

**MINISTRY OF HEALTH CARE REPUBLIC OF BELARUS
GOMEL STATE MEDICAL UNIVERSITY**

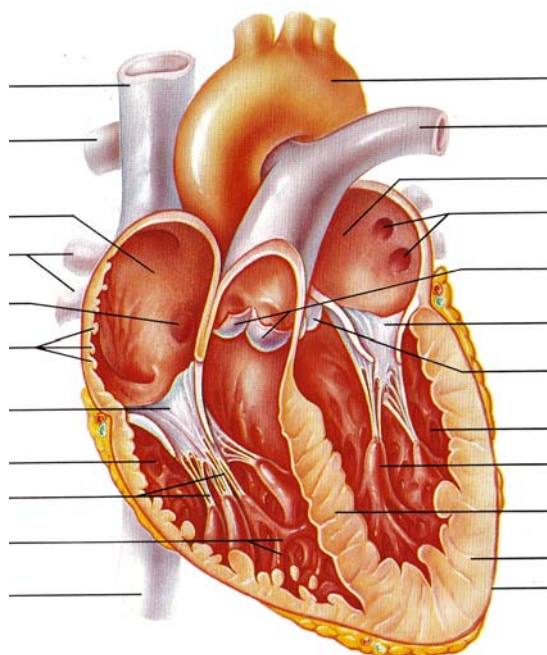
Normal Physiology Department

V. A. MELNIK, Y. I. BREL, N. B. SHEVKO, S. N. MELNIK

HUMAN PHYSIOLOGY

(Illustrations, tables, schemes)

**Educational-methodical guidance
for overseas students in English medium**



Gomel 2007

УДК 612 (076.5)
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Н 83

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Н 83 Human physiology (illustrations, tables, schemes): educational-methodical guidance for overseas students in English medium = Физиология человека: уч.-метод. пособие для иностранных студентов, обучающихся на английском языке / авт.: В. А. Мельник, Ю. И. Брель, Н. Б. Шевко, С. Н. Мельник; под ред. проф. Э. С. Питкевича; пер. на англ. яз. Ю. И. Брель, В. А. Мельника / Гомель: УО «Гомельский государственный медицинский университет», 2007. — 112 с.

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В пособии представлен наглядный материал по нормальной физиологии, для студентов факультета по подготовке специалистов для зарубежных стран, которые обучаются на английском языке.

Авторами представлены рисунки, таблицы, схемы по всем разделам нормальной физиологии. В конце пособия располагаются базовые константы физиологических систем в Международной системе физических единиц (СИ).

Утверждено и рекомендовано к изданию Центральным учебным научно-методическим советом Учреждения образования «Гомельский государственный медицинский университет» 6 марта 2007, протокол № 2.

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FOREWORD

The present guidance contains illustrative material in normal physiology, which can be used by the students of faculty of overseas students in English medium in Educational establishment «Gomel State Medical University» during preparation for the classes. All material corresponds to Program in Normal physiology for students in higher medical schools, No. 08-14/5941 approved by the Ministry of Health of the Republic of Belarus.

The guidance contains illustrations, tables and schemes for all the topics of normal physiology course.

Conclusion includes main constants of a healthy person.

The authors realize that because of the small volume of this guidance they didn't have the opportunity to include all the possible aspects of the normal physiology course. More wide information can be received from the list of literature given in the end of guidance.

Authors will be grateful to all who will make any comments upon this guidance; these will be taken into consideration contributed into development of a new edition of the guidance.

ПРЕДИСЛОВИЕ

Настоящее пособие представляет собой наглядный материал по нормальной физиологии, который может использоваться студентами факультета по подготовке специалистов для зарубежных стран УО «Гомельский государственный медицинский университет», обучающихся на английском языке при подготовке к занятиям. Материал пособия соответствует Программе по нормальной физиологии для студентов лечебно-профилактического факультета высших медицинских учебных заведений № 08-14/5941, утвержденной Министерством здравоохранения Республики Беларусь от 3 сентября 1997 г.

В иллюстрированном пособии авторами представлены современные схемы, таблицы и рисунки по всем разделам нормальной физиологии.

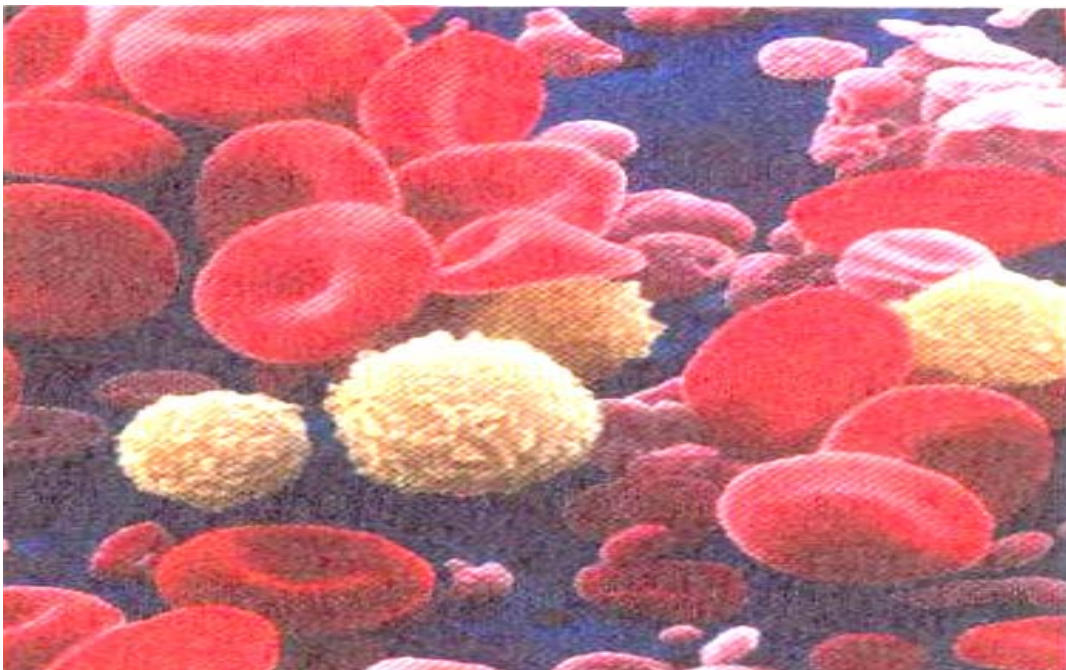
В заключении представлены основные константы здорового человека.

При этом авторы осознают, что в данном пособии в связи с небольшим его объемом не представилось возможным осветить подробно все аспекты рассматриваемых разделов нормальной физиологии. Более расширенные сведения можно получить из списка литературы расположенного в конце пособия.

Авторы будут весьма благодарны всем, кто сочтет возможным высказать свои критические замечания в адрес предлагаемого пособия, которые будут восприняты как выражение желания оказать помощь в его улучшении при последующим переиздании.

Unite 1

PHYSIOLOGY OF BLOOD SYSTEM

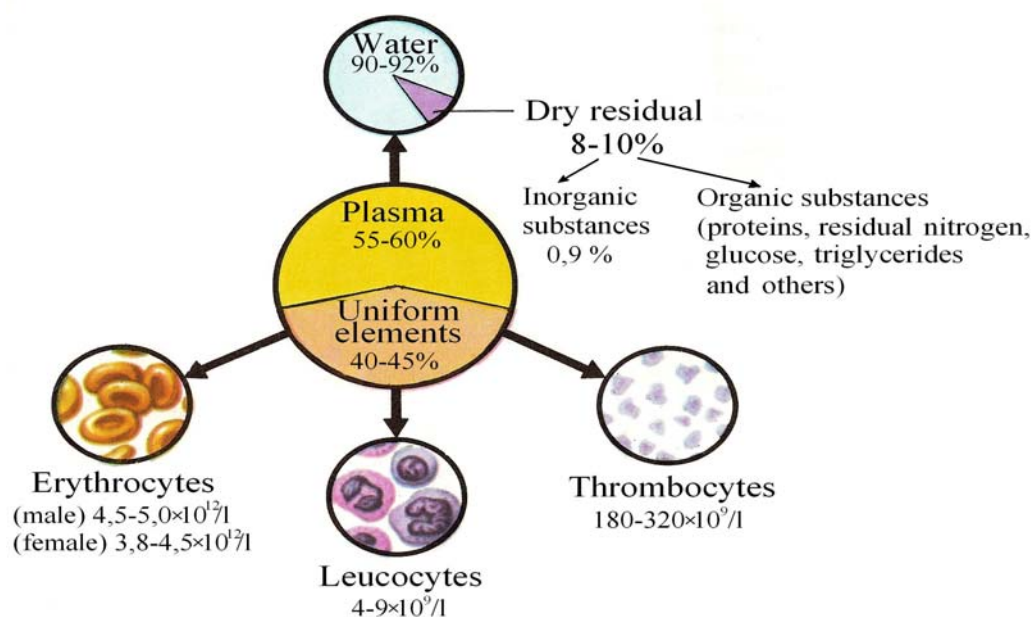


Blood along with interstitial fluid and lymph is an important component of the internal environment of an organism. Changes of physical and chemical properties of blood are the important mechanism in pathogenesis of many diseases and are used for diagnostics, evaluation of the efficiency of treatment and prognosis.

System of blood includes:

1. Blood (in vessels).
2. Organs of haemopoiesis (red bone marrow, lymph nodes, spleen, thymus gland).
3. Organs of blood destruction (liver, bone marrow, spleen).
4. Neurohumoral apparatus.

Blood consists of plasma and uniform elements (erythrocytes, thrombocytes, leucocytes).



Scheme 1.1. — The composition of blood
(by Korobkov A. V., Chesnokova S. A., 1986)

Table 1.1. — Changes of blood volume

Interrelation between uniform elements and plasma		Variant of volemia	Hematocrit number
		NORMOVOLEMIA	
UE 0,45%	plasma 0,55%	Simple	normal
UE 0,35%	plasma 0,65%	Oligocythemic	below normal
UE 0,55%	plasma,45%	Polycythemic	above normal
		HYPOVOLEMIA	
UE 0,45%	plasma	Simple	normal
UE 0,35%	plasma	Oligocythemic	below normal
UE 0,55%	plasma,45%	Polycythemic	above normal
		HYPERVOLEMIA	
UE 0,45%	plasma 0,55%	Simple	normal
UE 0,35%	plasma 0,65%	Oligocythemic	below normal
UE 0,55%	plasma,45%	Polycythemic	above normal

Note: UE — uniform elements of blood.

Blood basic functions:

1. Transport of various substances.
2. Respiratory function.
3. Trophic or nutritional function.
4. Excretory function.
5. Blood participates in thermoregulation.
6. Homeostatic function.
7. Blood provides water-salt exchange between blood and tissues.
8. Protective function.
9. Correlative function.
10. Blood maintains the constancy of base-alkaline state due to buffer systems.

Table 1.2. — Composition and functions of blood plasma

Components			Amount	Functions	
Water			90–92%	solvent for other substances	
Dry residual	Organic substances	Proteins:	albumins	45 g/l	<ul style="list-style-type: none">• form 80% of colloid-osmotic pressure• participate in regulation of water-salt balance.• participate in transport of many substances• bind some hormones• form protein reserve
			globulins	20–35 g/l	<ul style="list-style-type: none">• transport of hormones, vitamins, microelements• Immune function (γ-globulins)
			fibrinogen	3 g/l	<ul style="list-style-type: none">• participates in blood coagulation.
		Glucose		3,88–6,10 millimole/l	<ul style="list-style-type: none">• mainly energetic function
		Triglycerides		0,40–1,81 millimole/l	<ul style="list-style-type: none">• they are present in blood mainly in forms of lipoproteins and chylomicrons — forms which transport lipids to different organs and tissues
		Cholesterin		3,64–6,76 millimole/l.	
		Residual nitrogen		14,3–28,5 millimole/l	<ul style="list-style-type: none">• It consists mainly of end products of metabolism which are transported by blood to the organs of excretion
	Inorganic substances	Na^+ , K^+ , Ca^{2+} chlorides, phosphates, hydrocarbonates	0,9%	<ul style="list-style-type: none">• participate in maintenance of osmotic pressure;• participate in processes of excitation and contraction of cells• participate in coagulation of blood (Ca^{2+});• participate in the regulation of acid-base state	

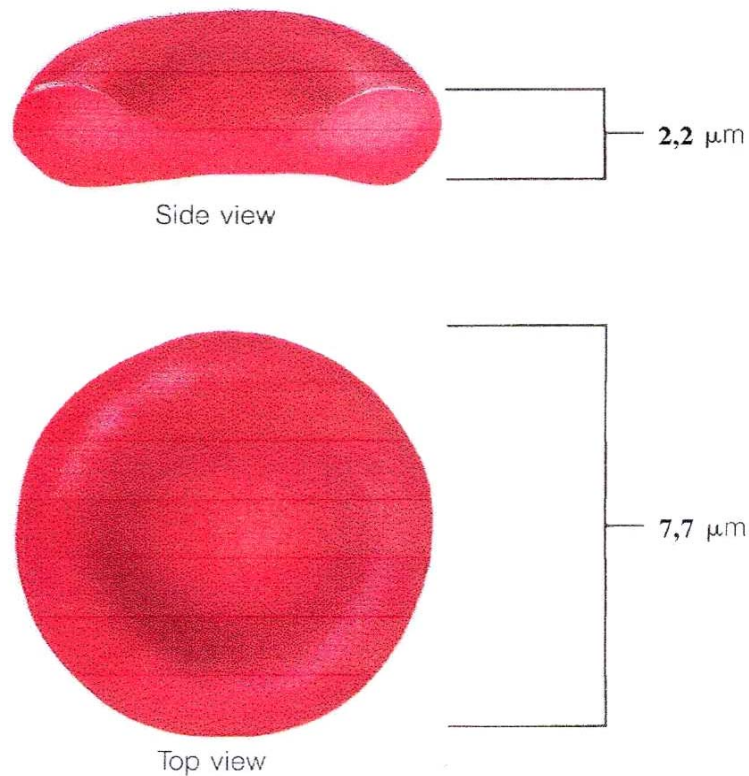


Figure 1.1. — The form and size of erythrocyte

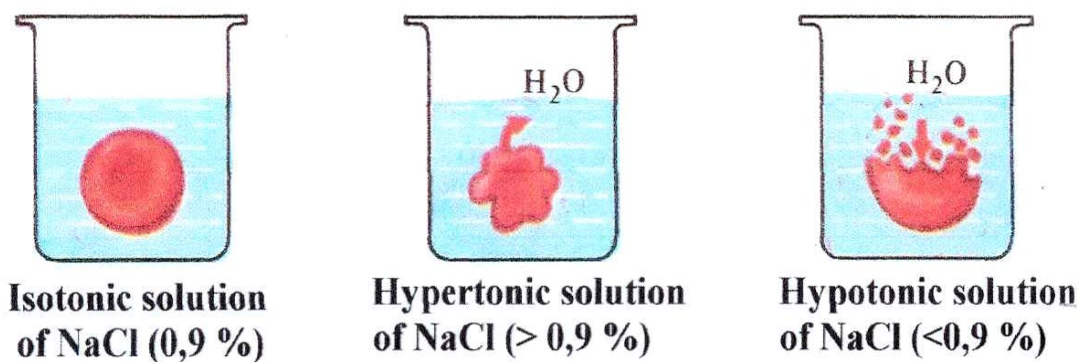


Figure 1.2. — The state of erythrocyte in solutions with different concentrations of NaCl (in hypotonic solution there is an osmotic hemolysis)
(by Korobkov A. V., Chesnokova S. A., 1986)

Functions of erythrocytes:

1. Transition of O_2 (due to hemoglobin).
2. Transition of CO_2 (due to hemoglobin).
3. Protective (absorption of harmful substances, production of antibiotic-eritrin).
4. Regulation of water-and-salt exchange.
5. Transition of nutrients.
6. Participation in regulation of erythropoiesis.

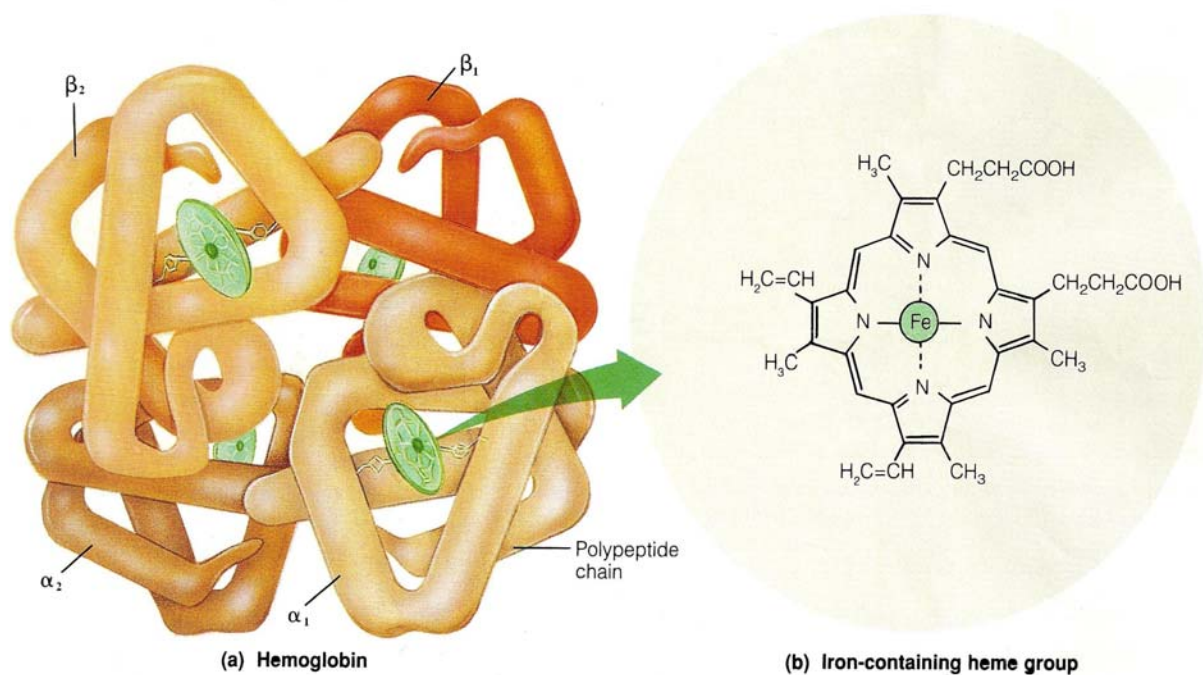


Figure 1.3. — Structure of hemoglobin (by Elaine N. Marieb, 1989)

(a) The intact hemoglobin molecule is composed of the protein globin bound to the iron-containing heme pigments. Each globin molecule has four polypeptide chains: two alpha (α) chains and two beta (β) chains. Each chain is complexed with a heme group, shown as a beaded structure.

(b) Structure of a single heme group.

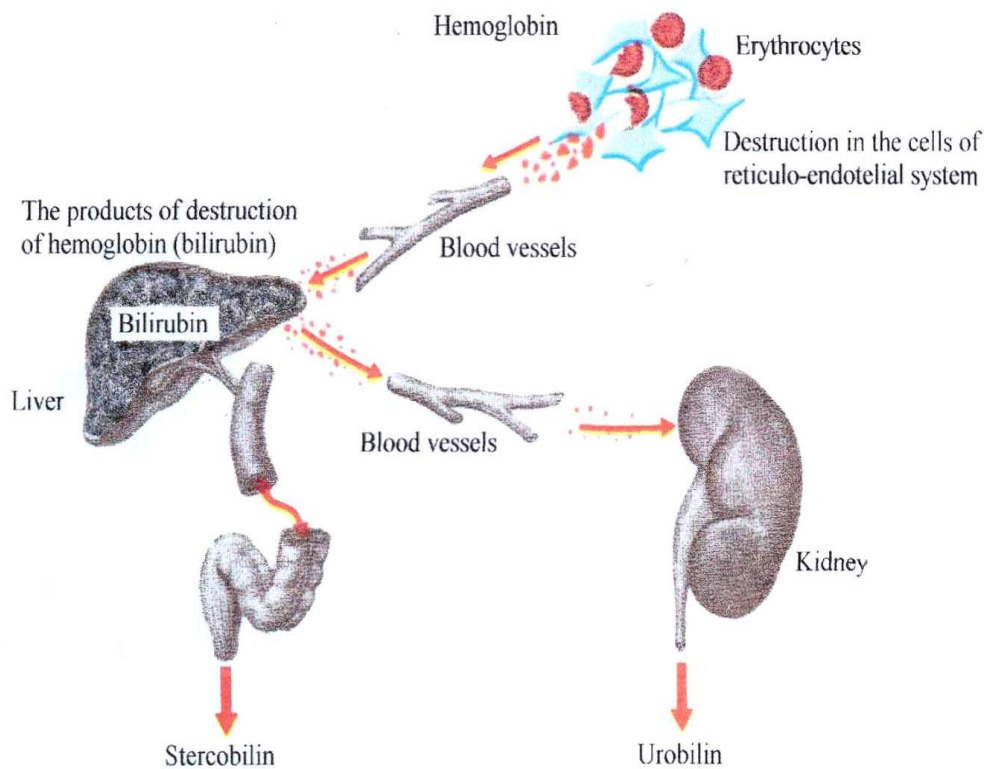

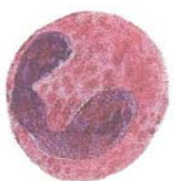
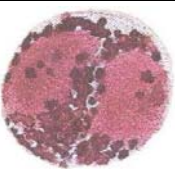

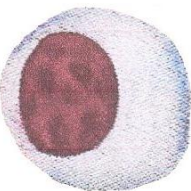

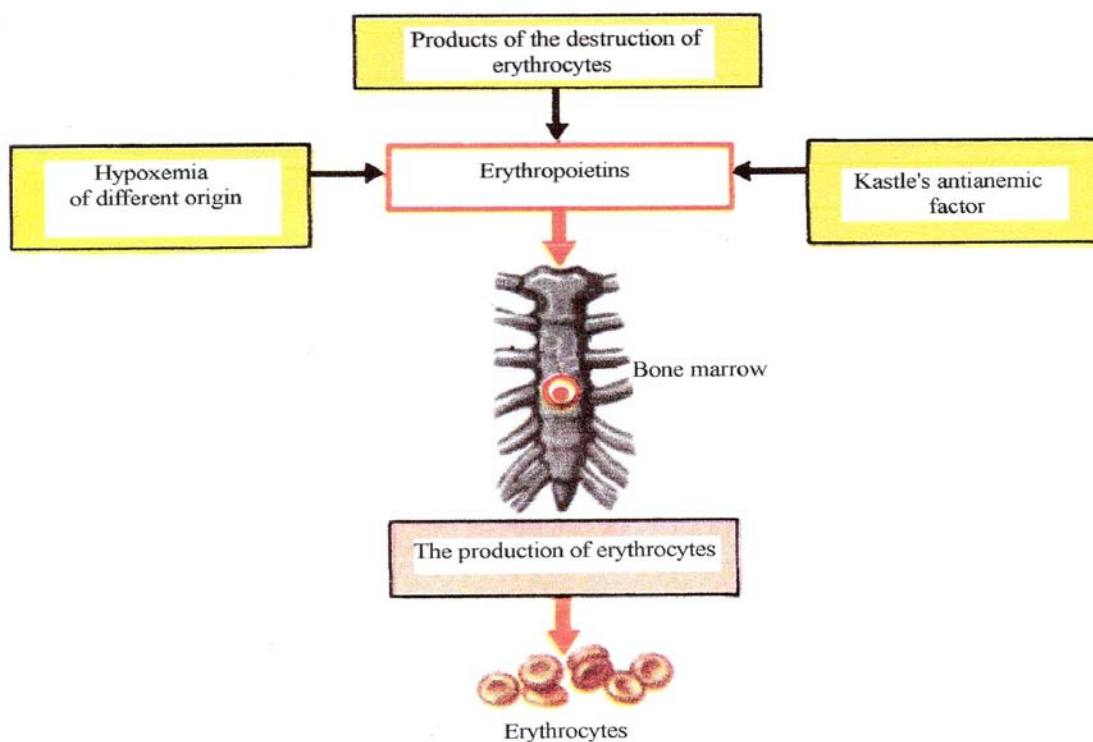


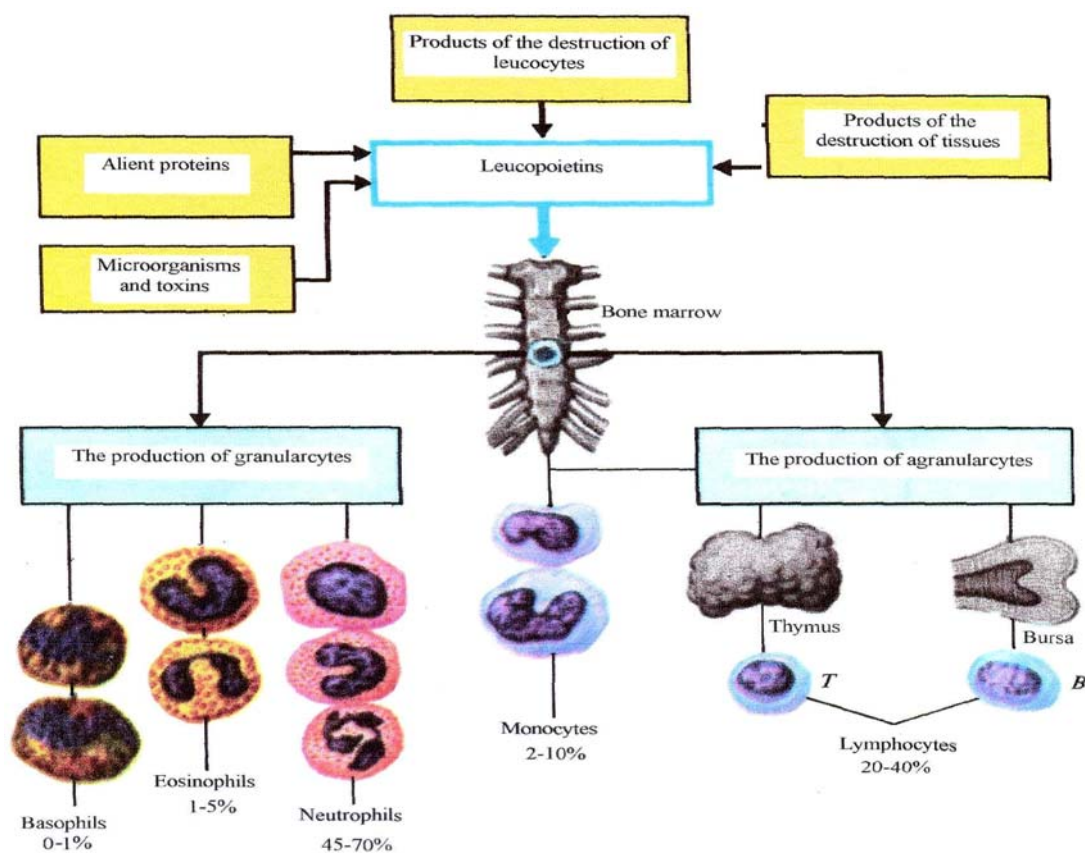
Figure 1.4. — The destruction of hemoglobin (by Korobkov A. V., Chesnokova S. A., 1986)

Table 1.3. — Classification of leucocytes

Type of cells		Amount (in %)	Functions	Morphology of cell
Granular leucocytes	<u>Neutrophils:</u> myelocytes metamyelocytes stab neutrophils segmentonuclear	0 0–1 1–5 45–70	They possess high bactericidal activity. They have receptors to IgG, to proteins of complement on their membrane.	 segmentonuclear neutrophil
	Eosinophils	1–5	<ul style="list-style-type: none"> • phagocytosis • neutralization of toxins of the albuminous nature • destruction of alien proteins and antigen-antibody complexes • production of plasminogen (participate in fibrinolysis) • cytotoxic effect on helminthes, their eggs 	 eosinophil
	Basophils	0–1	They produce histamine (it dilates capillaries). and heparin(it prevents coagulation of blood) they have receptors to IgE	 basophil
Agranular leucocytes	<u>Lymphocytes</u> <i>T - lymphocytes</i> (provide cellular immunity): a) T - helpers b) T - suppressors c) T - killers d) T - accelerators f) T-lymphocytes of immune memory <i>B - lymphocytes</i> (provide non-cellular immunity) a) plasma cells b) B - killers c) B - helpers d) B - suppressors f) B-lymphocytes of immune memory <i>Zero lymphocytes</i> (nature killers)	20–40	<ul style="list-style-type: none"> • antibody formation • destruction of alien cells • provide reaction of a transplant rejection • keep immune memory • destruction of own mutant cells • state of sensibilization 	 lymphocyte  plasma cell
	Monocytes	2–10	In tissues they turn into tissue macrophages and perform phagocytosis of microorganisms, died leucocytes, damaged cells of a tissue.	 monocyte



**Scheme 1.2. — Factors which stimulate erythropoiesis
(by Korobkov A. V., Chesnokova S. A., 1986)**



**Scheme 1.3 — Factors which stimulate leucopoiesis
(by Korobkov A.V., Chesnokova S.A., 1986)**

Table 1.4. — Blood groups of system AB0

Blood groups	Erythrocytes	Plasma or serum
	Agglutinin	Agglutinin
I (0)	0	α, β
II (A)	A	β
III (B)	B	α
IV (AB)	AB	0

Table 1.5. — Compatibility of various blood groups

Serum group	Erythrocyte group			
	I (0)	II (A)	III (B)	IV (AB)
I α, β	—	+	+	+
II β	—	—	+	+
III α	—	+	—	+
IV	—	—	—	—

Note: «+» — presence of agglutination (group incompatibility);
 «—» — absence of agglutination (group compatibility).

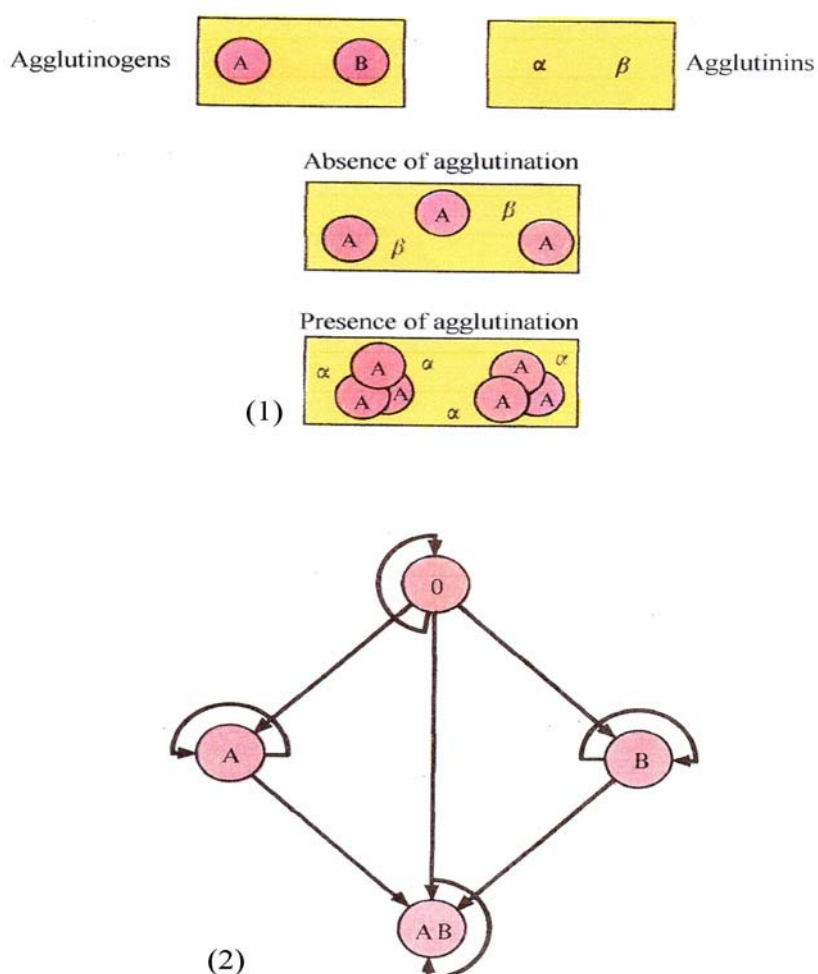


Figure 1.5. — Blood groups (by Korobkov A. V., Chesnokova S. A., 1986)

- (1) Mechanism of agglutination
 (2) Possible variants of transfusion of blood of different groups.

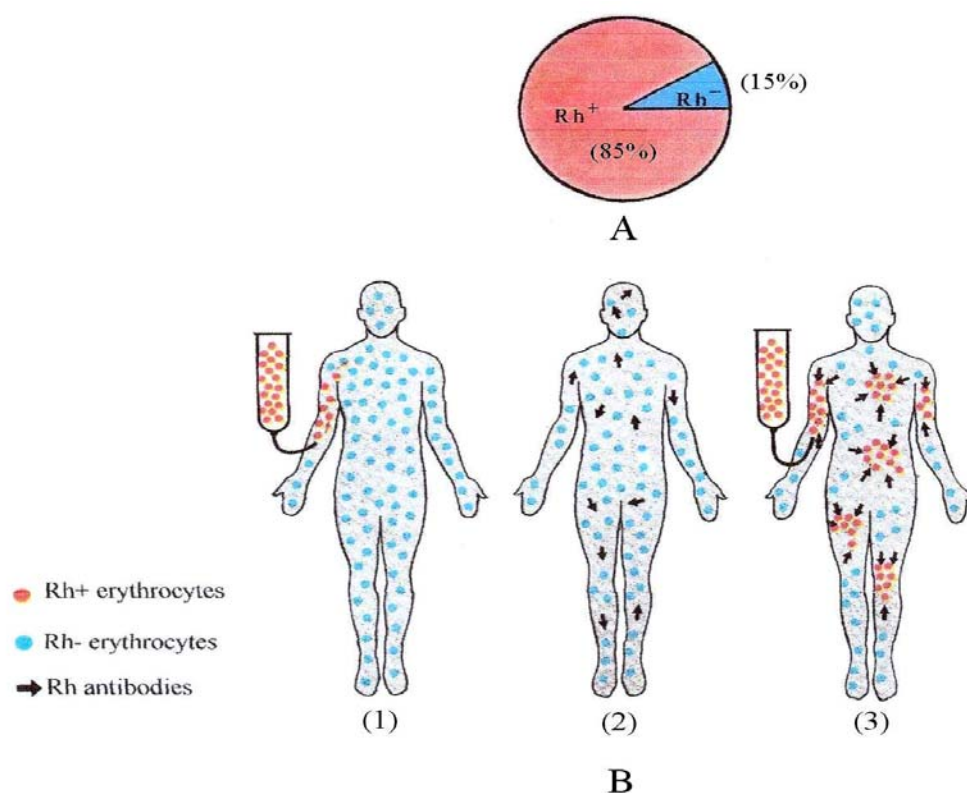


Figure 1.6. — Rhesus factor (Rh) (by Korobkov A. V., Chesnokova S. A., 1986)

A — frequency of Rh⁺ and Rh⁻ people. B — «Rhesus conflict».
 (1) — transfusion of Rh⁺ blood to Rh⁻ recipient. (2) — the production of Rh antibodies in the organism of recipient. (3) — the second transfusion of Rh⁺ blood to Rh⁻ recipient causes agglutination.

