy.

11. Accumulation is:

a) A decreased sensibility to a drug following by repeated administration;
b) An increased sensibility to a drug following by repeated administration;
c) An enhanced response to a drug following by repeated administration that results from its cumulation in the body;
d) Unusual drug reactions resulting from congenital enzyme defects;
e) An enhanced biotransformation of a drug following by repeated administration.

12. It is needed 25 mg of diuretic A or 50 mg diuretic of B to increase daily urine output by 2 l. Identify the correct statement:

a) Diuretic A is 2 times more effective than diuretic B;
b) Diuretic B is 2 times more effective than diuretic A;
c) Diuretic A is 2 times more potent (active) than diuretic B;
d) Diuretic B is 2 times more potent (active) than diuretic A;
e) Diuretics A and B are equipotent (active) but differ in efficacy.

13. It is established that ED50 value of diuretics A and B is 1,0 mg/kg. Besides, diuretic A increases daily urine output by 2 l at the highest tested dose and diuretic B — by 1 l. Identify the correct statement:

a) Diuretic A is 2 times more effective than diuretic B, potency (activity) is the same;

b) Diuretic B is 2 times more effective than diuretic A, potency (activity) is the same;

c) Diuretics A and B are equieffective but differ in potency (activity);

d) Diuretic B is 2 times more potent (active) than diuretic A;

e) Diuretics A and B are equieffective but differ in potency (activity).

14. What is tolerance?

a) Individual drug intolerance;

b) Decreased organism sensibility to drugs;

c) Increased organism sensibility to drugs;

d) Drug dependence.

15. Two drugs have opposite effects on the same receptor, it is called as:

a) Antidotism;

b) Physicochemical antagonism;

c) Physiologic antagonism;

d) Pharmacological antagonism;

e) Synergism.

16. Maximal effect is the measure of:

a) Potency (activity);

b) Efficacy;

c) Therapeutic index;

d) Safety;

e)Therapeutic range.

17. Which of these events appear only when drugs are used in combination?

a) Additive effect;

b) Antagonism;

c) Potentiation of action;

d) Sensibilization;

e) Tolerance;

f) Synergism;

g) Idiosyncrasy.

18. Arrange the drugs in descending order by potential hazard:

a) Drug A (TI = 900);

b) Drug B (TI = 10);

c) Drug C (TI = 50);

d) Drug D (TI = 300);

e) Drug E (TI = 100).

19. Arrange the drugs in ascending order by safety. LD50 is 500 mg for each, but ED50 values differ:

a) Drug A (ED50 = 0,01 mg);

b) Drug B (ED50 = 0,1 g);

c) Drug C (ED50 = 5 mg);

d) Drug D (ED50 = 50 mg).

20. Arrange the diuretic drugs in ascending order by efficacy:

a) Drug A (ED50=6,0 mg/kg – Emax=1000 ml/day);

b) Drug B (ED50=80 mcg/kg– Emax=3,0 l/day);

c) Drug C (ED50=0,2 mg/kg– Emax=2,0 l/day);

d) Drug D (ED50=0,01 g/kg – Emax=500 ml/day);

e) Drug E (ED50=10 mcg/kg– Emax=4,0 l/day).

21. Arrange the diuretic drugs in ascending order by potency (activity):

a) Drug A (ED50=0,2 mg/kg– Emax=2,0 l/day);
b) Drug B (ED50=80 mcg/kg– Emax=3,0 l/day);
c) Drug C (ED50=10 mcg/kg– Emax=4,0 l/day);
d) Drug D (ED50=0,01 g/kg – Emax=500 ml/day);
e) Drug E (ED50=6,0 mg/kg – Emax=1000 ml/day).

CHOLINOMIMETIC AND ANTICHOLINESTERASE DRUGS

1. Localization of N-cholinoreceptors:

a) Autonomic ganglions;
b) Postganglionic endings of parasympathetic nerves;
c) Endings of efferent nerve;
d) Chromaffin tissue of adrenal glands;
e) Sino-carotid zone.

2. N-cholinergic receptor is:

a) G-protein-coupled receptor;
b) Ligand-gated channel;
c) Transmembrane protein;
d) Nuclear receptor.

3. M-cholinergic receptor is:

a) G-protein-coupled receptor;
b) Ligand-gated channel;
c) Transmembrane protein;
d) Nuclear receptor.

4. After interaction with the receptor, acetylcholine is:

a) Enzymatically degraded in the synaptic cleft;
b) Eliminated from the body by the kidneys in unchanged form;
c) Metabolized primarily in the liver;
d) Enzymatically degraded in the presynaptic endings.

5. Acetylcholine is destroyed by:

a) Acetylcholinesterase;
b) Acetylcholinesynthase;
c) Acetylcholinearomathase;
d) Acetylcholine dehydrogenase;
e) Is not destroyed by enzymes.

6. Localization of M- cholinergic receptors:

a) Cells of effector organs near the end of postganglionic cholinergic fiber;
b) Neurons of sympathetic ganglions;
c) Neurons of parasympathetic ganglions;
d) Neurons of the spinal cord;
e) Carotid sinus;
f) Chromaffin cells of adrenal medulla;
g) Skeletal muscles.

7. Select M-cholinomimetics:

a) Pilocarpine;
b) Neostigmine;
c) Acetylcholine chloride;
d) Aceclidine;
e) Carbachol;
f) Pyridostigmine bromide;
g) Bethanechol.

8. Select N-cholinomimetics

a) Nicotine;
b) Cytisine;
c) Pilocarpine;
d) Aceclidine;
e) Bethanechol.

9. Select M, N-cholinomimetics of direct action

a) Acetylcholine chloride;
b) Carbachol;
c) Neostigmine;
d) Pyridostigmine bromide;
e) Donepezil.

10. Select M, N-cholinomimetics with indirect action

a) Acetylcholine chloride;
b) Carbachol;
c) Neostigmine;
d) Pyridostigmine bromide;
e) Donepezil.

11. Select Anticholinesterase drugs:

a) Neostigmine;
b) Pyridostigmine bromide;
c) Aceclidine;
d) Edrophonium chloride;
e) Carbachol;
f) Armin;
g) Donepezil.

12. Irreversible cholinesterase inhibitors are:

a) Pyridostigmine bromide;
b) Armin;
c) Donepezil;
d) Organophosphorous compounds;
e) Neostigmine.

13. Effects of acetylcholine are:

a) A decreased heart rate;
b) A decreased secretion of the bronchial glands and the digestive glands;
c) An increased secretion of the bronchial glands and the digestive glands;
d) A contraction of the bronchial muscles;
e) An increased intestine motility;
f) A hypersecretion of the sweat glands;
g) A hyporsecretion of the sweat glands.

14. The mechanism of reduction of ocular hypertension after pilocarpin application is:

a) Opening of the venous sinus, increased outflow of intraocular fluid from the anterior chamber of the eye;

b) Inhibition of the carbonic anhydrase and a decrease production of intraocular fluid.

15. Effects of M-cholinomimetics on the bronchi is:

a) Dilation of bronchi; c) Have no effect on the bronchi.
b) Bronchospasm;

16. Effects of M-cholinomimetics on heart rate is:

a) Increased heart rate;
b) Decreased heart rate;
c) Have no effect on heart rate.

17. Effects of M-cholinomimetics are:

a) A pupil dilatation (mydriasis);
b) A contraction of the pupil (miosis);
c) A decreased of intraocular pressure;
d) A spasm of accommodation;
e) A paralysis of accommodation.

18. Effects of pilocarpine are:

a) A decreases heart rate;
b) An increase in the secretion of the exocrine glands;
c) A decreased secretion of the exocrine glands;
d) Miosis;
e) A reduction of the tone of urinary bladder;
f) A decreased intraocular pressure;
g) An increased intraocular pressure;
h) A spasm of accommodation;
i) A paralysis of accommodation;
j) Mydriasis.

19. Aceclidine:

a) Increases the intraocular pressure;
b) Increases the intestinal tone;
c) Increases the secretion of the digestive glands;
d) Dilates the bronchi;
e) Causes the spasm of accommodation.

20. Acetylcholine chloride:

a) Decreases the intestinal tone;
b) Increase the secretion of the exocrine glands;
c) Increases the secretion of the bronchial glands;
d) Decreases the heart rate;
e) Causes bronchospasm.

21. Anticholinesterase drugs:

a) Inhibit the degradation of acetylcholine;
b) Activate the destruction of acetylcholine;
c) Stimulate the release of acetylcholine;
d) Inhibit acetylcholine release.

22. How do anticholinecterase drugs influence on the action of acetylcholine?

a) Potentiate;
b) Suppress;
c) Make it shorter;
d) Protract.

23. Effect of anticholinesterase drugs on skeletal muscle are:

a) Facilitation of the neuromuscular transmission;
b) Interruption of the neuromuscular transmission;
c) They do not act on neuromuscular transmission;
d) Raising of the muscle tone;
e) Reduction of the muscle tone;
f) They do not act on the muscle tone.

24. Effects of pyridostigmine:

a) Decreases secretion of digestive glands;
b) Bronchospasm;
c) Frequent urination;
d) Increases heart rate;
e) Decreases secretion of exocrine glands;
f) Facilitation of neuromuscular transmission;
g) Interrupt of neuromuscular transmission;
h) Raising of muscle tone;
i) Reduce muscle tone;
j) It does not effect on muscle tone;
k) Decreases the heart rate;
l) Depression of the A-V nodal activity;
m) Decreases the cardiac output;
n) Increases the A-V nodal activity;
o) Increase the cardiac output.

25. Indications for the anticholinesterase drugs:

a) Myasthenia;
b) Glaucoma;
c) Renal colic;
d) Intestinal atony;
e) Asthma;
f) Atony of urinary bladder.

26. Effects of nicotine:

a) Initiation of the inspiratory center;
b) An increase in the intestinal tone;
c) An increase in the heart rate;
d) Suppression of the inspiratory center;
e) A decrease in the intestinal tone.

27. Drugs that can be applied in the case of intestinal and urinary bladder atony:

a) Armin;
b) Pilocarpine;
c) Pyridostigmine bromide;
d) Donepezil;
e) Neostigmine;
f) Aceclidine;
g) Edrophonium chloride.

28. Drugs are used for the treatment of glaucoma:

a) Armin;
b) Pilocarpine;
c) Pyridostigmine bromide;
d) Donepezil;
e) Neostigmine;
f) Aceclidine;
g) Edrophonium chloride.

CHOLINERGIC ANTAGONIST (ANTICHOLINERGIC) DRUGS

1. Pirenzepine is:

a) Antagonist of M_1 receptors;
b) Antagonist of M_2 receptors;
c) Antagonist of M_3 receptors;
d) Agonist of M_1 receptors;
e) Non-selective antagonist of M-receptors.

2. Atropine is:

a) Antagonist of M_1 receptors;
b) Antagonist of M_2 receptors;
c) Antagonist of M_3 receptors;
d) Agonist of M_2 receptors;
e) Non-selective antagonist of M-receptors.

3. Darifenacine is:

a) Antagonist of M_1 receptors;
b) Antagonist of M_2 receptors;
c) Antagonist of M_3 receptors;
d) Agonist of M_3 receptors;
e) Non-selective antagonist of M-receptors.

4. Pipecuronium bromide is:

a) Antagonist of N_M receptors;
b) Antagonist of M_2 receptors;
c) Antagonist of M_3 receptors;
d) Agonist of M_1 receptors;
e) Antagonist of N_N receptors.

5. Trimethaphan is:

a) Antagonist of N_M receptors;
b) Antagonist of M_1 receptors;
c) Antagonist of M_2 receptors;
d) Agonist of M_3 receptors;
e) Antagonist of N_N receptors.

6. Select M-cholinergic antagonists:

a) Atropine;
b) Scopolamine;
c) Homatropine;
d) Trimethaphan;
e) Azamethonium bromide;
f) Darifenacine;
g) Tropicamide;
h) Pipecuronium bromide;
i) Suxamethonium chloride;
j) Trihexyphenidyl;
k) Pirenzepine;
l) Aprophen;
m) Atracurium.

7. N_N-cholinoblockers:

a) Atropine; c) Pirenzepine; e) Azamethonium bromide.
b) Pilocarpine; d) Trimethaphan;

8. N_M-cholinoblockers

a) Pipecuronium bromide; d) Neostigmine;
b) Pancuronium bromide; e) Pyridostigmine.
c) Atracurium;

9. Pharmacological effects of M-cholinergic antagonists:

a) Pupil dilatation (mydriasis) and loss of light reflex;
b) Decreasing of intraocular pressure;
c) Cycloplegia;
d) Bradycardia;
e) Tachycardia;
f) Decreased secretion of exocrine glands;
g) Decreased secretion of bronchial glands.

10. Effect of atropine on eye:

a) Contraction of circular muscle of the iris;
b) Relaxation of the ciliary muscle.

11. Effects of hyoscine hydrobromide on CNS:

a) CNS depression;
b) Pleasure emotions;
c) Paradoxal reaction with hallucinations in toxic doses;
d) No effect.

12. M-cholinergic antagonist used as bronchodilator:

a) Homatropine; d) Ipratropium bromide;
b) Pirenzepine; e) Tropicamide;
c) Trimethaphan; f) Darifenacine.

13. Selective M_3-cholinergic antagonists used to decrease tone of urinary bladder:

a) Propantheline bromide; d) Trepirium iodide;
b) Trihexyphenidyl; e) Suxamethonium chloride;
c) Darifenacine; f) Tolterodin.

14. Indications for administration of M-anticholinergic drug:

a) Intestinal atony;
b) Asthma;
c) Reflex bradycardia;
d) Renal and intestinal colics;
e) Hypersecretion of salivary and bronchial glands;
f) Gastric ulcer and duodenal ulcer;
g) Hypoacid gastritis;
h) Paralysis of accommodation.

15. Atropine:

a) Reduces the heart rate;
b) Increases the secretion of the salivary glands;
c) Decreases the secretion of the salivary glands;
d) Reduces the pupil (miosis);
e) Paralyses the urinary bladder and causes urinary retention;
f) Non-selectively blocks M-cholinergic receptors.

16. Ipratropium bromide:

a) Decreases the motility of the alimentary tact;
b) Decreases the secretion of the bronchial glands;
c) Increases the secretion of the bronchial glands;
d) Dilates the bronchi;
e) Causes bronchospasm.

17. Scopolamine:

a) Increases the intraocular pressure;
b) Intensifies the motility of the gastro-intestinal tract;
c) Increases the secretion of the digestive glands;
d) Relaxes the bronchial smooth muscle;
e) Causes spasm of accommodation.

18. Therapeutic uses of darifenacin:

a) Urinary disorders;
b) Reduction of urinary incontinence;
c) Glaucoma;
d) Decreased secretion of the digestive glands;
e) Bronchial asthma.

19. Therapeutic uses of pirenzepine:

a) Reduction of secretion of the digestive glands;
b) Peptic ulcer;
c) Relieving the urinary incontinence;
d) Bronchial asthma;
e) As mydriatic.

20. Tropicamide:

a) Reduces the intraocular pressure;
b) Increases the intraocular pressure;
c) Causes the spasm of accommodation;
d) Causes the paralysis of accommodation;
e) Reduces the pupil (miosis);
f) Causes pupil dilatation (mydriasis);

21. Therapeutic uses of tropicamide:

a) As mydriatic;
b) As cycloplegic (to prevent hypertrophy of ciliary muscle);
c) In patients with increased intraocular pressure;

d) Treatment of bronchospasm;
e) Treatment of the urinary incontinence.