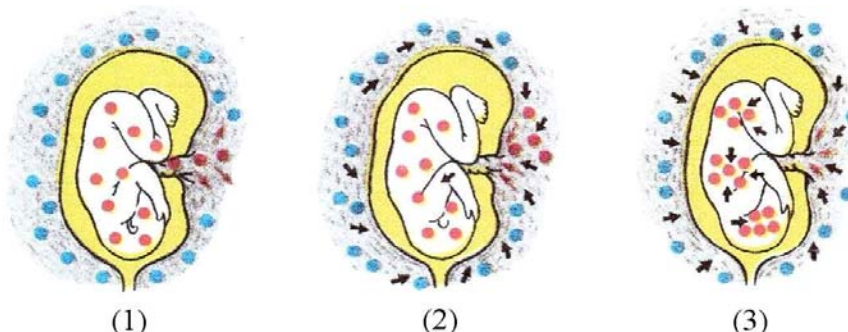
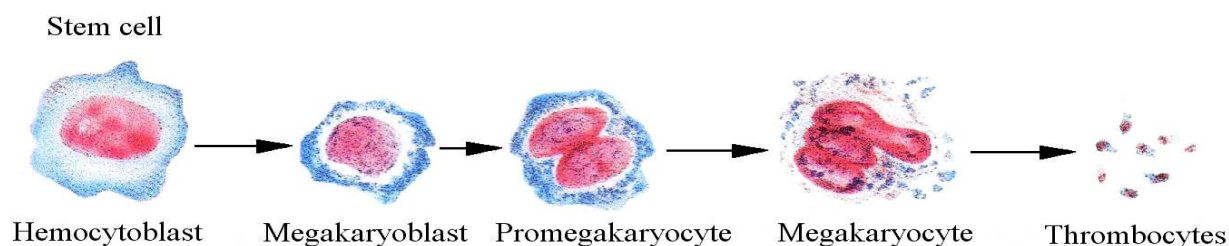


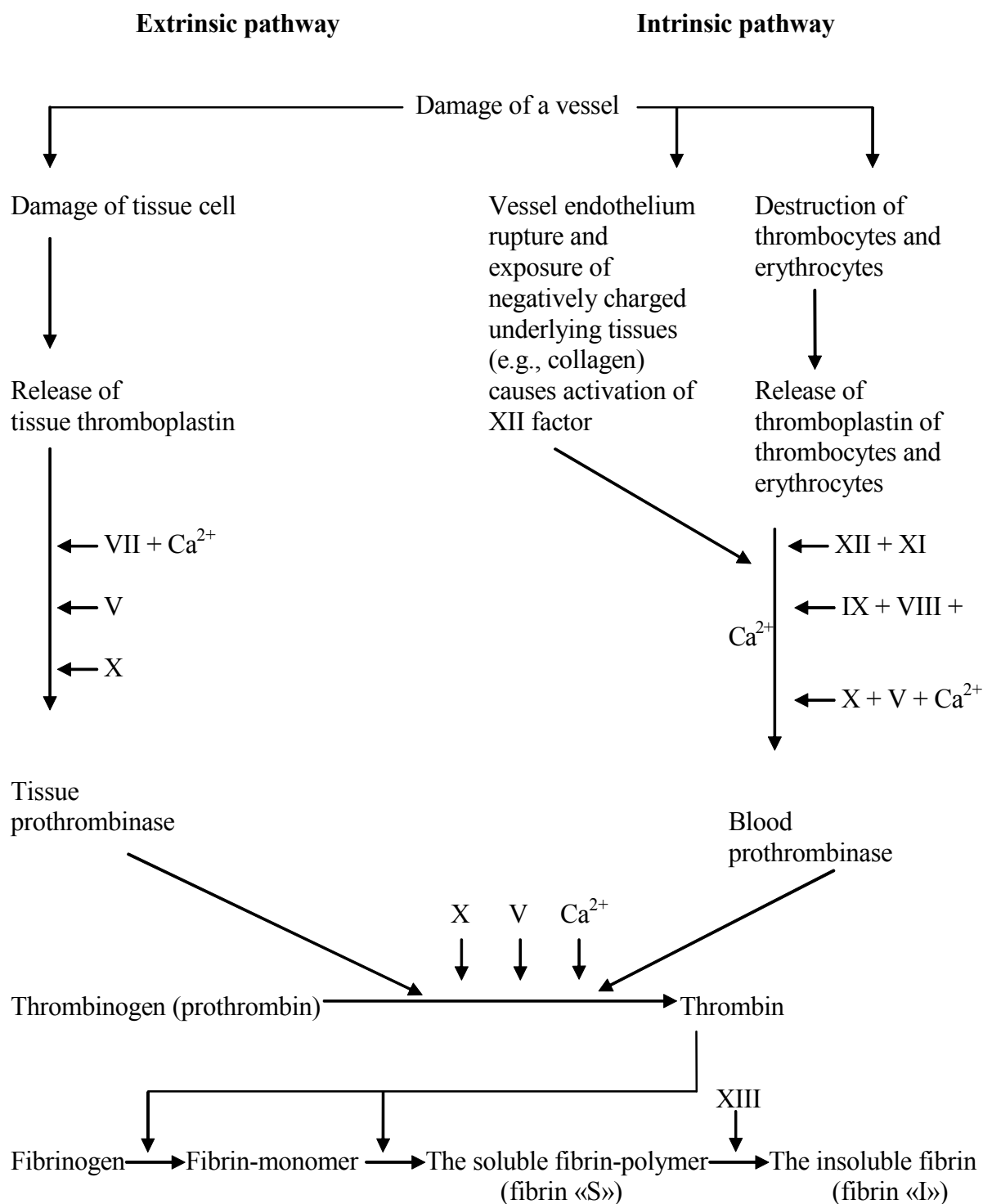
Figure 1.7. — Rhesus conflict between pregnant women and fetus (by Korobkov A. V., Chesnokova S. A., 1986)

I — immunization of Rh⁻ mother by Rh⁺ erythrocytes of fetus.
 II — the production of Rh antibodies in the organism of mother.
 III — agglutination of Rh⁺ erythrocytes of fetus by antibodies of mother.



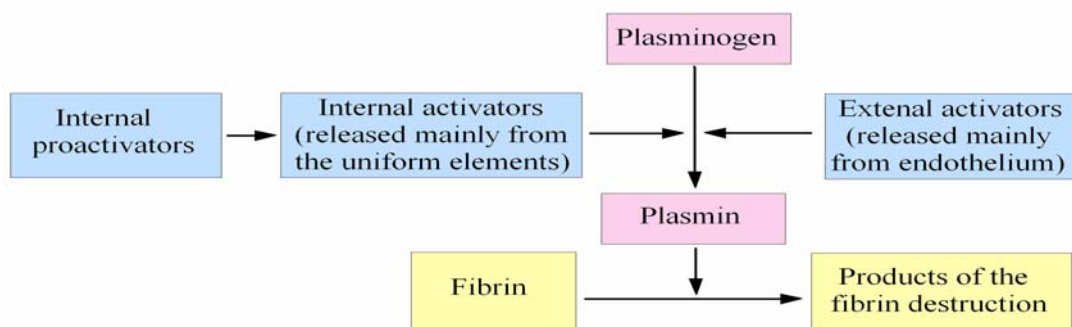
Scheme 1.4. — Genesis of thrombocytes (platelets) (by Elaine N. Marieb, 1989)

The stem cell (hemocytoblast) gives rise to cells that undergo several mitotic divisions unaccompanied by cytoplasmic division to produce the megakaryocytes. The cytoplasm of the megakaryocyte becomes compartmentalized by membranes, and the plasma membrane then fragments, liberating the thrombocytes.



Note: uniform elements of blood get stuck into fibrin nets, thus forming the blood thrombus. Such thrombus is subject to compression influenced by protein trombostenin. At compression of thrombus the periphery of the wound are closing.

Scheme 1.5. — Coagulating hemostasis



Scheme 1.6. — The activation of fibrinolysis (by K. Kluft, A. Gi, 1979)

Table 1.6. — Humoral factors which regulate haemopoiesis

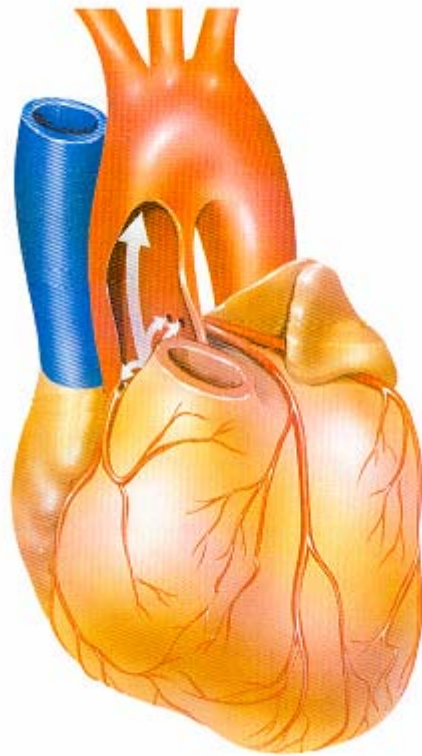
Humoral factors which regulate erythropoiesis		Humoral factors which regulate leucopoiesis		Humoral factors which regulate thrombocytopoiesis	
Factor	The role of the factor	Factor	The role of the factor	Factor	The role of the factor
Iron	In the structure of hemoglo- bin there is iron-containing heme group	Products of leu- kocytes destruc- tion	The more destruction of leucocytes is, the higher is their formation.	Throm- bocyto- poietins of short action.	They are formed in liver and stimulate release of thrombo- cytes into blood.
Vitamin B ₁₂	It is necessary for normal formation growth and of erythrocytes	Tissue destruc- tion products	They stimulate leu- copoiesis		
Kastle's anti- anemic fac- tor	It is necessary for the ab- sorption of vitamin B ₁₂ in the intestine	Microbes and their toxins.	They stimulate leu- copoiesis	Throm- bocyto- poietins of long action.	They are contained in blood plasma and stimulate for- mation of throm- bocytes in the bone marrow
Ascorbic acid	It promotes transmitting Fe ⁺⁺⁺ into Fe ⁺⁺ and absorp- tion of iron in the intestine	Leukopoietins	They stimulate leu- copoiesis		
Erythropoi- etins	They influence on cells- predecessors of erythro- cytes, stimulate their pro- liferation synthesis of he- moglobin	Adrenalin, hy- drocortisone	They cause leukocyto- sis due to release of leucocytes from blood depot (but glucocorti- coids decrease the for- mation of eosinophils and lymphocytes)		
Products of erythrocytes destruction	They stimulate erythropoi- esis (autoregulation). The amount of the destroyed erythrocytes is equal to that of newly formed erythro- cytes (self-control).	Interleukins	They stimulate leu- copoiesis		
Androgens adrenalin, thyroxin, somatotropic hormone	They stimulate erythrogenesis				
Estrogens	They decrease erythro- genesis				

Table 1.7. — Blood substituting solutions

Kind of the solution	Examples of the solutions	Positive property of the solutions	Negative property of the solutions	The cases of using the solutions
Salt solutions	Saline solution — (0,85–0,9% NaCl), Ringer-Lock's solution.	They do not cause allergic reactions (sensibilization)	They are quickly released from blood vessels	They can fill the amount of the lost blood within short period of time. They can be used for normalization of water-salt exchange and acid-base state
Synthetic colloid solutions	Reopoliglucin, Macrodex, Haemodez	They stay in the blood vessels for a long period of time They can bind toxic substances	They can cause allergic reactions (sensibilization)	They can fill the amount of the lost blood within long period of time. They can be used for improving of hemodynamics at the state of shock. They can be used at the state of intoxication.
Protein preparations	Solution of albumin (5%), Solution of gelatin (8%) Native, preserved and fresh frozen plasma	– // – – // –	The preparations of plasma can contain dangerous infections agents (for example – HIV, virus of hepatitis B)	– // – – // –
Preparations of blood	Preserved blood and plasma, Erythrocytes mass, Leukocytes (fresh), Thrombocytes (fresh)	Different kinds of preparations have special qualities according to the components of preparation	The preparations of blood can contain dangerous infections agents (for example – HIV, virus of hepatitis B). The preparations of blood have antigenic qualities and can cause posttransfusion complications.	Different kinds of preparations can be used according to what is necessary for a patient (for example at decreased amount of thrombocytes fresh thrombocytes can be transfused)

Unite 2

PHYSIOLOGY OF CARDIOVASCULAR SYSTEM



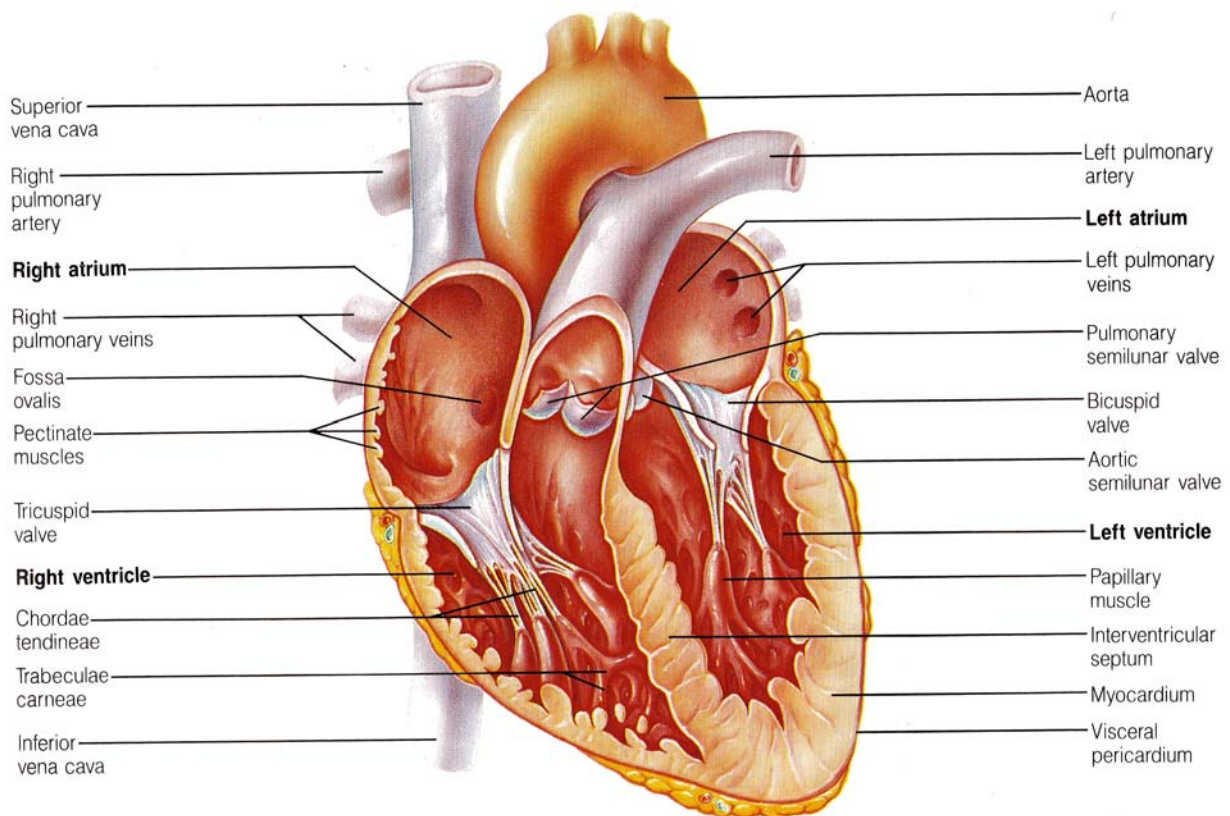


Figure 2.1. — Anatomy of the heart (frontal section showing inferior chambers and valves)
(by Elaine N. Marieb, 1989)

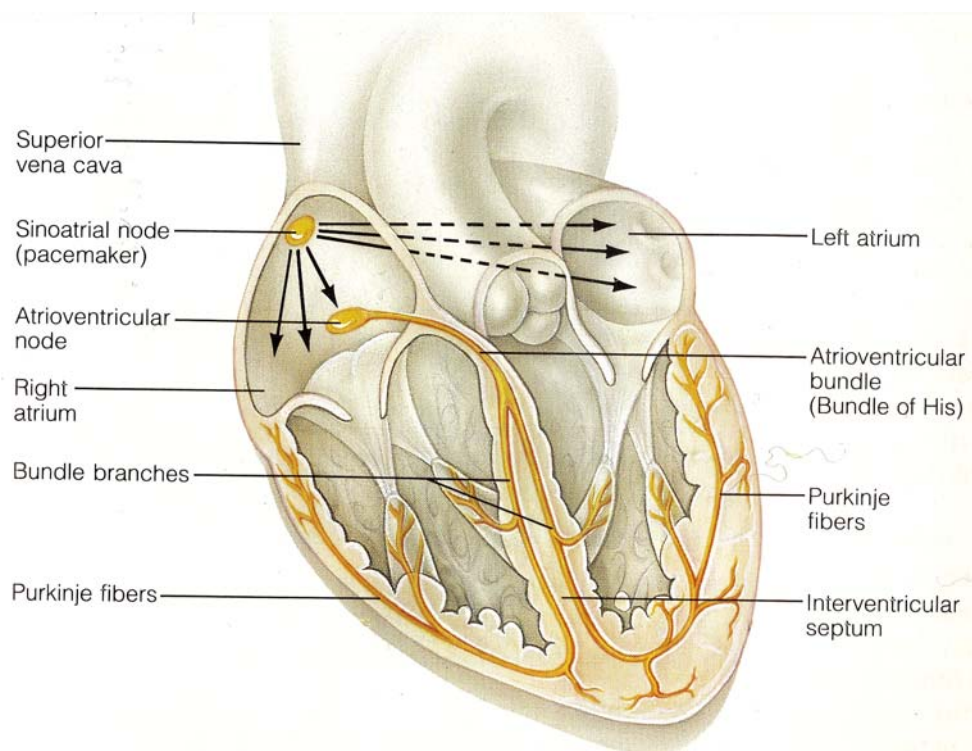
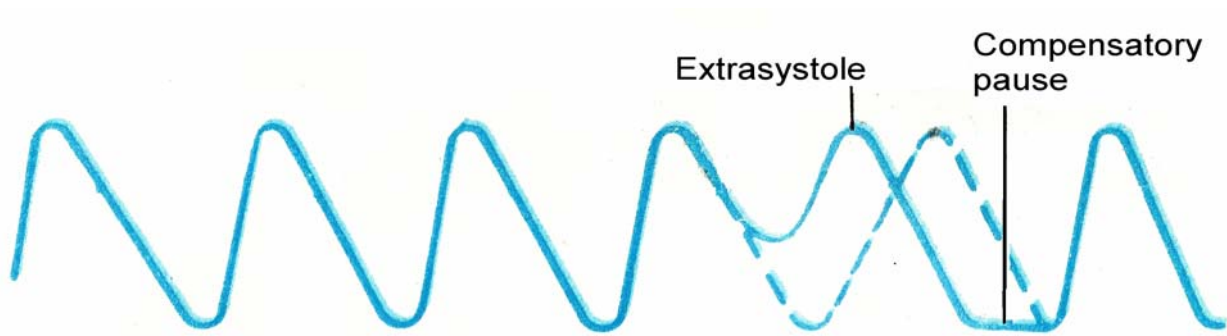
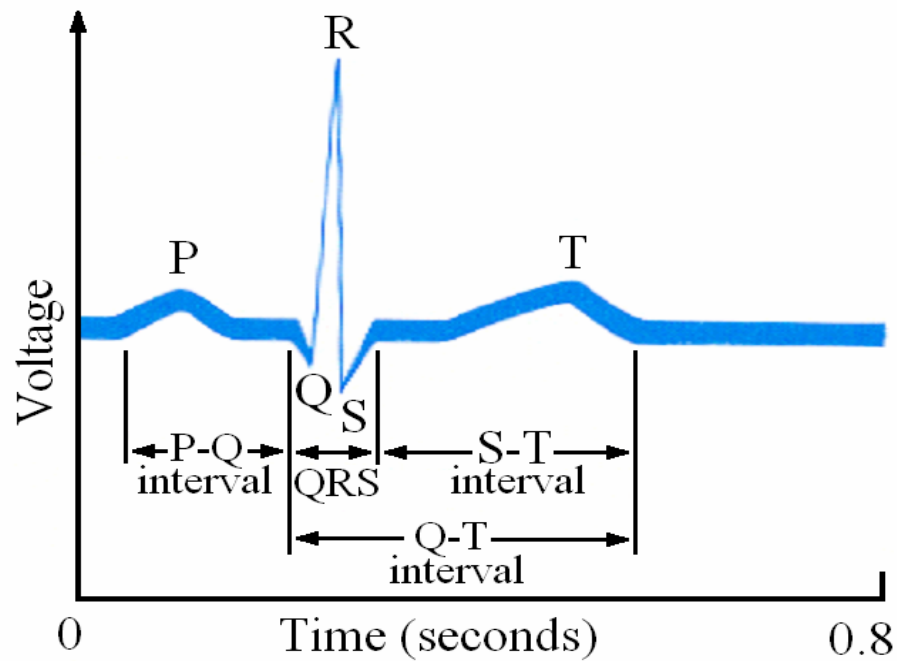


Figure 2.2. — The conduction system of the heart (by Elaine N. Marieb, 1989)

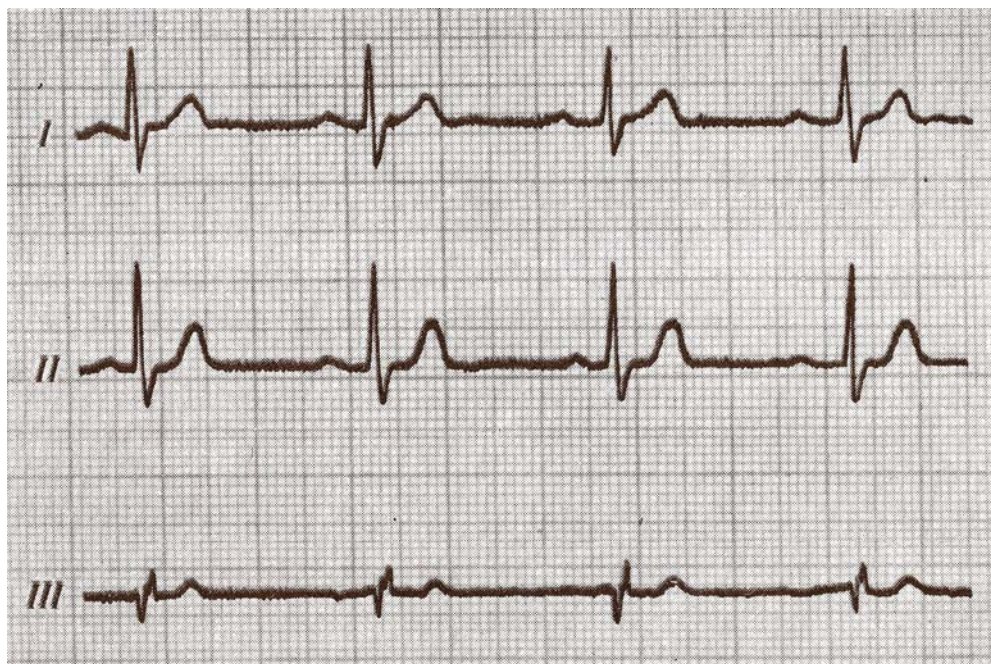
The depolarization wave is initiated by the sinoatrial node and then passes through the atrial myocardium to the atrioventricular node, bundle of His, the right and the left bundle branches, and the Purkinje fibers in the ventricular myocardium.



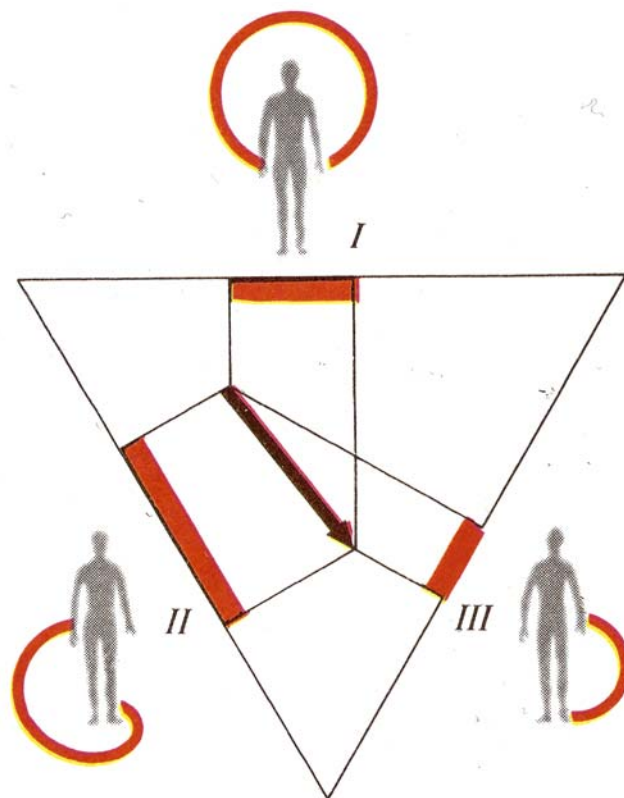
Scheme 2.1. — Extrasystole (schematic illustration)



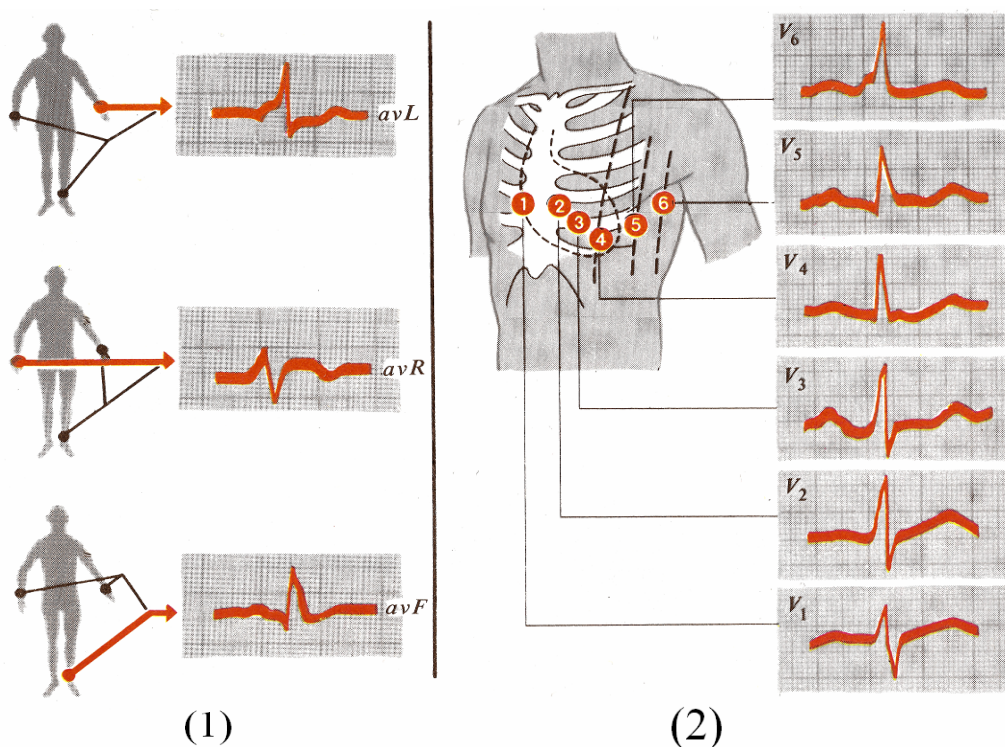
Scheme 2.2. — An electrocardiogram and its three positive and two negative waves



Scheme 2.3. — Electrocardiography (bipolar leads from extremities, Einthoven method)



Scheme 1.4 – Scheme which explains the difference between amplitudes of wave R in three bipolar (standard) leads (I, II, III) from extremities (Einthoven method) (by Korobkov A.V., Chesnokova S.A., 1986)

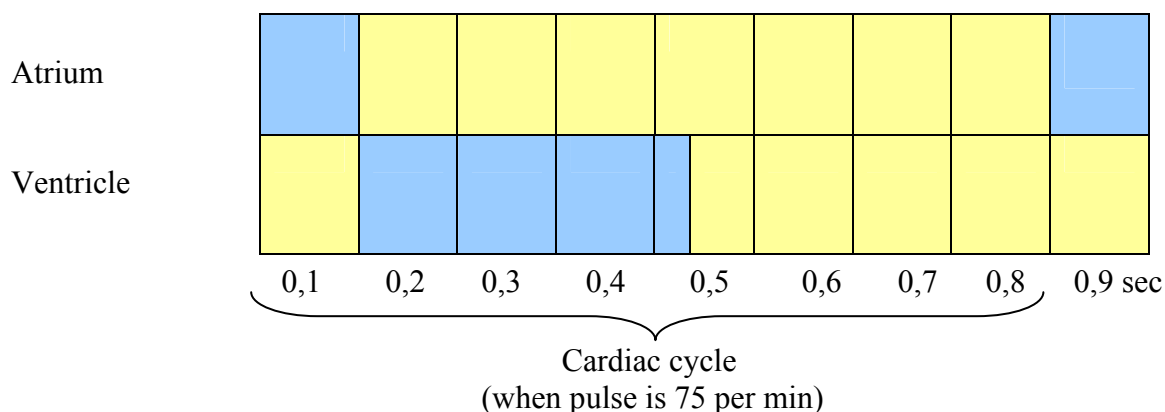


Scheme 2.5. — Electrocardiography (unipolar leads) (by Korobkov A. V., Chesnokova S. A., 1986)

- (1) — Unipolar leads from extremities (Goldberger's method)
- (2) — Unipolar thoracic (pre-cardiac) leads (Wilson method)

Table 2.1. — Periods and phases of cardiac cycle

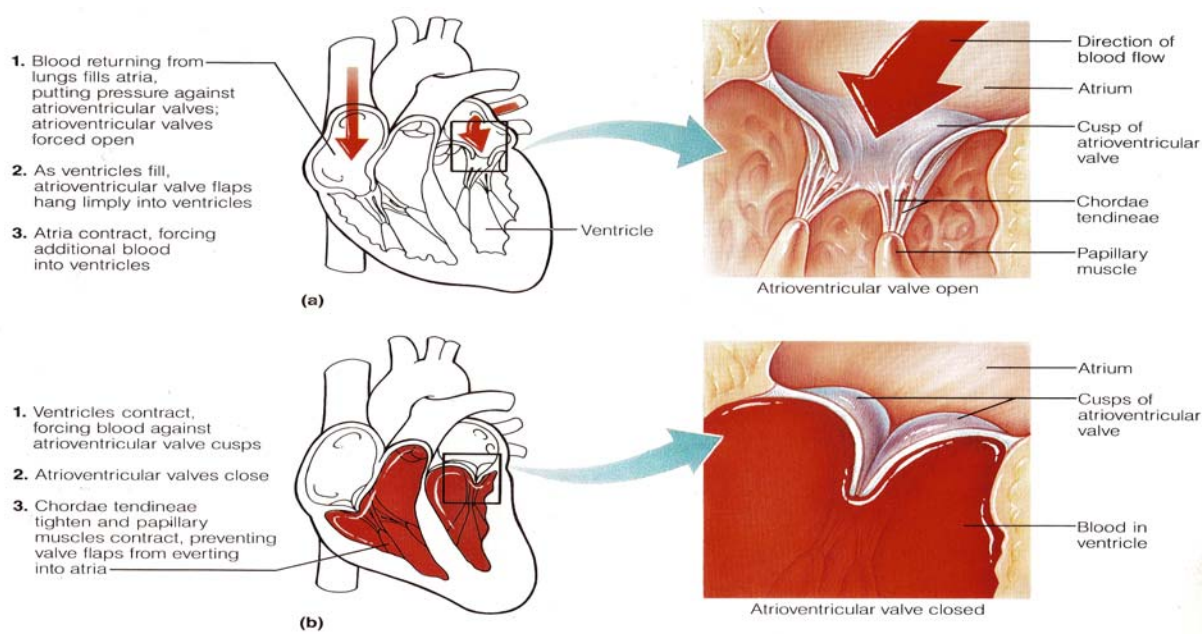
Systole of ventricles — 0,33 sec	Period of <i>extension</i> — 0,08 sec	Phase of <i>asynchronous</i> contraction — 0,05 sec
		Phase of <i>isometric</i> contraction — 0,03 sec
	Period of <i>expulsion</i> — 0,25 sec	Phase of <i>fast expulsion</i> of blood — 0,12 sec
		Phase of <i>slow expulsion</i> of blood — 0,13 sec
Diastole of ventricles — 0,47 sec	The <i>protodiastolic</i> period — 0,04 sec	
	The period of <i>isometric relaxation</i> — 0,08 sec	
	Period of <i>filling</i> of ventricles with blood — 0,25 sec	Phase of <i>fast</i> filling — 0,08 sec
		Phase of <i>slow</i> filling — 0,17 sec
	The <i>presystolic</i> period — 0,1 sec	



**Scheme 2.6. — Duration of systole and diastole of atriums and ventricles
(during one cardiac cycle)**

Systole is showed with the blue color.

Diastole is showed with the yellow color.