22. Atropine is used:

a) For the treatment of poisoning with anticholinesterase drugs;
b) For the treatment of sialorrhoea (hypersalivation);
c) Treatment of poisoning with overdosage of muscle relaxant drugs;
d) For the treatment of intestinal atony;
e) In patients with decreased body temperature.

23. Trihexyphenidyl is used:

a) For the treatment of parkinsonism;
b) For the treatment of bronchial asthma;
c) For cycloplegia during testing of refraction;
d) For the treatment of poisoning with muscle relaxant drugs.

24. Drugs applied in case of an overdosage of atropine are:

a) Pyridostigmine bromide;
b) Neostigmine;
c) Acetylcholine chloride;
d) Ipratropium bromide;
e) Pipecuronium bromide.

25. Choose the drugs that are used as cycloplegics (for testing of refraction or to prevent hypertrophy of ciliary muscle):

a) Ipratropium bromide;
b) Pilocarpine;
c) Homatropine;
d) Tropicamide;
e) Atropine.

26. Pharmacological effects of ganglionic blockers:

a) Hypotension (reduction of blood pressure);
b) Intensifying of the motility of the gastro-intestinal tract;
c) Decreased motility of the gastro-intestinal tract;
d) Mydriasis and paralysis of accommodation;
e) Bronchodilatation;
f) Bronchospasm;
g) Decreased secretion of the digestive glands.

27. Clinical applications for ganglionic blockers:

a) Arterial hypertension, hypertensive crisis;
b) Spasm of arteriols;
c) Gastric and duodenal ulcers;
d) For adjustable hypotonia;
e) Pulmonary edema;
f) Cerebral edema.

28. Side effect of ganglionic blockers are:

a) Postural hypotension;
b) Intestinal atony;
c) Miosis;

d) Paralysis of accommodation;
e) Xerostomia;
f) Frequent urination;
g) Intestinal atony;
h) Atony of the urinary bladder.

29. Aid measures in case of respiratory arrest caused by pipecuronium bromide:

a) Introduction of analeptics;
b) Introduction of anticholinesterase drugs;
c) Artificial lung ventilation.

30. Pipecuronium bromide:

a) Facilitates the neuromuscular transmission;
b) Interrupts the neuromuscular transmission;
c) Raises the muscle tone;
d) Reduces the muscle tone;
e) Has no effect on muscle tone.

31. The sequence of muscle relaxation after muscle relaxants application:

a) Hands, feet, limbs muscles;
b) Diaphragm;
c) Intercostal muscles;
d) Oculomotor muscles;
e) Neck and face muscles.

ADRENERGIC DRUGS

1. Specify selective α_1-adrenomimetic:

a) Epinephrine;
b) Dobutamine;
c) Ephedrine;
d) Phenylephrine;
e) Isoprenaline;
f) Salbutamol.

2. Specify selective α_2-adrenomimetic:

a) Amphetamine;
b) Terbutaline;
c) Clonidine;
d) Salmeterol;
e) Norepinephrine.

3. Specify α_1, α_2-adrenomimetic:

a) Norepinephrine;
b) Naphazoline;
c) Dopamine;
d) Isoprenaline;
e) Phenylephrine.

4. Specify sympatomimetic:

a) Phenylephrine;
b) Dobutamine;
c) Ephedrine;
d) Salbutamol;
e) Fenoterol.

5. Isoprenaline causes:

a) Stimulation of α- and β-receptors;

b) Blocking of α- and β-receptors;
c) Selective stimulation of β_1-receptors;
d) Selective stimulation of β_2-receptors;
e) Stimulation of β_1, β_2 and β_3-receptors;
f) Blocking of β_1, β_2 and β_3-receptors.

6. Salbutamol causes:

a) Stimulation of α- and β-receptors;
b) Blocking of α- and β-receptors;
c) Selective stimulation of β_1-receptors;
d) Selective stimulation of β_2-receptors;
e) Stimulation of β_1, β_2 and β_3-receptors;
f) Blocking of β_1, β_2 and β_3-receptors.

7. Localization of sympathetic part of peripheral nervous system:

a) Cranial outflow;
b) Thoracic outflow;
c) Thoracolumbar outflow;
d) Sacral outflow.

8. Localization of α_1-adrenoreceptors:

a) Bronchial smooth muscles;
b) Uterus;
c) Radial muscle of iris;
d) Circular muscle of iris;
e) Gastro-intestinal sphincters;
f) Pilo-motor smooth muscle;
g) Urinary sphincter;
h) Spleen capsule.

9. Localization of α_2 -adrenoreceptors:

a) Cardiac conduction system;
b) Presynaptic nerves;
c) Thrombocytes;
d) Adipose tissue;
e) Bronchial smooth muscle;
f) Radial muscle of iris.

10. Localization of β_1-adrenoreceptors:

a) Blood vessels;
b) Heart;
c) Cardiac conduction system;
d) Bronchial smooth muscle;
e) Uterus;
f) Juxtaglomerular apparatus.

11. Localization of β_2-adrenoreceptors:

a) Blood vessels;
b) Cardiac conduction system;
c) Bronchial smooth muscle;
d) Uterus;
e) Juxtaglomerular apparatus;
f) Blood vessels of skeletal muscle.

12. Localization of D_1-receptors:

a) Blood vessels;
b) Bronchial smooth muscle;
c) Mesenteric vessels;
d) Adipose tissue;
e) Blood vessels of the kindey;
f) Intestinal tract.

13. Effects associated with the activation of α_1-adrenoceptor:

a) Constriction of blood vessels;
b) Dilation of blood vessels;
c) Myosis;
d) Decreased blood pressure;
e) Reflex bradycardia;
f) An increase in tone GI sphincter;
g) Mydriasis;
h) An increase in arterial pressure.

14. Effects of activation of α_2-receptors:

a) An increase in NE release;
b) A decrease in NE release;
c) An increase in the heart rate;
d) Activation of platelet adhesion;
e) Decreased platelet adhesion;
f) Lipolysis inhibition.

15. Stimulation of β_1-adrenergic receptors causes the following changes in the indices of the heart:

a) An increase in the heart rate and myocardial contractility;
b) A decrease in the excitability;
c) An increase in automaticity and conduction velocity;
d) Decrease in automaticity and conduction velocity;
e) An increase in the cardiac output;
f) A decrease in the cardiac output;
g) A decrease in the heart rate and myocardial contractility;
h) An increase in excitability.

16. Effect of activation of β_1-receptors:

a) Increased renin secretion;
b) Decreased renin secretion;
c) Increased arterial pressure;
d) Decreased arterial pressure;
e) Bronchospasm;
f) Bronchodilation;
g) Increased basal metabolism;
h) Decreased basal metabolism;
i) Increased glycogenolysis;
j) Decreased glycogenolysis;
k) Lipolysis activation;
l) Lipolysis inhibition.

17. Effect of activation of β_2-receptors:

a) Increased heart rate;
b) Vasodilation;
c) Bronchodilation;
d) Increased tone and contractile activity of the myometrium;
e) Decreased tone and contractile activity of the myometrium;
f) Increased glycogenolysis.

18. Effect of activation of β_3-receptors:

a) Increased glycogenolysis;
b) Decreased glycogenolysis;
c) Lipolysis activation;
d) Increased blood free fatty acids;
e) Hyperglycemia;
f) Hypoglycemia.

19. Effect of activation of D_1-receptors:

a) Reduction of the tone of blood vessels in skeletal muscles, kidney, GIT, heart, CNS;

b) An increase in the tone of blood vessels in skeletal muscles, kidney, GIT, heart, CNS;

c) Increased heart rate;
d) Decreased heart rate.

20. Drugs are applied for the treatment of asthma:

a) Propranolol;
b) Ephedrine;
c) Norepinephrine;
d) Isoprenaline;
e) Xylometazoline;
f) Salbutamol;
g) Fenoterol.

21. Drugs are locally applied in rhinitis:

a) Propranolol;
b) Oxymetazoline;
c) Ephedrine;
d) Isoprenaline;
e) Phenylephrine;
f) Salbutamol;
g) Xylometazoline.

22. Drugs are used for the treatment of arterial hypotension:

a) Phenylephrine;
b) Epinephrine;
c) Ephedrine;
d) Salbutamol;
e) Dobutamine;

23. β_1-Agonists are used to treating the following diseases:

a) Hypotension;
b) Bronchial asthma;
c) Arrhythmia;
d) Atrioventricular heart block;
e) Congestive cardiac failure.

24. Correct statements about epinephrine:

a) It is the transmitter in the sympathetic system;
b) Synthesis of catecholamines begins with the amino acid tyrosine;
c) Mediate negative-feedback control on NE secretion;
d) The all epinephrine gets inactivation in liver by catechol-O-methyl-trans ferase (COMT).

25. Epinephrine has the following effects:

a) Cardiac stimulation;
b) Constriction blood vessels of the muscle;
c) Constriction blood vessels of the skin;
d) Bronchodilatation;
e) Hyperglycemia.

26. Epinephrine is used for:

a) Essential hypertension;
b) Anaphylactic shock;
c) Bronchial asthma;
d) Arteritis obliterans;
e) Cardiac resuscitation;
f) Hypoglycemia;
g) Extension of the duration of local anaesthesia.

27. Dopamine has the following features:

a) Stimulation of only dopamine-receptor;
b) Dilates renal blood vessels;

c) May cause severe heart failure with renal impairment;
d) Cross the BBB;
e) Route of administration is orally only.

28. Dopamine is used for treating the following diseases:

a) Congestive cardiac failure;
b) Essential hypertension;
c) Hypotension;
d) Cardiogenic shock;
e) Bronchial asthma;
f) Oligouric shock.

29. Correct statements about ephedrine:

a) Releases NE from sympathetic nerve endings;
b) Administer orally;
c) The duration of its action is less than epinephrine's one;
d) The onset of action is slower than epinephrine has;
e) It has a more pronounced effect on the central nervous system than epinephrine.

30. Drugs that can cause bronchodilation:

a) Epinephrine;
b) Ephedrine;
c) Phenylephrine;
d) Isoprenaline;
e) Salbutamol.

ADRENERGIC ANTAGONISTS

1. β_1-adrenergic antagonist, which additionally stimulates NO (nitrogen oxide) release:

a) Sotalol;
b) Nadolol;
c) Nebivolol;
d) Pindolol.

2. β_1, β_2-adrenergic antagonist with intrinsic sympathomimetic activity (ISA):

a) Metoprolol;
b) Pindolol;
c) Labetalol;
d) Phentolamine.

3. β_1-adrenergic antagonist with intrinistic sympathomimetic activity (ISA):

a) Tamsulosin;
b) Pindolol;
c) Propranolol;
d) Acebutolol.

4. Used to treat glaucoma:

a) Propranolol;
b) Yohimbine;
c) Timolol;
d) Guanethidine.

5. For the treatment of benign prostatic hyperplasia (BPH) used:

a) Phentolamine;
b) Prazosin;
Tamsulosin;
Carvedilol.

6. Alfa-adrenergic (both selective and non-selective) antagonists:

a) Nadolol;
b) Prazosin;
c) Labetalol;
d) Yohimbine;
e) Clonidine;
f) Phentolamine;
g) Metoprolol;
h) Tamsulosin;
i) Dihydroergotamine;
j) Guanethidine.

7. α_2-adrenergic antagonist:

a) Tamsulosin; c) Yohimbine;
b) Carvedilol; d) Timolol.

8. Beta-adrenergic (both selective and non-selective) antagonists:

a) Reserpine; e) Guanethidine; h) Doxazosin;
b) Prazosin; f) Terazosin; i) Atenolol;
c) Propranolol; g) Nadolol; j) Metoprolol.
d) Nebivolol;

9. Mixed-action (alfa and beta) adrenergic antagonists:

a) Guanethidine; c) Labetalol; e) Carvedilol;
b) Phentolamine; d) Timolol; f) Dihydroergotamine.

10. Sympatholytics:

a) Guanethidine; c) Prazosin; e) Sotalol.
b) Yohimbine; d) Reserpine;

11. α_1-adrenergic antagonists:

a) Nadolol; c) Phentolamine; e) Doxazosin;
b) Prazosin; d) Tamsulosin; f) Labetalol.

12. α_1, α_2-adrenergic antagonists:

a) Propranolol; c) Phentolamine; e) Dihydroergotamine.
b) Terazosin; d) Acebutolol;

13. β_1, β_2-adrenergic antagonists without intrinsic sympathomimetic activity (ISA):

a) Propranolol; e) Doxazosin; h) Guanethidine;
b) Phentolamine; f) Sotalol; i) Timolol;
c) Carvedilol; g) Prazosin; j) Phenylephrine.
d) Nadolol;

14. Selective β_1-adrenergic antagonists without intrinsic sympathomimetic activity (ISA):

a) Sotalol; e) Phentolamine; h) Propranolol;
b) Metoprolol; f) Bisoprolol; i) Nebivolol;
c) Atenolol; g) Timolol; j) Dihydroergotamine.
d) Reserpine;

15. α-adrenergic antagonists decrease:

a) Bronchi tone; b) Vascular tone;
c) Heart rate;
d) Blood pressure;
e) Smooth muscle tone in the neck of urinary bladder and prostatic urethra.

16. β-adrenergic antagonists decrease:

a) Heart rate; d) Myocardial contractility;
b) Bronchi tone; e) Automaticity;
c) Vascular tone; f) Secretion renin.

17. β-adrenergic antagonists may increase:

a) Heart rate;
b) Vascular tone;
c) Secretion of intraocular fluid;
d) Bronchi tone;
e) Activity of the myometrium;
f) Myocardial oxygen demand.

18. Effects of propranolol:

a) Decrease automaticity;
b) Atrioventricular conduction delay;
c) Release of glucose;
d) Decrease blood pressure;
e) Increase renin secretion;
f) Increase tone and contractile activity of the myometrium.

19. Timolol decrease:

a) Blood pressure;
b) Myocardial oxygen demand;
c) Activity and tone of the myometrium;
d) Automatism of heart;
e) Intraocular fluid;
f) Bronchi tone.

20. Labetalol increase:

a) Heart rate and contractility;
b) Bronchi tone (in patient with asthma);
c) Cardiac output;
d) Vascular tone;
e) Blood pressure.

21. Effects of reserpine:

a) Decrease blood pressure;
b) Bradycardia;
c) Reduces the secretion of gastric acid;
d) Increases the release of gastric acid;
e) Increase motion of the gastro-intestinal tract;
f) Induction CNS;
g) Sedation.

22. Indications for use of α-adrenergic antagonists:

a) Hypotension;
b) Arterial hypertension;
c) Spasms of peripheral blood vessels;
d) Pheochromocytoma;
e) Prostatic hyperplasia.

23. Indications for use of β-adrenergic antagonists:

a) Hypotension;
b) Arterial hypertension;
c) Atherosclerotic cardiovascular disease;
d) Delayed atrioventricular conduction;
e) Bronchial asthma;
f) Tachyarrhythmia.

24. Indications for use of labetalol:

a) Hypertensive crisis;
b) Arterial hypertension;
c) Tachyarrhythmia;
d) Open-angle glaucoma;
e) Pheochromocytoma.

25. Drugs for the treatment of arterial hypertension:

a) Doxazosin;
b) Aceclidine;
c) Metoprolol;
d) Physostigmine;
e) Phenylephrine;
f) Prazosin;
g) Ephedrine;
h) Labetalol;
i) Propranolol;
j) Reserpine.

26. Side effects of α-adrenergic antagonists:

a) Bronchospasm;
b) Tachycardia;
c) Depress A–V nodal activity;
d) Postural hypotension;
e) Mydriasis.