**Figure 2.3. — Operation of the atrioventricular valves of the heart
(by Elaine N. Marieb, 1989)**

(a) — The valves open when the blood pressure exerted on their atrial side is greater than that exerted on their ventricular side;

(b) — The valves are forced closed when the ventricles contract, moving their contained blood superiorly

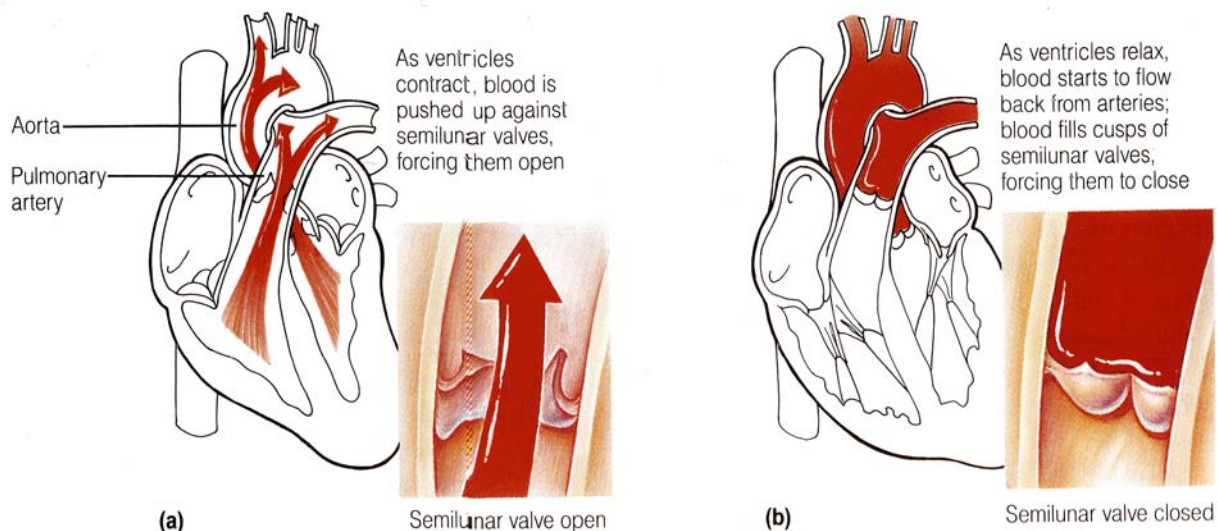


Figure 2.4. — Operation of the semilunar valves (by Elaine N. Marieb, 1989)

(a) — During ventricular contraction, the valves are open and their flaps are flattened against the artery walls;

(b) — When the ventricles relax, the backflowing blood fills the valve cusps and closes the valves

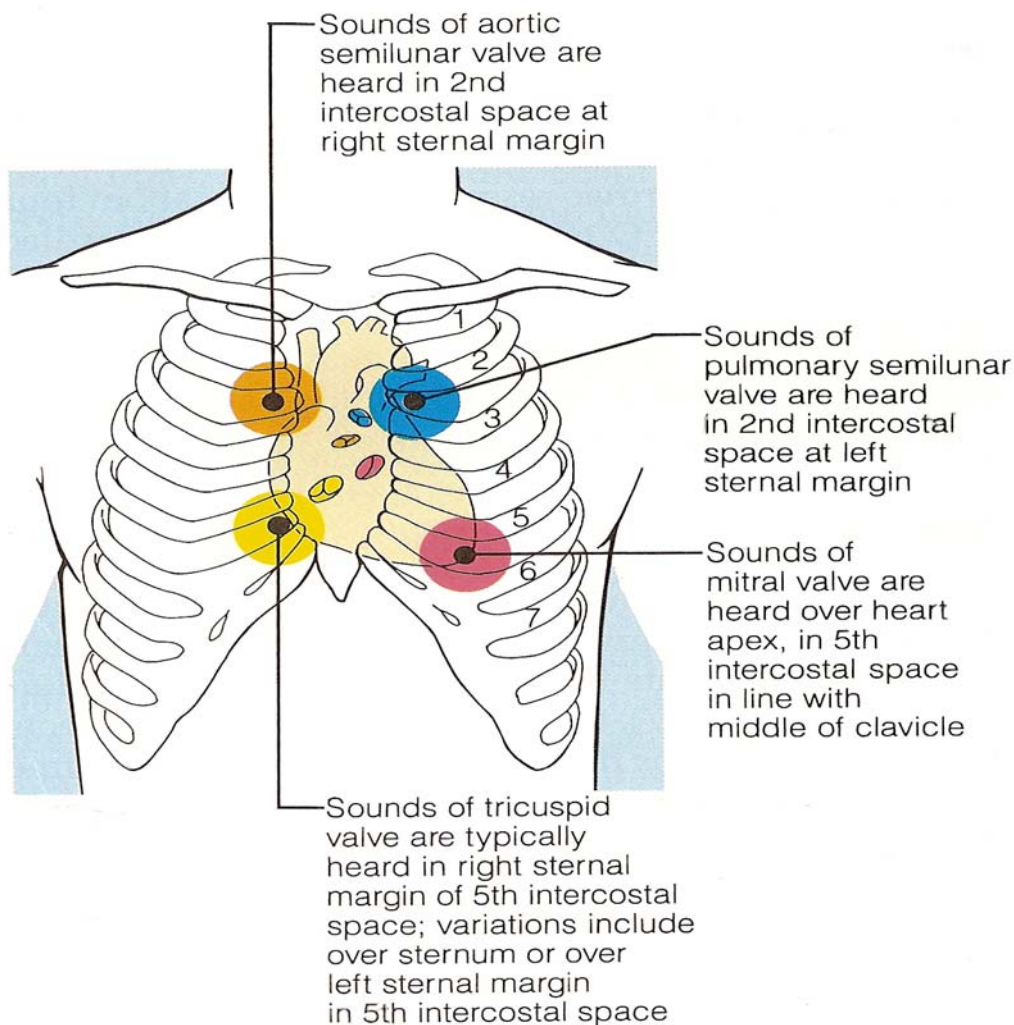


Figure 2.5 — Areas of the thoracic surface where the sounds of the heart can be best detected (by Elaine N. Marieb, 1989)

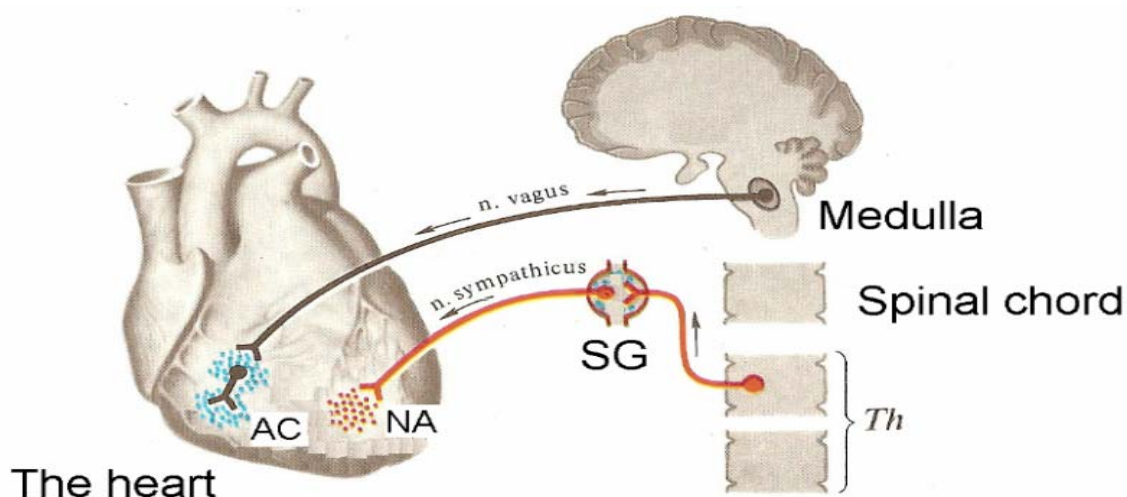
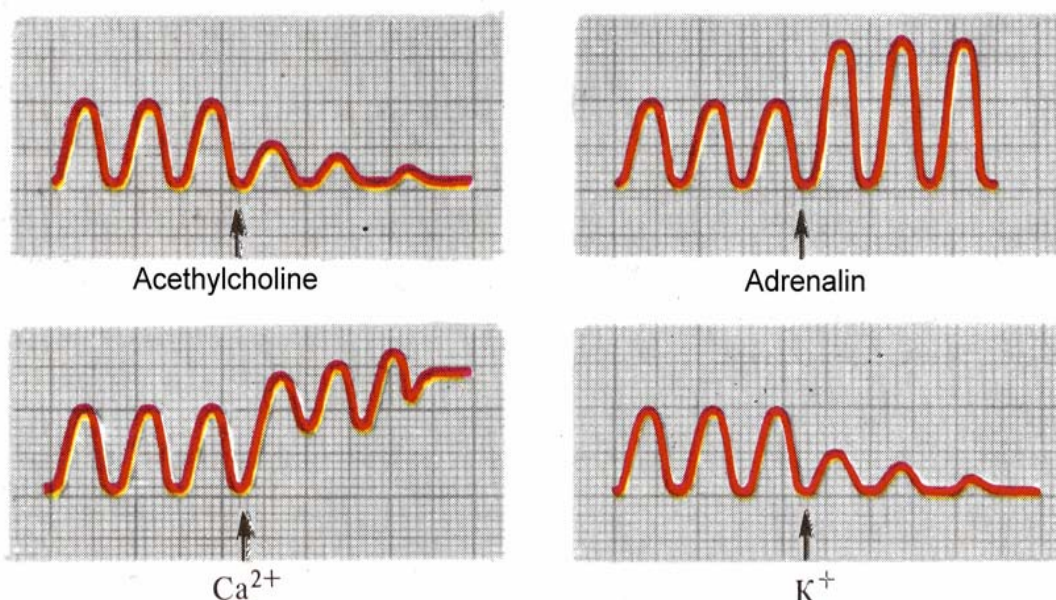


Figure 2.6 — Sympathetic and parasympathetic innervation of the heart
 (by Korobkov A. V., Chesnokova S. A., 1986)
 AC — acetylcholine; NA — noradrenalin; SG — sympathetic ganglion



Scheme 2.7. — The result of influence of increased amount of some humoral factors on the heart activity (arrows show the moment when the influence begins)
 (by Korobkov A.V., Chesnokova S.A., 1986)

Table 2.2. — Influence of the vegetative nervous system on the work of heart

The influence of vagus nerve	Influences of sympathetic nerve
1. Negative chronotropic effect (decreasing of rhythm of contractions).	1. Positive chronotropic effect (increasing of rhythm of contractions).
2. Negative inotropic effect (decreasing of amplitude of contractions).	2. Positive inotropic effect (increasing of amplitude of contractions).
3. Negative bathmotropic effect (decreasing of excitability of myocardium).	3. Positive bathmotropic effect (increasing of excitability of myocardium).
4. Negative dromotropic effect (decreasing of rate of excitation conduction in heart).	4. Positive dromotropic effect (increasing of rate of excitation conduction in heart).

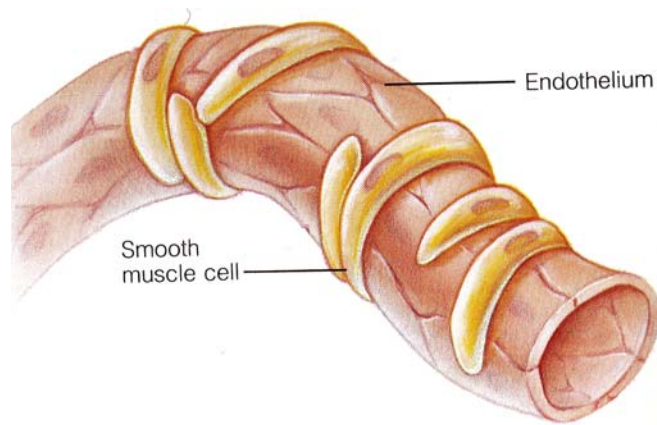


Figure 2.7. — Structure of a small arteriole (by Elaine N. Marieb, 1989)

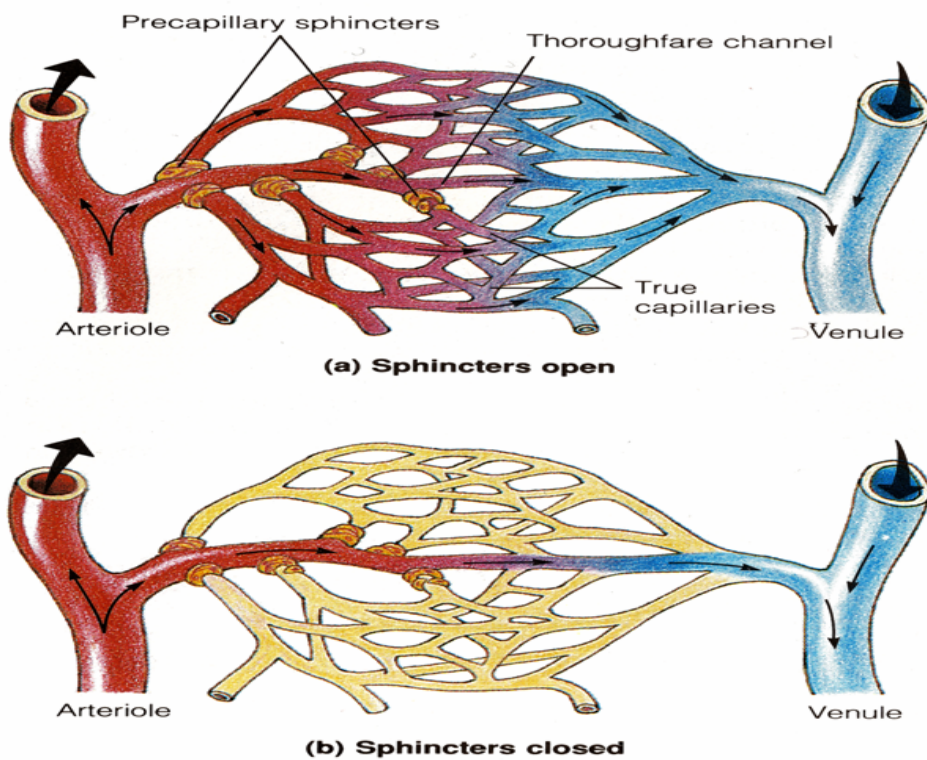


Figure 2.8. — Anatomy of a capillary bed (by Elaine N. Marieb, 1989)

Thoroughfare channels act as shunts to bypass the true capillaries when precapillary sphincters controlling blood entry into the true capillaries are constricted

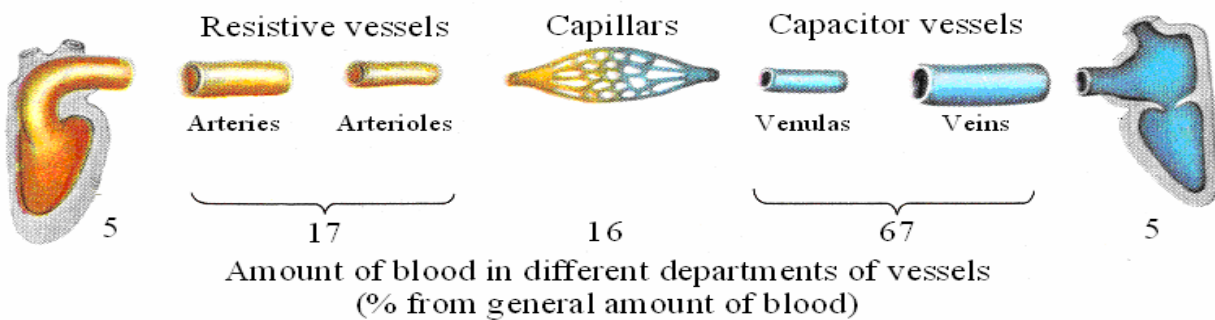


Figure 2.9. — Distribution of blood in different departments of vascular channel (by Korobkov A. V., Chesnokova S. A., 1986)

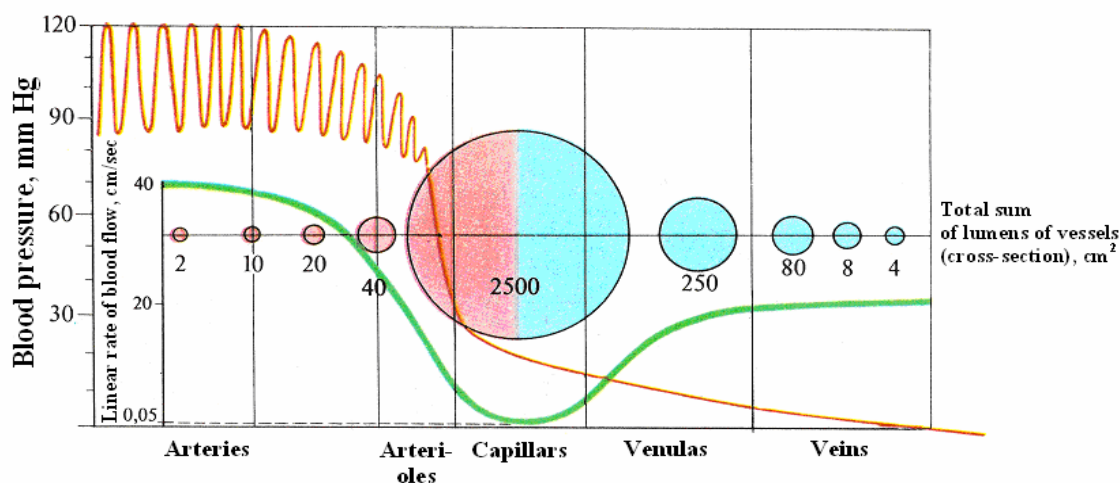


Figure 2.10. — The level of blood pressure, total sum of lumens of vessels and linear rate in different departments of vascular channel (by Korobkov A.V., Chesnokova S.A., 1986)

Factors that lead to increased systemic arterial blood pressure.

1. Pumping force of heart.
2. Peripheral resistance of vessels.
3. Volume of circulating blood.
4. Viscosity of blood.

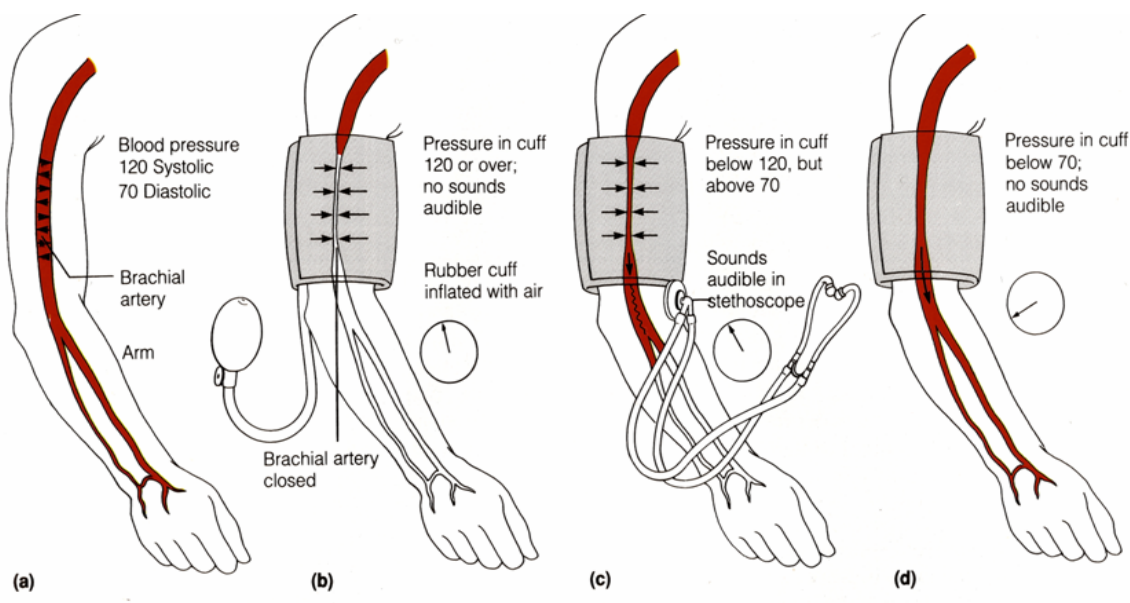
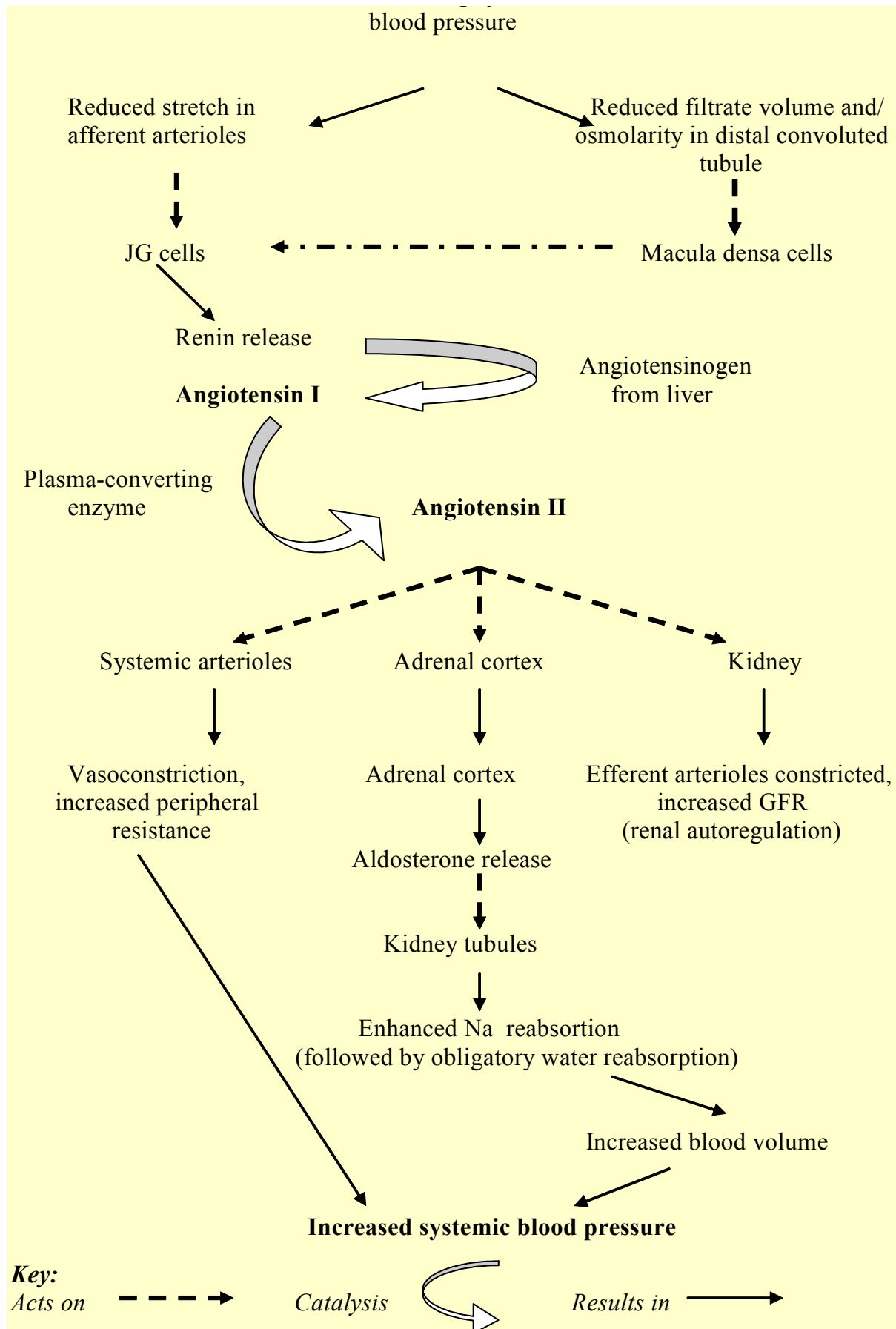


Figure 2.11. — Measurement of blood pressure (by Elaine N. Marieb, 1989)

- a) The course of the brachial artery of the arm. Assume of blood pressure 120/70 in a young healthy individual.
- b) The cuff of the sphygmomanometer is wrapped snugly around the arm just superior of the elbow and inflated until the cuff pressure exceeds the systolic blood pressure. At this point, blood flow into the arm is stopped and brachial pulse cannot be felt or heard.
- c) The pressure of the cuff is gradually reduced while the examiner listens (auscultates) carefully for sounds in the brachial artery with a stethoscope. The pressure read as the first soft tapping sound are heard (the first point at which a small amount of blood is spurting through the constricted artery) is recorded as the systolic pressure.
- d) As the pressure is reduced still further, the sounds become louder and more distinct, but when the artery is no longer constricted and blood flows freely, the sounds can now longer be heard. The pressure at which the sounds disappear is recorded as a diastolic pressure.



Scheme 2.8. — Flowchart illustrating the role in regulating blood pressure under conditions of declining systemic blood pressure (by Elaine N. Marieb, 1989)

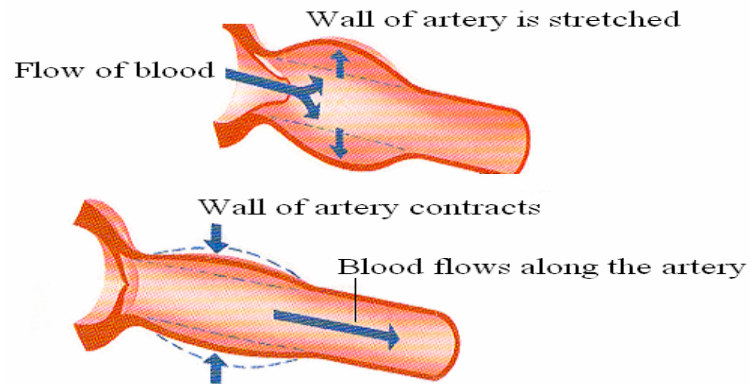
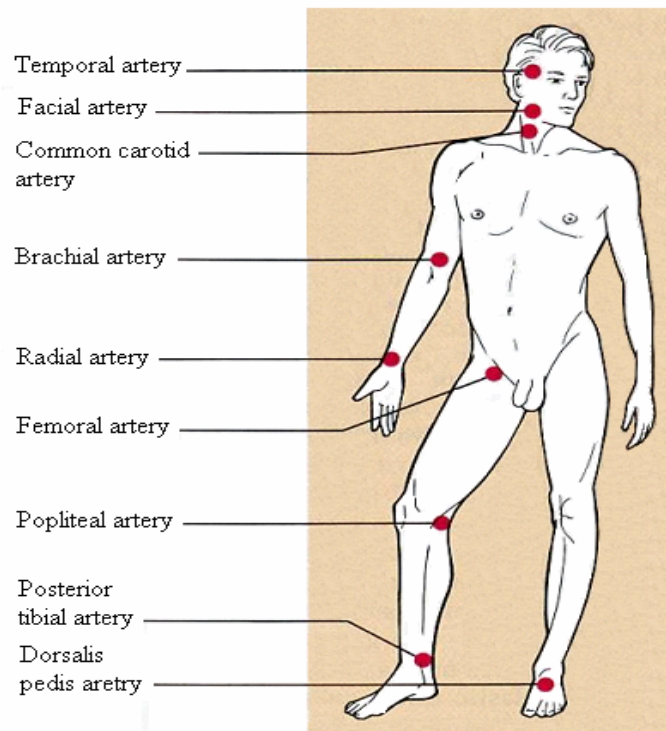


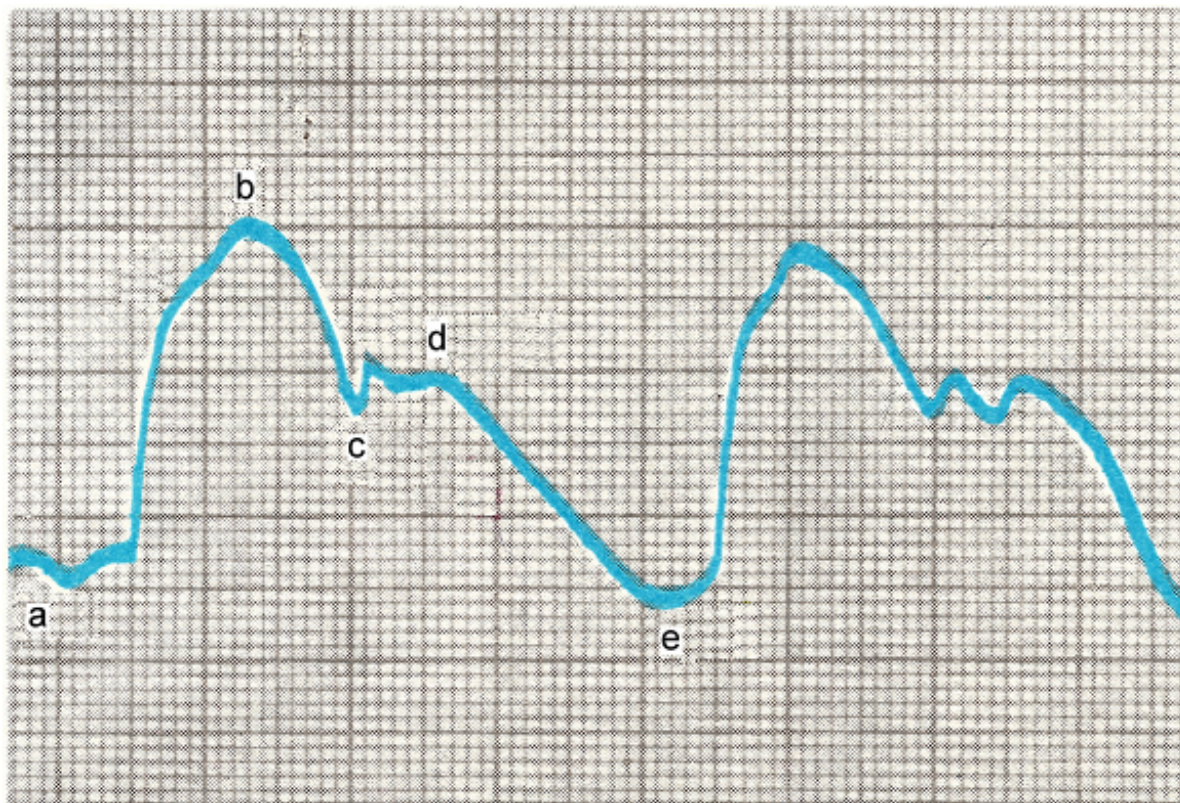
Figure 2.12. — The forming of pulse wave

Table 2.4. — Characteristics of arterial pulse

Characteristics of pulse	Norm	Deviations	Method of definition
Heart rate:	normal (60–80 per minute)	slow, rapid	It is determined by counting of pulse within 1 minute
Rhythm:	rhythmic	arrhythmic	It is determined by comparing of duration of the intervals between the pulse beats or R-R intervals on the ECG
Filling (height)	good	satisfactory, weak, thready pulse	It is determined by the height the arterial wall rising (palpated volume of the artery under the fingers) during systole.
Strain	moderate,	firm, mild pulse	It is determined by effort of squeezing of an artery which should be made for the pulse disappearance.
Rate	normal,	rapid, slow pulse	It is determined by the rate of rising and lowering of the arterial wall.



**Figure 2.13. — Body sites where the pulse is most easily palpated
(by Elaine N. Marieb, 1989)**

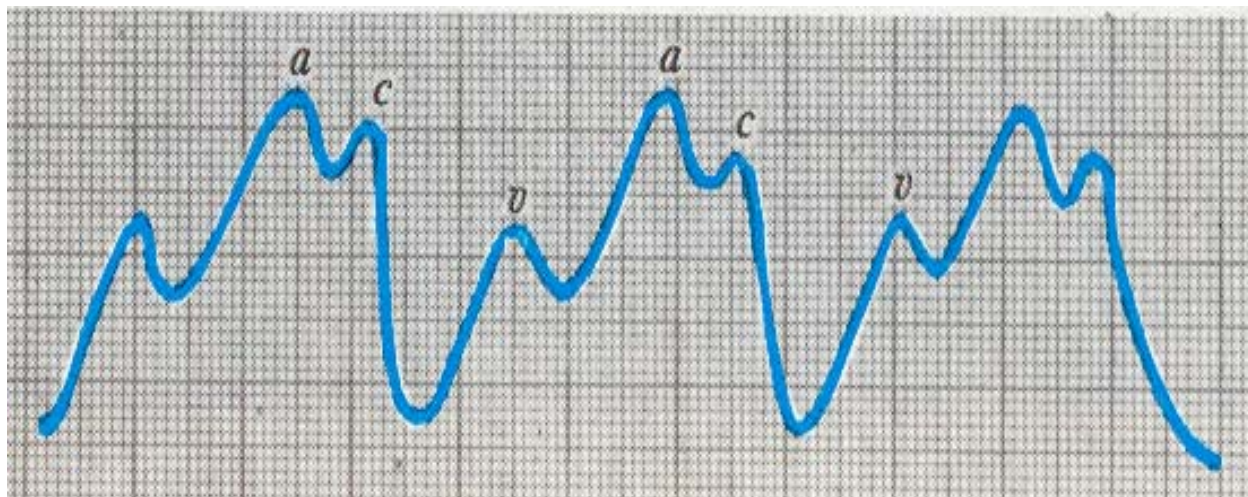


Scheme 2.9. — Sphygmogram and its components
(by Korobkov A. V., Chesnokova S. A., 1986)

a–b — Anacrotism

d–e — Catacrotism

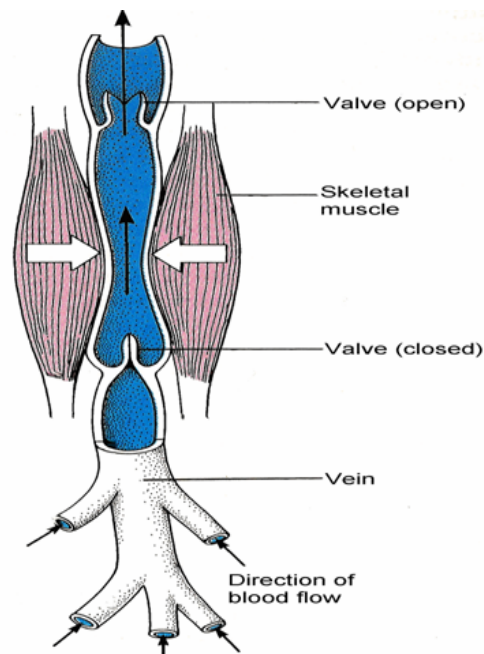
c–d — Dicrotic wave



Scheme 2.10. — Phlebogram and its components
(a-wave, c-wave, v-wave)

Factors, which ensure the blood-flow in veins

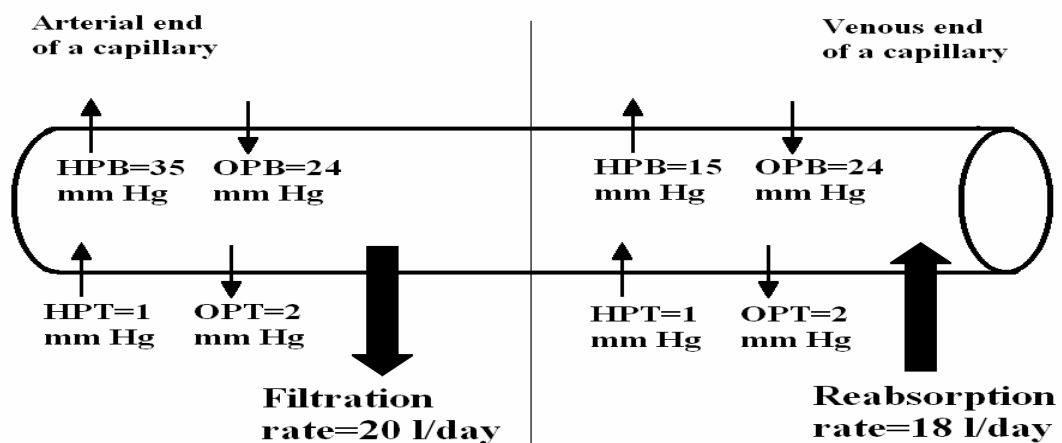
1. Suction action of thorax (decreasing of atmospheric pressure in thorax during inspiration promotes veins distention).
2. «Muscular pump» (squeezing of veins at contraction of skeletal muscles pushes blood to heart; presence of valves on internal surface of some veins prevents inverse blood flow)
3. Suction action of heart (due to moving of atrioventricular septum at systole of ventricle).
4. Peristaltic contraction of walls of some veins (2–3 times a minute).