27. Side effects of β_1,β_2-adrenergic antagonists:

a) Bradycardia;
b) Depress A–V nodal activity;
c) Vasoconstriction;
d) May cause bronchospasm;
e) Decrease tone and contractile activity of the myometrium;
f) Intestinal atony.

28. Side effects of β_1-adrenergic antagonists:

a) Bradycardia;
b) Depress A–V nodal activity;
c) Increase cardiac failure;
d) Vasoconstriction;
e) Bronchospasm;
f) Increase tone and contractile activity of the myometrium.

29. Drugs that cause postural hypotension:

a) Prazosin;
b) Phentolamine;
c) Propranolol;
d) Atenolol;
e) Labetalol.

30. Side effects of α,β-adrenergic antagonists:

a) Postural hypotension;
b) Bradycardia;
c) Depress A–V nodal activity;
d) Increase cardiac failure;
e) May cause bronchospasm;
f) Vasoconstriction.

DIURETIC DRUGS

1. Localization of action of thiazide and thiazide-like diuretics in nephron:

a) Proximal renal tubules;
b) The ascending part of Henle's loop;
c) Distal renal tubules (final part);

d) Distal renal tubules (initial part);
e) Collector renal tubules.

2. Localization of action of furosemide and bumetanide in nephron:

a) Proximal renal tubules;
b) The ascending part of Henle's loop;
c) Distal renal tubules;
d) Collector renal tubules;
e) The descending part of Henle's loop.

3. Localization of action of potassium-sparing diuretics in nephron:

a) Proximal renal tubules;
b) The ascending part of Henle's loop;
c) Distal renal tubules (initial part);
d) Collector renal tubules;
e) Glomerulus.

4. Localization of action of osmotic diuretics in nephron:

a) Acting on the all nephron;
b) The ascending part of Henle's loop;
c) Distal renal tubules (initial part);
d) Collector renal tubules;
e) Only proximal renal tubules.

5. Pharmacodynamic features of hydrochlorthiazide:

a) Inhibits reabsorption of Na^+, Cl^-, HCO_3^- ions;
b) Remains K^+-ions in the organism;
c) Effects lasts for 4–8 hours;
d) Effects lasts more than 24 hours;
e) Increases the action of antihypertensive drugs;
f) Increases the reabsorption of Ca^{2+} ions.

6. Properties of furosemide are as follows:

a) Low speed of onset;
b) Short duration of the effect (2–4 hours);
c) High diuretic potency;
d) Decreasing of the blood pressure;
e) Increasing of the reabsorption of Ca^{2+} и Mg^{2+} ions;
f) Acts on the proximal renal tubules.

7. Properties of spironolactone:

a) Decreases K^+ ions excretion;
b) Delays the Na^+ ions excretion;
c) Has high efficacy;
d) High speed of onset;
e) Blocks the synthesis of aldosterone;
f) Clinical uses include condition of increased aldosterone secretion.

8. Properties of acetazolamide:

a) Decreases K^+ ions excretion;
b) Inhibits the reabsorption of Na^+, Cl^-, HCO_3^- ions;
c) Clinical uses include condition of increased aldosterone secretion;
d) Clinical uses include glaucoma;
e) Long-term application can causes the acidosis;
f) Deafness is typical adverse effect.

9. Mannitol:

a) Inhibits Na^+-K^+-$2Cl^-$co-transporter in the thick ascending part of Henle's loop;
b) Increases osmotic pressure in the renal tubules;
c) Can be used as dehydrator;
d) Is indicated for forced diuresis;
e) Is indicated in the case of chronic heart failure;
f) Is well absorbed in the intestine.

10. The following drugs can be used for the treatment of arterial hypertension:

a) Hydrochlorothiazide;
b) Furosemide;
c) Mannitol;
d) Spironolactone;
e) Acetazolamide;
f) Indapamide.

11. The following drugs can be used for forced diuresis:

a) Bendroflumethiazide;
b) Triamterene;
c) Spironolactone;
d) Mannitol;
e) Furosemide;
f) Metolazone.

12. Choose the practical combinations of diuretics:

a) Furosemide+acetazolamide;
b) Hydrochlorthiazide+amiloride;
c) Amiloride+triamterene;
d) Triamterene+chlorthalidone;
e) Spironolactone+hydrochlorthiazide;
f) Mannitol+acetazolamide.

13. Hydrochlorthiazide and furosemide can be combined for the following purposes:

a) Prophylaxis of hypercalcemia;
b) Prophylaxis of hypokalemia;
c) Increasing the duration of action;
d) Changing the pH of the urine;
e) Inhibition of the secretion of aldosterone.

14. Indications of loop diuretics:

a) Edema, caused by heart failure;
b) Pulmonary edema;
c) Acute hypercalcemia;
d) Hypokalemia caused by thiazide diuretics;
e) Brain edema;
f) Elevated antidiuretic hormone.

15. Indications of thiazide diuretics:

a) Nephrogenic diabetes insipidus;
b) Hypertension;
c) Congestive heart failure;
d) For forced diuresis;
e) Idiopathic calciuria;
f) Toxic pulmonary edema.

ANTIHYPERTENSIVE DRUGS

1. Arterial blood pressure is directly proportionate to:

a) Cardiac output and peripheral vascular resistance;
b) Heart rate and peripheral vascular resistance;
c) Stroke volume and heart rate;
d) Cardiac output and heart rate;
e) All answer choices are not correct.

2. What antihypertensive drug can block the production of renin?

a) Prazosin; c) Captopril; e) Diazoxide;
b) Metoprolol; d) Sodium nitroprusside; f) Clonidine.

3. What diuretic should be prescribed in case of hypertensive crises complicated by pulmonary edema?

a) Furosemide; c) Triamterene; e) Bendroflumethiazide;
b) Indapamide; d) Mannitol; f) Chlortalidone.

4. Targets of antihypertensive drugs are:

a) β-adrenergic receptors;
b) α_2-adrenergic receptors;
c) I_1-imidazoline receptors;
d)α_1-adrenergic receptors;
e) angiotensin-II receptors;
f) N_m-cholinergic receptors.

5. Mechanisms of hypotensive action of diuretics:

a) Reduction of the circulating blood volume;
b) Increase in the synthesis of vasolitic prostaglandins in the kidney;
c) Reduction of the vessel response to vasoconstrictors;
d) For some diuretics – direct vasolytic action;
e) A decrease in the heart rate.

6. Typical side-effects of thiazides and thiazide-like diuretics:

a) Electrolyte disturbances;
b) Dry cough, rashes;
c) Swellings;
d) Hyperglycemia;
e) Hyperlipidemia;
f) Hyperuricemia.

7. Counter indications of ACE-inhibitors:

a) Pregnancy;
b) Bilateral renal artery stenosis;
c) Hyperpotassemia;
d) Heart failure;
e) Hypopotassemia.

8. Clonidine:

a) Has analgesic activity;
b) Is precursor of norepinephrine;
c) Rapid infusion can lead to a shortly increased blood pressure;
d) Has effects of anxiolytic, sedative drug and amnesia;
e) Can treat withdrawal symptoms in opioid and alcohol addicts.

9. Non-selective β-adrenergic blockers shouldn't be applicated in patients with bronchial asthma and chronic obstruction pulmonary disease because of:

a) Block of β_2-adrenergic receptors can lead to bronchospasm;
b) Stimulation of gland secretion;
c) Intensification of pulmonary blood supply;
d) Negative influence on gas exchange;
e) Inhibition of the cells respiration.

10. Methyldopa:

a) Is first-line antihypertensive drug during pregnancy;
b) Can cause orthostatic hypotension;
c) Is used for relief of hypertensive crises;
d) Has the same final effect as clonidine;
e) Does not pass through blood-brain barrier.

11. The main aims of treatment of arterial hypertension:

a) Reduce blood pressure to lower the point of 140/90 mmHg;
b) Prevention of eventual end-organ damage (heart, kidney, brain);
c) Prevention of cardiovascular complications, increasing the life expectancy;
d) Relief the hypertensive crises, everything else does not matter;
e) Keep blood pressure at the level of feeling well, without complaints.

12. During the treatment of arterial hypertension with α-adrenergic antagonists can be:

a) Reflex tachycardia;
b) Bradycardia;
c) Increased plasma concentrations of very-low-density lipoproteins;
d) Decreased sympathetic influence;
e) Improvement of blood supply in peripheral arteries.

13. Ganglionic blockers can be used in case of:

a) Long-term treatment of arterial hypertension;
b) Relief of hypertensive crises;
c) Controlled hypotension;

d) Increase in blood pressure in patients with collapse;
e) Ganglionic blockers do not change the blood pressure.

14. What is the mechanism of action of calcium channel blockers (one answer)?

a) Interact with membrane phospholipid and inhibit ion transport;
b) Block the Na^+/K^+ ATPase in smooth muscles and heart;
c) Interact with definite domen of calcium L-type channel;
d) Decrease the Ca^{2+} influx as a result of interactions with sodium-channels;
e) Disturb the actin-myosin interaction.

15. Side-effects of vasodilating calcium channel blockers:

a) Ankle swellings
b) Head ache
c) Bradycardia
d) Reflex tachycardia

ANTIANGINAL AND HYPOLIPIDEMIC DRUGS

1. Atenolol:

a) Cardioselective β-adrenergic antagonists;
b) Has intrinsic symphatomimetic activity;
c) Pass through blood-brain barrier;
d) Dilate coronary vessels;
e) Can be used for relief of angina attacks.

2. Verapamil:

a) Can be applicated to treat vasospastic (or variant) angina pectoris;
b) Speed up the conduction through the AV node;
c) Increase the heart rate;
d) Dilate all vessels except coronary;
e) Is used for relief of angina attacks.

3. Mechanism of antianginal effect of isosorbide mononitrate:

a) Blocks the calcium channels;
b) Activates the potassium channels;
c) Releases of nitric oxide (NO);
d) Blocks β-adrenergic receptors;
e) Blocks α-adrenergic receptors.

4. Define the antianginal drugs:

a) Metoprolol;
b) Clonidine;
c) Isosorbide mononitrate;
d) Enalapril;
e) Indapamide;
f) Amlodipin.

5. β-adrenergic antagonists:

a) Dilate coronary vessels;
b) Dilate large veins, decrease the amount of blood returned to the heart;
c) Increase the myocardial oxygen supply;
d) Decrease the myocardial oxygen demand;
e) Decrease heart rate and contractility.

6. Propranolol:

a) Selective β_1-adrenoreceptor blockers;
b) Antagonist with intrinsic sympathomimetic activity;
c) Can cause bronchospasm;
d) Passes into CNS, causes depression;
e) Dilates coronary vessels.

7. Metoprolol:

a) Cardioselective β-adrenergic antagonist;
b) Passes through blood-brain barrier;
c) Dilates coronary vessels;
d) Does not change heart rate;
e) Causes «coronary steal phenomenon».

8. Side-effects of propranolol:

a) Disturbance of atrioventricular conduction;
b) Bronchospasm;
c) Depression, sedation, sleeplessness;
d) An increase in blood pressure;
e) An increase in intraocular pressure.

9. The preload and the afterload are decreased by:

a) Metoprolol;
b) Verapamil;
c) Nitroglycerin;
d) Isosorbide mononitrate;
e) Trinitrolong.

10. Reflex tachycardia is caused by:

a) Isosorbide dinitrate;
b) Metoprolol;
c) Nifedipine;
d) Verapamil;
e) Amlodipin.

11. Atrioventricular conduction can be disturbed by:

a) Nitroglycerin;
b) Atenolol;
c) Verapamil;
d) Trimetazidine;
e) Molsidomine.

12. Amlodipin:

a) Is vasodilating calcium channel blocker;
b) Has antiarrhythmic activity;
c) Causes increased plasma concentrations of very-low-density lipoproteins;
d) Has antihypertensive activity;
e) Can cause reflex tachycardia.

13. Nicorandil:

a) Is a nicotinamide nitrate ester;
b) Decreases the preload and afterload;
c) Potassium channels activator;
d) Is the first-line drug for relief of angina attack;
e) Blocks β-adrenergic receptors.

14. Common properties of propranolol and verapamil:

a) Decrease force of myocardial contraction;
b) Decrease myocardial oxygen demand;
c) Cause coronary steal phenomenon;
d) Inhibit atrioventricular conduction;
e) Can cause bronchospasm.

15. First-line drugs for pain relief in case of myocardial infarction:

a) Morphine; c) Fentanyl; e) Validol.
b) Metamizole; d) Keterolac;

DRUGS USED FOR THE TREATMENT OF HEART FAILURE

1. ACE inhibitors are the first-line drugs in the treatment of chronic heart failure because of:

a) Retard remodeling and cardiac hypertrophy;
b) Deftly manage with control of drug plasma concentration;
c) Improvement of pump heart function, that's why improvement of clinical symptoms;
d) High tolerability and low cost;
e) They can be applied one time a day.

2. The main benefit of β-adrenergic antagonists in the treatment of chronic heart failure:

a) Reduction of heart remodeling and improvement of prognosis;
b) Improvement of clinical symptoms and quality of life;
c) An increase of pump heart function;
d) High tolerability and low cost; e) Monotherapy.

3. Correct assumptions about diuretic usage in the treatment of chronic heart failure:

a) Indication is clinical symptoms of congestion (start with class II failure);
b) Loop diuretics are prefer;
c) Reduce the heart remodeling;
d) Improve the prognosis because retard the progress of chronic heart failure;
e) Pulse-therapy is effective only.

4. The main groups of drugs in the treatment of chronic heart failure:

a) Renin-angiotensin system inhibitors;
b) Diuretic drugs;
c) Cardiac glycosides;
d) β-adrenergic antagonists;
e) Vasodilators;
f) Calcium channel blockers.

5. Miscellaneous groups of drugs in the treatment of chronic heart failure:

a) Cytoprotective agents;
b) Diuretic drugs;
c) Antiplatelet drugs;
d) β-adrenergic antagonists;
e) Vasodilators;
f) Calcium channel blockers.

6. For the following ACE inhibitors improvement of prognosis in the treatment of chronic heart failure are provided:

a) Trandalopril; c) Enalapril; e) Lisinopril;
b) Captopril; d) Ramipril; f) Fosinopril.

7. Potassium chloride is indicated in the treatment of digoxin toxicity because of:

a) High level of potassium inhibits glycoside's binding to Na^+-K^+-ATPase;
b) High level of potassium induces glycoside's binding to Na^+-K^+-ATPase;
c) High level of potassium increases Ca^{2+} level in myocyte cells;
d) High level of potassium induces conduction from atriums to ventricles;
e) Potassium chloride is counter-indicated in the treatment of digoxin toxicity.

8. Effects of the treatment of chronic heart failure with cardiac glycosides:

a) Improve of prognosis; d) Improve quality of life;
b) Slow down the progression of disease; e) Extend life span.
c) Clinical benefits;

9. Angiotensin-converting-enzyme inhibitors with long-term action (can be applicated one time a day):

a) Captopril; c) Lisinopril; e) Trandolapril.
b) Amlodipine; d) Ramipril;

10. Cardioselective β-adrenergic antagonists:

a) Bisoprolol; c) Carvedilol; e) Atenolol.
b) Metoprolol; d) Propranolol;

11. Drugs increasing myocardial contractility and are phosphodiesterase inhibitors:

a) Dopamine; c) Milrinone; e) Vesnarinone.
b) Dobutamine; d) Enoximone;

12. Effective measures in the treatment of digoxin toxicity are:

a) Infusion of unithiol;
b) Infusion of potassium chloride;
c) Treatment of AV-block with atropine;
d) Treatment with ventricle arrhythmias with lidocaine;
e) Renal dialysis;
f) Infusion of drugs containing Ca^{2+}.