**Figure 2.14. — Relationship of skeletal muscle activity to venous valve function
(by Elaine N. Marieb, 1989)**

Table 2.5. — Classification and functions of capillars

Type of capillars	Structure of wall of capillars	Features of functioning	Places of localization
Somatic type	Continuous endothelial and basal envelope, big number of the smallest pores (4–5 nm in diameter)	The walls of this type of capillars are permeable for water and mineral substances	Skeletal and unstriped muscles Adipose and connective tissue Lungs Cerebral cortex
Visceral type	The capillars of this type has fenestrations (holes) with diameter — 0.1 μm . Fenestrations are frequently covered with the thinnest membrane.	The walls of this type of capillars are permeable for fluids and macromolecules	Kidney Digestive canal Endocrine glands
Sinusoid type	Basal membrane is absent partially; endothelial envelope is irregular, with big interstitial lumens.	The walls of this type of capillars are permeable for fluids, blood cells, macromolecules	Bone marrow Liver Spleen.



Scheme 2.11. — Forces which determine direction of fluid flows in capillary bed

HPB = hydrostatic pressure of blood in the capillary
 OPB = oncotic pressure of blood in the capillary
 HPT = hydrostatic pressure of tissues (in the interstitial fluid)
 OPT = oncotic pressure of tissues (in the interstitial fluid)
 Small arrows show the direction of action of a pressure.
 Big arrows show the direction of fluid flows.

Notice that filtration rate is 20 l/day and reabsorption rate is 18 l/day, and 2 l of fluids per day gets into lymphatic vessels.

Effective filtration pressure = (HPB + OPT) – (HPT + OPB) = (35 mm Hg + 2 mm Hg) — (1 mm Hg + 24 mm Hg) = 12 mm Hg

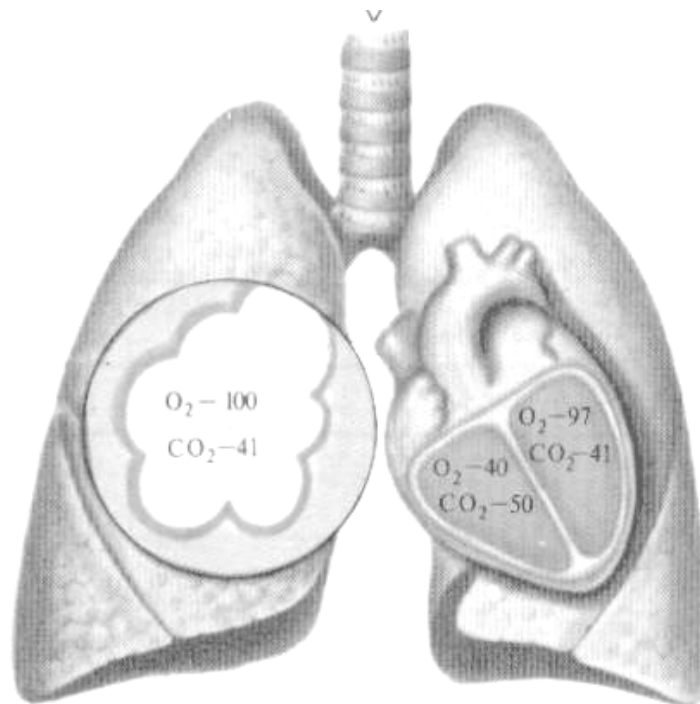
Effective reabsorption pressure = (HPB + OPT) – (HPT + OPB) = (15 mm Hg + 2 mm Hg) — (1 mm Hg + 24 mm Hg) = – 8 mm Hg

Table 2.6. — Regulation of vascular tonus

Nervous mechanism			Humoral mechanism	
Afferent part	Central part	Efferent part	Vasoconstrictors	Vasodilators
<u>Angioreceptors</u> (situated in the walls of vessels): 1) baroreceptors (they respond to the distention of walls of vessels); 2) chemoreceptors (they are sensitive to O ₂ , CO ₂ , H ⁺ presence in blood).	<u>Vasomotor center</u> (it includes various levels of CNS): 1) thoracic and lumbal segments of spinal chord (vasoconstrictor centers); 2) the vasomotor center of medulla (this is the main center of regulation of vascular tonus and arterial pressure); 3) the pressor and depressor zones in hypothalamus; 4) the cortex of cerebrum (its participation in regulation of vascular tonus is proved by method of conditioned reflexes).	1) <u>Sympathetic nerve fibers</u> produce vasoconstriction (but dilate vessels of heart and skeletal muscles). 2) <u>Parasympathetic nerves</u> produce vasodilatation (but constrict the vessels in heart).	1) <u>The hormones suprarenal glands</u> : • Adrenalin (it is mainly vasoconstrictor, but it dilates vessels of skeletal muscles, unstriated muscles of bronchus); • Noradrenalin (it produces vasoconstriction); • Aldosterone (it changes sensitivity of walls of vessels to action of adrenalin and noradrenalin). 2) <u>Vasopressin</u> (the hormone of neurohypophysis; it is mainly vasoconstrictor, but it dilates cerebral and cardiac vessels) 3) <u>Renin</u> (it is produced by uxtaglomerular apparatus of kidneys; it helps in production of Angiotensin I, which then turns into Angiotensin II — vasoconstrictor).	Biologically-active substances and local hormones: • Histamin • Bradykinin

Unite 3

PHYSIOLOGY OF RESPIRATORY SYSTEM



Respiration — set of the processes providing receipt of O_2 by the organism, its delivery and consumption by tissues and excretion of the respiration end-product CO_2 into environment.

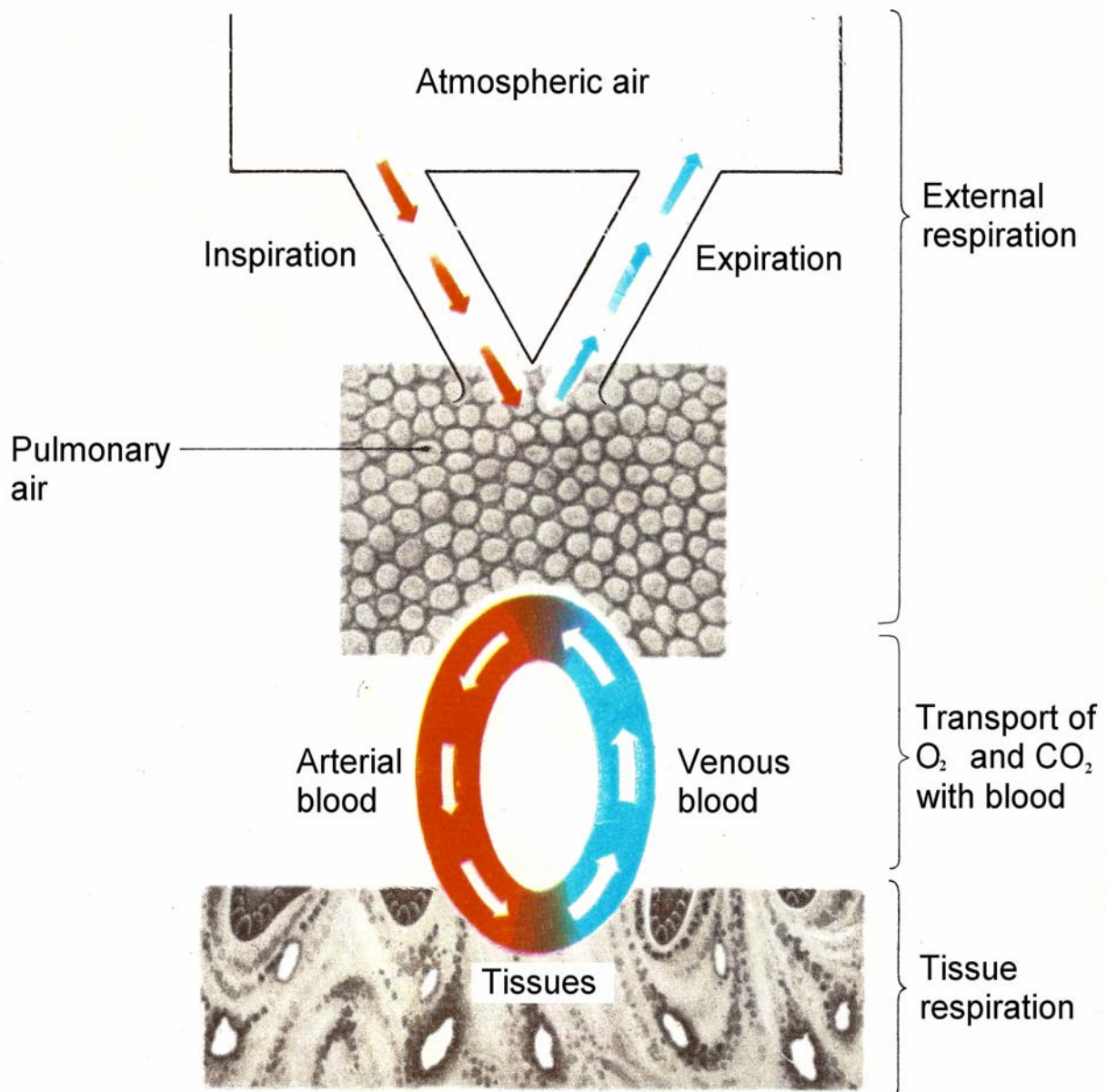


Figure 3.1 — Process of gas exchange between the environment and the organism
(by Korobkov A. V., Chesnokova S. A., 1986)

Adaptive features of lungs which help to perform the function of gas exchange

1. The presence of air and blood channels. They are separated by the thinnest (0,004 mm) membrane through which diffusion of gases occurs.
2. The respiratory area of lungs is 50–90 m².
3. The presence of special – small – circle of blood circulation.
4. The presence of elastic tissue in lungs which ensure expanding and falling of lungs at inspiration and expiration.
5. The presence of supporting cartilage tissues (as cartilage of bronchi) in respiratory ways (cartilage tissues prevent falling of respiratory ways and promote fast and easy passage of air).

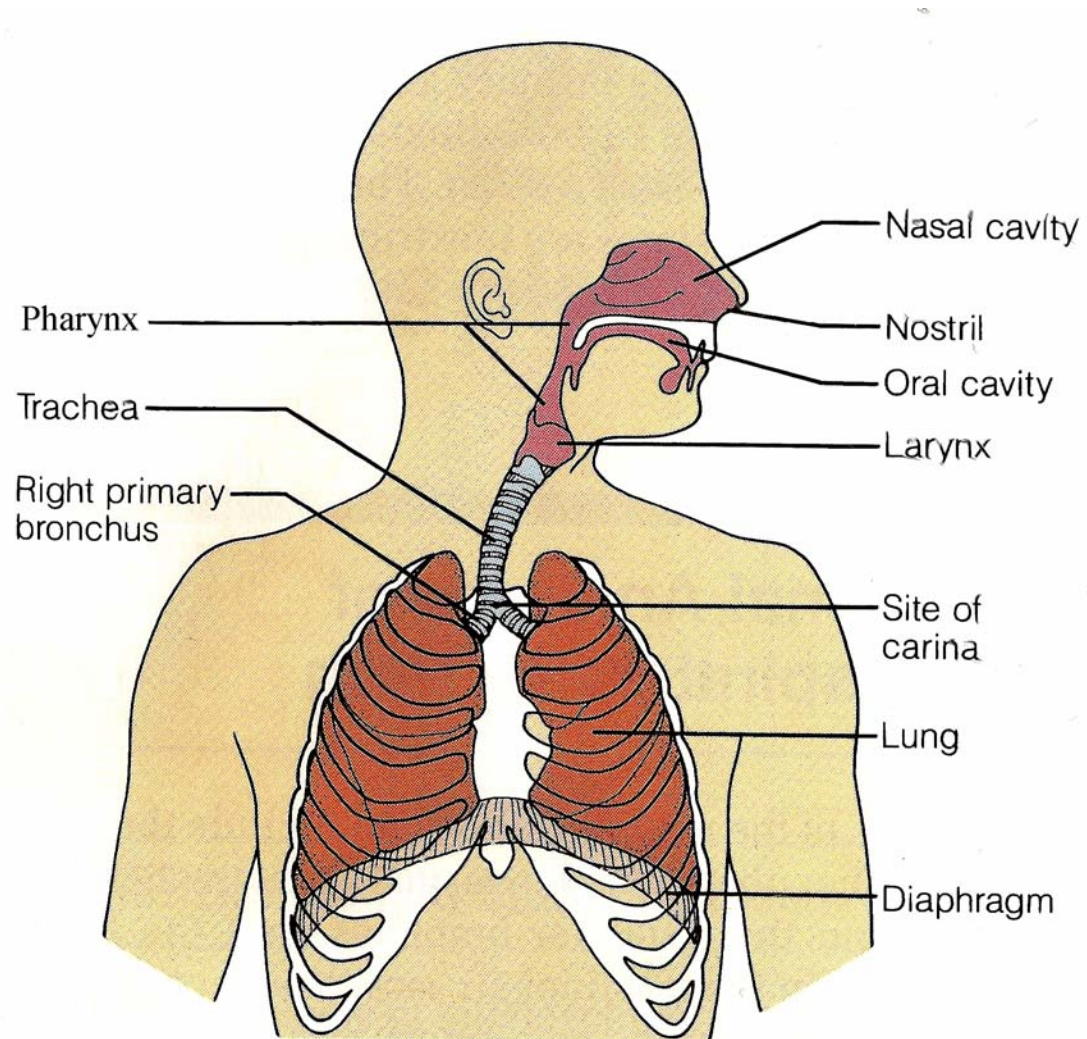


Figure 3.2. — Organs of the respiratory system (by Elaine N. Marieb, 1989)

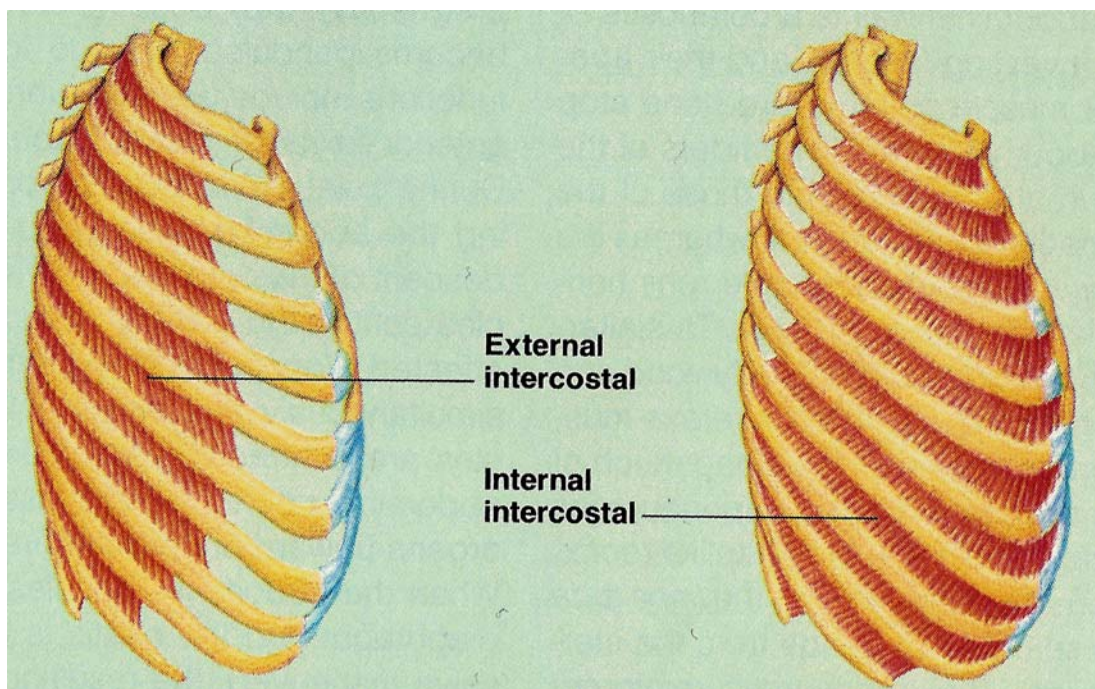


Figure 3.3. — Intercostal muscles (by Elaine N. Marieb, 1989)

Table 3.1. — Respiratory movements

Phase of respiratory cycle	The mechanism of changing of thorax volume	Describing of the mechanism
Inspiration	Raising of ribs	Contraction of main inspiration muscles: <ul style="list-style-type: none"> • external intercostal muscles; • internal intercartilaginous muscles. Contraction of auxiliary inspiration muscles (at forced respiration): <ul style="list-style-type: none"> • greater and smaller pectoral muscles; • scalene muscle; • sternocleidomastoid muscle; trapezius muscle, etc.
	Movement of diaphragm	At inspiration diaphragm flattens (moves down) as result of contraction of its muscular fibers.
Expiration	Decreasing of thorax volume due to factors which are not connected with contraction of muscles	Decreasing of thorax volume due to: <ul style="list-style-type: none"> • weight of thorax; • elasticity of costal cartilages; • elasticity of lungs; • the pressure of organs of abdominal cavity on the diaphragm.
	Movement of diaphragm	Diaphragm relaxes and adjoins the internal wall of thorax.
	At forced respiration decreasing of thorax volume due to contraction of some muscles	Contraction of main expiration muscles: <ul style="list-style-type: none"> • internal intercostal muscles. Contraction of auxiliary expiration muscles: <ul style="list-style-type: none"> • backbone flexors; • abdominal muscles.

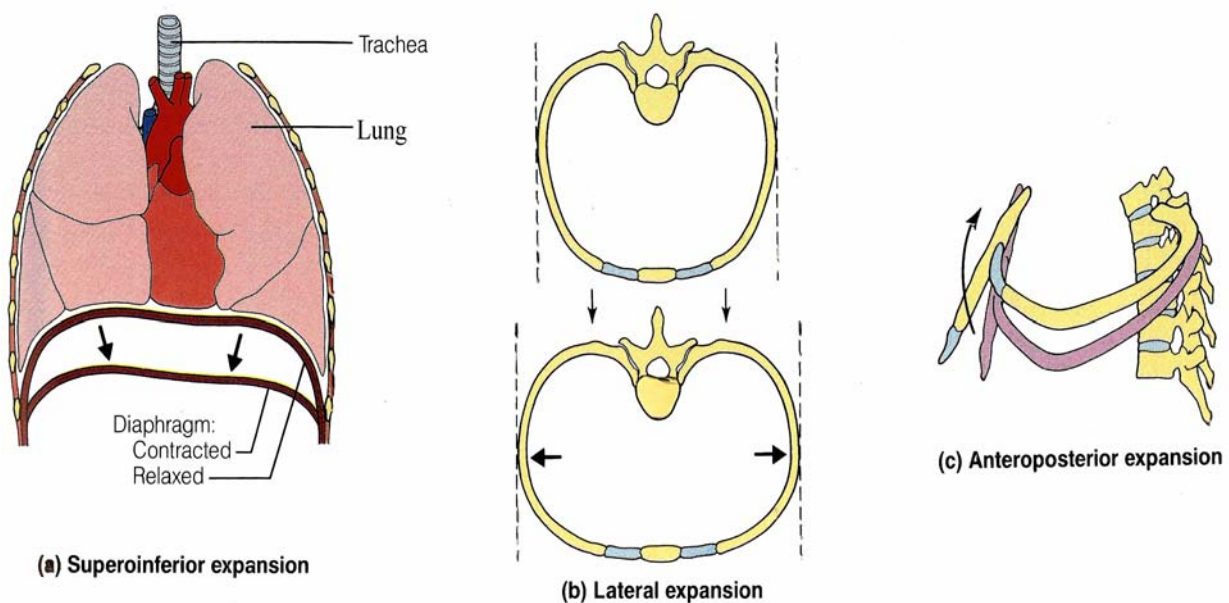


Figure 3.4. — Changes in thoracic volume during breathing
(by Elaine N. Marieb, 1989)

(a-c) Ways in which the volume of the thorax is increased during inspiration. The diaphragm descends as it contracts, increasing the superiorinferior dimension (a). Due to the contraction of the external intercostal muscles the ribs are elevated, the thorax expands laterally (b) and in the anterioposterior plane (c).

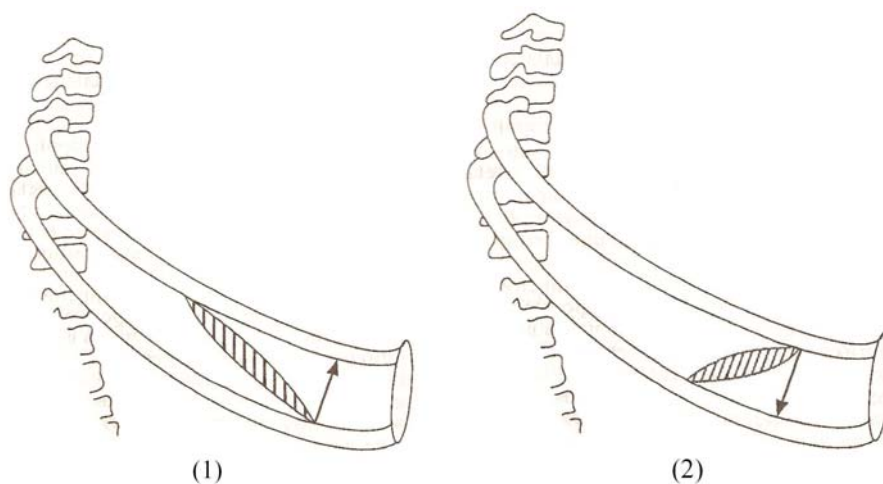
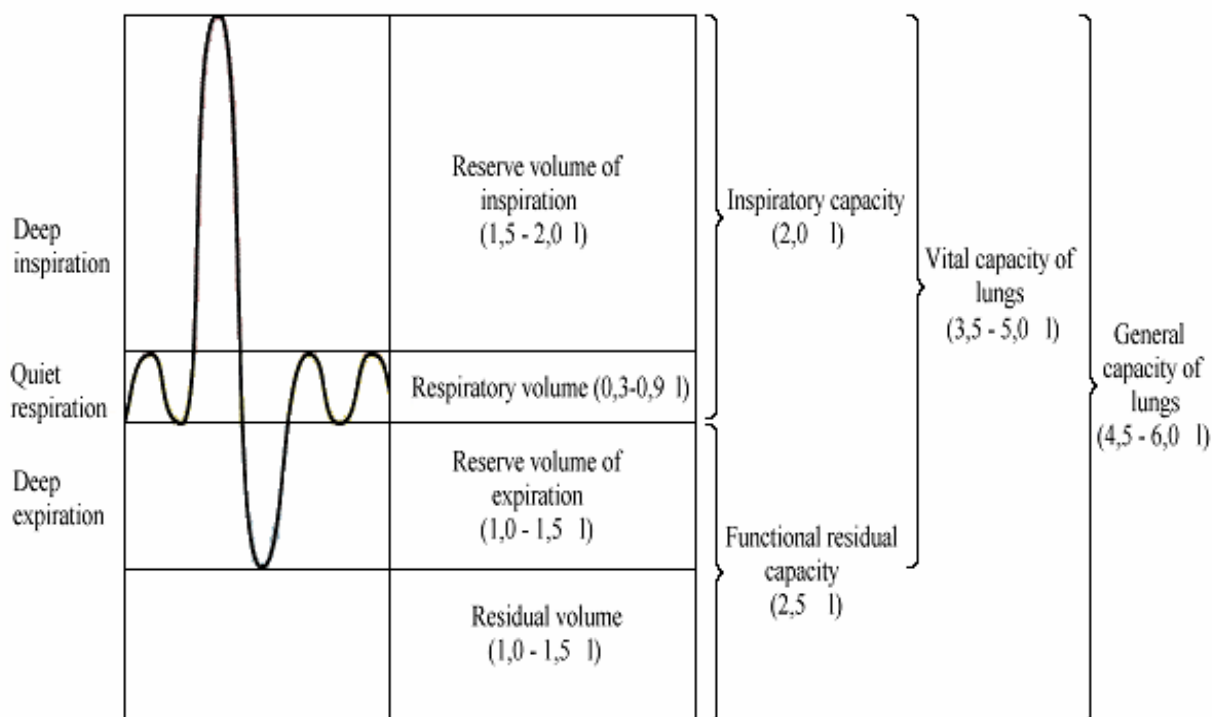


Figure 3.5 — Mechanism of rising or lowering the ribs due to contraction of intercostal muscles

- (1) — at contraction of external intercostal muscles ribs rise;
 (2) — at contraction of internal intercostal muscles ribs lower;



Scheme 3.1. — Spirographic record. Pulmonary volumes and capacities

Table 3.2. — There are 4 respiratory volumes and 4 capacities of lungs

Volumes	Capacities
Respiratory volume	Inspiratory capacity
Reserve volume of inspiration	Vital capacity
Reserve volume of expiration	General capacity of lung
Residual volume	Functional residual capacity

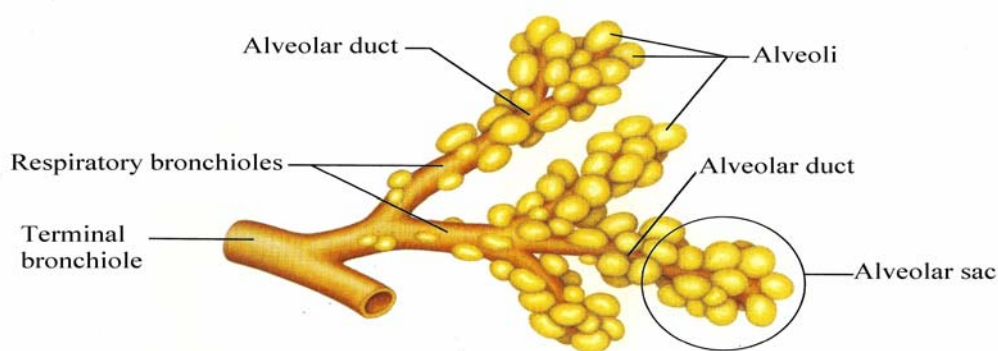


Figure 3.6. — Diagrammatic view of the respiratory structures

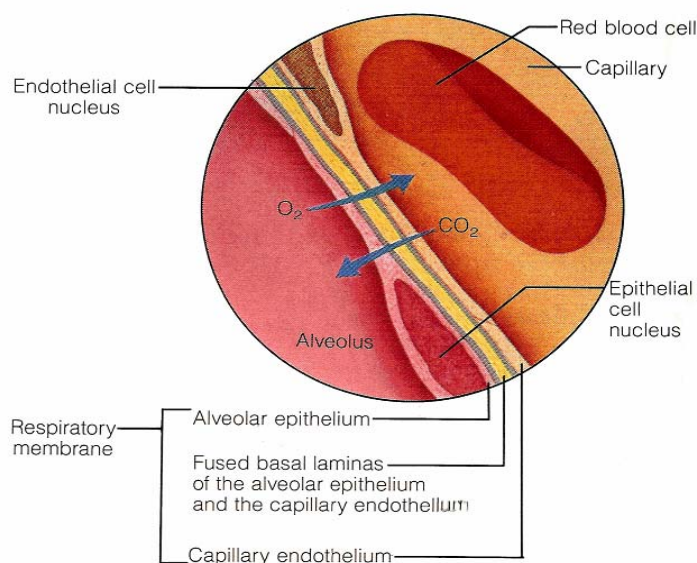


Figure 3.7. — Anatomy of the respiratory membrane (by Elaine N. Marieb, 1989)

Table 3.3. — Structure of air (in %)

Air	O_2	CO_2
Inhaled	21.0	0.02—0.03
Exhaled	16.0	4.5
Alveolar	14.0	5.5

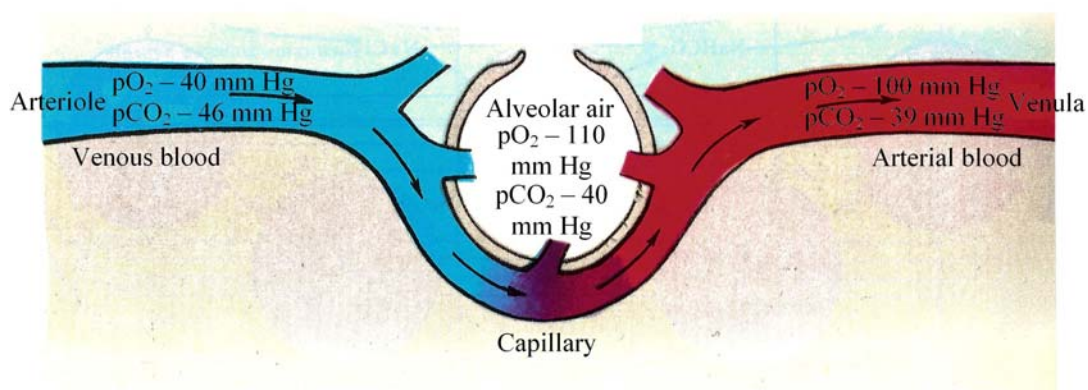


Figure 3.8. — Gas exchange between the alveolus and capillary (by Korobkov A. V., Chesnokova S. A., 1986)

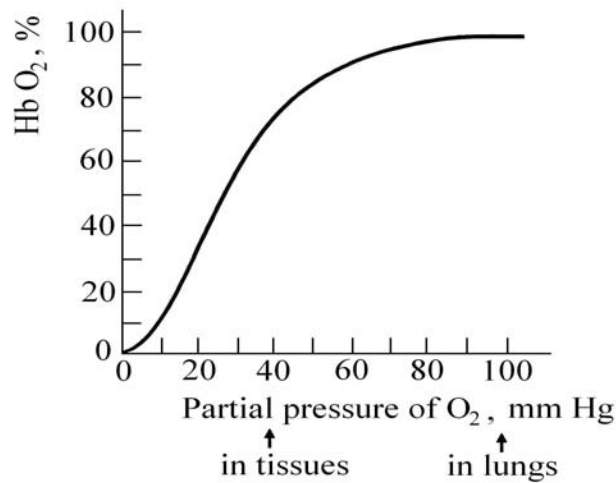


Figure 3.9. — Oxyhemoglobin dissociation curve
Percent of HbO₂ is shown at different oxygen partial pressures

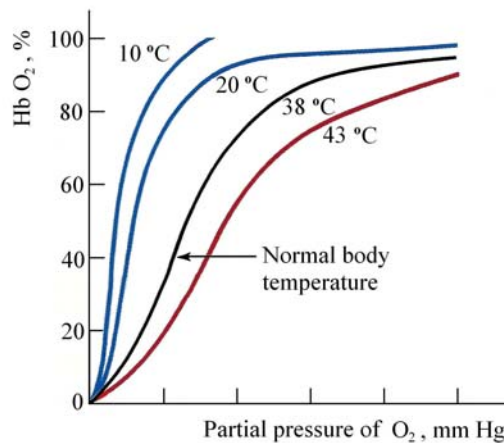


Figure 3.10. — Effects of temperature on the hemoglobin dissociation curve
Oxygen unloading is accelerated at conditions of increased temperature, resulting in a shift to the right of the dissociation curve

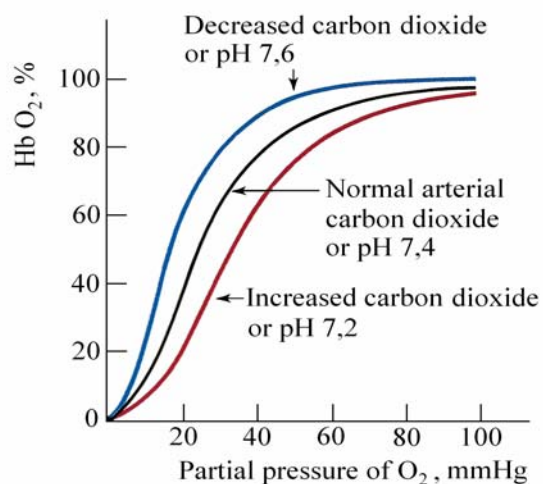
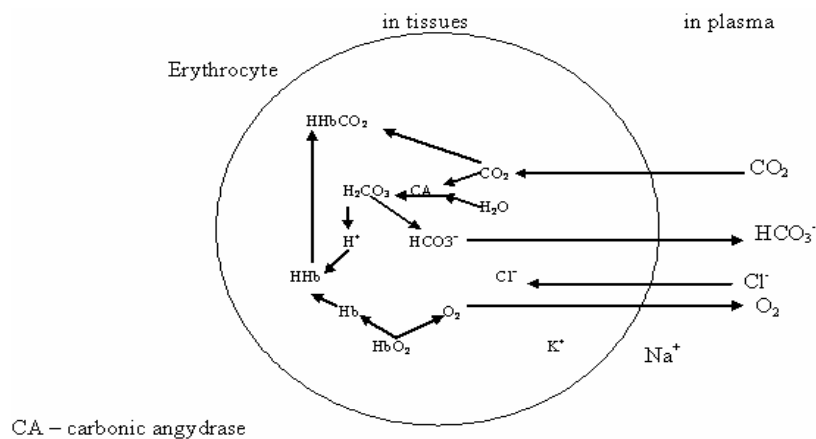
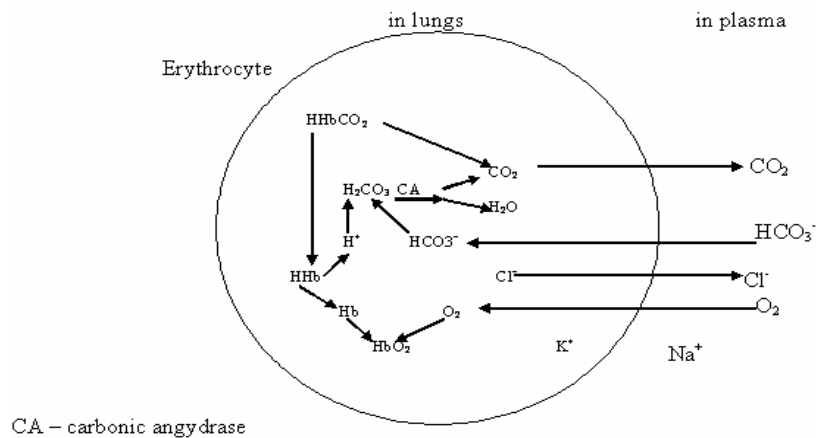


Figure 3.11. — Effects of partial pressure of CO₂ and blood pH on the hemoglobin dissociation curve
Oxygen unloading is accelerated at conditions of increased partial pressure of CO₂ and decreased pH, resulting in a shift to the right of the dissociation curve.



CA – carbonic anhydrase



CA – carbonic anhydrase

Scheme 3.2. — Transport of CO_2

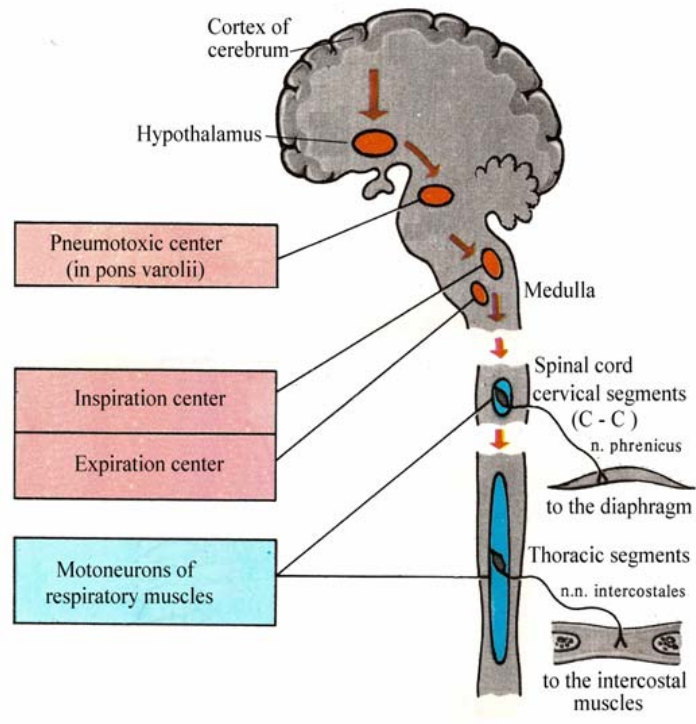


Figure 3.12. — Respiratory center (its components) and efferent nerves
(by Korobkov A. V., Chesnokova S. A., 1986)

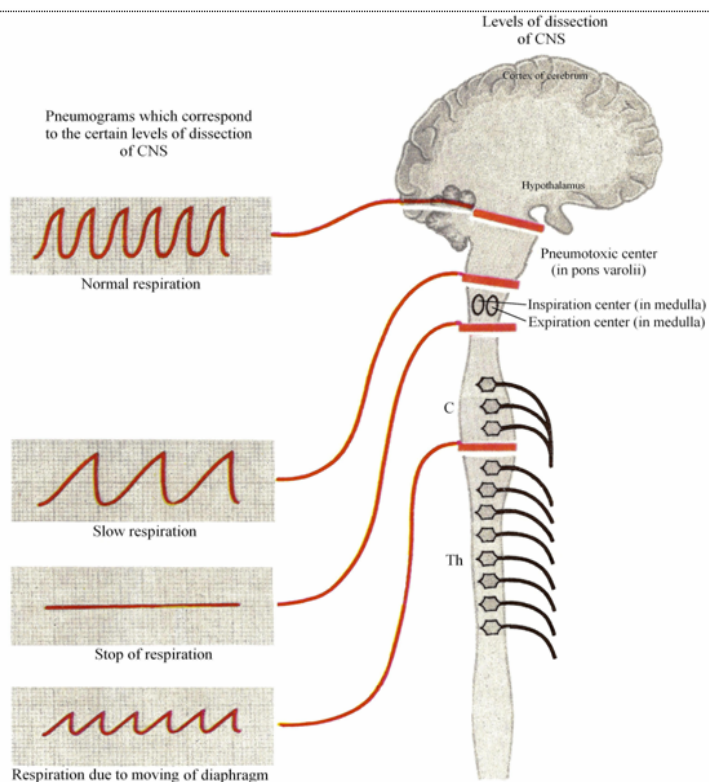


Figure 3.13. — Influence of dissection of different levels of central nervous system on respiration (by Korobkov A. V., Chesnokova S. A., 1986)

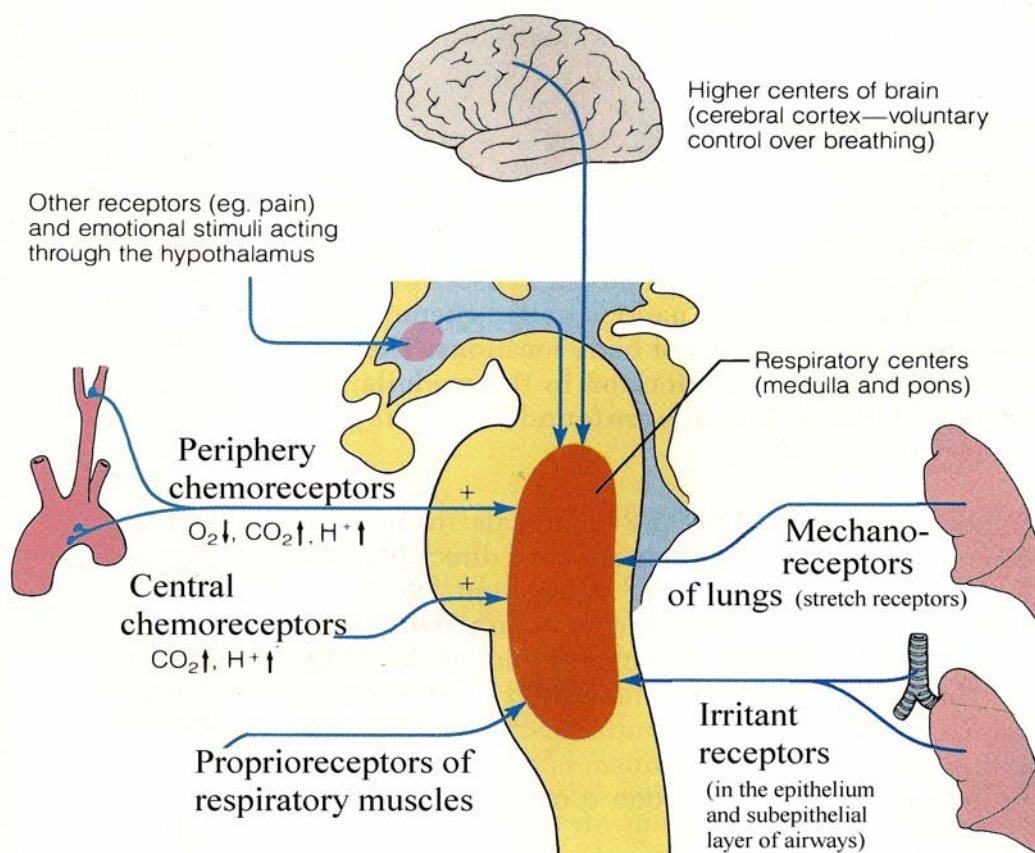


Figure 3.14. — Neural and chemical influences on the respiratory centers in medulla (by Elaine N. Marieb, 1989)

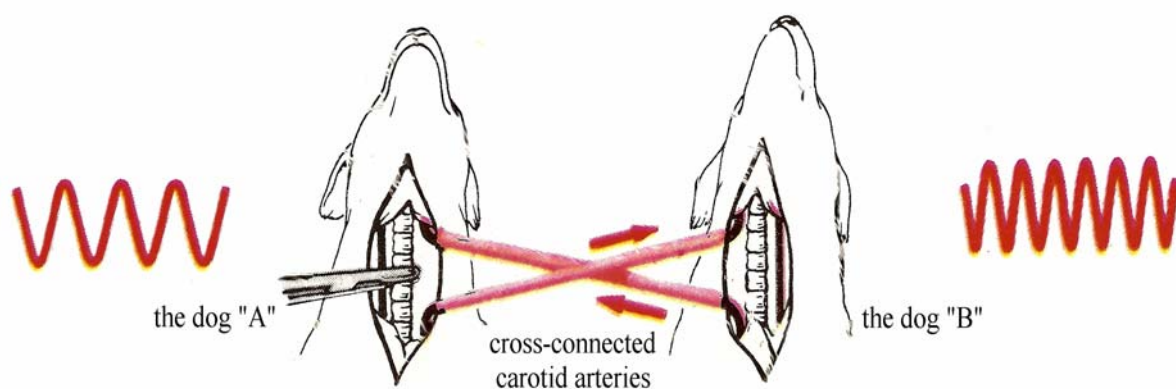


Figure 3.15. — The Frederico test with cross-circulation
(by Korobkov A. V., Chesnokova S. A., 1986)

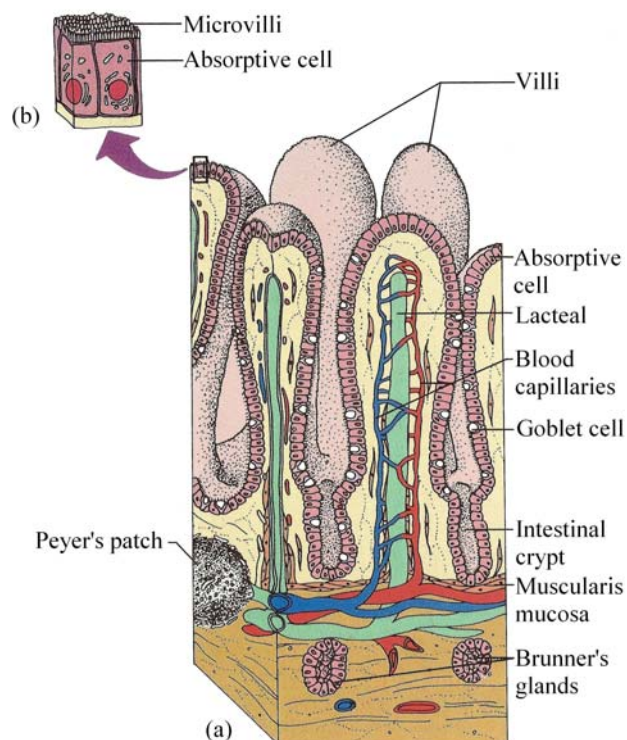
Pinching of the trachea of the dog «A» causes hyperpnoea in the dog «B»;
hyperpnoea of the dog «B» causes decreasing of ventilation rate in the dog «A».

Table 3.4. — Critical zones of hypoxia (at respiration at the lowered atmospheric pressure)

Name of a zone	Altitude	Changes in the functioning of the organism
Neutral zone	up to 2000 m	Physiological functions practically do not suffer
Zone of complete compensation	2000–4000 m	<ul style="list-style-type: none"> • Ventilation of lungs increases (due to stimulation of carotid and aortal chemoreceptors). • Heart rate increases. • Systolic and minute volume of blood increase. • Blood pressure increases. • Physical and mental work capacity is reduced a bit.
Zone of incomplete compensation (zone of danger)	4000–7000 m	<ul style="list-style-type: none"> • Signs of mountain disease are developed: apathy or euphoria, short-breathing, tachycardia, giddiness, vomiting, headache. • Muscular twitching appears. • Blood pressure decreases. • Work capacity is reduced, ability to decision-making and reactions is affected. • Consciousness is fogged.
Critical zone	>7000 m	<ul style="list-style-type: none"> • pO_2 in alveolar air becomes lower than critical threshold (30–35 mm Hg) • Cramps, loss of consciousness, breathlessness and disturbance blood circulation dangerous for life can happen. If such state lasts for a long period of time, affection of central nervous system and death happen.

Unite 4

PHYSIOLOGY OF DIGESTIVE SYSTEM



Digestion is the physical and chemical processing of food.

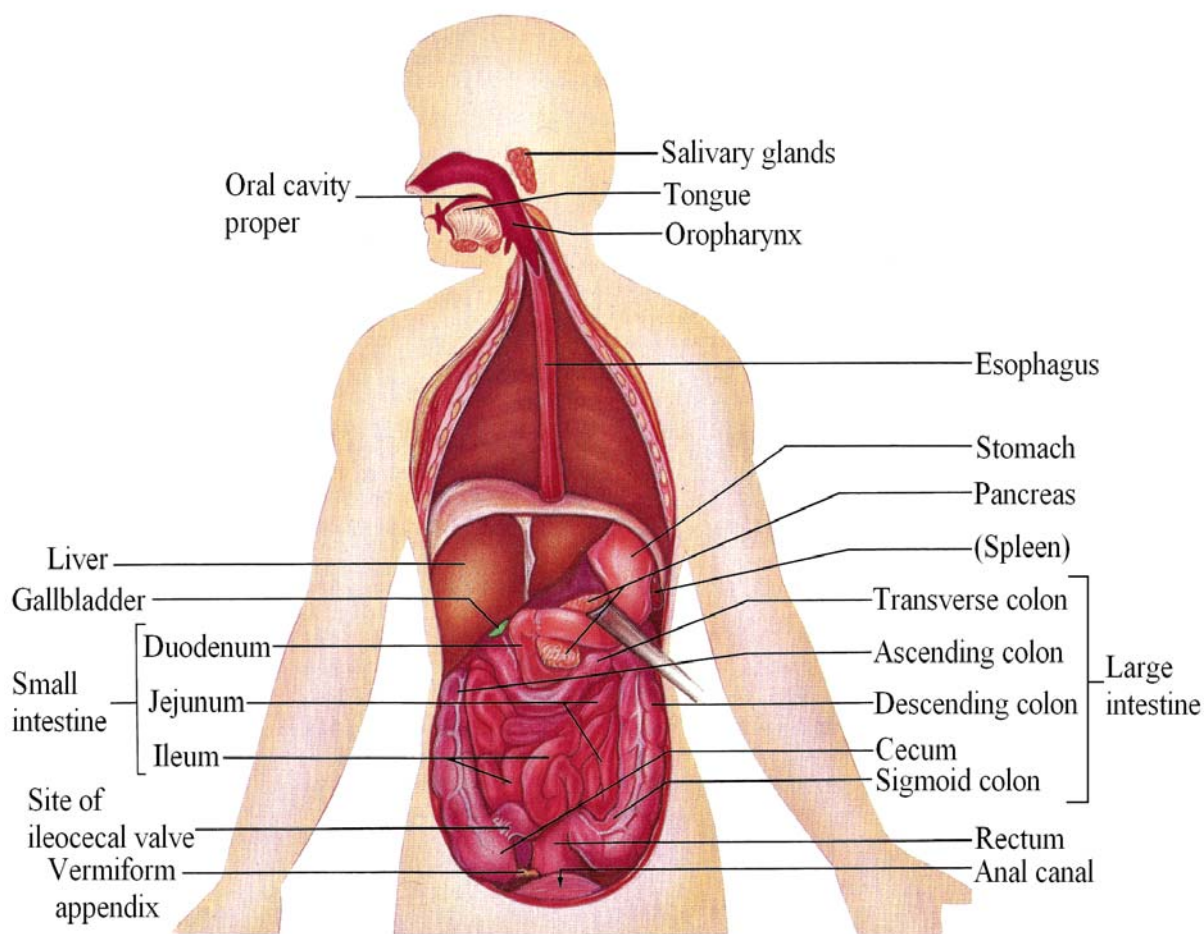


Figure 4.1. — Organs of the alimentary canal and related accessory digestive organs (by Elaine N. Marieb, 1989)

Table 4.1. — Theories of hunger and satiation

Name of a theory	The factor which causes the appearance of hunger or satiation
Local theory of hunger	Hunger appears due to signals from the stomach which is not filled with food (in such empty stomach periodical contractions of its walls happen).
Glucostatic theory	Hunger appears due to the decreasing of glucose concentration in blood.
Aminoacidostatic theory	Hunger appears due to the decreasing of the amino acids concentration in blood.
Lipostatic theory	Hunger appears due to mobilization of fats from fat depot.
Liponeurostatic theory	Hunger appears due to stimulation of food center by the signals from fat depot during the mobilization of fat.
Thermostatic theory	Satiation appears due to suppression of food center because of rising of blood temperature during eating.
Hydrostatic theory	Decrease of water resources of the organism reduces consumption of food.
Metabolic theory	This theory unites all mentioned theories. The amount of intermediary products of Krebs's cycle in blood influence on the food center.

Kinds of satiation

1) Sensory (initial) satiation — it appears before the absorption of digestion products (nutrients) into blood and has the complex reflex nature.

2) Metabolic (secondary) satiation — it appears due to absorption of digestion products (nutrients) into blood.

Table 4.2. — Structure of saliva (pH 7.4–8.0)

Organic components			Inorganic components
Digestive enzymes		Other organic substances	Na ⁺ K ⁺ Ca ²⁺ Mg ²⁺ Chlorides, Carbonates Phosphates (and others)
Name of the enzyme	Role of the enzyme	Mucin Lysozyme Callecrein Proteins Amino acids Creatinine	
<u>Alpha-amylase</u>	It splits carbohydrates (polysaccharides — starches, glycogen) with formation of dextrines, disaccharides (maltose) and partially glucose)		
<u>Proteinases (cathepsines, salivain, glandulin),lipase, alkaline and acidic phosphatases</u>	The activity of this enzymes in saliva is insignificant		

Saliva also contains water

Functions of saliva:

1. Moistens and dilutes food;
2. Promotes gustatory approbation of food;
3. Enzymes of saliva provide hydrolysis of carbohydrates;
4. Protects mucous membrane;
5. Due to mucin food lump is formed;
6. The lysozyme carries out bacteriostatic action (factor of nonspecific protection);
7. The saliva partially neutralizes acidic products getting into oral cavity;
8. Contact of proteins with saliva provides their best digestion;
9. The saliva contains biologically active substances — callicrein, parotin.

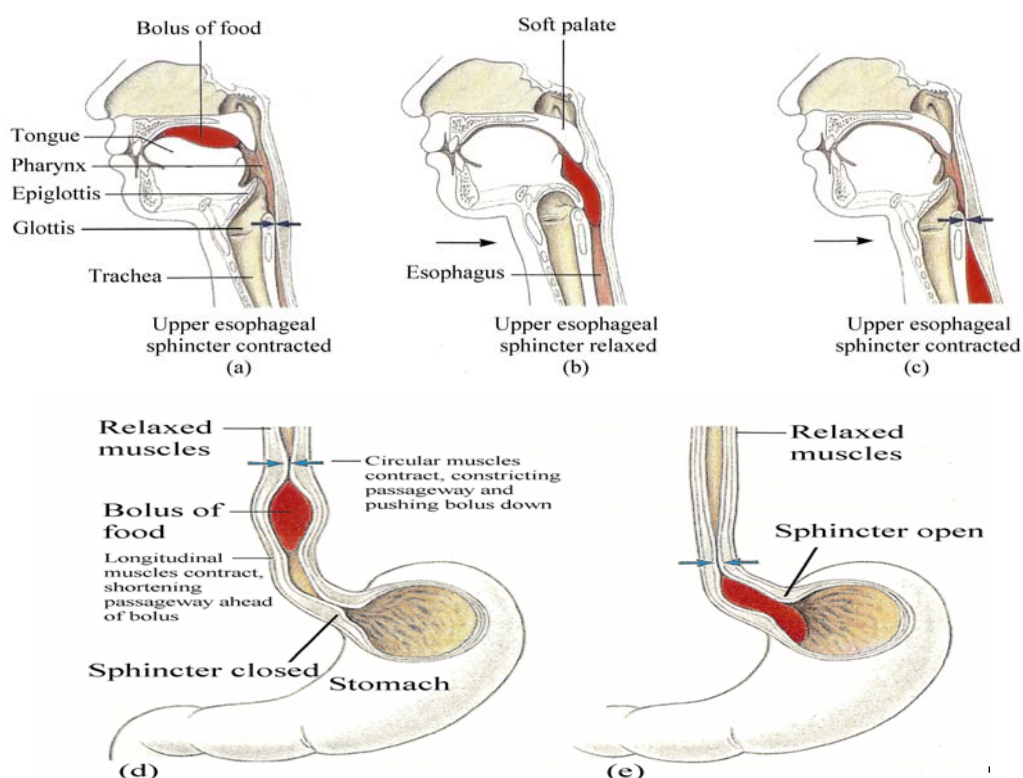


Figure 4.2. — Deglutition (swallowing) (by Elaine N. Marieb, 1989)

The process of swallowing consists of voluntary (buccal) and involuntary (pharyngeal- esophageal) phases. (a) During the buccal phase, the tongue rises and presses against the hard palate; in so doing, it forces the food bolus into the oropharynx. Once food enters the pharynx, the involuntary phase of swallowing begins. (b) Food passage into respiratory passageways is prevented by the rising of the uvula and larynx and relaxation of the upper esophageal sphincter to allow food entry the esophagus. (c) The constrictor muscles of the pharynx contract, forcing food into the esophagus inferiorly, and the upper esophageal sphincter contracts once food entry has occurred. (d) Food is conducted along the length of the esophagus to the stomach by peristaltic waves. (e) The gastroesophageal sphincter opens, and food enters the stomach.

Table 4.3. — Functions of stomach

Not digestive functions	Digestive functions
1. Participation in regulation of erythropoiesis (due to Castle's intrinsic factor). 2. Participation in metabolism. 3. Excretory function. 4. Endocrine function (due to presence of endocrine cells which form hormones and hormone-like products of digestive system). 5. Protective function (due to antibacterial function of HCl).	1. Deposition of food. 2. Mixing of food. 3. Chemical (enzymatic) processing of food. 4. Portion evacuation of partially digested food into duodenum. 5. Adsorption of some products of digestion.

Table 4.4. — Structure of gastric juice (pH 1.5–1.8)

Organic components		Inorganic components
Digestive enzymes		Hydrochloric acid Na^+ K^+ Ca^{2+} Mg^{2+} Chlorides Sulphates, Bicarbonates (and others)
Name of the enzyme	Role of the enzyme	
<u>Pepsin and gastricsin</u> (they are secreted in inactive form — pepsinogens — and act in the presence of HCl)	They split proteins with the formation of large polypeptides	
<u>Chymosin</u>	It coagulates proteins of milk	
<u>Gelatinase</u>	It splits gelatin	
<u>Lipase</u>	It splits fats (especially emulsified fat of milk at breast feeding of a child), The activity of this enzyme in gastric juice is insignificant.	

Gastric juice also contains water

Functions of HCl:

1. Activation of pepsinogen into pepsin.
2. Denaturation of proteins (eases the hydrolysis of proteins)
3. Antibacterial function.
4. Decalcification of bones.
5. Regulation of gastric motor activity.
6. Stimulation of formation of hormones (HCl comes into duodenum, and stimulates the production of such duodenal hormones as secretin and pancreaticozymine).
7. Regulation of evacuation of chymus (performing of obturator pyloric reflex: when the chymus from stomach comes into duodenum, pH in duodenum becomes more acid due to HCl of gastric juice, pyloric sphincter contracts and stops evacuation of chymus from the stomach; after the neutralizing of acid by the hydrocarbonates of duodenal juice pH in duodenum rises and pyloric sphincter opens).

Table 4.5. — Neural and hormonal mechanisms that regulate the release of gastric juice

Phase of stomachal secretion	Mechanism of stimulation of secretion	Example of an experiment which proves the presence of the phase	Physiological role of the phase	Reflex arch
«cerebral» phase	conditioned reflexes	View and smell of food leads to the secretion of gastric juice, which can be received in dogs with fistula of stomach	Stomach is prepared to reception of food	1) visual, auditory, olfactory receptors; 2) cortex of cerebrum; 3) hypothalamus; 4) nuclei of vagus nerve in medulla; 5) stomach glands
	unconditioned reflexes	The so-called «imaginary» feeding of esophagotomy dogs with fistula of stomach (in such dog food does not get into the stomach, but, in 5–10 min after beginning of feeding secretion of gastric juice begins)	Stomach is prepared to reception of food	1) receptors of oral cavity, esophagus; 2) nuclei of vagus nerve in medulla; 3) stomach glands
stomachal phase	unconditioned reflexes	Introduction of food or some solutions into stomach through fistula, or irritation of mechanoreceptors of stomach produce secretion of gastric juice.	Correction of the amount and structure of gastric juice according to the properties taken food	1) chemoreceptors and mechanoreceptors of mucous membrane of the stomach; 2) nuclei of vagus nerve in medulla; 3) stomach glands. 4) Regulation of secretion of stomach glands is made by nerve and humoral (gastrin) mechanisms
intestinal phase	unconditioned reflexes	Introduction of some kinds of food (meat bouillon, cabbage juice, hydrolysates of proteins) into small intestine produces secretion of gastric juice.	Correction of the amount and structure of gastric juice according to the properties of partially digested food which came into the intestine	1) chemoreceptors and mechanoreceptors of mucous membrane of intestine; 2) nuclei of vagus nerve in medulla; 3) stomach glands. Regulation of secretion of stomach glands is made by nerve and humoral (secretin and cholecystokinin — pancreaticozymmin) mechanisms

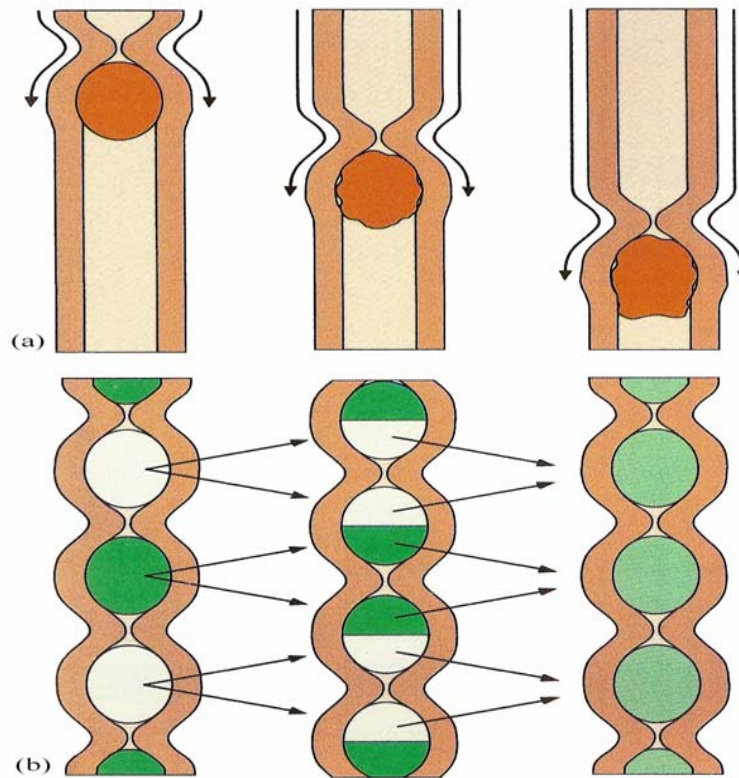


Figure 4.3. — Peristalsis and segmentation (by Elaine N. Marieb, 1989)

(a) In peristalsis adjacent segments of the intestine (or other alimentary tract organs) alternately contract and relax, which results in the movement of food along the tract distally.

(b) In segmentation, nonadjacent segments of the intestine alternately contract and relax. Because the active segments are separated by inactive regions, the food is moved forward and then backward; this results in mixing of the food rather than food propulsion.

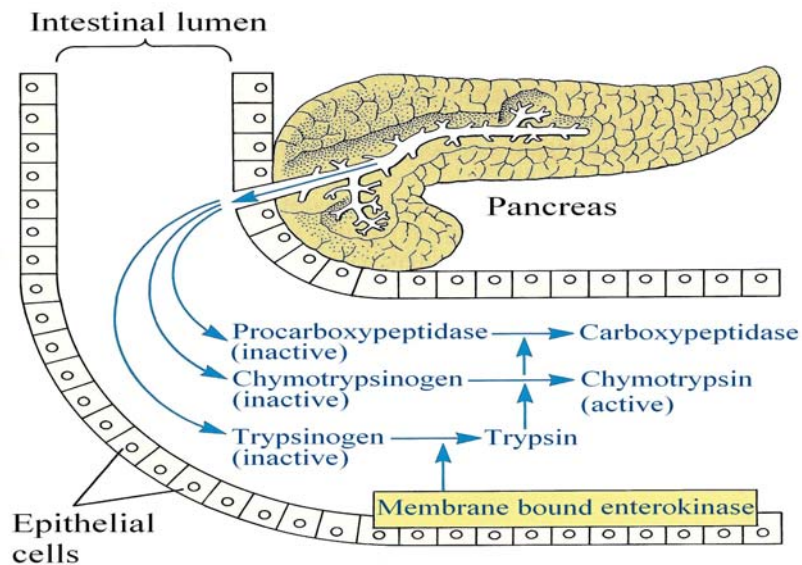


Figure 4.4. — Activation of pancreatic proteases in the small intestine (by Elaine N. Marieb, 1989)

Pancreatic proteases are secreted in an inactive form and are activated in duodenum. Enterokinase, membrane — bound (brush border) intestinal enzyme activates trypsinogen to the active trypsin form. Trypsin, itself a proteolytic enzyme, then activates procarboxypeptidase and chymotrypsinogen.

Table 4.6. — Structure of pancreatic juice (pH 7.8–8.4)

Organic components		Inorganic components
Digestive enzymes		Bicarbonates (cause the alkalinity of juice) Chlorides Na ⁺ K ⁺
Name of enzyme	Role of enzyme	
<u>Trypsin</u> (synthesized in an inactive form — trypsinogen, which turns into trypsin in the duodenum under action of its enzyme enterokinase; Ca ²⁺ accelerate the process)	They split proteins and large polypeptides with the formation of small peptides and aminoacids	
<u>Chymotrypsin</u> (synthesized in inactive form — chymotrypsinogen, which is activated by trypsin)		
<u>Elastase</u> (synthesized in inactive form — proelastase, which is activated by trypsin)		
<u>Carboxypeptidases A and B</u> (synthesized in inactive form — procarboxypeptidases A and B, which are activated by trypsin)	They act on C — end connections of proteins and peptides	
<u>Lipase</u>	They split fats (emulsified by action of salts of cholic acids) with the formation of monoglycerides and fatty acids	
<u>Phospholipase</u> (synthesized in inactive form — prophospholipase, which is activated by trypsin)		
<u>Esterase</u>		
<u>Alpha-amylase</u>	It splits carbohydrates: (polysaccharides) with the formation of oligosaccharides, disaccharides, monosaccharides	
<u>Ribonuclease and deoxvribonuclease</u>	They split nucleic acids with the formation of nucleotides	

Pancreatic juice also contains water

Table 4.7. — Structure and functions of bile (pH 7.3–8.0)

Organic components of bile	Inorganic components of bile	Functions of bile
<u>Cholic acids</u> — cholic and chenodeoxycholic acids — and their salts (in the bile they are contained as compounds with glyocol and taurine; glycocholic acids — 80% and taurocholic acids — 20%) <u>Cholic pigments</u> (bilirubin and biliverdin) <u>Cholesterin</u> <u>Fatty acids</u> <u>Mucin</u> <u>Proteins and amino acids</u>	Na ⁺ K ⁺ Ca ²⁺ Mg ²⁺ chlorides bicarbonatesphosphates (and others)	1. Emulsification of fats, which promote their hydrolysis. 2. Strengthens action of lipolytic and amylolytic enzymes. 3. Strengthens motility of intestine. 4. Participates in neutralization of acidic products which have come from the stomach. 5. Promotes adsorption of fatty acids, liposoluble vitamins, cholesterin, amino acids and salts of calcium. 6. Inhibits decay process in the intestine. 7. Protective function (bile has bacteriostatic action). 8. Stimulates biligenesis

Table 4.8. — Structure of juice of small intestine (pH 5.05–7.07)

Organic components		Inorganic components
Digestive enzymes		Na^+ K^+ Ca^{2+} Chlorides Bicarbonates Phosphates
Name of enzyme	Role of enzyme	
Protein-digesting enzymes: <u>enterokinase, peptidases (dipeptidase, aminopeptidase)</u>	They split small peptides, dipeptides with the formation of mainly aminoacids	
Carbohydrate-digesting enzymes: <u>amylase, lactase, saccharase</u>	They split dextrins and oligosaccharides with the formation of mainly monosaccharides	
Fat-digersting enzymes: <u>lipase, phospholipase</u>	They split fats with the formation of monoglycerides and fatty acids	
<u>Alkaline phosphatase</u>	It splits residue of phosphoric acid from the organic ether compounds of phosphoric acid	
<u>Nuclease</u>	It splits nucleotides with the formation of N-containing bases, ribose, deoxyribose, phosphate	
		Mucus Proteins Amino acids Urea Non-destroyed epithelial cells and fragments of cells

Juice of small intestine also contains water

Table 4.9. — Hormones and hormone-like products that act in digestion

Hormone	Site of production	Target	Effect
Gastrin	Stomach mucosa	Stomach Ileocecal valve	Causes gastric glands to increase secretory activity; most pronounced effect is on HCl secretion. Relaxes ileocecal valve
Serotonin	Stomach mucosa	Stomach	Causes contraction of stomach muscle
Histamine	Stomach mucosa	Stomach	Activates parietal cells to release HCl
Somatostatin	Stomach mucosa	Stomach Pancreas Small intestine	Inhibits gastric secretion of all products; inhibits gastric motility and emptying. Inhibits secretion of pancreas. Inhibits intestinal absorption.
Intestinal gastrin	Duodenal mucosa	Stomach	Stimulates gastric glands
Secretin	Duodenal mucosa	Stomach Pancreas Liver	Inhibits gastric gland secretion during gastric phase of secretion. Increases output of pancreatic juice rich in bicarbonate ions. Increases bile output.
Cholecystokinin	Duodenal mucosa	Stomach Pancreas Gallbladder Sphincter of Oddi	Inhibits gastric gland secretion during gastric phase of secretory activity. Increases output of enzyme-rich pancreatic juice. Stimulates organ to contract and expel stored bile. Relaxes Oddi's sphincter to allow entry of bile and pancreatic juice into duodenum.
Gastric inhibitory peptide	Duodenal mucosa	Stomach	Inhibits gastric gland secretion during gastric phase.
Enterogastrone	Duodenal mucosa	Stomach	Inhibits motility of stomach smooth muscle.