13. Excess of dose over mean therapeutic dose of dopamine can cause:

a) An increase in peripheral vascular resistance;
b) A decrease in blood pressure;
c) Arrhythmias;
d) Tachycardia;
e) Orthostatic collapse;
f) Angina attack in patients with chronic heart failure.

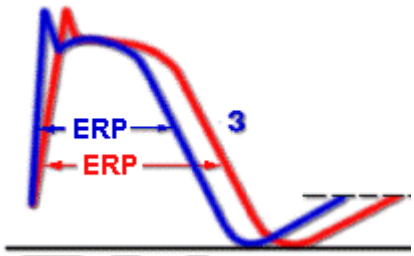
14. Counter indications of cardiac glycosides:

a) Tachyarrhythmical form of continuous arrhythmia;
b) Heart failure;
c) Supraventricular tachycardia;
d) AV block;
e) Ventricular extrasystole;
f) Bradycardia.

15. Unithiol can be used in the treatment of digoxin toxicity because:

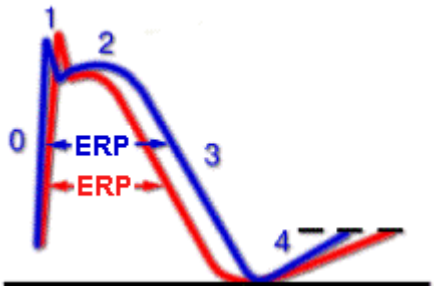
g) Stimulates of function of troponin complex proteins in cardiomyocytes;
h) Force the metabolism of glycosides in the liver;
i) Derease the Ca^{2+} influx in cardiomyocytes;
j) Recover the SH-groups of Na^{+}-K^{+}-ATPase in cardiomyocytes.

ANTIARRYTHMIC DRUGS



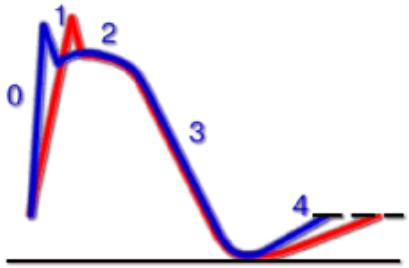
1. This picture shows the change of action potential during the treatment of antiarrhythmic drugs of the class:

a) IB; b) IA; c) IC; d) II; e) IV.



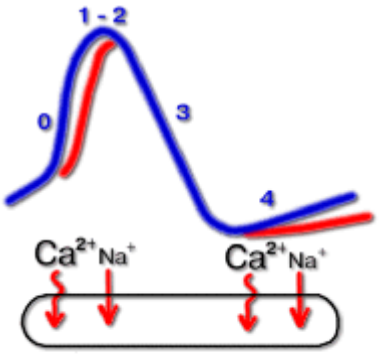
2. **This picture shows the change of action potential during the treatment of antiarrhythmic drugs of the class:**

a) IB; b) IA; c) IC; d) III; e) IV.



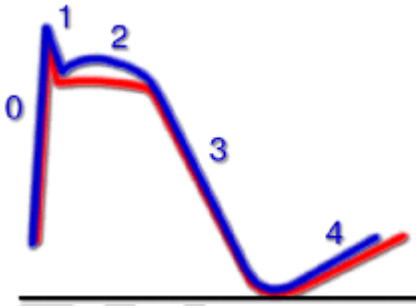
3. **This picture shows the change of action potential during the treatment of antiarrhythmic drugs of the class:**

a) IB; b) IA; c) IC; d) II; e) III.



4. **This picture shows the change of action potential during the treatment of antiarrhythmic drugs of the class:**

a) IB; b) IA; c) IC; d) III; e) IV.



5. **This picture shows the change of action potential during the treatment of antiarrhythmic drugs of the class:**

a) IB; b) IA; c)IC; d) II; e) IV.

6. Define correct assertions about antiarrhythmic drugs with class IV:

a) By blocking voltage-gated sodium channels they slow the phase 0 of action potential;

b) They block calcium channels;

c) They slow conduction through SA and AV nodes;

d) They facilitate the potassium channels gating, it leads to shortening of effective refractory period;

e) They block β_1-adrenergic receptors, it decreases automatism of SA and AV nodes.

7. Define correct assertions about antiarrhythmic drugs with class IA:

a) By blocking voltage-gated sodium channels they slow the phase 0 of action potential;

b) By blocking potassium channels they prolong repolarization and effective refractory period;

c) They slow conduction through SA and AV nodes by blocking calcium channels;

d) They facilitate the potassium channels gating, it leads to shortening of effective refractory period;

e) They block β_1-adrenergic receptors, it decreases automatism of SA and AV nodes.

8. Define correct assertions about antiarrhythmic drugs with class IB:

a) By blocking voltage-gated sodium channels they slow the phase 0 of action potential;

b) By blocking potassium channels they prolong repolarization and effective refractory period;

c) They slow conduction through SA and AV nodes by blocking calcium channels;

d) They facilitate the potassium channels gating, it leads to shortening of effective refractory period;

e) They block β_1-adrenergic receptors, it decreases automatism of SA and AV nodes.

9. Define correct assertions about antiarrhythmic drugs with class IC:

a) By blocking voltage-gated sodium channels they slow the phase 0 of action potential;

b) By blocking potassium channels they prolong repolarization and effective refractory period;

c) They slow conduction through SA and AV nodes by blocking calcium channels;

d) They do not change the duration of effective refractory period;

e) They block β_1-adrenergic receptors, it decreases automatism of SA and AV nodes.

10. Define correct assertions about antiarrhythmic drugs with class II:

a) By blocking voltage-gated sodium channels they slow the phase 0 of action potential;

b) By blocking potassium channels they prolong repolarization and effective refractory period;

c) They slow conduction through SA and AV nodes by blocking calcium channels;

d) They block β_1-adrenergic receptors;

e) They decrease automatism of SA and AV nodes.

11. What antiarrhythmic drugs bind with voltage-gated sodium channels firmly?

a) Antiarrhythmic drugs with class IA;

b) Antiarrhythmic drugs with class IB;

c) Antiarrhythmic drugs with class IC;

d) All antiarrhythmic drugs with class I;

e) Antiarrhythmic drugs with class I do not bind with sodium channels at all.

12. Antiarrhythmic drugs that dissociate from the channel with rapid kinetics are:

a) Drugs with class IA;

b) Drugs with class IB;

c) Drugs with class IC;

d) All antiarrhythmic drugs with class I;

e) Antiarrhythmic drugs with class I do not bind with sodium channels at all.

13. Amiodarone:

a) Blocks voltage-gated sodium channels (slows the phase 0 of action potential)

b) By blocking potassium channels it prolongs repolarization and effective refractory period

c) Blocks calcium channels

d) It facilitates the potassium channels gating, it leads to shortening of effective refractory period

e) It blocks β_1-adrenergic receptors, it decreases automatism of SA and AV nodes

14. Side effects of amiodarone:

a) AV block;

b) Dysfunction of thyroid gland;

c) Corneal microdeposits;

d) A gray-blue skin discoloration;

e) Photosensibilization, photodermatitis;

f) Arterial hypertension.

15. Side effects of drugs with class II:

a) Bronchospasm;

b) Bradycardia;

c) An increase in blood pressure;

d) AV block;

e) Heart failure;

f) An increase in intraocular pressure.

GENERAL ANESTHETICS. ETHYL ALCOHOL. ANTICONVULSANTS. ANALGETICS

1. Definition of general anesthesia includes all the following except of:

a) Analgesia;
b) Amnesia;
c) Psychostimulation;
d) Skeletal muscle relaxation;
e) Unconsciousness.

2. Minimal Alveolar Concentration (MAC) of inhaled anesthetics is:

a) Concentration of inhaled anesthetics in inspired gas to prevent a response to a surgical incision over 50 % (effect of analgesia occurs);

b) Concentration of inhaled anesthetics in inspired gas to prevent a response to a surgical incision in the proximity of 100 % (effect of analgesia occurs);

c) Concentration of inhaled anesthetics in the blood causing apnea in the proximity of 50 %;

d) Concentration of inhaled anesthetics in inspired gas causing surgical anesthesia in the proximity of 50 %.

3. Ideal anesthetic drug should:

a) Induce slow general anesthesia and be rapidly reversible upon discontinuation;

b) Induce rapid general anesthesia and be slowly reversible upon discontinuation;

c) Induce rapid general anesthesia and be rapidly reversible upon discontinuation;

d) Induce slow general anesthesia and be slowly reversible upon discontinuation;

e) Speed of induction of general anesthesia make no difference.

4. Type of general anesthesia, based on combination of general anesthetics with drugs potentiated them (opioid analgesics, anxiolytics, skeletal muscle relaxants and others) is:

a) Mixed anesthesia;
b) Potentiated anesthesia;
c) Basis anesthesia;
d) Induction of anesthesia;
e) Neuroleptanalgesia.

5. Method of general anesthesia beginning that provides rapid, safety and effective loss of consciousness, analgesia and skeletal muscle relaxation:

a) Mixed anesthesia;
b) Potentiated anesthesia;
c) Basis anesthesia;
d) Induction of anesthesia;
e) Neuroleptanalgesia.

6. Type of general anesthesia occurring by usage of two or more general anesthetics at the same time is:

a) Mixed anesthesia;
b) Potentiated anesthesia;
c) Basis anesthesia;
d) Induction of anesthesia;
e) Neuroleptanalgesia.

7. Features of halothane:

a) Has high narcotic activity;
b) General anesthesia occurs rapidly — in 3–5 minutes;
c) Mild stage of excitement;
d) Recovery is rapid;
e) Explosive.

8. Side-effects of halothane:

a) Tachycardia;
b) Bradycardia;
c) Arrhythmias;
d) An increase in blood pressure;
e) Hypotension.

9. Nitrous oxide:

a) Has high narcotic activity;
b) Has low narcotic activity;
c) Has high analgesic activity;
d) Is poor skeletal muscle relaxant;
e) Is non-irritant;
f) Has little effect on inner organs.

10. Features of propofol:

a) General anesthesia occurs rapidly — in 30–40 seconds;
b) Duration of action is 3–10 minutes;
c) Duration of action is 1,5–3 hours;
d) Recovery is rapid;
e) Has severe depression of consciousness after recovery.

11. Features of thiopentone sodium:

a) Has a rapid onset;
b) Mild stage of excitement;
c) Severe stage of excitement;
d) Duration of general anesthesia is 20–30 minutes;
e) Stimulation of vasomotor and respiratory centers.

12. Side effects of ketamine:

a) A decrease in blood pressure;
b) An increase in blood pressure;
c) Tachycardia;
Hallucinations after recovery;
Bradycardia.

13. Features of ketamine:

a) Noncompetitive antagonist of NMDA-receptors;
b) Causes deep surgical anesthesia;
c) Causes immobility, loss of consciousness and analgesia;
d) Has a little effect on skeletal muscle tone;
e) Causes marked relaxation of skeletal muscles.

14. When halothane causes hypotension, to restore pressure cannot be used:

a) Epinephrine;
b) Phenylephrine;
c) Norepinephrine;
d) Ephedrine;
e) Atropine.

15. Morphine acts on antinociceptive system in the following way:

a) Stimulates the synthesis of opioid peptides;
b) Intensify the release of opioid peptides;
c) Stimulates the opioid receptors;
d) Block the inactivation of opioid receptors;
e) Block the presynaptic opioid receptors.

16. The opioid antagonist is:

a) Naloxone; c) Clonidine; e) Ibuprofen.
b) Droperidol; d) Nefopam;

17. Mechanism of vomiting upon the application of morphine:

a) Irritation of receptors of stomach mucosal membrane;
b) Intracranial hypertension;
c) Excitement of chemoreceptors emetic trigger zone;
d) Acting on vestibular system;
e) Stimulation of pharynx mechanoreceptors.

18. What drug can be combined with phentanyl for the purpose of neuroleptanalgesia:

a) Acetylsalicylic acid; c) Paracetamol; e) Pyracetam.
b) Droperidol; d) Diazepam;

19. Features of narcotic analgetics:

a) Increase respiratory volume; d) Cause drug dependence;
b) Relieve pain of any genesis; e) Have anti-inflammatory activity.
c) Facilitate sleep onset;

20. Mechanisms of obstipation caused by morphine:

a) Block of motilin receptors;
b) Inhibition of secretion of digestive glands;
c) Spasm of intestine sphincters;
d) Inhibition of intestinal peristalsis;
e) A decrease in intestinal smooth muscle tone.

21. Features of nonnarcotic analgetics:

a) Relieve pain of any genesis;
b) Decrease respiratory volume;
c) Cause drug dependence;
d) Relieve pain of inflammatory genesis;
e) Have anti-inflammatory activity;
f) Have antipyretic activity.

22. Peripheral COX inhibitors:

a) Ibuprofen; c) Keterolac; e) Paracetamol.
b) Acetylsalicylic acid; d) Metamizol;

23. Features of acetylsalicylic acid are:

a) Is pain reliever;
b) Anti-inflammatory activity;
c) Antipyretic activity;
d) Antiplatelet action;
e) Cough reduction.

24. Features of paracetamol:

a) Pain reliever;
b) Anti-inflammatory activity;
c) Antipyretic activity;
d) Antiplatelet action;
e) Inhibition of intestinal peristalsis.

25. Features of ibuprofen:

a) Pain reliever;
b) Anti-inflammatory activity;
c) Inhibition of intestinal peristalsis;
d) Emetogenic activity;
e) Anticonvulsant action.

26. Features of keterolac:

a) Antipyretic activity;
b) Anti-inflammatory activity;
c) Stimulation of intestinal peristalsis;
d) Diuretic activity;
e) Analgesic activity.

27. Features of metamizole:

a) Pain reliever;
b) Antipyretic activity;
c) Causes miosis;
d) Sedative-hypnogenic activity;
e) Antiemetic activity.

28. Drugs that are counter indicated in case of intracranial hypertension:

a) Ketamine;
b) Morphine;
c) Phentanyl;
d) Propofol;
e) Thiopental sodium.

ANXIOLITIC AND SEDATIVE-HYPNOGENIC DRUGS. ANTIPSYCHOTISC

1. Anxiolitic effect is:

a) Ability to induce sleep;
b) Raising of mood;
c) Stimulation of CNS;
d) Reduction of depression;
e) Reduction of anxiety.

2. Sedative-hypnogenic effect is:

a) Appearance of colorful dreaming;
b) Deficiency of dreaming;
c) Reduction of depression;
d) Sedation and facilitation of sleep onset;
e) Raising of mood.

3. Anxiolitic effect can be useful in the following situations:

a) Decreased requirement of sleep;
b) Panic;
c) Psychic excitement;
d) Sleepiness;
e) Brain ischemia.

4. Sedative-hypnogenic effect can be useful in the following situations:

a) Decreased requirement of sleep;
b) Sleeplessness;
c) Sleepiness;
d) Brain ischemia;
e) Psychic excitement.

5. Melatonin can be applied in the case of:

a) Decreased requirement of sleep;
b) Clock zone changing for correction of biorhythmies;
c) Sleepiness;
d) Brain ischemia;
e) Psychic excitement.

6. Effects of barbiturates:

a) Diarrhea;
b) Leukopenia;
c) Suppression of respiration;
d) Anesthesia;
e) Anticonvulsant activity;
f) Bronchospasm;
g) Gastrointestinal ulcers;
h) Suppression of vasomotor center;
i) Myorelaxation;
j) Hearing disturbance;
k) Antiplatelet effect;
l) Antipyretic effect;
m) Facilitation of the sleep onset;
n) Reduction of the pain;
o) An increase in the respiratory volume;
p) Antipsychotic activity.

7. Effects of benzodiazepines:

a) An increase in bronchi tonus;
b) Hematopoiesis disturbance;
c) Anticonvulsant activity;
d) An increase in gastrointestinal motility;
e) Hearing disturbance;
f) Sedative effect;
g) Hypnogenic effect;
h) An increase in the respiratory volume;
i) A decrease in the tone of skeletal muscles;
j) A decrease in the anxiety;
k) Anti-inflammatory effect.

8. Features of buspirone:

a) Has hypnogenic effect;
b) Reduction of anxiety;
c) Does not cause significant sedative effect;
d) Anticonvulsant activity;
e) Is muscle relaxant;
f) Driving is not recommended upon the application of this drug;
g) Causes myorelaxation;
h) Effect occurs immediately after drug administration;
i) Hepatic metabolism is typical.

9. Mechanisms of muscle tone reduction upon the application of benzodiazepines:

a) Calcium depletion in the sarcolemma;

b) Inhibition of GABA-dependent regulation of muscle tone in the spinal cord;

c) Phosphodiesterase inhibition in the muscle fibers;

d) Block of neuromuscle transmission (high doses);

e) Accumulation of lactic acid in the muscle fiber.

10. Anticonvulsant activity of benzodiazepines is determined by:

a) Hypnogenic effect;

b) Inhibition of primary seizure pattern;

c) Increasing of limbic system activity;

d) A decrease in cortex structures excitability;

e) Suppression of centers of medulla oblongata.

11. Hypnogenic activity of benzodiazepines is determined by:

a) Activation of epiphysis function;

b) Decrease of spontaneous activity of CNS;

c) Decrease of metabolic activity of CNS;

d) A decrease in cortex structures excitability;

e) Facilitation of NMDA-dependent signal flow in the neuronal network.

12. Mechanisms of action of benzodiazepines:

a) An increase in duration of GABA-dependent chloric channel opening;

b) An increase in rate of GABA-dependent chloric channel opening;

c) Inhibition of GABA-dependent ion channel;

d) An increase in effectiveness of GABA-dependent synaptic inhibition;

e) Direct activation of GABA-receptor.

13. Mechanisms of action of barbiturates:

a) An increase in duration of GABA-dependent chloric channel opening;

b) An increase in rate of GABA-dependent chloric channel opening;

c) Inhibition of GABA-dependent ion channel;

d) An increase in effectiveness of GABA-dependent synaptic inhibition;

e) Direct activation of GABA-receptor.

14. Define the sedative drugs without anxiolytic effect:

a) Alprazolam;

b) Diazepam;

c) Nitrazepam;

d) Diphenhydramine;

e) Promethazine.

15. Features of zolpidem:

a) Driving is not recommended upon the application of this drug;

b) Causes mild myorelaxation;

c) Effect occurs immediately after drug administration;

d) Acts on GABA-dependent signal transmission;

e) Suppresses respiratory center;

f) Driving can be recommended upon the application of this drug;
g) Significant residual effect is typical;
h) Effect occurs slowly (in one week);
i) Is antagonist of serotonin receptors;
j) Low toxic.

16. Antipsychotic drugs are applied in the following cases:

a) Ischemic stroke;
b) Depression;
d) Opioid withdrawal syndrome;
c) Schizophrenia.

17. Antipsychotic drugs are effectively the most in case of:

a) Panic disorder;
b) Manic depressive psychosis;
c) Positive symptoms;
d) Sleepiness;
e) Brain ischemia.

18. Antipsychotic drugs cause:

a) Colorful dreaming;
b) Hallucination;
c) Memory improvement;
d) Supression of positive symptoms in case of psychosis;
e) Sleep.

19. The main properties of neuroleptics (antipsychotic drugs):

a) Intensify the GABA-dependent suppression of CNS;
b) Block the dopamine receptors;
c) Activate the serotonin receptors;
d) Block the Mcholinergic receptors;
e) Inhibit the NMDA-receptors;
f) Block the αadrenergic receptors;
g) Activate the Mcholinergic receptors.

20. Features of antipsychotic drugs:

a) Increase the agitation in patients with schizophrenia;
b) Decrease the skeletal muscle tone;
c) Increase the anxiety in health people;
d) Reduce the anxiety;
e) Reduce the vomiting;
f) Induce psychic excitement;
g) Cause extrapyramidal disorder;
h) Increase the prolactin secretion;
i) Are effective in patients with Parkinson's disease;
j) Can cause euphoria.

21. Side effects of neuroleptics (antipsychotic drugs):

a) Hypertension;
b) Sleepiness;
c) Restlessness (akathisia);
d) Decreased libido in men;

e) Tardive dyskinesia (extrapyramidal symptoms);
f) Gynecomastia;
g) Increased libido in women.

22. Effects of neuroleptics associated with acting on M-cholinergic receptors:

a) Extrapyramidal symptoms;
b) Impotention;
c) Sleeplessness;
d) Constipation;
e) Paralysis of accommodation.

23. Effects of neuroleptics associated with acting on α-adrenoreceptors:

a) Giddiness;
b) Gynecomastia;
c) Orthostatic hypotension;
d) Obstipation;
e) Increased libido in women.

24. Effects of neuroleptics associated with acting on dopamine receptors in extrapyramidal system:

a) Decreased libido in men;
b) Obstipation;
c) Tardive dyskinesia;
d) Restlessness (akathisia);
e) Sleepiness.

25. Effects of neuroleptics associated with acting on dopamine receptors in hypothalamus:

a) Orthostatic hypotension;
b) Restlessness (akathisia);
c) Increased libido in women;
d) Gynecomastia in men;
e) Tardive dyskinesia.

26. Effects of neuroleptics associated with acting on prolactin secretion:

a) Gynecomastia in men;
b) Ejaculation disorder;
c) Induction of lactation;
d) Increased libido in women;
e) Parkinson's syndrome.

ANTIDEPRESSANTS. PSYCHOSTIMULANTS. NOOTROPIC DRUGS AND TONICS

1. Set up a correspondence between the pharmacological group:

a) Antidepressant, serotonin reuptake inhibitors;
b) Antidepressant, norepinephrine reuptake inhibitor;
c) Antidepressant, MAO inhibitor;
d) Neuroleptic;
e) Normothymic**.**

and drug:

1) Amitriptyline;
2) Fluoxetine;
3) Clozapine;
4) Carbamazepine;
5) Moclobemid.

2. Normothymic (antimanic) drugs can be administered in case of:

a) Panic disorder;
b) Manic-depressive psychosis;
c) Schizo-affective psychosis;
d) Sleppiness;
e) Brain ischemia.

3. Supposed mechanisms of antimanic activity of lithium salts:

a) Inhibition of Na^+, K^+-ATPase activity of sodium pump in the neuronal membrane;
b) Shift of secondary messengers activity;
c) Block of D_2-receptors;
d) Shift of cation distribution in intra-and intercellular compartments;
e) Modification of neuromediators releasing: norepinephrine, dopamine, etc.

4. Side effects of lithium salts:

a) Raising of arterial blood pressure;
b) Hypertrophy of thyroid gland;
c) Nephrogenic diabetes insipidus;
d) Secondary immunodeficiency;
e) Parkinson's disease.

5. Antidepressants can be administered in case of:

a) Panic disorder;
b) Endogenous depression;
c) Sleppiness;
d) Brain ischemia;
e) Psychic excitement.

6. Mechanism of action of tricyclic antidepressants:

a) Direct activation of adrenergic receptors;
b) Nonselective inhibition of monoamines reuptake (epinephrine, norepinephrine);
c) Block the inactivation of norepinephrine by MAO;
d) Selective inhibition of norepinephrine reuptake;
e) Block the inactivation of norepinephrine by COMT.

7. Set up a correspondence between antidepressants:

a) Sertraline;
b) Amitriptyline;
c) Moclobemide;
d) Tianeptine;
e) Mirtazapine.

and their mechanisms of action:

1) MAO inhibitor;
2) Serotonin reuptake inhibitor;
3) Strenghtens neuronal serotonin reuptake;
4) Inhibitor of presynaptic α_2-adrenergic receptor;
5) Norepinephrine reuptake inhibitor.

8. Features of tricyclic antidepressants:

a) An increase in arterial blood pressure;
b) Obstipation and urinary retention;
c) Relive the pain, potentiate the analgesics;

d) Increase the exercise tolerance;
e) Weight gain.

9. Biochemical effects of MAO inhibitors (group of antidepressants):

a) Inhibition MAO activity in presynaptic terminals;
b) Inhibition MAO activity in postsynaptic terminals;
c) Inhibition MAO activity in synaptic cleft;
d) An increase in mediator concentration in vesicles;
e) An increase in mediator concentration in synaptic cleft.

10. Effects of MAO inhibitors:

a) Cachexia;
b) Decreased blood pressure;
c) Sexual dysfunction, loss of libido;
d) Lack of the significant sedation;
e) Alcohol decreases the sedative effect of this drugs.

11. Correct affirmation about tricyclic antidepressants:

a) Are administered once a day as usual;
b) Clinical effect occurs in 2–3 weeks of daily application;
c) Are administered three and more times a day because of short half-life time;
d) Clinical effect occurs in first few days;
e) Drug effect ends in a few days after delay.

12. Correct assertions about serotonin reuptake inhibitors:

a) Are administered once a day as usual;
b) Functional accumulation is typical;
c) Are administered parenterally mainly;
d) Clinical effect occurs in first few days;
e) Side effects occur in first few days.

13. Features of MAO inhibitors:

a) Functional accumulation is typical;
b) Clinical effect occurs in 2–3 weeks of daily application;
c) Combination with serotonin reuptake inhibitors is recommended;
d) May cause sleeplessness;
e) Side effects occur in first few days.

14. What symptom may appear while eating tyramine containing food (red vine, cheese, etc), and taking MAO inhibitors?

a) Severe hypotension;
b) Obstipation;
c) Bronchospasm;
d) Hypertensive crisis;
e) Insulin resistance.

15. Combination of what drugs may cause the «serotonin syndrome»:

a) MAO inhibitors and serotonin reuptake inhibitors;
b) Tricyclic antidepressants and serotonin reuptake inhibitors;

c) Two drugs of serotonin reuptake inhibitors;
d) Phenelzine and и fluoxetine;
e) Fluoxetine and doxepin.

16. Select the antidepressants:

a) Buspirone;
b) Fluoxetine;
c) Flumazenil;
d) Tianeptine;
e) Moclobemide;
f) Sertraline;
g) Amobarbital.

17. Nootropic drugs:

a) Reduce the anxiety;
b) Facilitate the sleep onset;
c) Stimulate the immune system;
d) Improve cognitive skills;
e) Increase the brain resistance to hypoxia.

18. Effects of piracetam:

a) Increased physical performance with a single dose;
b) An increase in mental capacity with the use of single dose;
c) Do not act on mental capacity with the use of single dose;
d) Memory improvement in patients with brain disorder;
e) Learning improvement in patients with organic brain disorder.

19. Indications of nootropic drugs:

a) For rapid stimulation of mental capacity;
b) For rapid increasing of physical endurance;
c) Correction of posttraumatic mental disorders in children and adults;
d) Correction of mental disorders caused by cerebrovascular disturbance;
e) Prophylaxis of Parkinson's disease.

20. Define adaptogens:

a) Tianeptine;
b) Pantocrin;
c) Ginseng tincture;
d) Piracetam;
e) Eleutherococ liquid extract.

21. Choose analeptics:

a) Caffeine sodium benzoate;
b) Mezocarb;
c) Bemegride;
d) Aethimisol;
e) Doxapram.

22. Correct assertions about aethimisol:

a) Causes the bronchospasm;
b) Increases the concentration of glucocorticosteroids in blood plasma;
c) Stimulates the respiratory center;
d) Suppresses the respiratory center;
e) Can be used as analeptic.

23. Correct assertions about bemegride:

a) Causes the psychomotor agitation in high doses;

b) Can be used in case of poisoning with barbiturates and general anesthetics;

c) Stimulates the respiratory center;

d) Is used as stimulator of gastrointestinal motility;

e) Is administered parenterally;

f) Is administered orally.

HORMONAL AND ANTI-HORMONAL DRUGS

1. Tetracosactide is effective stimulator of secretion of:

a) Glucocorticoids;
b) Androgenic steroids;
c) Thyroxine;
d) Norepinephrine;
e) Insulin.

2. The excessive secretion of parathyroid hormone may cause:

a) Exophtalm («bulging eyes»), tachycardia, raised body temperature;

b) Apyretic tetanus, cataract, psychosis;

c) Hypoglycemia, raised body temperature;

d) Water retention, raised blood pressure, increase in glucose concentration;

e) Suppression of immune system.

3. Drug is applied in case of decreased level of thyroid hormones:

a) Propylthiouracil;
b) Thiamazole;
c) Levothyroxine sodium;
d) Teriparatide;
e) Radioactive iodine.

4. Antithyroid drugs are administered for the treatment of:

a) Hypothyroid infantilism;
b) Congenital myxedema;
c) Loss of sexual power;
d) Hypothyroid status;
e) Thyrotoxicosis.

5. Hypoglycemic drugs that is the sulfonylurea derivate:

a) Glybenclamide;
b) Acarbose;
c) Metformin;
d) Glucagon;
e) Gliclazide.

6. Select the correct assertion about calcitonin:

a) It increases the calcium absorption from intestine;

b) It increases a bone decalcination;

c) It increases the calcium concentration in the blood plasma;

d) Is administered in patients with acute hypocalcemia;

e) Is applied in case of osteoporosis.

7. The following drugs are the hypothalamic hormones and their synthetic analogues:

a) Thyrotropin;
b) Sermorelin;
c) Oxytocin;
d) Octreotide;
e) Somatropi (growth hormone);
f) Gonadorelin.

8. Posterior pituitary lobe hormone drugs and their synthetic analogues are:

a) Melatonin;
b) Oxytocin;
c) Goserelin;
d) Urofollitropin;
e) Desmopressin.

9. Correct assertion about desmopressin are:

a) It is a vasopressin derivate;
b) It has diuretic activity;
c) Can be applied for labor induction;
d) Is used in case of diabetes insipidus;
e) Can be administered in patients with diabetes type II.

10. Properties of thiamazole:

a) Inhibits the synthesis of thyroid hormones;
b) Can be applied in case of hyperthyroid status;
c) Can be administered in patients with hypothyroidism (goiter);
d) Has goitrogenic activity;
e) Inhibits the synthesis of thyrotropin alfa.

11. Mechanisms of hypoglycemic activity of insulin are:

a) An increase in glucose uptake by insulin dependent tissue;
b) An increase in peripheral glucose disposal;
c) Activation of glycogenolysis;
d) Induction of lipolysis;
e) Inhibition of gluconeogenesis.

12. Side effects of insulin preparations are:

a) Loss of appetite;
b) Hypoglycemia;
c) Allergic reactions;
d) Dyspeptic disturbances;
e) Arterial hypertension.

13. Drug is used in patients with diabetes insipidus:

a) Terlipressin;
b) Oxytocin;
c) Desmopressin;
d) Urofollitropin;
e) Furosemide.

14. Physiological insulin antagonists:

a) Glucagon;
b) Epinephrin;
c) Acarbose;
d) Glucocorticoids;
e) Rosiglitasone.

15. Mechanism of action of biguanides:

a) Inhibition of gluconeogenesis in the liver;
b) Induction of insulin secretion by the β-cells of pancreas;
c) An increase in glucose utilization by muscles and fat tissue;

d) A decrease in glucose absorption in the intestine;
e) Induction of glycogenolysis.