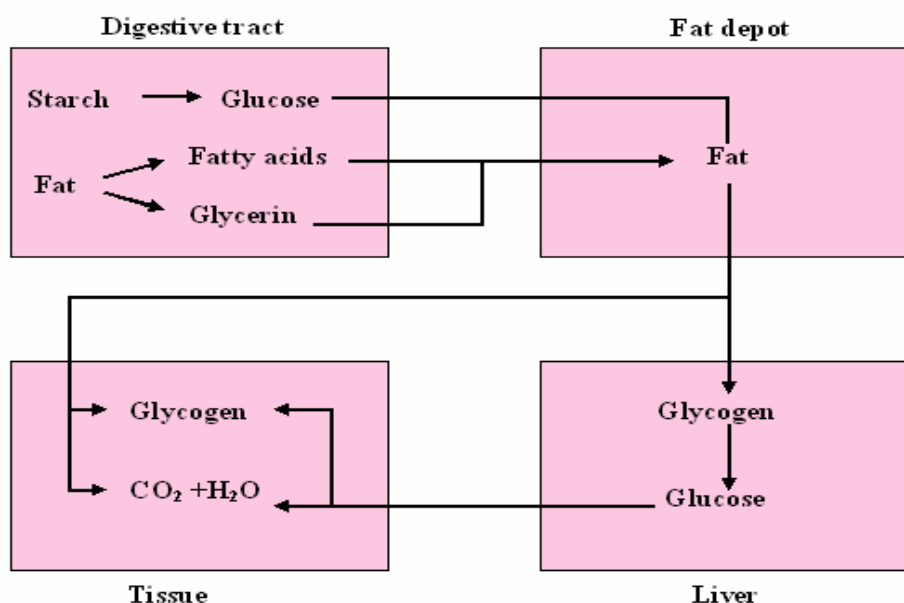
Table 4.10. — Humoral regulation of activity of gastrointestinal tract

The kind of activity	Stimulators	Inhibitors
Secretion of saliva	Acetylcholine, histamine, callicrein, toxins, CO ₂ , parotin	Noradrenalin, adrenalin
Secretion of gastric juice	Gastrin, acetylcholine, bombesin, motilin	Noradrenalin, adrenalin, decreasing of pH, secretin, cholecystokinin, gastric inhibitory peptide, somatostatin, serotonin
Motor activity of stomach	Gastrin, motilin, serotonin, insulin, acetylcholine	Secretin, cholecystokinin, gastric inhibitory peptide, noradrenalin, adrenalin, enterogastrone
Secretion of the juice of small intestine	Products of digestion of proteins, fats, pancreatic juice, HCl, gastric inhibitory peptide, motilin	Somatostatin
Motor activity of small intestine	Vegetable food, fats, vasopressin, oxytocin, bradikinin, serotonin, histamine, gastrin, motilin, cholecystokinin	Secretin, gastric inhibitory peptide
Secretion of pancreatic juice	Acetylcholine, HCl, secretin, cholecystokinin, gastrin, serotonin, insulin, salts of bile acids	Adrenalin, noradrenalin, glucagons, calcitonin, gastric inhibitory peptide, somatostatin
Excretion of pancreatic juice	Cholecystokinin	Carboxypeptidases A and B
Secretion of bile	Bile, Serotonin, glucagon, gastrin, cholecystokinin, prostaglandins	—
Excretion of bile	Eggs, meat, fats, cholecystokinin, gastrin, secretin, bombesin	Glucagon, calcitonin, gastric inhibitory peptide
Motor activity of large intestine	Rude food, vegetable food	Serotonin, adrenalin, glucagon

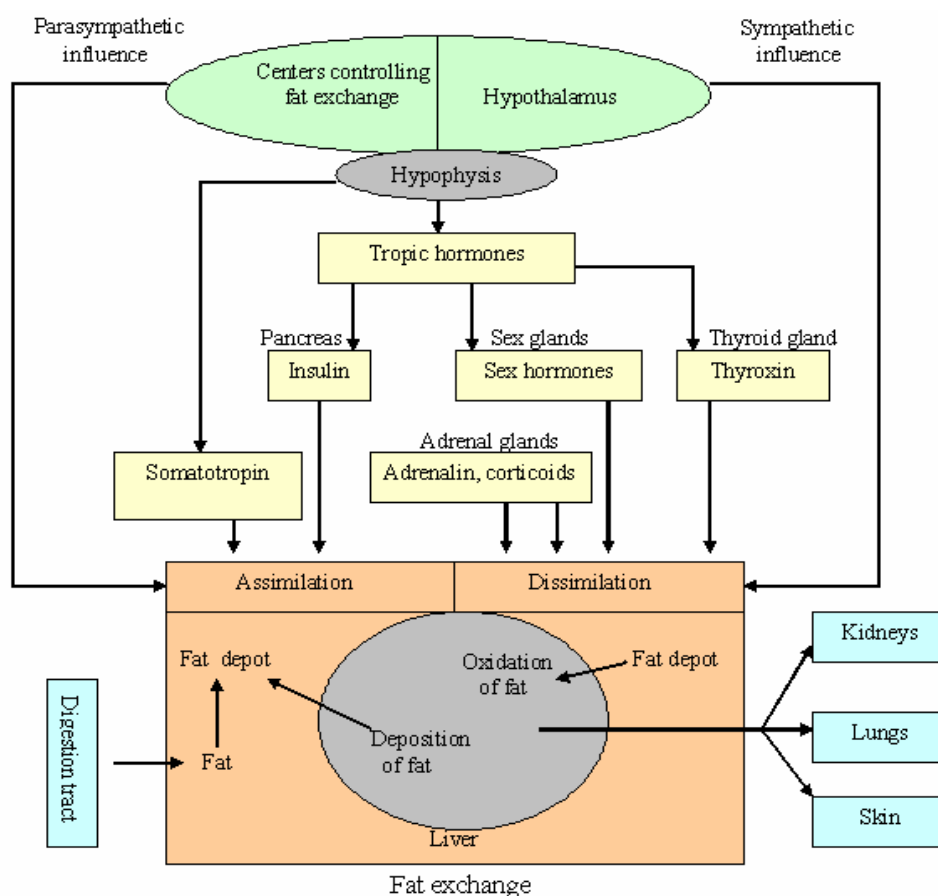
Unite 5

**METABOLISM.
ENERGY METABOLISM.
THERMOREGULATION**

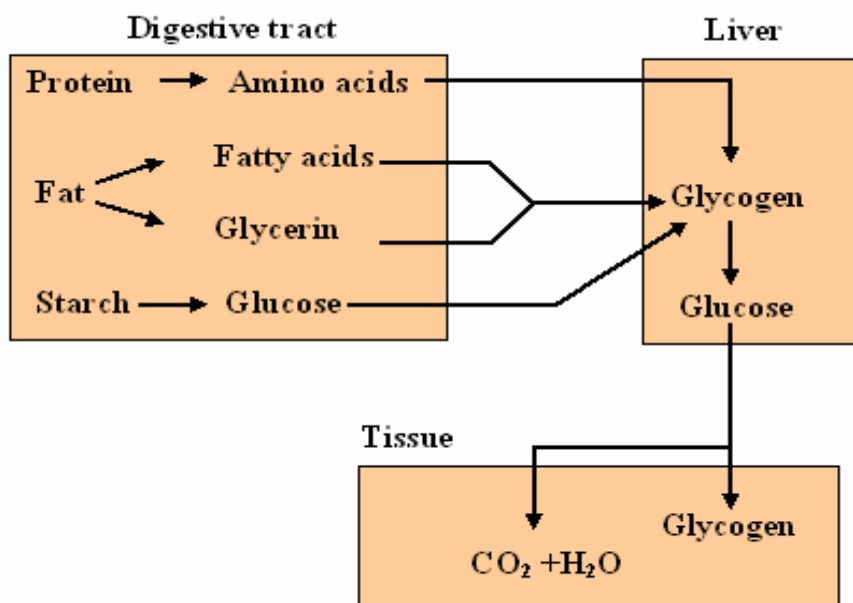
Metabolism — it includes all physical, chemical and physiological processes, which provide reception and delivery of energy to cells, organs and tissues from exo- and endogenous sources, ensuring of plastic needs of the organism for regenerating of structures and excretion of metabolism products from the organism.



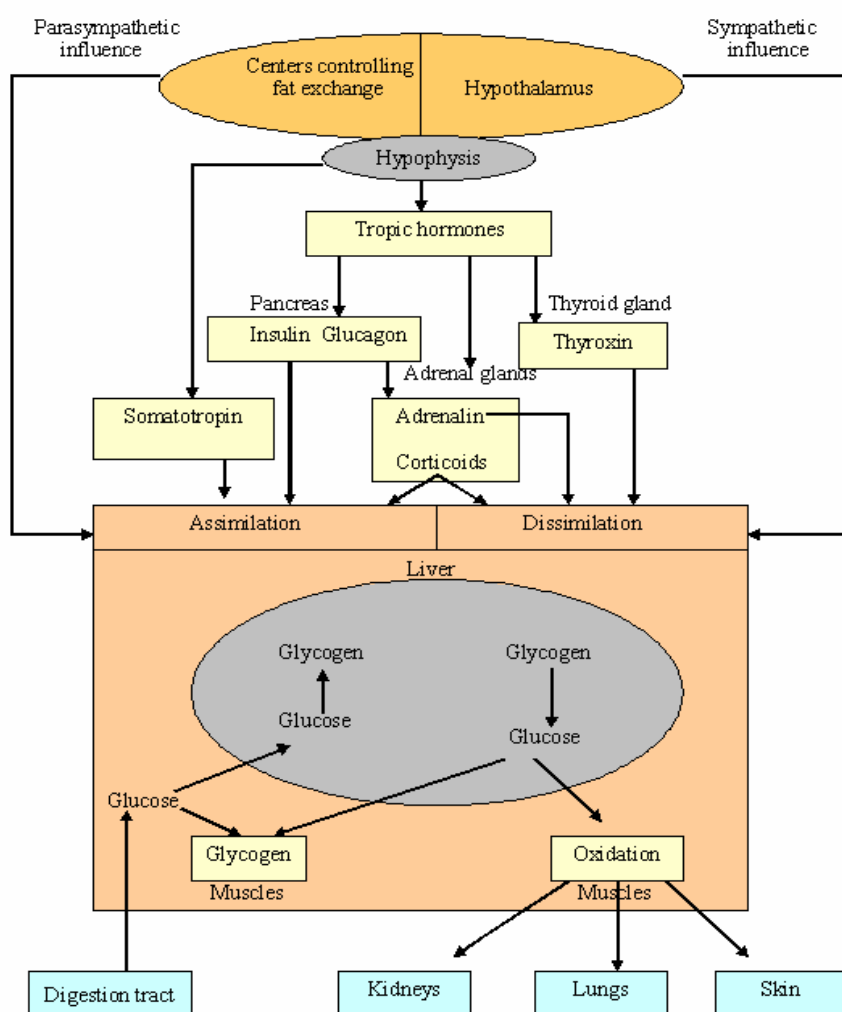
Scheme 5.1. — Fat exchange (by A. Ginetsinskiy, 1956)



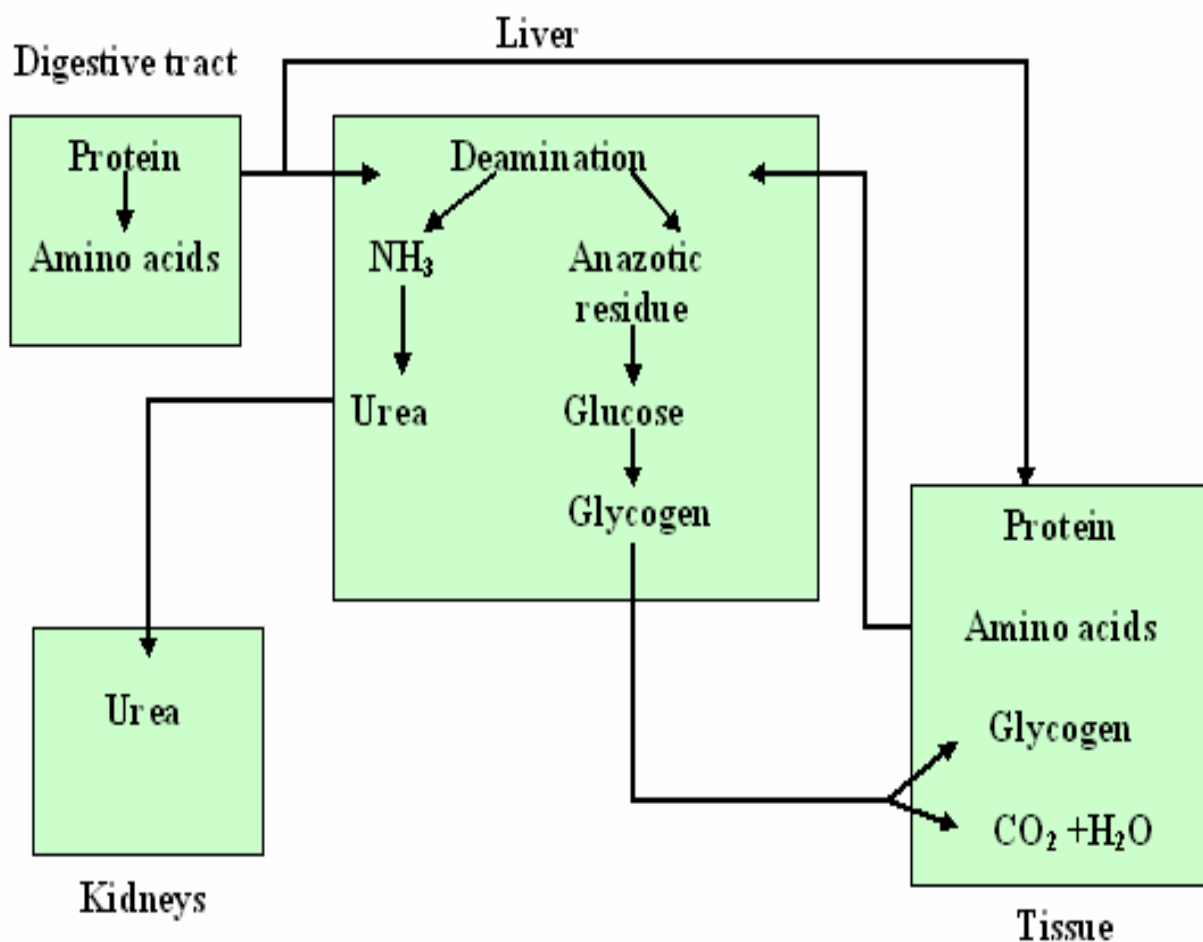
Scheme 5.2. — Regulation of fat exchange (by Korobkov A. V., Chesnokova S. A., 1986)



Scheme 5.3. — Carbohydrate exchange (by A. Ginetsinskiy, 1956)



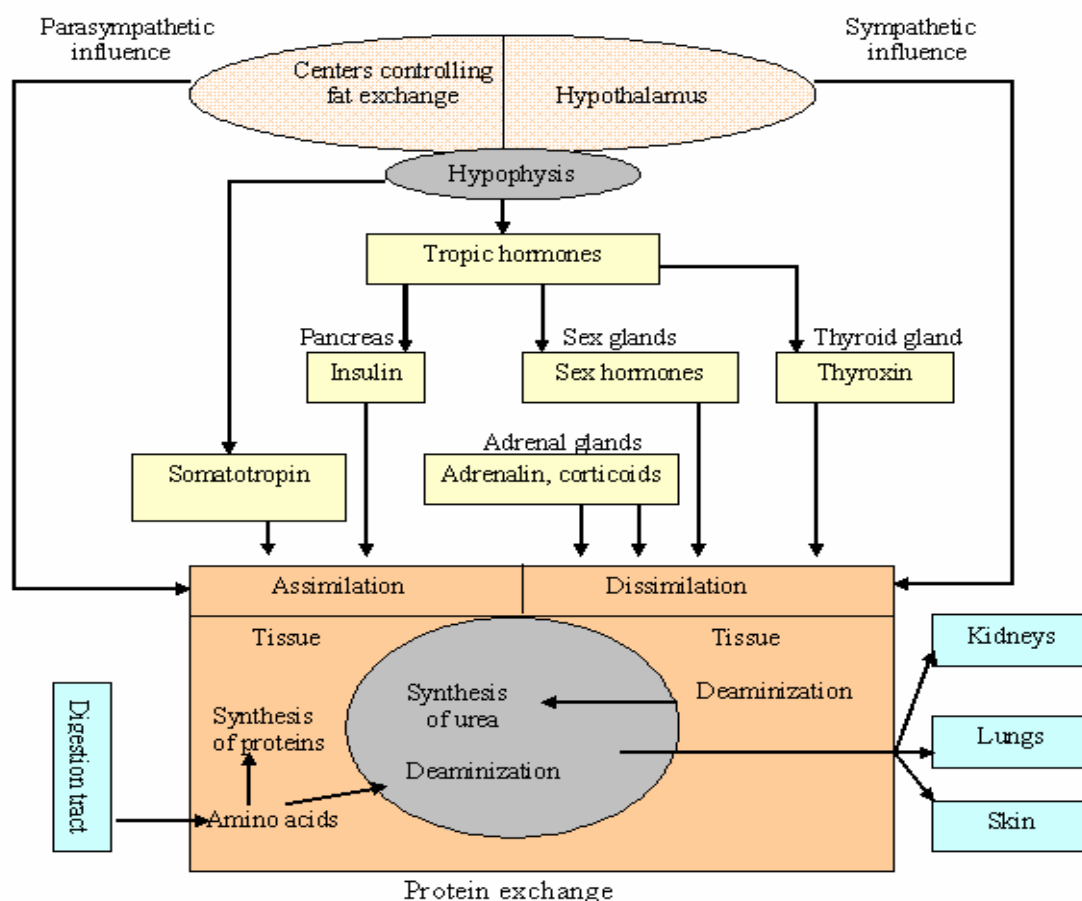
Scheme 5.4. — Regulation of carbohydrate exchange (by Korobkov A. V., Chesnokova S. A., 1986)



Scheme 5.5. — Protein exchange (by A. Ginetsinskiy, 1956)

Table 5.1. — Nitrogen balance

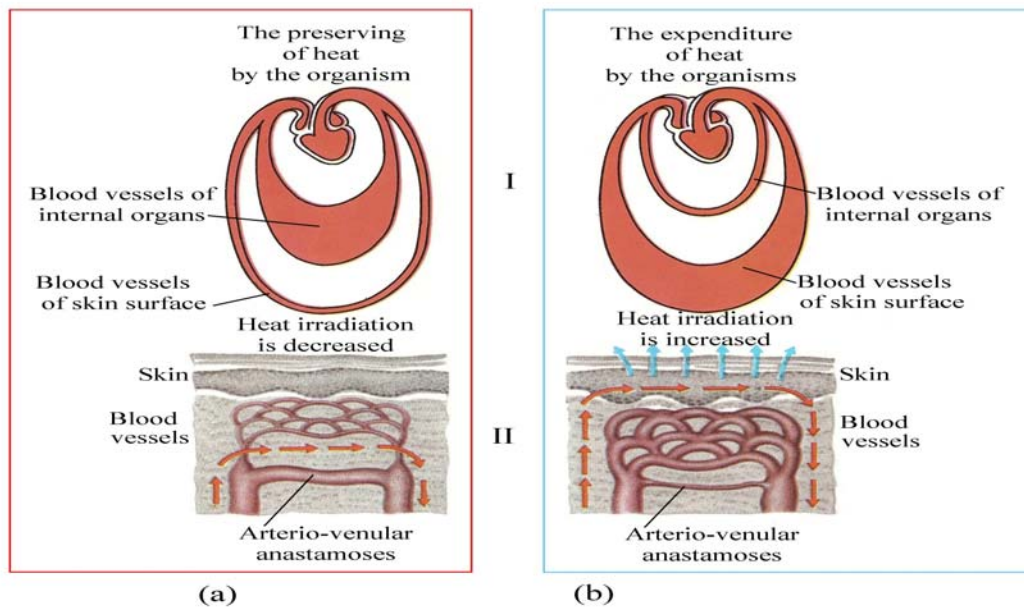
Kind of balance	Characteristics	Example
Nitrogen equilibrium	The amount of nitrogen consumed with food is equal to the amount of nitrogen excreted from the organism (the amount of synthesized protein is equal to the amount of destructed protein)	It is observed in adults
Positive nitrogen balance	The amount of nitrogen consumed with food is more than the amount of nitrogen excreted from the organism (synthesis of protein predominates over destruction of protein)	It is observed: <ul style="list-style-type: none"> • in children at growth, • during recovery, • at pregnancy, at increased sports activity.
Negative nitrogen balance	If the amount of nitrogen excreted from the organism is more than the amount of nitrogen consumed with food (destruction of protein predominates over synthesis of protein)	It is observed: <ul style="list-style-type: none"> • at protein starvation, • in old people, • during serious diseases.



Scheme 5.6. — Regulation of protein exchange
(by Korobkov A. V., Chesnokova S. A., 1986)

Table 5.2. — Methods of definition of energy consumption of organism

Name of method		Devices used for measuring	Principle of method
Direct calorimetry		Special chambers — biocalorimeters (which are thermoisolated from the environment).	It is based on direct and complete calculating of amount of heat released by the organism. Temperatures of water coming into the chamber and flowing from it are measured and the difference between these temperatures is calculated.
Indirect calorimetry	Complete gas analysis	At the open respiratory method of Douglas — Choldane airproof sack is used.	It is based on the definition of the amount of oxygen inspired and carbonic gas expired during certain interval of time. The expired air is collected into airproof sack with following definition of its total amount and amount of oxygen and carbonic gas in it by means of gas analyzers. Then calculation of heat production is made.
	Incomplete gas analysis		It is based on the definition of only the amount of oxygen inspired with following calculation of heat production. This method is used for definition of energy consumption when person is in conditions of relative rest.



Scheme 5.7. — The mechanisms of heat irradiation by the organism at the conditions of cold (a) and heat (b) ((by Sherington, 1897)

I — redistribution of blood between the vessels of internal organs and vessels of skin surface.
II — redistribution of blood in the vessels of skin.

Table 5.3. — Mechanisms of thermoregulation

Kinds of thermoregulation	Name of the mechanism	Characteristics of the mechanism
Chemical thermoregulation (heat production)	Shivering thermogenesis	Irregular involuntary tonic contractions of muscles.
	Not shivering thermogenesis	<ul style="list-style-type: none"> • The increasing of metabolic processes in internal organs; • The increasing of metabolic processes in brown adipose tissue; • The increasing of thermogenesis after taking food (at disintegration of proteins, fats and carbohydrates).
Physical thermoregulation (heat irradiation)	Thermal irradiation	It happens in the form of electromagnetic waves
	Convection	If the skin is warmer than surrounding air, the air layer adjacent to the skin is heated up, rises and displaced by colder air.
	Heat conductivity	It is transition of heat from one subject to another at immediate contact with a body surface. Biological tissues serve as isolators (for example, fats).
	Evaporation	Evaporation of water (sweat) from surface of skin happens with the use of energy for transition of fluid into steam.
	Changing of lumens of skin vessels	Dilatation of skin vessels increases heat irradiation, and constriction of skin vessels decreases heat irradiation.
	Changing of respiration rate	Expired air carries heat. When respiration becomes frequent, heat irradiation increases.

Table 5.4. — Regulation of constancy of body temperature

Receptors	Spinal cord	Hypothalamus	Cortex of cerebrum
Thermoreceptors («cold» and «thermal»). They are situated in skin, dermal and hypodermic vessels, and in CNS.	In the spinal cord some centers of thermoregulatory reflexes are situated (it participates in regulation of shivering thermogenesis and in regulation of lumen of skin vessels).	The centers of heat irradiation are situated in the region of anterior nuclei of hypothalamus; the centers of heat production are situated in the lateral-dorsal region of hypothalamus.	It is possible to form conditioned reflex of rising of temperature. Rising of body temperature can happen under influence of hypnosis, at mental diseases, hysteria, and also in actors and in students during examinations.

Table 5.5. — The hormones of endocrine glands

Endocrine glands	Hormone	Target	Effects	Effects of hyposecretion and hypersecretion
Thyroid gland	Triiodothyronine and tetraiodothyronine (T3,T4)	All organism	Accelerates metabolism and consumption of oxygen by tissue	Hyposecretion: Cretinism (at childhood) myxedema Hypersecretion: thyrotoxicosis
Parathyroid glands	Parathormone	Bone, kidneys, digestive tract	Increases the level of calcium in blood. Simultaneously reduces concentration of inorganic phosphates in blood, and increases their excretion with urine.	Hyposecretion: tetany Hypersecretion: osteoporosis
Beta-cells of pancreas	Insulin	All organism	Decreases the level glucose in the blood	Hyposecretion: diabetes mellitus Hypersecretion: hypoglycemic coma
Alpha-cells of pancreas	Glucagon	Liver	Promotes splitting of glycogen and increase the level of glucose in blood. Simultaneously, glucagon stimulates synthesis of glycogen in liver.	Hyposecretion: pathological syndrome: dermatitis, anemia.
Cortex of adrenal gland	Mineralocorticoids (aldosterone)	Kidneys	Increases the amount of Na ⁺ in blood (due to increasing of reabsorption of Na ⁺ in kidneys) and decreased the amount of K ⁺ in blood; Sodium reabsorption is accompanied with water reabsorption, and due to this blood volume and blood pressure increases.	Hyposecretion: Addison's disease Hypersecretion: aldosteronism
	Glucocorticoids (cortisol)	All organism	Promote gluconeogenesis and hyperglycemia; mobilize fats for energy metabolism; stimulate protein catabolism; assist body to resist stress factors; depress inflammatory and immune responses.	Hyposecretion: Addison's disease Hypersecretion: Cushing's disease
	Gonadocorticoids (mainly androgens)		May be responsible for female libido and source of estrogen after menopause	Hypersecretion: virilization of females

Endo- crine glands	Hormone	Target	Effects	Effects of hyposecretion and hypersecretion
Medullary layer of adrenal gland	Adrenaline Noradrenaline	Myocardium, smooth muscles of arterioles, liver, skeletal muscles, fatty tissue Arterioles	Increases heart rate and blood pressure (due to vasoconstriction), stimulate lipolysis and glycogenolysis Increase blood pressure by promoting vasoconstriction	Hypersecretion: hypertension
Ovaries	Estrogens, Progesterone	Female genital organs, mammary gland, uterus, all organism	Stimulate development of secondary sex characteristics; provide cyclic process in uterus and ovaries, mammary glands	Hyposecretion: hypogonadism
Testes	Testosterone	Male genital organs, all organism	Stimulate development of secondary sex characteristic and normal function	Hyposecretion: hypogonadism

Table 5.6. — Hormones of hypophysis

Hormone	Target	Effects	Effects of hyposecretion and hypersecretion
Hormones of anterior lobe of the hypophysis			
Growth hormone	Body cells, mainly bone and muscle	Stimulates somatic growth; mobilizes fats	Hyposecretion: pituitary dwarfism (pituitary nanism) in children. Hypersecretion: gigantism in children; acromegaly in adults.
Thyroid-stimulating hormone	Thyroid gland	Stimulates thyroid gland to release thyroid hormone	Hyposecretion: cretinism in children, myxedema in adults. Hypersecretion: symptoms of Graves's disease (Basedow's disease).
Adrenocorticotrophic hormone	Adrenal cortex	Promotes release of glucocorticoids and androgens	Hyposecretion: Rare Hypersecretion: Cushing's disease.
Follice-stimulating hormone	Ovaries and testes	In females: stimulates ovarian follicle maturation and estrogen production. In males: stimulates sperm production.	Hyposecretion: failure of sexual maturation Hypersecretion: no important effects
Luteinizing hormone	Ovaries and testes	In females: triggers ovulation and stimulates ovarian production of progesterone. In males: promotes testosterone production	Hyposecretion: failure of sexual maturation Hypersecretion: no important effects
Prolactin	Breast secretory tissue	Promotes lactation	Hyposecretion: decrease of milk production in nursing women. Hypersecretion: galactorrhea; absence of menses in females; impotency in males.

Hormone	Target	Effects	Effects of hyposecretion and hypersecretion
Hormones of intermediate lobe of the hypophysis			
Intermedin	Melancytes	Stimulates pigment production	Hyposecretion: albinism Hypersecretion: hyperpigmentation
Posterior pituitary hormones			
Oxytocin	Uterus	Stimulates uterine contractions (especially at delivery). Initiates milk excretion from breast	Hyposecretion: powerless delivery
Antidiuretic hormone	Kidneys	Stimulates reabsorption of water in kidney	Hyposecretion: diabetes insipidus Hypersecretion: unknown

Table 5.7 — Vitamins

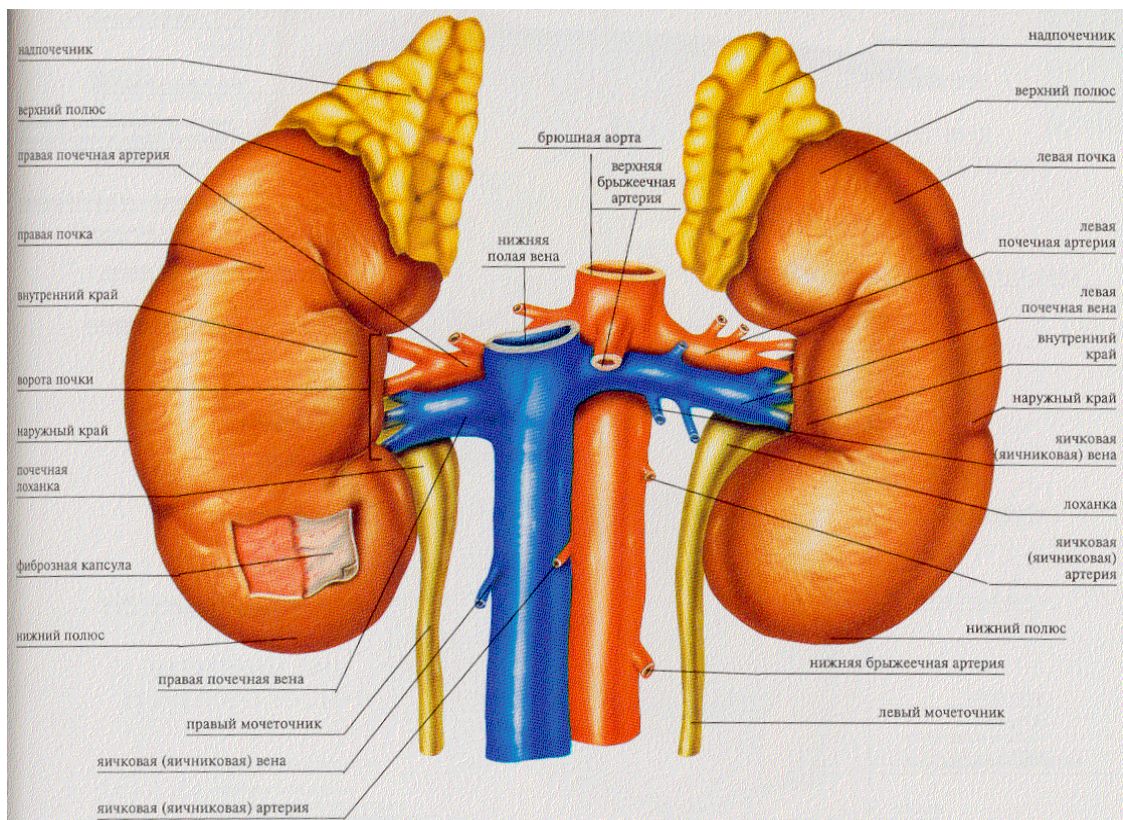
Vitamin	Sources	Physiological role	Problems	
			Excesses	Deficits
A (retinol)	Formed from provitamin carotene in intestine, liver, kidneys. Carotene is found in yellow and green leafy vegetables; vitamin A is found in fish liver, oils, egg yolk, liver oils, fortified foods	Required for synthesis of photoreceptor pigments of rods and cones, integrity of skin and mucosae, normal tooth and bone development; normal reproductive capabilities; acts with vitamin E to stabilize cell membranes	Massive doses induce toxicity: nausea, vomiting, anorexia, headache, hair loss, bone and joint pain, bone fragility, enlargement of liver and spleen	Night blindness; epithelial changes: dry skin and hair, skin sores; increased respiratory, digestive, urogenital infections; drying of conjunctiva; clouding of cornea
D (antirachitic factor)	Vitamin D ₃ is produced in skin by irradiation of 7-dehydrocholesterol by UV light; active form produced by chemical modification of vitamin D ₃ in liver, then in kidneys	Increases calcium blood levels by enhancing absorption of calcium; mobilizes calcium from bones	Massive doses induce toxicity: vomiting, diarrhea, weight loss, calcification of soft tissues, renal damage	Faulty mineralization of bones and teeth; rickets in children, osteomalacia in adults; poor muscle tone, restlessness, irritability
E (antisterility factor)	Vegetable oils, margarine, whole grains, dark-green leafy vegetables	It is an antioxidant; may help to prevent oxidation of vitamins A and C in intestine; in tissues, decreases oxidation of unsaturated fatty acids, thus helps maintain integrity of cell membranes	Thrombophlebitis, hypertension; slow wound healing	Extremely rare, precise effects uncertain: possible hemolysis of RBCs, macrocytic anemia; fragile capillaries
K (coagulation vitamin)	Mostly synthesized by coliform bacteria in large intestine; food sources: green vegetables, cabbage, cauliflower, pork liver	Essential for formation of clotting proteins and some other proteins made by liver; as intermediate in electron transport chain, participates in oxidative phosphorylation in all body cells	None known, not stored in appreciable amounts	Easy bruising and bleeding (prolonged clotting time)