16. Put in the right order the action of steroid hormones:

a) Activation of translation;
b) Binding with specific receptors;
c) Transport in the nucleus;
d) Transport in the cell;
e) Correlation with the genome;
f) Induction of the transcription.

17. Gestagen drugs:

a) Induce the ovulation;
b) Inhibit the contractive activity of myometrium;
c) Are used for the maintenance of pregnancy;
d) Stimulate the development of secondary sex characteristics;
e) Are applied in the contraceptive pills.

18. Estrogen drugs:

a) Stimulate the development of secondary sex characteristics;
b) Cause the hyperplasia of endometrium;
c) Are applied in case of deficiency of ovarian function;
d) Are in composition of combined contraceptive pills;
e) Cause osteoporosis.

19. Put in the right order of action of steroid hormones:

a) Correlation with the genome;
b) Regulation of the transcription;
c) Activation of translation;
d) Transport in the cell;
e) Binding with specific receptors in the cytoplasm of the cell;
f) Transport the ligand-bound receptor complex in the nucleus.

20. Adverse effects of glucocorticoids are:

a) Behavioral changes, anxiety;
b) Sleeplessness, acute psychosis;
c) Weakness, apathy;
d) A decrease in the convulsive threshold;
e) Vestibulo-cochlear disoders.

21. Define the correct assertions about prednisolone:

a) Supresses the synthesis of endogenous glucocorticoids;
b) Has severe hypotension activity;
c) More than half of dosage is applied in the morning if prednisolone is used as anti-inflammatory and anti-allergic drug;
d) Applied dosage is uniformly distributed if prednisolone is used as anti-inflammatory and anti-allergic drug;
e) Has immunostimulatory activity.

22. Mineralocorticoids have the following properties:

a) Increase the reabsorption of sodium ions and water in the renal tubules;

b) Increase the elimination of potassium ions;
c) Increase the diuresis;
d) Can cause the arterial hypertension;
e) Can be applied in patients with Addison disease.

23. Set up a corresponds between groups

a) Anabolic steroids; c) Estrogenes; e) Mineralocorticoids.
b) Androgenes; d) Glucocorticoids;

and hormone drugs (each element in the right column can be used only once)

1. Testosterone; 4. Desoxycortone
2. Diethylstibestrol; 5. Mometasone
3. Nandrolone;

24. Glucocorticoids can be used as … drugs:

a) Anti-allergic; d) Catabolic;
b) Hyperglycemic; e) Immunosuppressive.
c) Anti-inflammatory;

25. Side effects of glucocorticoids:

a) Growth impairment in children;
b) Menstrual disorders (secondary amenorrhea);
c) Acceleration of sexual maturation;
d) Disturbance of glucose tolerance;
e) Hyperthyroidism.

26. Choose the correct assertions about tetracosactide:

a) Is synthetic analogue of corticotrophin;
b) Immunogenic activity is weak;
c) Is administered in case of Cushing' syndrome;
d) Is applied in patients with secondary adrenal insufficiency.

27. Select the side effects of glucocorticoids:

a) Negative nitrogen balance; d) Raised appetite;
b) Hypoglycemia; e) Obesity.
c) Hyperlipidemia;

28. Properties of anabolic steroids:

a) Inhibit the protein synthesis;
b) Can be applied in case of cachexia (pantotrophia);
c) Decrease the muscle mass;
d) Are administered in case of osteoporosis;
e) Can cause masculinization in women.

29. The following drug has intensed mineralocorticoid activity (sodium and water retention and intensification of potassium elimination):

a) Dexamethasone; c) Momethasone; e) Methylprednisolone.
b) Hydrocortisone; d) Prednisolone;

ANTI-INFLAMMATORY DRUGS

1. The main mechanism of anti-inflammatory action of NSAIDs:

a) Stabilization of mast cell membranes, inhibition of the release of mediators of allergy and inflammation;

b) Suppression of prostaglandin synthesis by inhibition of cyclooxygenase;

c) Suppression of prostaglandin synthesis by inhibition of phospholipase A2;

d) Suppression lipoxygenase activity with reduced production of leukotrienes;

e) Destruction of mediators of inflammation.

2. The main side effects of nonselective cyclooxygenase inhibitors are:

a) Ulceration of the gastrointestinal tract;
b) Immunosuppression;
c) Inhibition of kidney function (nephrotoxic effect);
d) Cardiotoxic action;
e) Impairment of protein, fat and carbohydrate metabolism.

3. The main pharmacodynamic effects of non-steroidal anti-inflammatory drugs are:

a) Antipyretic;
b) Anabolic;
c) Anti-inflammatory;
d) Analgesic;
e) Immunosuppressive;
f) Immunostimulatory.

4. Select NSAIDs with low selectivity for COX-2:

a) Indomethacin;
b) Celecoxib;
c) Acetylsalicylic acid (analgesic and antipyretic doses);
d) Naproxen;
e) Valdecoxib;
f) Diclofenac.

5. Features of celecoxib:

a) It is equally inhibits COX-1 and COX-2;
b) Has weak ulcerogenic effect;
c) There is a risk of thromboembolic cardiovascular complications;
d) Is less potent than acetylsalicylic acid for anti-inflammatory efficacy;
e) Abnormal liver function requires correction dosing regimen.

6. Features are typical for non-steroidal anti-inflammatory drugs:

a) Poor tolerability;
b) Suppression of inflammation of any nature;
c) Combination of anti-inflammatory, analgesic and antipyretic action;
d) Reduction of the production of endogenous glucocorticosteroids;
e) Inhibition of cyclooxygenase activity.

7. Features of salicylates:

a) Have a gastrotoxic effect;

b) Causes hyperglycemia;

c) In low doses, platelet aggregation is inhibited;

d) May cause bronchospasm;

e) Suppress the migration of phagocytes to the focus of inflammation, inhibit phagocytosis.

8. Steroidal anti-inflammatory drugs:

a) Suppress the production of endogenous glucocorticosteroids;

b) Have an immunosuppressive effect;

c) Only have anti-inflammatory, analgesic and antipyretic effects;

d) Causes ulceration of the gastrointestinal tract;

e) Block the synthesis of inflammatory mediators.

9. Specify the effects of steroidal anti-inflammatory drugs:

a) Anti-inflammatory;

b) Immunostimulatory;

c) Immunosuppressive

d) Anti-allergic;

e) M-cholinoblocking.

10. Mechanism of anti-inflammatory effect of glucocorticosteroids:

a) Decrease in the synthesis of prostaglandins and leukotrienes due to inhibition of the activity of phospholipase A2;

b) Selective suppression of prostaglandin synthesis, due to inhibition of cyclooxygenase activity;

c) Inhibition of COX-2 production;

d) Suppression of cellular mechanisms of inflammation (impairment of migration of macrophages and neutrophils in the focus of inflammation);

e) Immunosuppressive action — disturbance of proliferation and differentiation of immunocompetent cells, antibodies, cytokines, inflammatory mediators.

11. Beclomethasone:

a) Glucocorticosteroid for topical application;

b) Glucocorticosteroid for systemic use;

c) Inhibition of the synthesis of endogenous glucocorticosteroids is significant;

d) Used in aerosol dosage forms;

e) It is used for the treatment of bronchial asthma and vasomotor rhinitis.

12. Features of prescribing glucocorticosteroids as anti-inflammatory and antiallergic agents:

a) Most of the daily dose is prescribed in the morning hours;

b) Most of the daily dose is prescribed in the evening hours;

c) The daily dose is evenly distributed;

d) Cancel gradually, slowly lowering the dose;

e) Canceled at the same time.

13. Irreversible consequences of GCS application:

a) Reduced resistance to infections;
b) Deceleration of tissue regeneration;
c) Subcapsular cataract;
d) Teratogenic effect; e) Steroid diabetes.

14. Mechanism of anti-gout action of allopurinol:

a) Inhibition of reabsorption of uric acid in renal tubules;
b) Disruption of biosynthesis of uric acid from hypoxanthine;
c) Suppression of phagocytosis and ejection of inflammatory mediators;
d) Acceleration of biotransformation of uric acid;
e) Covalent binding and excretion of uric acid.

15. Mechanism of anti-gout action of sulfinpyrazone:

a) Inhibition of xanthine oxidase;
b) Enhancement of uric acid secretion in renal tubules;
c) Decrease in reabsorption of uric acid in renal tubules;
d) Acceleration of biotransformation of uric acid;
e) Covalent binding and excretion of uric acid.

ANTI-ALLERGIC DRUGS. DRUGS AFFECTING THE RESPIRATORY SYSTEM

1. Effects of antihistamines of the 1st generation:

a) Antiemetic effect;
b) Sedative effect on the central nervous system;
c) Potentiation the action of drugs for general anesthesia, opioid analgesics and anesthetics;
d) Stimulation of peristalsis;
e) Constriction of small arterioles.

2. Distinctive features of antihistamines of the 2nd generation from the 1st generation:

a) High selectivity to H_1-histamine receptors;
b) Long duration of action;
c) Less pronounced sedative effect;
d) Less chance of the development of tolerance;
e) Minor efficiency;
f) Reduce glucose tolerance.

3. Side effects of antihistamines of the 1st generation associated with their M-cholinoblocking action:

a) Dry mouth; d) Bradycardia;
b) Urine retention; e) Paralysis of accommodation;
c) Constipation; f) Activation of catabolism.

4. Most probable side-effects after parenteral administration of antihistamines:

a) Hypotension; d) Bradycardia;
b) Tachycardia; e) Tachycardia with hypertension;
c) Hypertension; f) Tradicardia with hypotension.

5. The most suitable medicines for the treatment of mild allergic reactions of immediate type (pruritus, urticaria):

a) Epinephrine; d) Clemastine;
b) Cromoglycic acid; e) Prednisolone;
c) Diphenhydramine; f) Loratadine.

6. Set correspondence between groups:

a) Histamine receptor antagonist;
b) Inhibitor of the action of mediators of allergy;
c) Interleukins;
d) Stabilizers of mast cell membranes;
e) Leukotriene receptor antagonists.

and drugs

1) Diphenhydramine; 4) Nedocromil;
2) Zafirlukast; 5) Fenspiride.
3) Betaleikin;

7. Specify antihistamines without M-cholinoblocking action:

a) Difenhydramine; d) Fexofenadine;
b) Loratadine; e) Desloratadine.
c) Promethazine;

8. Specify antihistamines, which can be taken once a day:

a) Clemastine; d) Diphenhydramine;
b) Loratidine; e) Cetirizine.
c) Hifenadine;

9. Restore the mechanism of development of a delayed-type allergic reaction:

a) Production of interleukin-1 by macrophages;
b) Antigen killing, topical repair (or progression of immune inflammation);
c) Induction of transformation of T-lymphocytes into effector cells;
d) Antigen receipt, its recognition and capture by macrophages;
e) The interaction of effector cells with other immune cells;
f) Assignment of mediators of allergy and inflammation, attraction of immunocompetent cells to the outbreak;
g) Activation of T-helpers;
h) Production of interleukin-2 by T-helpers.

10. Restore the mechanism of development of an allergic reaction of an immediate type:

a) Primary recognition of antigen by immunocompetent cells;

b) Interaction of antigen with mast cells having specific sites of its binding;

c) Clinical manifestations of an allergic reaction of immediate type;

d) Degranulation of the mast cell with the release of mediators of allergy and inflammation;

e) Production of antibodies (IgE) and its presentation on the surface of mast cells;

f) Second contact with antigen.

11. Drugs for treatment of delayed-type allergic reactions:

a) Preparations of gold;

b) Glucocorticoids;

c) Leukotriene receptor antagonists;

d) Inhibitors of proliferation;

e) Stabilizers of mast cell membranes;

f) Penicillamine;

g) Antihistamines.

12. Restore the molecular mechanism of action of methotrexate:

a) Antagonism with folic acid;

b) Inhibition of differentiation and proliferation of immunocompetent cells;

c) Improvement of clinical symptoms;

d) Immunosuppression, inhibition of remodeling of connective tissue;

e) Inhibition of the synthesis of nucleic acids and proteins.

13. Restore the sequence of actions to assist in anaphylactic shock:

a) Administration of glucocorticosteroids, preferably intravenously;

b) Discontinuation of the ingestion of an allergen (epinephrine topically);

c) Symptomatic therapy (bronchodilators, pacemakers, antihistamines, respiratory analeptics, etc.);

d) Maintenance of systemic arterial pressure and work of the heart (epinephrine systemically).

14. The mechanism of anti-allergic effect of glucocorticoids:

a) Reduction of immunocompetent cells;

b) Bockade of histamine receptors;

c) Stabilization of mast cell membranes;

d) A decrease in the synthesis of immunoglobulins;

e) Suppression of migration of immunocompetent cells.

15. Zafirlukast:

a) Reduces vascular permeability;

b) Suppresses bronchial secretion and reduces the viscosity of sputum;

c) It is used for the relief of bronchospasm;

d) Reduces the swelling of the bronchial mucosa;

e) Is a leukotriene receptor antagonist;

f) It is an antihistamine drug of the 1st generation.

16. This drug dilates the bronchi by reducing parasympathetic effects:

a) Aminophylline; c) Atropine; e) Salmerotol.
b) Epinephrine; d) Isoprenaline;

17. This drug has a bronchodilator effect due to stimulation of beta2-adrenoreceptors:

a) Aminophylline; c) Iprorotropium; e) Montelukast.
b) Beclomethasone; d) Isoprenaline;

18. This drug has a bronchodilator effect by suppressing the release of mediators of allergy:

a) Cromoglycic acid; c) Atropine; e) Salmerotol.
b) Epinephrine; d) Isoprenaline;

19. The antitussive drugs include:

a) Cromoglycic acid; c) Atropine; e) Beclomethasone.
b) Epinephrine; d) Dextromethorphan;

20. For the relief of bronchospasm is used:

a) Epinephrine; c) Salmerotol; e) Acetylcysteine.
b) Cromoglycic acid; d) Tiotropium;

21. For the prevention of bronchospasm used:

a) Epinephrine; c) Salmeterol; e) Atropine.
b) Isoprenaline; d) Salbutamol (in aerosol);

22. A side effect of adrenergic bronchodilators is:

a) Tachycardia; d) Bronchospasm;
b) Bradycardia; e) Peripheral vasospasm.
c) Increased blood pressure;

23. Salbutamol is contraindicated in:

a) Atrioventricular blockade; d) Bronchospasm;
b) Extrasystoles; e) Anaphylactic shock.
c) Preterm labor activity;

24. Bronchodilators from the M-cholinoblockers group are contraindicated in:

a) Bradycardia; d) Diarrhea;
b) Atrioventricular blockade; e) Hyperacid gastritis.
c) Glaucoma;

25. Side effects of topical application of glucocorticosteroids in the treatment of pulmonary diseases:

a) Reduced tolerance to the respiratory tract infections;
b) Increased resistance to respiratory infections;
c) Hypoglycaemia;
d) Complete adrenal insufficiency;
e) Atrophy of bronchial mucosa.

26. Acetylcysteine:

a) Reflexively stimulates the secretion of the bronchial glands;

b) Has a direct stimulating effect on bronchial glands;

c) Reduces the viscosity of sputum due to destruction of disulfide bonds of proteoglycans;

d) Inhibits cough reflex;

e) Relaxes the smooth muscles of the bronchi.

27. Therapeutic action of ganglionic blockers at pulmonary edema caused by:

a) Tissue dehydration;

b) Diuretic effect;

c) Anti-inflammatory effect;

d) Reduce the pressure in the pulmonary circulation;

e) Reduce the load on the heart.

28. Drugs with bronchodilator action:

a) M-cholinoblockers; c) Ganglio-blockers; e) Beta-agonists.

b) M-cholinomimetics; d) Beta-blockers;

29. Unlike atropine, ipratropium bromide:

a) Selectively blocking m-cholinergic receptors of the bronchi;

b) It is used only by inhalation;

c) Has a slight resorptive effect;

d) Does not affect the secretion of bronchial glands;

e) Contraindicated in glaucoma.

30. Codeine:

a) Inhibits the cough reflex;

b) Reduces the tone of the muscles of the bronchi;

c) Has analgesic properties;

d) Has a sedative effect;

e) May induce drug dependence;

f) Stimulates intestinal motility.

31. The following statements are true:

a) Propranolol can cause bronchospasm;

b) Salbutamol causes tachycardia;

c) Blockers of H_1-histamine receptors used in the treatment of allergic rhinitis;

d) Codeine does not have analgesic activity;

e) Prolonged use of α-adrenergic agonists leads to the development of rhinitis.

32. Principles of pharmacotherapy of pulmonary edema:

a) Pressure reduction in the pulmonary circulation;

b) Stimulation of the center of breathing;

c) Suppressing the foaming of the transudate;

d) Elimination of hypoxia;

e) Dehydration of respiratory tract tissues;

f) Inhibition of the cough center.

33. Medications used to treat bronchial asthma:

a) Blockers of leukotriene receptors;
b) Blockers release of mediators of allergy from mast cells;
c) Beta-adrenoreceptor agonists;
d) Alpha-adrenoreceptor agonists;
e) Local decongestants;
f) Glucocorticosteroids.

34. For the treatment of bronchial asthma use:

a) Bemegrid;
b) Salmeterol;
c) Beclomethasone;
d) Tiotropium;
e) Zafirlukast;
f) Xylometazoline.

35. The allergic component in bronchial asthma is suppressed by:

a) Tiotropium;
b) Salbutamol;
c) Nedocromil;
d) Theophylline;
e) Budesonide;
f) Ketotifen.

SYNTHETIC ANTIMICROBIAL DRUGS

1. Mechanism of action of sulfonamides:

a) Drug molecules are reduced by anaerobic microbes to metabolites interfering with nucleic acid replication;
b) Inhibition of nucleic acid replication, complexation with microbial metalloenzymes;
c) Folic acid synthesis inhibition in bacterial cells;
d) Nitro-group of the drugs is reduced by anaerobic microbes and protozoic cells to metabolites causing DNA damage;
e) Bacterial topoisomerase II (DNA-gyrase) and IV inhibition.

2. Sulfonamides are:

a) Bacteriostatic;
b) Bactericidal;
c) Fungicidal;
d) Virucidal.

3. Trimethoprim is:

a) Bacteriostatic;
b) Bactericidal;
c) Fungicidal;
d) Virucidal.

4. Co-trimoxazole is:

a) Bacteriostatic;
b) Bactericidal;
c) Fungicidal;
d) Virucidal.

5. Sulfonamides may cause:

a) Bone marrow depression (anemia, leucopenia);
b) Hearing loss and visual disturbances;
c) Allergic reactions;
d) Cristaluria and nephrolithiasis;
e) Dyspepsia, hepatotoxicity.

6. Co-trimoxazole may cause:

a) Bone marrow depression (neutropenia, anemia, thrombocytopenia);
b) Nausea, vomiting, glossitis, stomatitis;
c) Thrombosis;
d) Allergic reactions (rash, Stevens-Johnson syndrome);
e) Tachyarrhythmia.

7. Phtalylsulfathiazole is used only for the treatment of intestinal infections (bacterial dysentery, enterocolitis) because:

a) It is superior to other sulfonamides in its activity against intestinal pathogens;
b) Almost is not absorbed in GIT;
c) Decreases intestinal peristalsis;
d) Restores intestinal microflora;
e) Well absorbed in GIT, excreted with bile.

8. Co-trimoxazole:

a) Is bacteriostatic;
b) Is bactericidal;
c) Is inferior to other sulfonamides in its spectrum of activity;
d) Has a wider range of activity than sulfonamides;
e) Comparing to sulfonamides bacterial resistance develops more slowly.

9. Antimicrobial spectrum of co-trimoxazole:

a) Has a broader spectrum of activity than sulfonamides;
b) Nocardia spp., Moraxella spp., Pneumocysts;
c) Toxoplasma spp., Haemophilus influenza;
d) Pseudomonas aeruginosa;
e) Mycobacterium tuberculosis;
f) Mycoplasma spp., Rickettsia spp.

10. Antimicrobial spectrum of sulfonamides:

a) Extremely broad; b) Relatively narrow;
b) Toxoplasma spp, Haemophilus influenza;
c) Shigella spp., Staphylococcus spp. (most strains);
d) Treponema pallidum;
e) Most fluoroquinolone-resistant microbes.

11. Mechanism of action of 8-oxyquinoline derivatives:

a) Drug molecules are reduced by anaerobic microbes to metabolites interfering with nucleic acid replication;
b) Inhibition of nucleic acid replication, complexation with microbial metalloenzymes;
c) Folic acid synthesis inhibition in bacterial cells;
d) Nitro-group of the drugs is reduced by anaerobic microbes and protozoic cells to metabolites causing DNA damage;
e) Bacterial topoisomerase II (DNA-gyrase) and IV inhibition.

12. 8-Oxyquinoline derivatives are:

a) Nitroxoline; c) Metronidazole; e) Furazolidone.
b) Nalidixic acid; d) Chlorquinaldol;

13. Quinolones are:

a) Nalidixic acid; c) Oxolinic acid; e) Trimethoprim.
b) Lomefloxacin; d) Fusidic acid;

14. Fluoroquinolones are:

a) Norfloxacin; c) Metronidazole; e) Lomefloxacin.
b) Ciprofloxacin; d) Ofloxacin;

15. Mechanism of action of fluoroquinolones:

a) Drug molecules are reduced by anaerobic microbes to metabolites interfering with nucleic acid replication;

b) Inhibition of nucleic acid replication, complexation with microbial metalloenzymes;

c) Folic acid synthesis inhibition in bacterial cells;

d) Nitro-group of the drugs is reduced by anaerobic microbes and protozoic cells to metabolites causing DNA damage;

e) Bacterial topoisomerases II (DNA-gyrase) and IV inhibition.

16. Fluoroquinolones are

a) Bacteriostatic; c) Fungicidal;
b) Bactericidal; d) Virucidal.

17. Fluoroquinolones may cause:

a) Anorexia, nausea, vomiting, alteration in taste;
b) Nephritis, nephrolithiasis;
c) Allergic reactions (rash, angioedema), photosensitization;
d) Headache, vertigo, sleep disorder;
e) Tendinitis, juvenile arthropathy.

18. Nitroxoline:

a) Has a broad spectrum of activity;
b) Affects only gram-negative bacteria;

c) Almost is not absorbed from GIT, that is why it is used for intestinal infections;

d) Well absorbed from GIT, eliminated by renal excretion as unchanged drug, used for treating urinary infections;

e) Is bacteriostatic.

19. Antimicrobial spectrum of fluoroquinolones:

a) Broad;
b) Narrow, only gram-negative bacteria are sensitive;
c) Narrow, only gram-positive bacteria are sensitive;
d) Treponema pallidum;
e) Chlamydia, mycoplasma; f) Mycobacterium tuberculosis.

20. Ciprofloxacin:

a) Has a broad spectrum of activity;

b) Affects only gram-positive bacteria;

c) Is used for intestinal infections (typhoid fever, paratyphoid fever, dysentery);

d) Well absorbed from GIT, passes through BBB;

e) Contraindicated in pregnant and nursing women.

21. 5-Nitroimidazole derivatives are:

a) Norfloxacin; c) Metronidazole; e) Nitrofurantoin.

b) Nalidixic acid; d) Tinidazole;

22. Mechanism of action of 5-nitroimidazole derivatives:

a) Drug molecules are reduced by anaerobic microbes to metabolites interfering with nucleic acid replication;

b) Inhibition of nucleic acid replication, complexation with microbial metalloenzymes;

c) Nitro-group of the drugs is reduced by anaerobic microbes and protozoic cells to metabolites causing DNA damage;

d) Bacterial topoisomerase II (DNA-gyrase) and IV inhibition.

23. 5-Nitroimidazole derivatives are

a) Bacteriostatic; c) Fungicidal;

b) Bactericidal; d) Virucidal.

24. 5-Nitroimidazole derivatives may cause:

a) Nausea, vomiting, stomatitis, metallic taste;

b) Hepatitis, liver cirrhosis;

c) Allergic reactions (rash, angioedema);

d) Urine discoloration (reddish-brown);

e) Disulfiram-like reactions when taken together with alcohol.

25. Antimicrobial spectrum of 5-nitroimidazole derivatives:

a) Affect only aerobic bacteria; d) Amoebae;

b) Anaerobic bacteria; e) Trichomonas spp.;

c) Ultra-broad; f) Lamblia spp.

26. Nitrofurans are:

a) Nitrofurantoin; c) Fusidic acid; e) Furazolidone.

b) Tnidazole; d) Ofloxacin;

27. Nitrofurans may cause:

a) Headache, nausea, vertigo;

b) Malignant hyperthermia;

c) Peripheral neuropathy;

d) Bone marrow depression (anemia, leucopenia);

e) Liver injury (hepatitis, cholestasis).

28. Antimicrobial spectrum of nitrofurans:

a) Anaerobic bacteria;
b) Broad;
c) Escherichia coli, Shigella spp., Salmonella spp., Vibrio cholera;
d) Pseudomonas aeruginosa, Proteus spp., Klebsiella spp.;
e) Trichomonas spp.;
f) Lamblia spp.

ANTIBIOTICS, PART I

1. Antimicrobial combination therapy is used:

a) For the prevention of resistant bacterial strains development;
b) To enhance antimicrobial effect;
c) To broaden antibacterial spectrum of activity;
d) To enhance antimicrobial effect of a bacteriostatic antibiotic it is necessary to add bactericidal one;
e) To decrease the toxicity of certain antibiotics.

2. The most common causative agents of superinfections:

a) Clostridium difficile;
b) Candida fungi;
c) Mycobacterium tuberculosis;
d) Chlamydia;
e) Pseudomonas aeruginosa.

3. The causes of antibiotic therapy inefficiency:

a) Resistance of a pathogen to antibiotics;
b) Concurrent administration of vitamins;
c) Viral infections;
d) Dosage regime violation;
e) Incorrect antibiotic combinations.

4. Basic principles of chemotherapy:

a) Early start of chemotherapy;
b) Pathogen identification;
c) In life-threatening conditions broad-spectrum antibiotics may be used before pathogen identification has been completed;
d) Full-course of chemotherapy unless pathogen eradication is achieved;
e) Carry out chemotherapy until symptoms have resolved;
f) The use of the most effective and safest antimicrobial drugs;
g) Combination chemotherapy to increase the efficacy of the treatment or minimize the development of antibiotic resistant microbes.

5. Beta-Lactam antibiotics interfere with:

a) Cell wall synthesis;
b) Plasma membrane permeability;
c) Protein synthesis on ribosomes;
d) RNA synthesis;
e) All listed variants.

6. Benzylpenicillin preparations typically cause:

a) Agranulocytosis; d) Hearing loss and vestibular disturbances;
b) Anemia; e) Nephrotoxicity;
c) Allergic reactions; f) Dysbacteriosis.

7. Penicillins show little activity or ineffective against:

a) Treponema pallidum;
b) Actively growing bacterial cells;
c) Meningococci;
d) Resting bacterial cells.

8. First-line antibiotic for the treatment of infections caused by Pseudomonas aeru-ginosa:

a) Benzylpenicillin; c) Chloramphenicol; e) Tetracycline.
b) Piperacillin; d) Erythromycin;

9. First-line antibiotic for the treatment of meningococcal meningitis:

a) Amphotericin B; d) Streptomycin;
b) Benzylpenicillin sodium salt; e) Nystatin.
c) Chloramphenicol;

10. Most appropriate antibiotic for treating infections in pregnancy:

a) Streptomycin; c) Benzylpenicillin; e) Chloramphenicol.
b) Tetracycline; d) Gentamicin;

11. Identify the correct statements about cephalosporins:

a) Cephalosporins are bactericidal towards multiplying bacteria;
b) Both cephalosporins and penicillins have the same spectrum of activity;
c) There is cross-sensitivity between penicillins and cephalosporins;
d) Cephalosporins are resistant to staphylococcal beta-lactamases (1st and 2nd generation), gram-negative bacteria (3rd and 4th generation).

12. Most active drugs against Pseudomonas spp.:

a) First-generation cephalosporins;
b) Second-generation cephalosporins;
c) Third-generation cephalosporins;
d) Fourth-generation cephalosporins.

13. The greatest ability to penetrate into the cerebrospinal fluid is for:

a) First-generation cephalosporins;
b) Second-generation cephalosporins;
c) Third-generation cephalosporins;
d) Fourth-generation cephalosporins.

14. Characteristic features of aztreonam:

a) Has a narrow spectrum of activity;
b) Is inactivated by beta-lactamases;
c) Resistant to beta-lactamases;
d) Inhibits RNA synthesis on ribosomes;

e) Inhibits microbial cell wall synthesis;
f) Is administered orally;
g) Is administered parenterally.

15. Characteristic features of imipenem:

a) Has a narrow spectrum of activity;
b) Has a broad spectrum of activity;
c) Is bacteriostatic;
d) Is bactericidal;
e) Inhibits RNA synthesis on ribosomes;
f) Inhibits microbial cell wall synthesis;
g) Is inactivated by beta-lactamases;
h) Resistant to beta-lactamases;
i) Is administered orally;
j) Is administered parenterally.

ANTIBIOTICS, PART II

1. Characteristic features of tetracyclines:

a) Have a broad spectrum of activity;
b) Affect predominantly gram-negative bacteria;
c) Are bactericidal;
d) Are bacteriostatic;
e) Slow resistance development;
f) Fast resistance development;
g) Inhibit protein synthesis on ribosomes;
h) Inhibit cell wall synthesis;

2. Tetracyclines are the drugs of choice for:

a) Coccal infections;
b) Bacillary dysentery;
c) Brucellosis;
d) Tularemia;
e) Rickettsial infections;
f) Typhoid fever;
g) Syphilis;
h) Cholera;
i) Plague;
j) Typhoid fever.

3. Tetracyclines may cause:

a) Anemia;
b) Dyspepsia;
c) Hearing loss;
d) Dysbacteriosis;
e) Liver injury;
f) Allergic reactions;
g) Visual disturbances.

4. Characteristic features of chloramphenicol:

a) Has a broad spectrum of activity;
b) Affects predominantly gram-positive bacteria;
c) Is bactericidal;
d) Is bacteriostatic;

e) Slow resistance development;
f) Fast resistance development.

5. Chloramphenicol is the drug of choice for:

a) Typhoid fever and other salmonellosises;
b) Coccal infections;
c) Spotted fever and other Rickettsial infections;
d) Cholera;
e) Bacillary dysentery;
f) Amebial dysentery.

6. Chloramphenicol may cause:

a) Agranulocytosis;
b) Anemia;
c) Collapse;
d) Hearing loss;
e) Dysbacteriosis;
f) Allergic reactions.

7. Characteristic features of streptomycin:

a) Has a broad spectrum of activity;
b) Affects predominantly gram-positive bacteria;
c) Is bactericidal;
d) Is bacteriostatic;
e) Interferes with mRNA attachment and causes misreading of the genetic code;
f) Interferes with plasma membrane permeability;
g) Well absorbed from GIT;
h) Poorly absorbed from GIT.