Vitamin	Sources	Physiological role	Problems	
			Excesses	Deficits
C (ascorbic acid)	Fruits and vegetables	Acts in hydroxylation reaction in formation of nearly all connective tissues; in conversion of tryptophan to serotonin; in conversion of cholesterol to bile salts; helps to protect vitamins A and E dietary fats from oxidation; enhances iron absorption and use; required for conversion of folacin to its active form	Result of megadoses; enhanced mobilization of bone minerals and blood coagulation; exacerbation of gout, kidney stone formation	Defective formation of intercellular cement; fleeting joint pains, poor tooth and bone growth; poor wound healing, increased susceptibility to infection; extreme deficit causes scurvy
B₁ (thiamin)	Lean meats, liver, eggs, whole grains, leafy green vegetables, legumes	Part of coenzyme co-carboxylase, which acts in carbohydrate metabolism; required for transformation of pyruvic acid to acetyl CoA, for synthesis of pentose sugars and acetylcholine; for oxidation of alcohol	None known	Beriberi: decreased appetite; gastrointestinal disturbances; peripheral nerve changes indicated by weakness of legs, cramping of calf muscles, numbness of feet; heart enlarges, tachycardia
B₂ (riboflavin)	Widely varying sources such as liver, yeast, egg white, whole grains, meat, poultry, fish, legumes, major source is milk	Present in body as co-enzymes FAD and FMN both of which act as hydrogen acceptors in body; also is a component of amino acid oxidases	None known	Dermatitis; cracking of lips at corners; lips and tongue become purple-red and shiny; ocular problems: light sensitivity, blurred vision.
Niacin (nicotinamide)	Diets that provide adequate protein usually provide adequate niacin because amino acid tryptophan is easily converted to niacin; preformed niacin provided by poultry, meat, fish; less important sources: liver, yeast, peanuts, potatoes, leafy green vegetables	Constituent of NAD and NADP, coenzymes involved in glycolysis, oxidative phosphorylation, fat breakdown; inhibits cholesterol synthesis	Result of megadoses; hyperglycemia; vasodilation leading to flushing of skin, tingling sensations; possible liver damage gout	Pellagra: listlessness, headache, weight loss, loss of appetite; progresses to soreness and redness of tongue and lips; nausea, vomiting, diarrhea; photosensitive dermatitis, neurological symptoms also occur.
B₆ (pyridoxine)	Meat, poultry, fish; less important sources: potatoes, tomatoes, spinach	Active form is coenzyme pyridoxal phosphate which functions in several enzyme systems involved in amino acid metabolism, also required for conversion of tryptophan, for glycolysis, for formation of antibodies and hemoglobin	Depressed deep tendon reflexes, numbness, loss of sensation in extremities	Infants: nervous irritability, convulsions, anemia, vomiting, weakness, abdominal pain; adults: seborrhea lesions around eyes and mouth

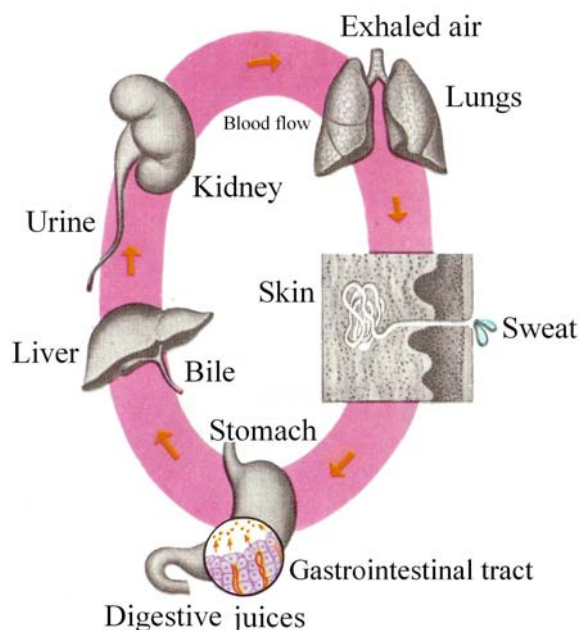
Vitamin	Sources	Physiological role	Problems	
			Excesses	Deficits
Pantothenic acid	Name derived from Greek panthos meaning every where; widely distributed in animal foods, whole grains, legumes; liver, yeast, egg yolk, meat especially good sources; some produced by entetic bacteria	Functions in form of coenzyme A in reactions that remove or transfer acetyl group, e.g.; formation of acetyl CoA from pyruvic acid, oxidation and synthesis of fatty acids; also involved in synthesis of steroids and heme of hemoglobin	None known	Symptoms vague: loss of appetite, abdominal pain, mental depression, pains in arms and legs, muscle spasms, neuromuscular degeneration
Biotin	Liver, egg yolk, legumes, nuts; some synthesized by bacteria in gastrointestinal tract	Function as coenzyme for a number of enzymes that catalyze carboxylation, decarboxylation, deamination reactions; essential for reactions of Krebs's cycle, for formation of purines and nonessential amino acids, for use of amino acids, for use of amino acids for energy	None known	Scaly skin, muscular pains, pallor, anorexia, nausea, fatigue; elevated blood cholesterol levels
B₁₂ (cyano-cobalamin)	Liver, meat, poultry fish, dairy foods except butter, eggs; not found in plant foods	Functions as coenzyme	None known	Pernicious anemia, signified by pallor, anorexia, dyspnea, weight loss, neurological disturbances
Folic acid (folacin)	Liver, deep-green vegetables, yeast, lean beef, eggs, veal, whole grains; synthesized by enteric bacteria	Basis of coenzymes that act in synthesis of methionine and certain other amino acids, choline, DNA; essential for formation of red blood cells	None known	Macrocytic or megaloblastic anemia; gastrointestinal disturbances; diarrhea

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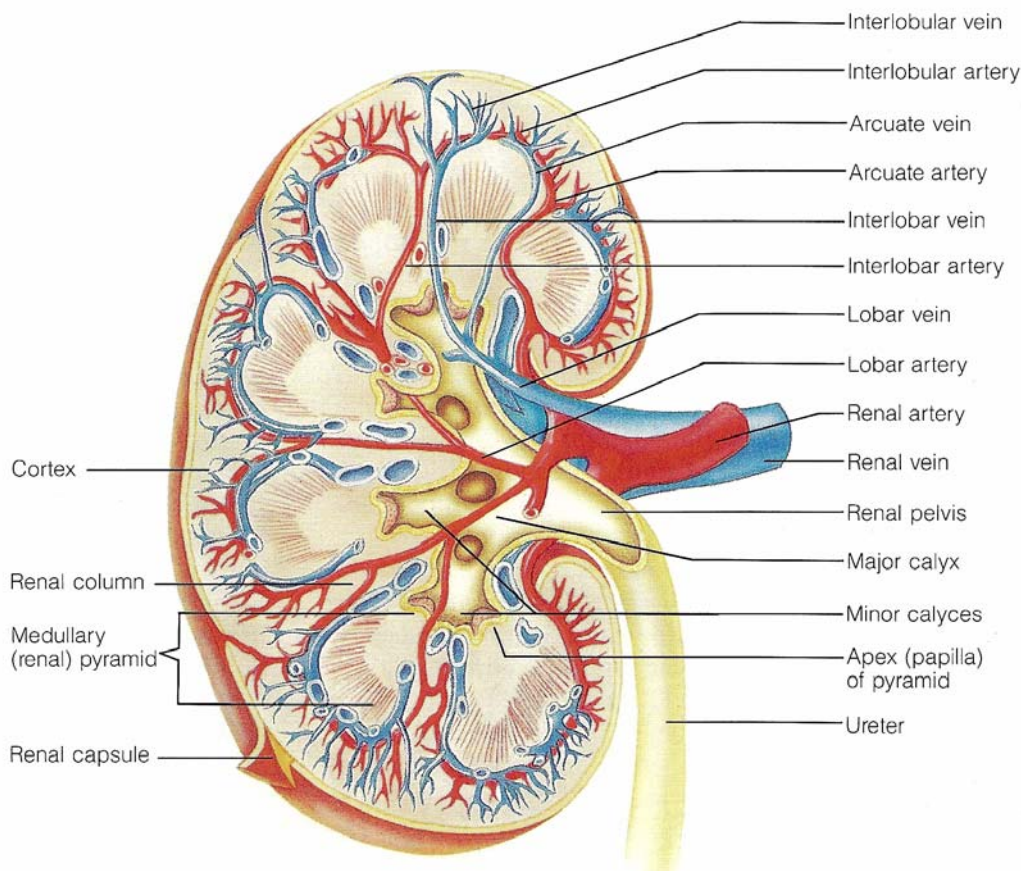
PHYSIOLOGY OF EXCRETION



Excretion is important process of homeostasis, it provides purifying of organism from products of exchange which cannot be used further by an organism (CO_2 and H_2O , foreign, toxic substances and series of other substances).



**Figure 6.1. — Organs, which participate in excretory processes
(the processes of purifying of blood from metabolism products)
(by Korobkov A. V., Chesnokova S. A., 1986)**



**Figure 6.2. — Internal anatomy of the kidney
(diagrammatic view of a coronally sectioned kidney, illustrating major blood vessels)
(by Elaine N. Marieb, 1989)**

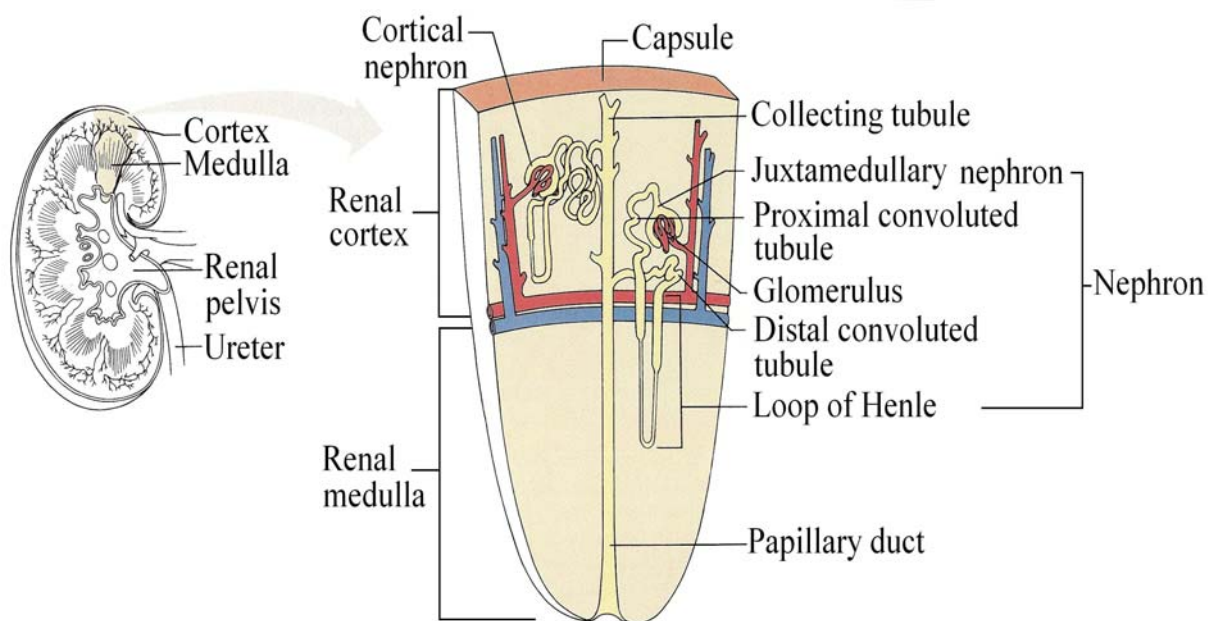


Figure 6.3. — Wedge-shaped section (lobule) of kidney tissue, indicating the locations of nephrons in the kidney (by Elaine N. Marieb, 1989)

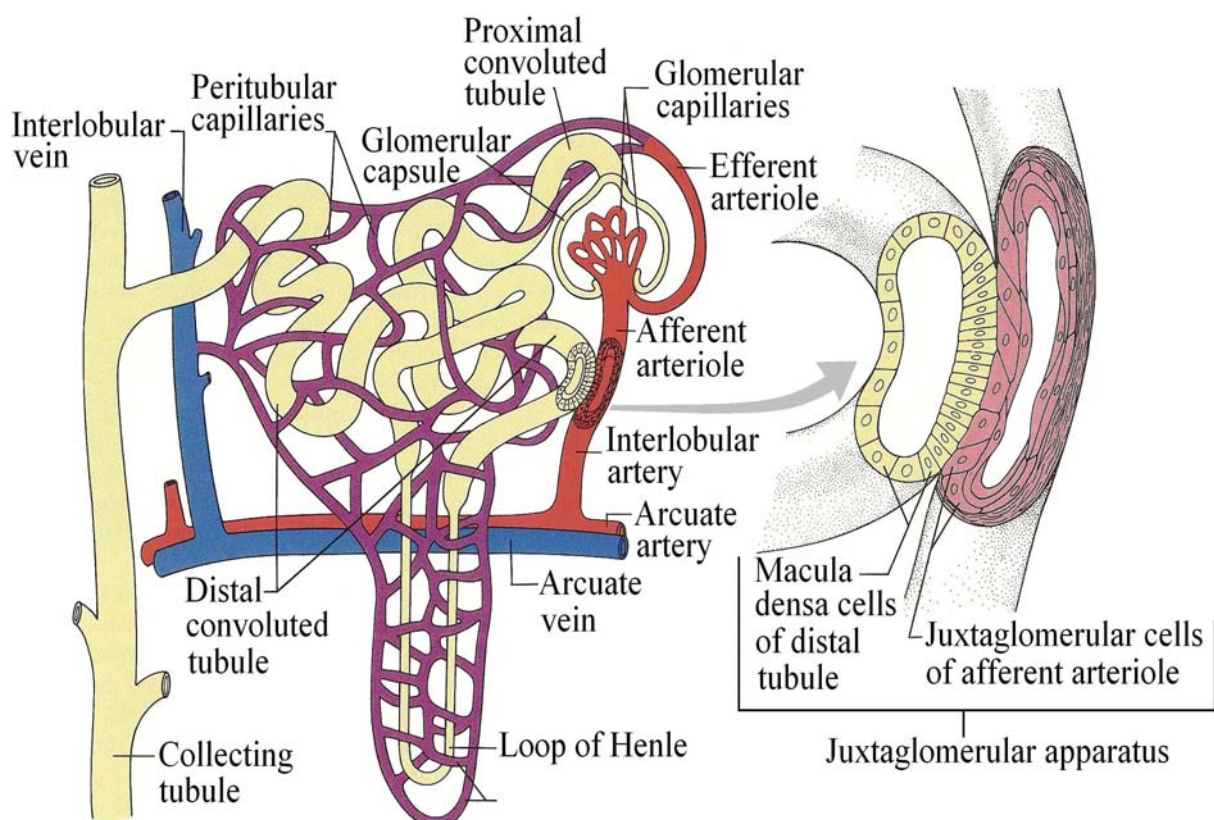


Figure 6.4. — Structure of a juxtamedullary nephron and its associated capillaries (by Elaine N. Marieb, 1989)

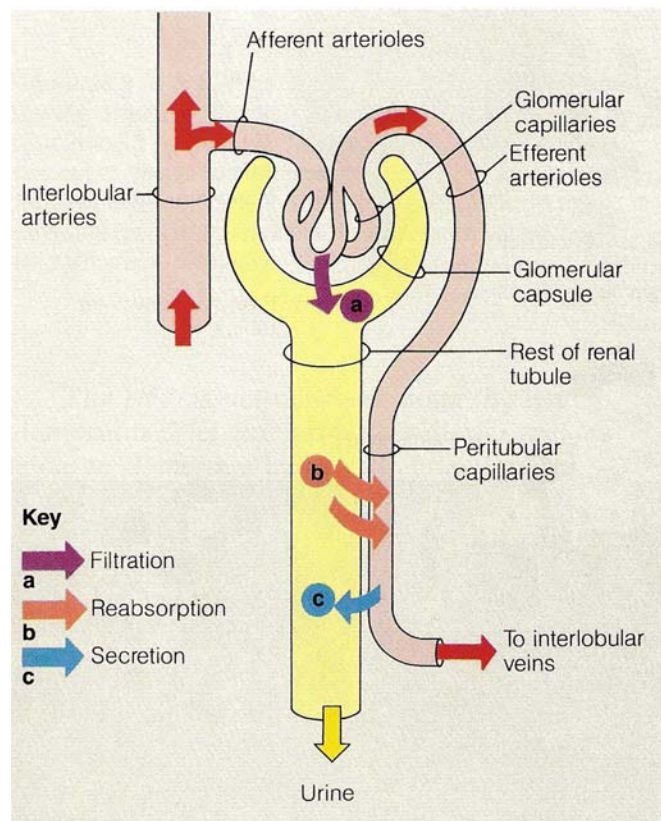


Figure 6.5. — The processes which take place in a nephron (by Elaine N. Marieb, 1989)

The three major mechanisms by which the kidney adjust the composition of plasma are:
 (a) glomerular filtration, (b) tubular reabsorption, (c) tubular secretion.

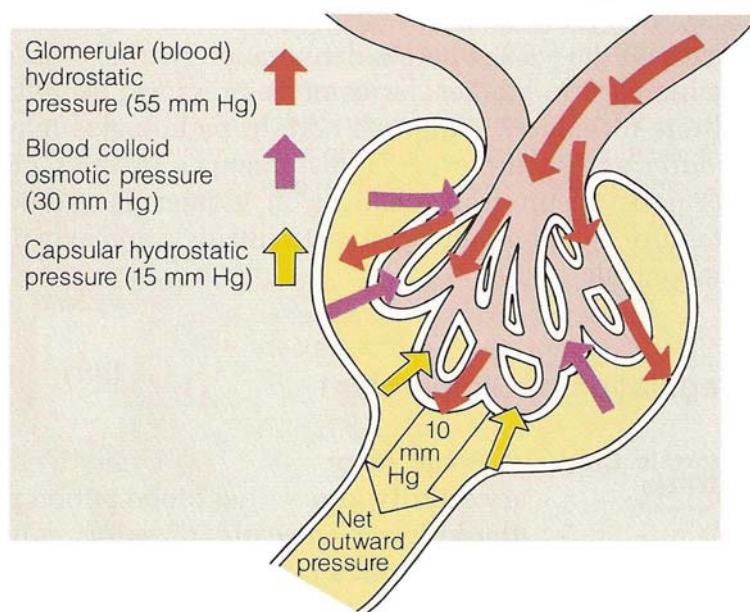


Figure 6.6. — Forces that determine glomerular filtration and the effective filtration pressure (by Elaine N. Marieb, 1989)

The glomerular hydrostatic (blood) pressure is the major factor forcing fluids and solutes out of the blood. This is opposed by the colloid osmotic pressure of the blood and the hydrostatic pressure that exists within the glomerular capsule. The pressure values cited in the diagram are approximate.

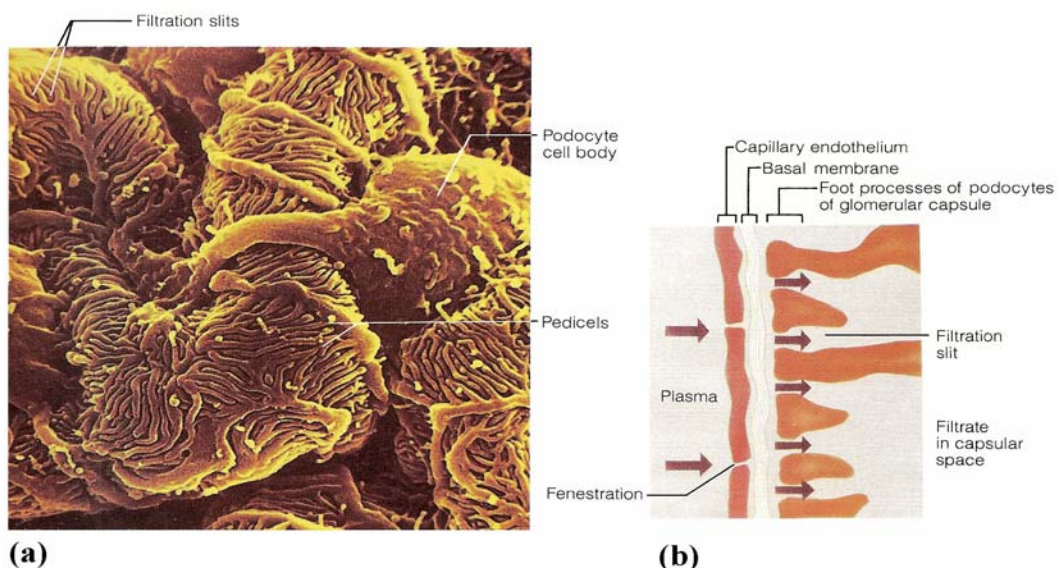


Figure 6.7. — The filtration membrane (by Elaine N. Marieb, 1989)

The filtration membrane in renal capsule consists of three layers:

- 1). the endothelium of glomerular capillary;
- 2). the basal membrane;
- 3). the layer of epithelial cells which cover Bowman's capsule (the cells of this layer are called podocytes).

(a) Scanning electron micrograph of the layer of podocytes. Filtration slits between the podocyte foot processes are evident (39,000 X)

(b) Diagrammatic view of a section taken through the filtration membrane showing all three structural elements

Effective filtration pressure (EFP).

Glomerular filtration depends on the effective filtration pressure (EFP).

$$EFP = HPB - (OPB + HPF);$$

HPB — hydrostatic pressure of blood in capillaries of glomerulus (it is equal to 60–90 mm Hg);

OPB — oncotic pressure of proteins of blood plasma (it is equal to 30 mm Hg);

HPF — hydrostatic pressure of fluid in Bowman's capsule (it is equal to 20 mm Hg).

$$EFP = 70 \text{ mm. Hg} - (30 \text{ mm Hg} + 20 \text{ mm Hg}) = 20 \text{ mm Hg}.$$

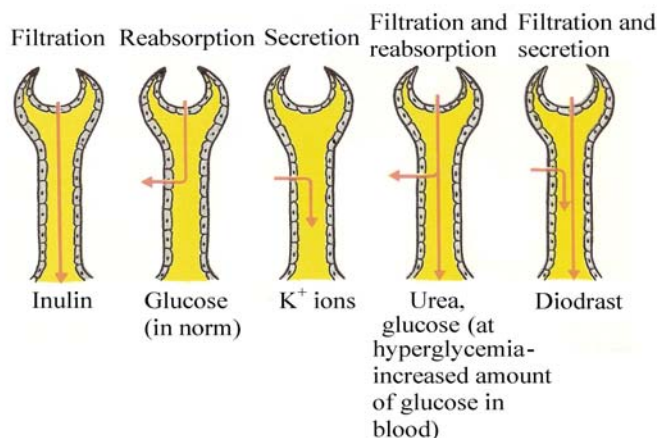
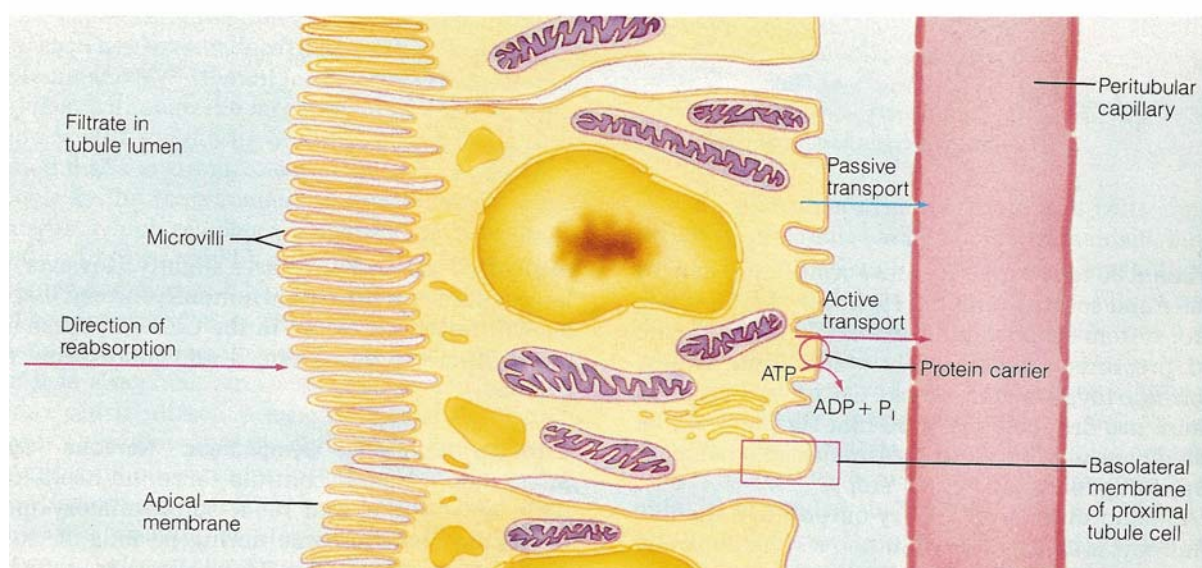


Figure 6.8. — Processes, which take place in the tubules of kidney, when different components of urine pass through the tubules (by Korobkov A. V., Chesnokova S. A., 1986)

Table 6.1. — Reabsorption capabilities of different segments of the renal tubules and collecting tubules

Proximal convoluted tubule	Sodium ions (Na^+) Virtually all nutrients (glucose, amino acids, vitamins) Cations (K^+ , Mg^{2+} , Ca^{2+} , and others) Anions (Cl^- , HCO_3^-) Water Urea and lipid – soluble solutes Small proteins
Loop of Henle: Descending loop Ascending loop	Water Na^+ and Cl^-
Distal convoluted tubule	Na^+ Anions
Collecting tubule	Water Urea



**Figure 6.9. — Directional movement of reabsorbed substances
(by Elaine N. Marieb, 1989)**

During reabsorption substances pass from the filtrate through the tubule cell's apical membrane and then through the tubule cell to its basolateral membrane. From there the substances move through the basolateral membrane into the interstitial fluid and then into the peritubular capillary blood.

Mechanisms of reabsorption

There are two ways for reabsorption of substance from tubule lumen into interstitial fluid.

- 1) para-cellular way (between the cells);
- 2) trans-cellular (through the cell; substances should overcome two plasma membranes of the tubule cell).

Main molecular mechanisms of reabsorption

- 1) endocytosis;
- 2) passive transport (for example, diffusion);
- 3) active transport (transmission of substances through membranes against concentration and electrochemical gradients with energy consumption), which can be divided into two types:

— primary-active transport - transmission of substances due to energy of cellular metabolism (energy received immediately at splitting of molecules ATP).

— secondary- active transport of substances against concentration gradient, but without using energy of cell immediately on this process (reabsorption of a substance happens with the help of special transmitting agent which should attach Na^+ ion ;this complex (transmitting agent + organic substance + Na^+) promotes moving of substances through membrane and its entering inside of cell).

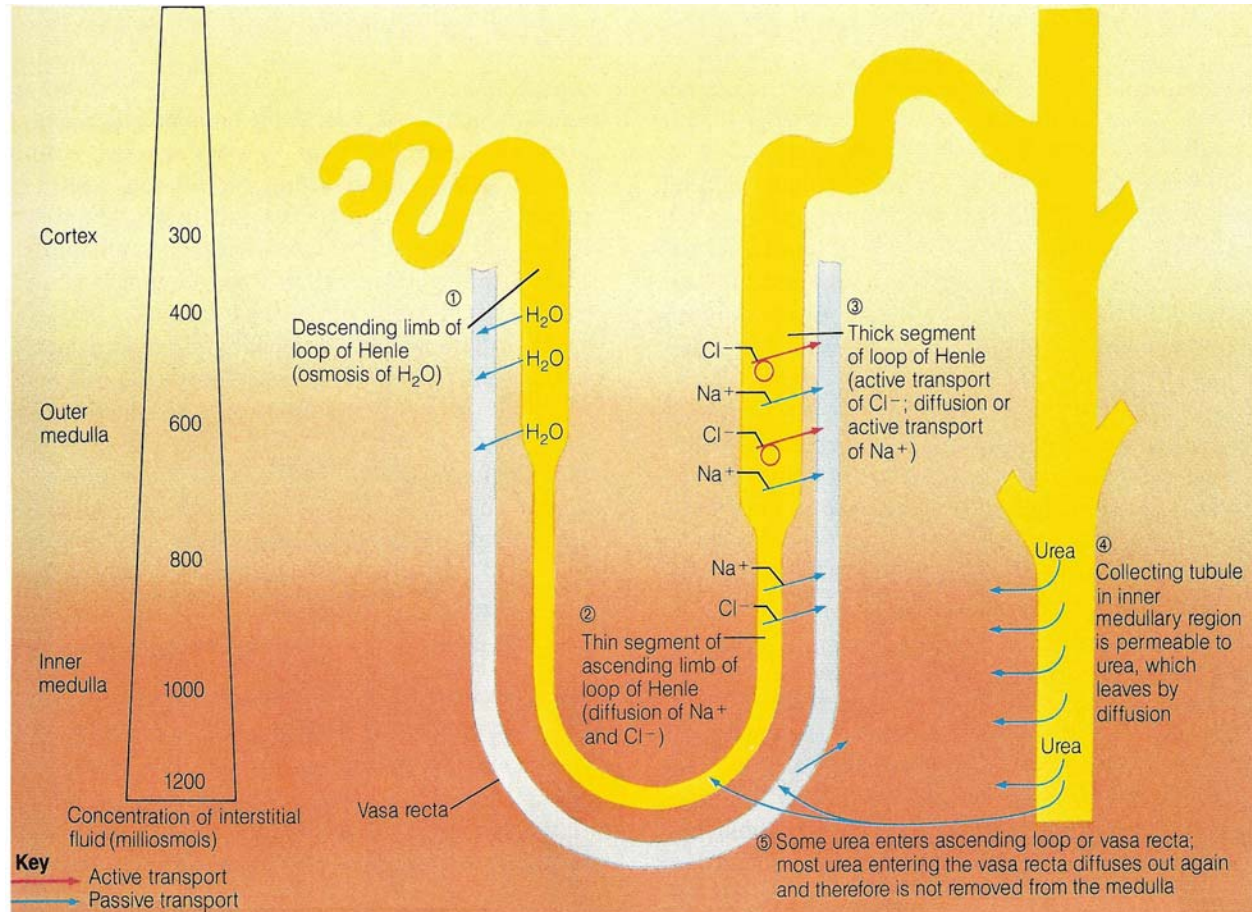
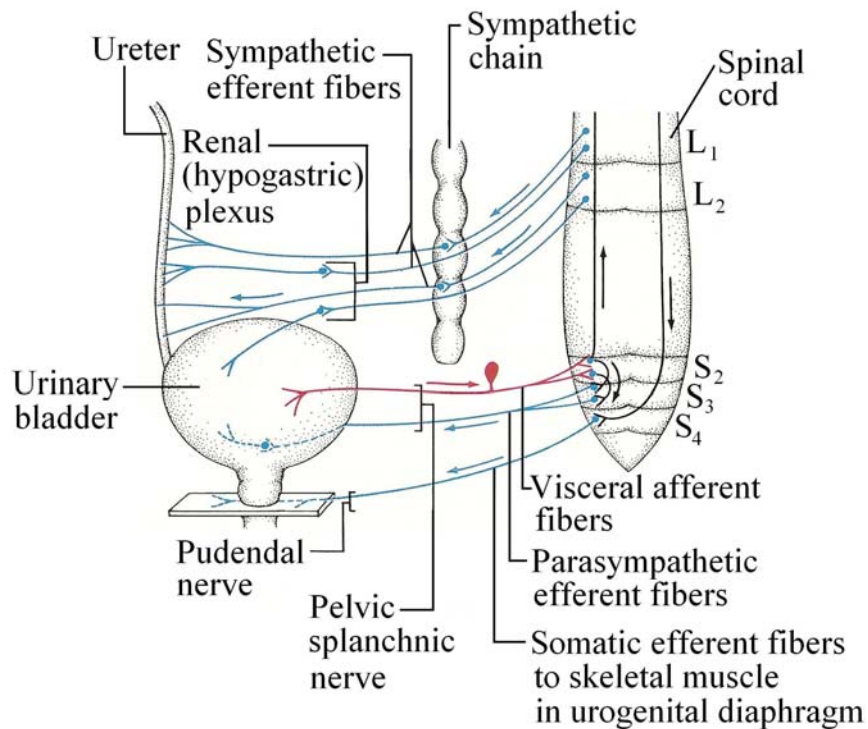


Figure 6.10. — The mechanism of establishing and maintaining the osmotic gradient (by Elaine N. Marieb, 1989)

1. The descending limb of the loop of Henle is relatively impermeable to solutes and freely permeable to water,
2. The thin segment of the ascending limb of the loop of Henle is freely permeable to sodium and chloride, poorly permeable to urea, and impermeable to water.
3. The thick segment of the ascending limb of the loop of Henle has an active transport mechanisms for chloride (and probably sodium) ions.
4. The collecting tubules in the deep medullary regions are permeable to urea.
5. The vasa recta removes very little urea, thus maintaining the osmotic gradient. Most of the urea that enters the vasa recta diffuses back out again.

If antidiuretic hormone is released, it courses the pores of the collecting tubules to enlarge so that water passes easily into the interstitial space. When the filtrate flows through the collecting tubules and is subjected to the hyperosmolar conditions in the medulla, water rapidly leaves the filtrate and exits the collecting tubules. Depending of the amount of antidiuretic hormone released (which is keyed to the level of body hydration), urine concentration may rise as high as 1200mOsm, the concentration of the interstitial fluid in the deepest part of the medulla.

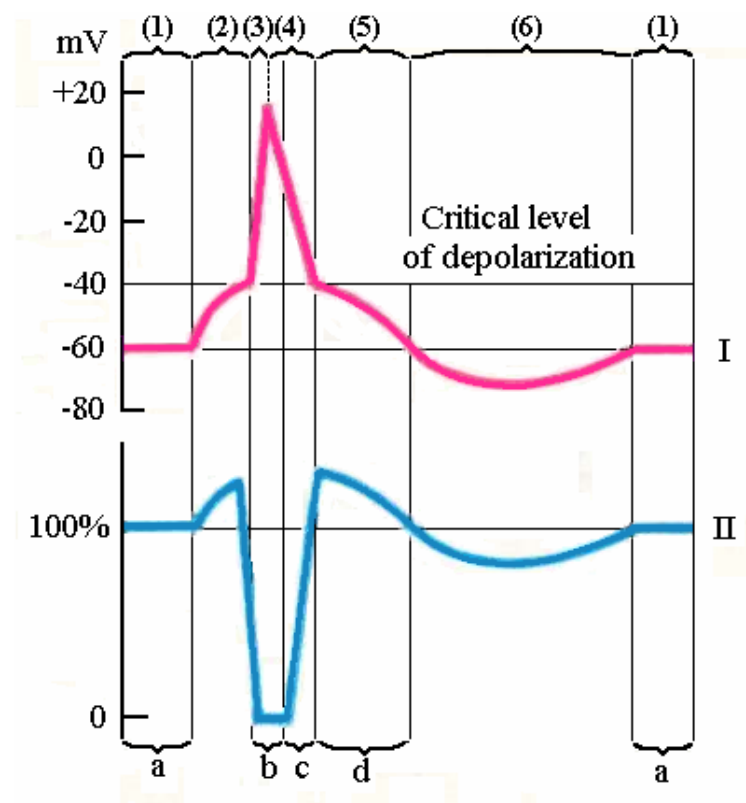


**Figure 6.11. — Micturition (urination) reflex arc pathway
(by Elaine N. Marieb, 1989)**

Stretching of the bladder wall by urine causes afferent impulses to be transmitted to the sacral region of the spinal cord. Efferent impulses are delivered to the bladder detrusor muscle and the internal sphincter via parasympathetic fibers of the pelvic nerves. The pudendal nerve serves the skeletal muscle fibers of the voluntary external sphincter (largely in the pelvic diaphragm). The role of sympathetic efferents is controversial.