8. Streptomycin is the drug of choice for:

a) Tuberculosis;
b) Typhoid fever;
c) Plague;
d) Tularemia;
e) Bacillary dysentery;
f) Syphilis;
g) Gonorrhea.

9. Streptomycin may cause:

a) Allergic reactions;
b) Anemia;
c) Liver injury;
d) Hearing loss;
e) Vestibular disturbances;
f) Dysbacteriosis;
g) Kidney injury.

10. Neomycin is used for:

a) Wound infections, phlegmon, abscesses caused by Staphylococci, Streptococci and Pseudomonas aeruginosa;
b) Rickettsial infections;
c) Tuberculosis;
d) Candidiasis;
e) Bowel preparation before surgery.

11. Third generation aminoglycosides are:

a) Streptomycin;
b) Tobramycin;
c) Neomycin;
d) Kanamycin;
e) Gentamicin;
f) Netilmycin;
g) Amikacin.

12. Characteristic features of polymyxins:

a) Have a broad spectrum of activity;
b) Affect predominantly gram-negative bacteria;
c) Are bactericidal;
d) Are bacteriostatic;
e) Interfere with plasma membrane structure and functioning;
f) High efficacy against intracellular pathogens;
g) Active against Pseudomonas aeruginosa.

13. Polymyxin B is used for:

a) Syphilis;
b) Pseudomonas aeruginosa caused infections;
c) Tuberculosis;
d) Bowel preparation before surgery;
e) Rickettsial infections;
f) Candidiasis.

14. Characteristic features of lincosamides:

a) Have a broad spectrum of activity;
b) Affect predominantly gram-positive bacteria;
c) Inhibit cell wall synthesis;
d) Inhibit protein synthesis on ribosomes;
e) Acquired resistance develops rapidly;
f) Drugs of choice for the treatment of osteomielitis.

15. Characteristic features of lincosamides:

a) Have a broad spectrum of activity;
b) Affect predominantly gram-positive bacteria;
c) Are bacteriostatic;
d) Are bactericidal;
e) Acquired resistance develops slowly;
f) Drugs of choice for treating osteomielitis.

16. Lincosamides may cause:

a) Dyspepsia;
b) Allergic reactions;
c) Pseudomembranous colitis;
d) Liver injury;
e) Respiratory arrest (on fast i/v administration);
f) Collapse;
g) Thrombocytopenia.

17. Characteristic features of vancomycin:

a) Has a broad spectrum of activity;
b) Affect predominantly gram-positive bacteria;
c) Is bactericidal;
d) Is bacteriostatic;
e) Inhibits bacterial cell wall synthesis;
f) Inhibits RNA synthesis on ribosomes;
g) Well absorbed from GIT;
h) Poor GIT absorption.

18. Vancomycin may cause:

a) Kidney injury;
b) BP decrease;
c) BP increase;
d) Seizures;
e) Thrombophlebitis;
f) Deafness.

19. Antimicrobial combination therapy is used:

a) For the prevention of resistant bacterial strains development;
b) To enhance antimicrobial effect;
c) To broaden antibacterial spectrum of activity;
d) To enhance antimicrobial effect of a bacteriostatic antibiotic it is necessary to add bactericidal one;
e) To decrease the toxicity of certain antibiotics.

20. Synergistic antibiotic combinations are:

a) Penicillins + aminoglycosides;
b) Cephalosporins + aminoglycosides;
c) Aminoglycosides + carbenicillin;
d) Macrolides + tetracyclines;
e) Gentamicin + amikacin;
f) Ampicillin + oxacillin.

21. The most common causative agents of superinfections:

a) Clostridium difficile;
b) Candida fungi;
c) Mycobacterium tuberculosis;
d) Chlamydia;
e) Pseudomonas aeruginosa.

22. The causes of antibiotic therapy inefficiency:

a) Resistance of a pathogen to antibiotics;
b) Concurrent administration of vitamins;
c) Viral infections;
d) Dosage regime violation;
e) Incorrect antibiotic combinations.

23. Aminoglycosides used for the treatment of infections caused by gentamicin-resistant bacteria:

a) Streptomycin;
b) Amikacin;
c) Neomycin;
d) Tobramycin;
e) Kanamycin.

24. High synovial fluid concentrations are produced by:

a) Clindamycin;
b) Erythromycin;
c) Nystatin;
d) Lincomycin;
e) Cefuroxime;
f) Phenoxymethylpenicillin.

ANTIFUNGAL DRUGS. ANTIPROTOZOAL DRUGS

1. Nystatin-sensitive microorganisms:

a) Causative agents of systemic mycoses (Histoplasma spp. etc.);
b) Causative agents of dermatomycoses;
c) Yeast-like fungi (Candida spp.);
d) Gram-positive bacteria;
e) Gram-negative bacteria.

2. Identify the correct statements about nystatin:

a) Is well absorbed from GIT;
b) Is not absorbed from GIT;
c) Highly toxic;
d) Has a low toxicity;
e) Is used for the treatment of systemic mycoses;
f) Is used for the treatment of superficial mycoses.

3. Amphotericin B resistant microorganisms :

a) Causative agents of systemic mycoses (Histoplasma spp. etc.);
b) Causative agents of dermatomycoses;
c) Yeast-like fungi (Candida spp.);
d) Mold fungi (Aspergillus spp.);
e) Trypanosomes.

4. Identify the correct statements about amphotericin B:

a) Antimycotic spectrum of activity is similar to that of nystatin;
b) Antimycotic spectrum of activity is wider than nystatin's;
c) Good GIT absorption;
d) Has a high toxicity;
e) Is used for the treatment of dermatomycoses;
f) Is used for the treatment of systemic mycoses.

5. Ketoconazole-resistant microorganisms :

a) Causative agents of systemic mycoses (Histoplasma spp. etc.);
b) Causative agents of dermatomycoses (Microsporum spp.);
c) Viruses;
d) Yeast-like fungi (Candida spp.);
e) Mold fungi (Aspergilla spp.).

6. Identify the correct statements about clotrimazole:

a) Antimycotic spectrum of activity is similar to that of nystatin;

b) For topical use;
c) For topical and systemic use;
d) Is used for the treatment of dermatomycoses;
e) Is used for the treatment of systemic mycoses;
f) Good GIT absorption.

7. Identify the correct statements about fluconazole:

a) Well absorbed from GIT;
b) Not absorbed from GIT;
c) Is used for the treatment of systemic mycoses;
d) Has a low toxicity;
e) Inhibits the fungal steroid synthesis pathway;
f) The drug of choice in immunocompromised patients.

8. Griseofulvin-sensitive microorganisms :

a) Causative agents of systemic mycoses (Histoplasma spp. etc.);
b) Causative agents of dermatomycoses (Microsporum spp.);
c) Yeast-like fungi (Candida spp.);
d) Mold fungi (Aspergilla spp.);
e) Protozoa (amebas, leischmanias).

9. Identify the correct statements about griseofulvin:

a) Good GIT absorption;
b) Fungistatic;
c) Provides fast antifungal effect;
d) High concentrations are achieved in the cells producing keratin;
e) Is used for the treatment of systemic candidiasis;
f) Is used for the treatment of dermatomycoses.

10. A drug used for the prevention of candidiasis resulting from broad-spectrum antibiotics:

a) Amphotericin B; c) Nystatin; e) Clotrimazole.
b) Griseofulvin; d) Metronidazole;

11. First-line antibiotic for the treatment of mycoplasmosis and chlamydial infections is:

a) Erythromycin; c) Gentamicin; e) Vancomycin.
b) Cefuroxime; d) Carbenicillin;

12. Effective against preerythrocytic forms of Plasmodium malariae:

a) Chloroquine; c) Pyrimethamine; e) Mefloquine.
b) Quinine; d) Primaquine;

13. Effective against paraerythrocytic forms of Plasmodium malariae:

a) Quinine; c) Pyrimethamine; e) Mefloquine.
b) Primaquine; d) Chloroquine;

14. Effective against sexual forms of Plasmodium malariae:

a) Mefloquine; c) Quinine; e) Primaquine.
b) Methotrexate; d) Chloroquine;

15. Drugs used for the prevention of malaria transmission (community protection measures):

a) Affect preerythrocytic forms of Plasmodium malariae;
b) Affect erythrocytic forms of Plasmodium malariae;
c) Affect gametes;
d) Affect paraerythrocytic forms of Plasmodium malariae.

16. Drugs used for treating of malaria (to eliminate clinical symptoms):

a) Affect preerythrocytic forms of Plasmodium malariae;
b) Affect erythrocytic forms of Plasmodium malariae;
c) Affect gametes;
d) Affect paraerythrocytic forms of Plasmodium malariae.

17. Pyrimethamine (including combinations with sulfonamides) is used for:

a) Malaria treatment;
b) Individual chemoprophylaxis of malaria;
c) Prevention of malaria relapses;
d) Prevention of malaria transmission;
e) Amoebiasis; f) Toxoplasmosis.

18. Drugs active against luminal amebas:

a) Diloxanide; d) Emetine;
b) Quiniofone; e) Tetracyclines;
c) Chloroquine; f) Metronidazole.

19. Drugs effective against amebas residing in the colonic mucosa:

a) Quiniofone; c) Emetine; e) Metronidazole.
b) Chloroquine; d) Doxycycline;

20. Drugs for the treatment of trichomoniasis:

a) Policresulen; d) Trichomonacid;
b) Chloroquine; e) Tinidazole;
c) Metronidazole; f) Furazolidone.

21. Drugs for the treatment of giardiasis

a) Mepacrine; d) Metronidazole;
b) Chlorquinaldol; e) Chloroquine;
c) Furazolidone; f) Tinidazole.

22. Identify the correct statements about mefloquine:

a) Causes arrhythmias;
b) Used for the treatment of malaria symptoms;
c) Used for the individual chemoprophylaxis of malaria;
d) Has a low toxicity;
e) Used for the prevention of malaria transmission.

23. Identify the correct statements about chloroquine:

a) The drug of choice for the treatment of all types of malaria;
b) Used for the treatment of malaria symptoms;

c) Less toxic than other antimalarial agents;
d) Has a high toxicity;
e) Used for the prevention of malaria transmission.

24. Identify the correct statements about primaquine:

a) The drug of choice for the eradication of intrahepatic plasmodia;
b) Used only for the treatment of malaria symptoms;
c) Active against hypnozoites;
d) Highly toxic;
e) Used for the prevention of malaria transmission.

25. Identify the correct statements about metronidazole:

a) Used for the treatment of malaria;
b) Used for the treatment of amebiasis;
c) Used for the treatment of syphilis;
d) Produces disulfiram-like reaction with alcohol;
e) Used for the treatment of giardiasis.

26. Identify the correct statements about tinidazole:

a) Used for the prevention of malaria transmission;
b) Used for the treatment of trichomoniasis;
c) Used for the treatment of all types of malaria;
d) Produces disulfiram-like reaction with alcohol;
e) Used for the treatment of toxoplasmosis.

ANTIMYCOBACTERIAL DRUGS. ANTIVIRAL DRUGS

1. First-line anti-tuberculosis drugs:

a) Isoniazid; c) Rifampicin; e) PASA.
b) Streptomycin; d) Ethambutol;

2. Second-line anti-tuberculosis drugs:

a) PASA; c) Rifampicin; e) Cycloserine.
b) Ethionamide; d) Isoniazid;

3. The most effective anti-tuberculosis drugs (WHO classification):

a) Pyrazinamide; c) Rifampicin; e) Isoniazid.
b) Kanamycin; d) Streptomycin;

4. Multi-drug resistant tuberculosis is resistant:

a) To isoniazid only;
b) To rifampicin only;
c) To ethionamide and rifampicin;
d) To isoniazid and rifampicin;
e) To streptomycin and isoniazid.

5. Antibiotics with anti-tuberculosis activity:

a) Tetracyclines; c) Streptomycin; e) Cycloserine.
b) Vancomycin; d) Rifampicin;

6. Identify the correct statements about isoniazid:

a) One of the most effective anti-tuberculosis drugs;
b) Has a broad antimicrobial spectrum;
c) Affects M. tuberculosis and M. leprae;
d) Blocks the synthesis of mycolic acids (components of the mycobacterial cell wall);
e) Causes peripheral neuropathy.

7. Identify the correct statements about rifampicin:

a) One of the most effective anti-tuberculosis drugs;
b) Has a broad antimicrobial spectrum;
c) Inhibits DNA-dependent RNA-polymerase;
d) Resistance develops slowly;
e) Passes through blood-brain barrier.

8. Identify the correct statements about ethambutol:

a) Affects predominantly M. tuberculosis;
b) Has a broad antimicrobial spectrum;
c) Inhibits the synthesis of mycobacterial cell wall;
d) Causes retrobulbar neuritis;
e) First-line anti-tuberculosis drug.

9. Identify the correct statements about streptomycin:

a) Has a broad antimicrobial spectrum;
b) Affects only M. tuberculosis;
c) Inhibits protein synthesis on ribosomes;
d) Resistance develops rapidly;
e) For parenteral use.

10. Features of M. tuberculosis as a target for chemotherapy:

a) Mycolic acids are the constituents of the mycobacterial cell wall;
b) Sensitive to environmental factors and disinfectants;
c) Resistance to chemotherapeutic agents develops slowly;
d) Intracellular localization;
e) Able to persist in the host organism due to L-forms.

11. Have anti-influenza activity:

a) Rimantadine; c) Saquinavir; e) Acyclovir.
b) Oseltamivir; d) Interferons;

12. Broad-spectrum antiviral agents:

a) Interferons; d) Protease inhibitors;
b) Nucleoside analogs; e) Neuraminidase inhibitors.
c) Interferon inducers;

13. Anti-HIV drugs:

a) Zidovudine; c) Acyclovir; e) Saquinavir.
b) Stavudine; d) Rimantadine;

14. Antiherpetic agents:

a) Acyclovir; c) Idoxuridine; e) Rimantadine.
b) Zidovudine; d) Butaminophen;

15. Used for the treatment of cytomegalovirus infection:

a) Ganciclovir; c) Didanosine; e) Rimantadine.
b) Foscarnet; d) Acyclovir;

16. Identify the correct statements about acyclovir:

a) Purine nucleoside analogue;
b) Inhibits viral DNA-polymerase;
c) Passes through blood-brain barrier;
d) Does not pass through blood-brain barrier;
e) Effective predominantly against Herpes simplex and Herpes zoster.

17. Identify the correct statements about foscarnet:

a) Non-nucleoside analogue of pyrophosphate;
b) Inhibits viral DNA-polymerase;
c) Inhibits the penetration of viruses into cells;
d) Effective against Herpes zoster virus and cytomegalovirus;
e) Used for treating HIV.

18. Identify the correct statements about rimantadine:

a) Aminoadamantane derivative;
b) Inhibits the release of viral genome;
c) Inhibits viral RNA synthesis;
d) Effective against Influenza virus A;
e) Administered orally.

19. Identify the correct statements about ribavirin:

a) Inhibits viral RNA and protein synthesis;
b) Inhibits viral neuraminidase;
c) Interferes with the assembly process;
d) Effective against Influenza virus, Respiratory syncytial virus;
e) Used orally, by inhalations, intravenously.

20. Identify the correct statements about zidovudine:

a) Absorbed from GIT;

b) Inhibits HIV reverse transcriptase, prevents the transcription of viral RNA into DNA;

c) Inhibits HIV proteases, prevents the synthesis of viral structural proteins and enzymes;

d) Causes bone marrow depression;
e) Effective against all RNA-containing viruses.

CONTENTS

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