Unite 7

PHYSIOLOGY OF EXCITABLE TISSUES



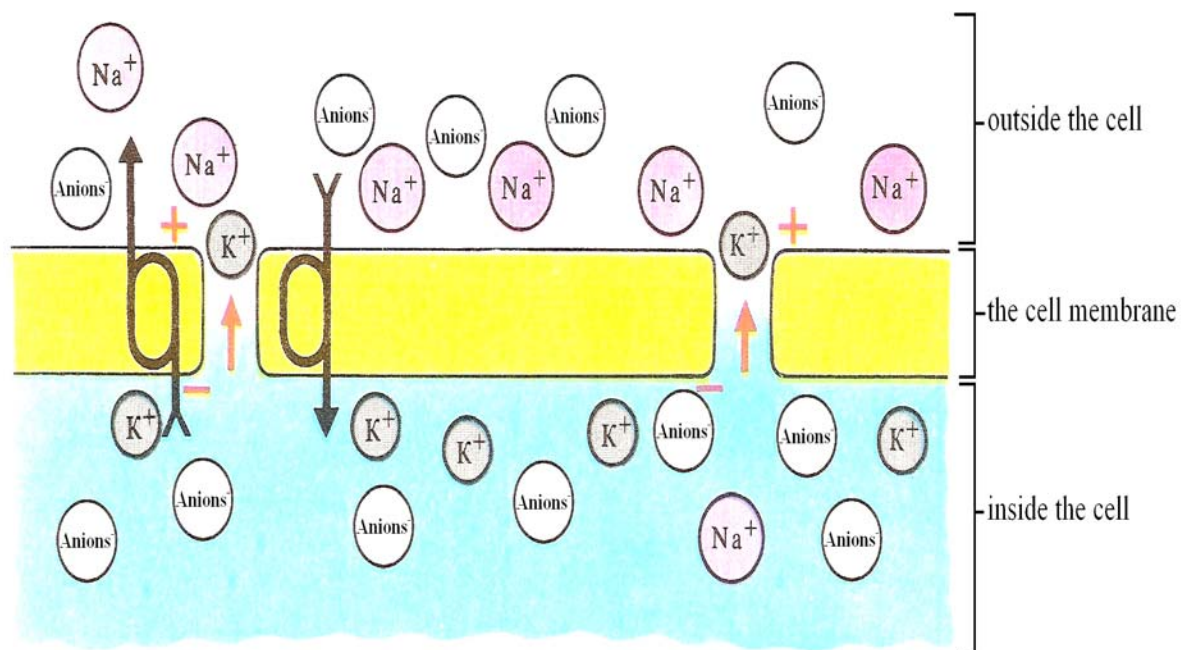


Figure 7.1. — Membrane resting potential
(by Korobkov A. V., Chesnokova S. A., 1986)

External surface of the cell membrane is charged positively «+» and the internal surface of the cell membrane - negative «-» The difference of charges between the external and internal membranes is called membrane resting potential.

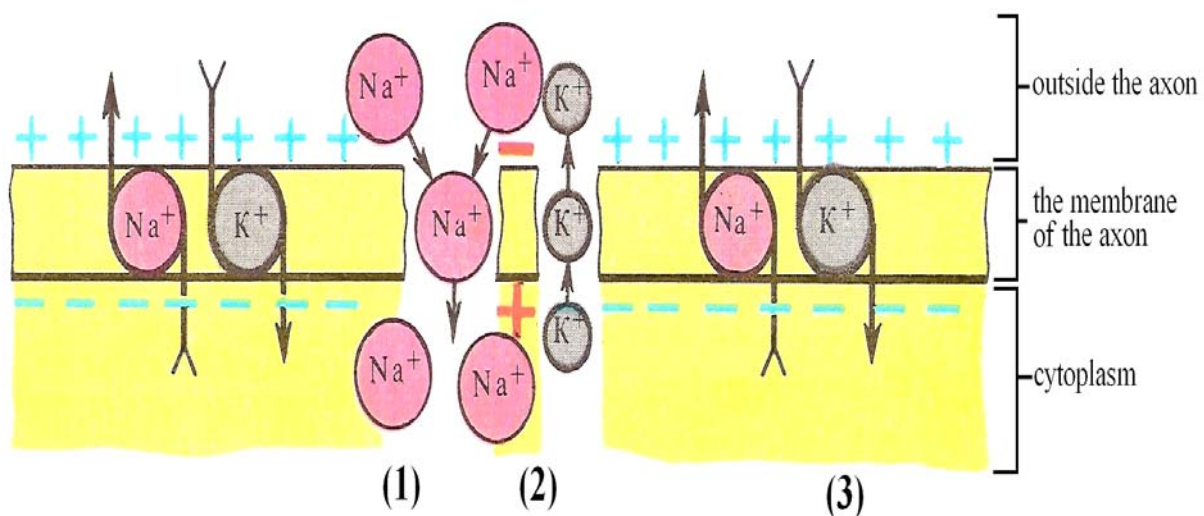


Figure 7.2. — Membrane action potential (by K. Kullande, 1968)

(1) Cell membrane becomes permeable for ions of Na^+ and they enter inside the cell. The internal surface of membrane becomes positive charged and the external surface of the membrane becomes negative charged. Change of charges on the internal and external surfaces of the membrane corresponds to depolarization phase.

(2) Sodium channels close, and potassium channels which have been partially closed, open. The ions of K^+ go out of the cell. This action potential phase is called repolarization.

(3) The action of $\text{Na}^+ - \text{K}^+$ -pump is restored.

Regeneration of membrane resting potential is observed.

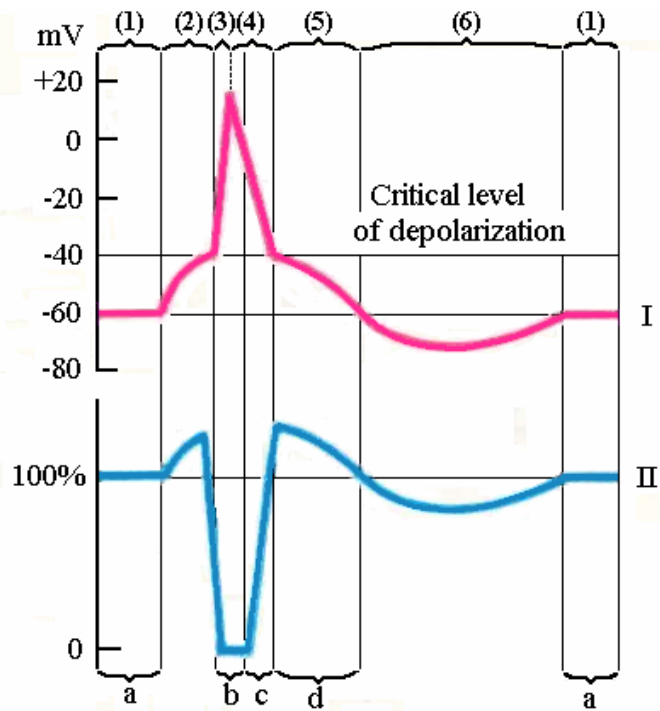


Figure 7.3. — Changes of the membrane potential and excitability during excitation (by Korobkov A. V., Chesnokova S. A., 1986)

I — Changes of the membrane potential:

(1) Membrane resting potential

Phases of membrane action potential:

(2) Slow depolarization;

(3) Fast depolarization.

(4) Fast repolarization;

(5) Slow repolarization

(6) Hyperpolarization

II — Changes of excitability:

(a) Normal excitability

(b) Absolute refractory period

(c) Relative refractory period

(d) Supernormal or exaltation period

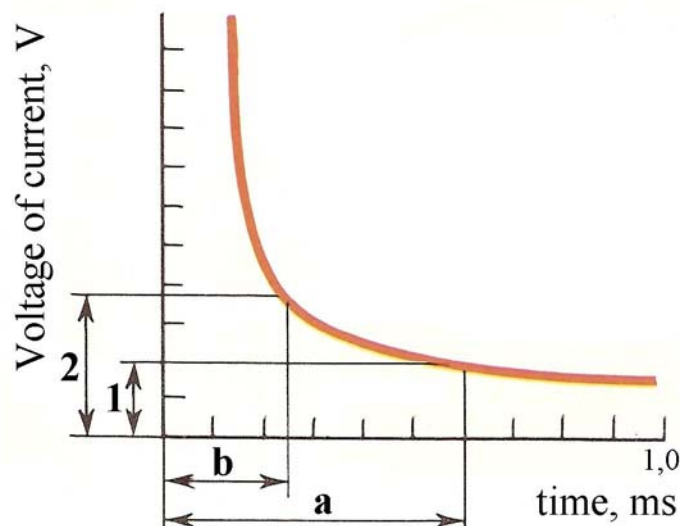


Figure 7.4. — The curve «force-duration» (by Lapić and others, 1926)

1 — rheobase; 2 — double rheobase;

a — available time; b — chronoxy

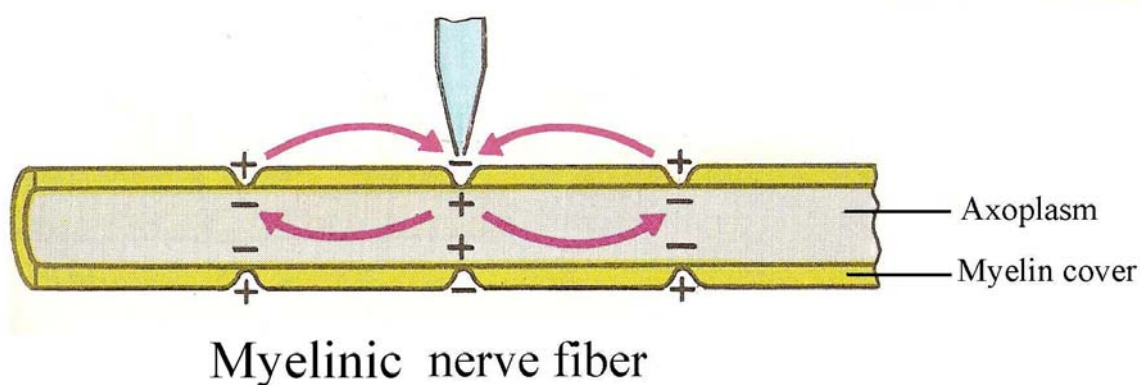
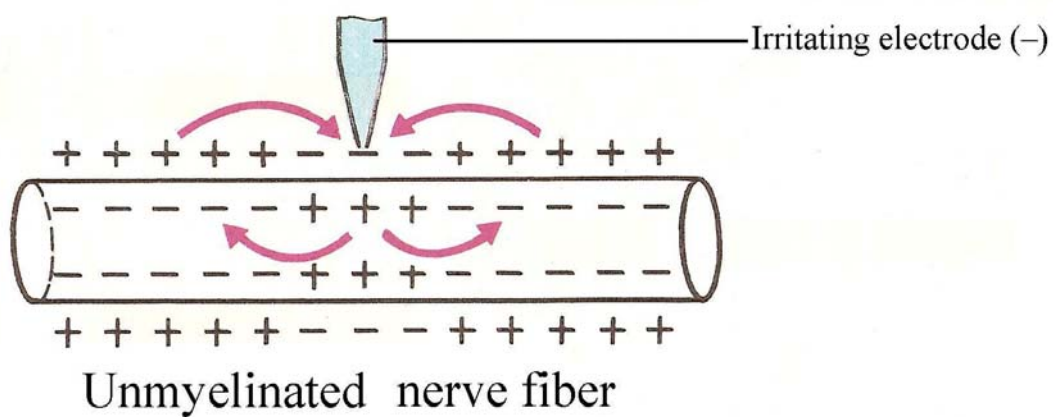
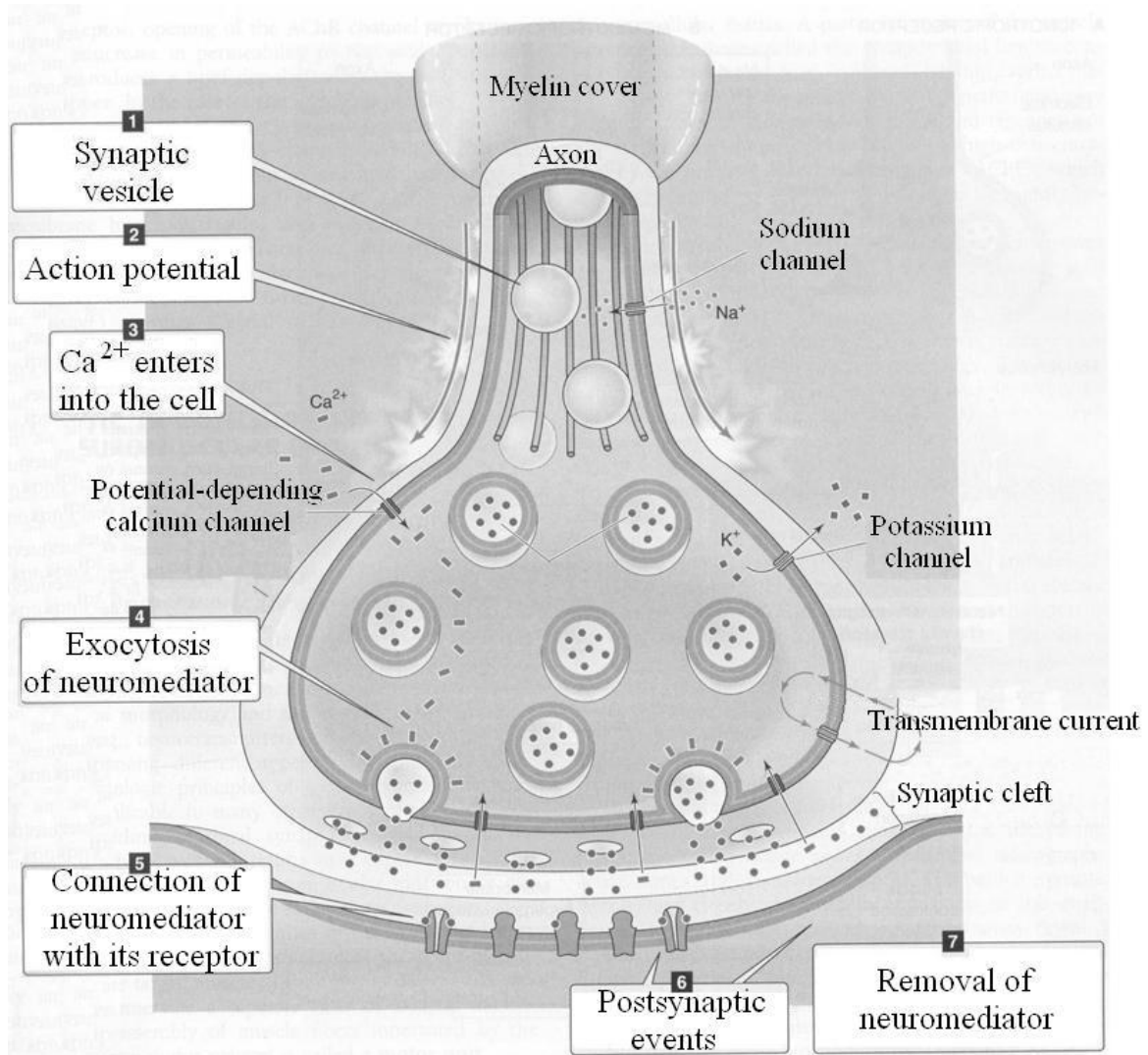


Figure 7.5. — Conduction of excitation in nerve fibers (G. Bendoll, 1970)

Table 7.1. — Properties of different nervous fibers

Type of fibers	Diameter, mcm	Speed of conduction, m/sec	Functions
Aα	13–22	70–120	Efferent fibers conduct excitation to skeletal muscles, afferent fibers conduct excitation from muscle receptors
Aβ	8–13	40–70	Afferent fibers conduct excitation from touch and tendinous receptors
Aγ	4–8	15–40	Afferent fibers conduct excitation from touch and pressure receptors, efferent fibers conduct excitation to skeletal spindles
B	1–3	3–14	Preganglionic fibers of vegetative nervous system
C	0.5–1.0	0.5–1.0	Postganglionic fibers of vegetative nervous system, afferent fibers conduct excitation from pain, temperature and pressure receptors

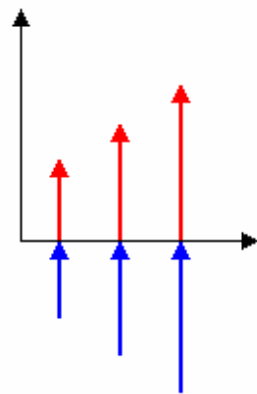


**Figure 7.6. — Stages of signal transmission in synapse
(by Orlov R.S., Nozdrachev A. D., 2005)**

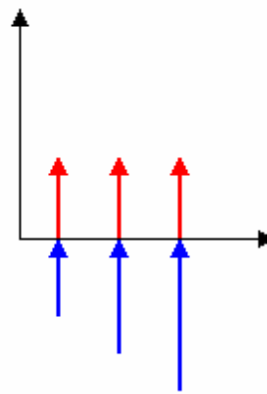
Classification of synapses

1. By the way of transmission:
 - chemical synapses;
 - electrical synapses.
2. By the type of excreted mediator chemical synapses are divided into:
 - adrenergic (adrenalin is mediator);
 - cholinergic (acetylcholine is mediator).
3. By effect:
 - excitants;
 - inhibitors.
4. By location:
 - axoaxonic synapse;
 - axosomatic synapse;
 - axodendritic synapse;
 - dendrodendritic synapse;
 - somatodendritic synapse.

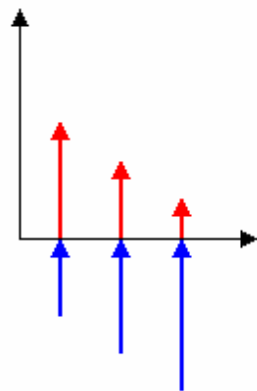
Normal response



Equaling phase



Paradoxical phase



Inhibitory phase

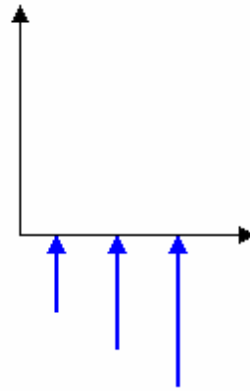


Figure 7.7. — Parabiosis (by Vvedensky)

The arrows above show the force of irritant (current)

The arrows below show the force of response (the force of muscular contraction)

The state of decreasing of lability is called parabiosis. There are 3 phases of parabiosis:

1. **Equaling phase.** If to irritate nerve fibril with irritants of various force (weak and strong), the response of a muscle will be always identical.
2. **Paradoxical phase.** In this case strong irritants induce weak response, weak irritants — on the contrary — strong one.
3. **Inhibitory phase.** Both strong and weak irritants cannot induce response.

In the human organism muscular tissue according to structure and physiological properties is divided into 3 types:

1. Skeletal.
2. Unstriated.
3. Cardiac.

All types of muscles possess some properties:

1. Excitability.
2. Conduction.
3. Contractility, — change of length or strain, — and ability to relaxed.

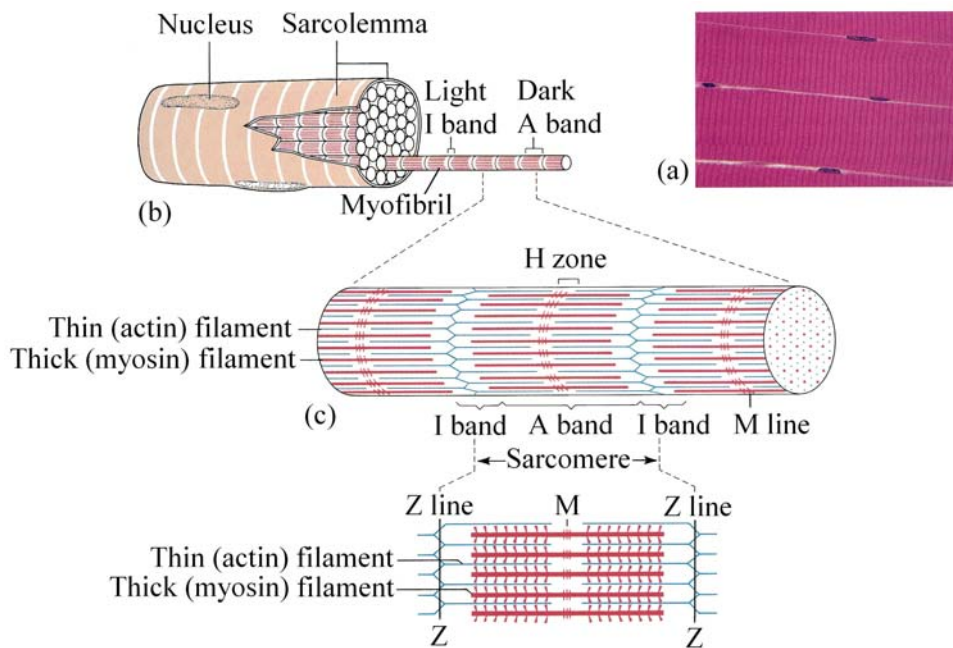


Figure 7.8. — Microscopic anatomy of a muscle fiber
(by Elaine N. Marieb, 1989)

- (a) Photomicrograph of two isolated skeletal muscle cells (250x).
- (b) One myofibril is shown extending from the cut end of a muscle fiber.
- (c) A small portion of one myofibril is enlarged to show the myofilaments responsible for the banding pattern. Each sarcomere, or contractile unit, extends from one Z line to the next.

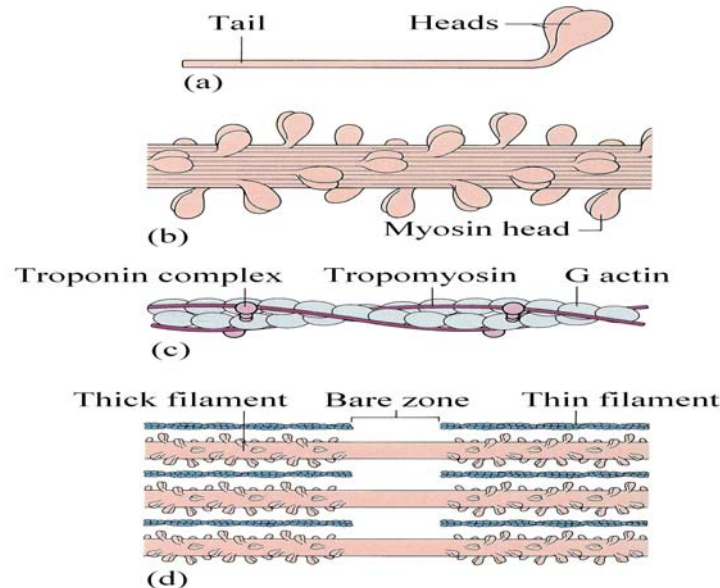


Figure 7.9. — Myofilament composition in skeletal muscle
(by Elaine N. Marieb, 1989)

- (a) An individual myosin molecule has a stalklike tail, from which two «heads» protrude.
- (b) Each thick filament consists of many myosin molecules.
- (c) A thin filament contains two strands of F actin twisted together. Each strand is made up of G actin subunits. Tropomyosin molecules coil around the F actin. A troponin complex is attached to each tropomyosin molecule.
- (d) Arrangement of the filaments in a sarcomere (longitudinal view).

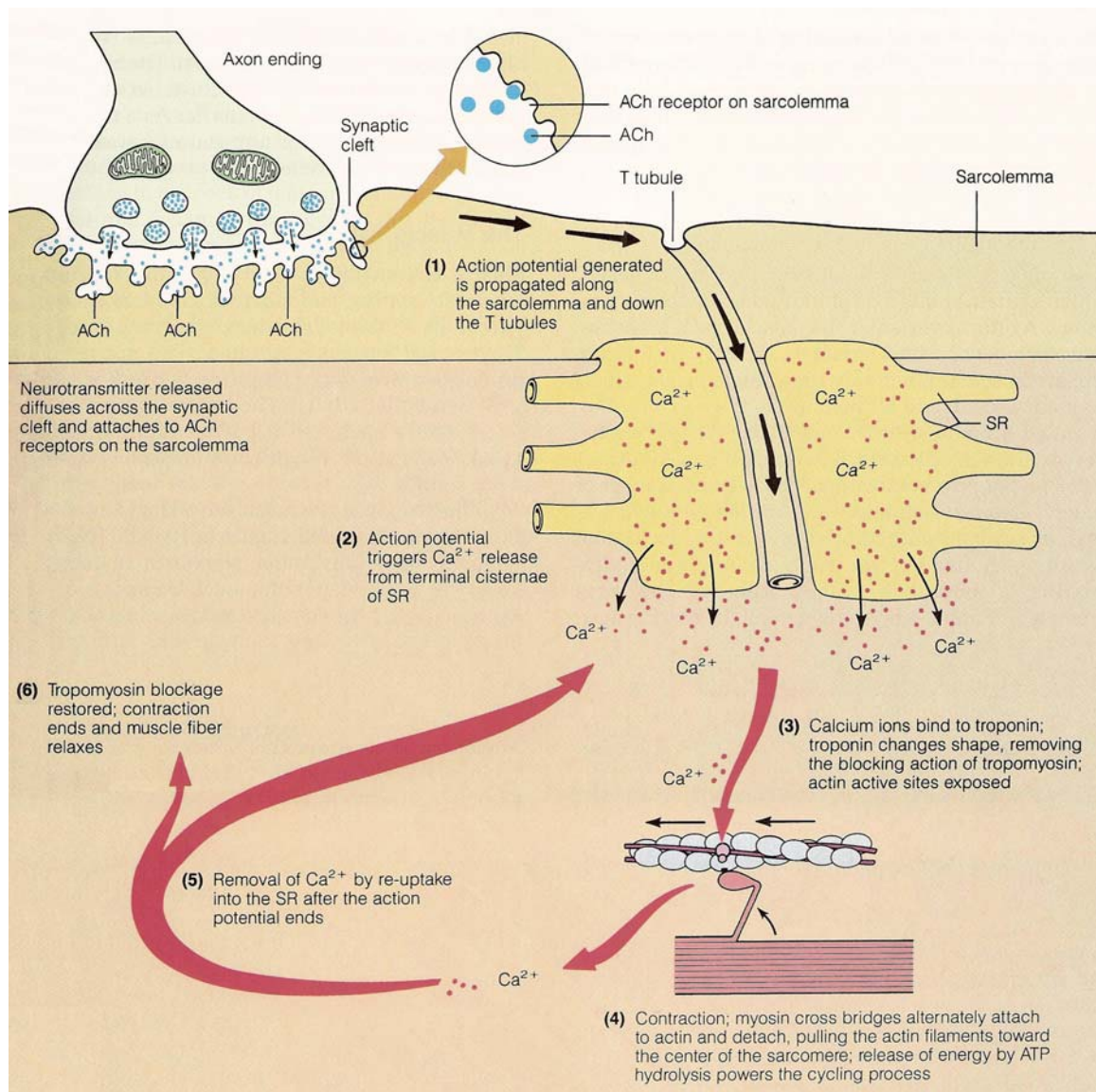


Figure 7.10. — Sequence of events in excitation-contraction coupling
(by Elaine N. Marieb, 1989)

Events (1) through (5) indicate the sequence of events in the coupling process. As shown in the flow of events to the left, contraction continues until the calcium signal ends.

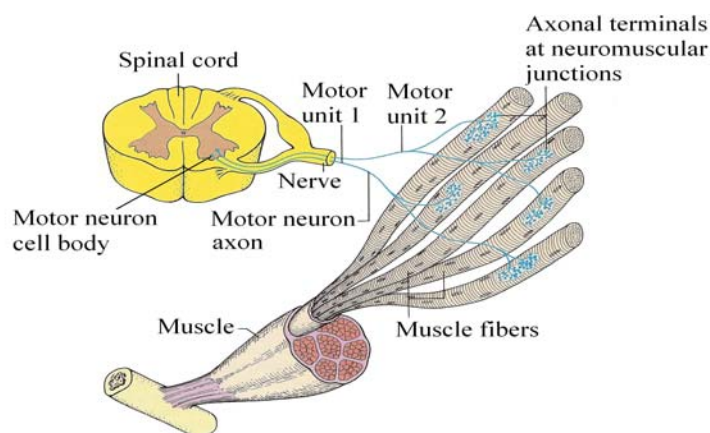


Figure 7.11. — Motor units
(schematic view of portions of two motor units)
(by Elaine N. Marieb, 1989)

The cell bodies of the motor neurons reside in the spinal cord, and their axons extend to the muscle. Within the muscle, each axon divides into a number of axonal terminals, which are distributed to muscle fibers scattered through the muscle.

Each motor unit consists of a motor neuron and all of the muscular fibers it innervates.

Table 7.2. — Forms and types of muscle contractions

Form	Type	Characteristics of the type	Example
Dynamic form	Isotonic type	Muscle is shortened but does not change its strain	Walking
	Eccentric type	If a load on a muscle is more than its strain, the muscle is stretched, but does not change its strain	Lowering of a heavy object
Static form	Isometric type	Muscle changes its strain but does not change length	Maintenance of a posture or overcoming of terrestrial attraction.
Auxotonic or mixed	—	Muscle change its strain and length	Most of contractions are mixed.

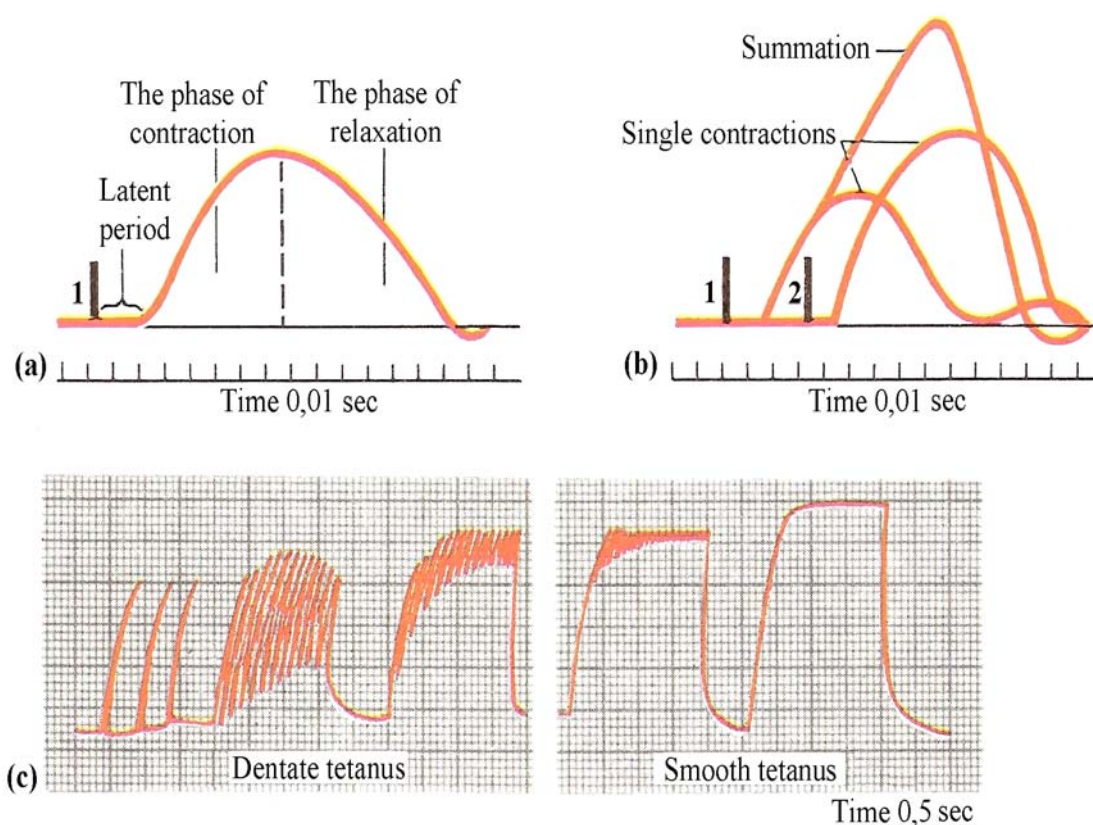


Figure 7.12. — Single muscle contraction (a), summation (b), tetanus (c)
(by Korobkov A.V., Chesnokova S.A., 1986)

1 — the moment of giving of the first irritant signal
2 — the moment of giving of the second irritant signal

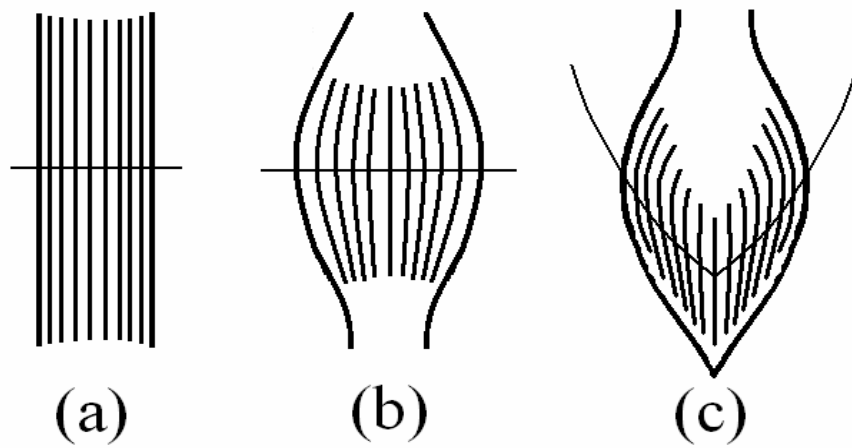
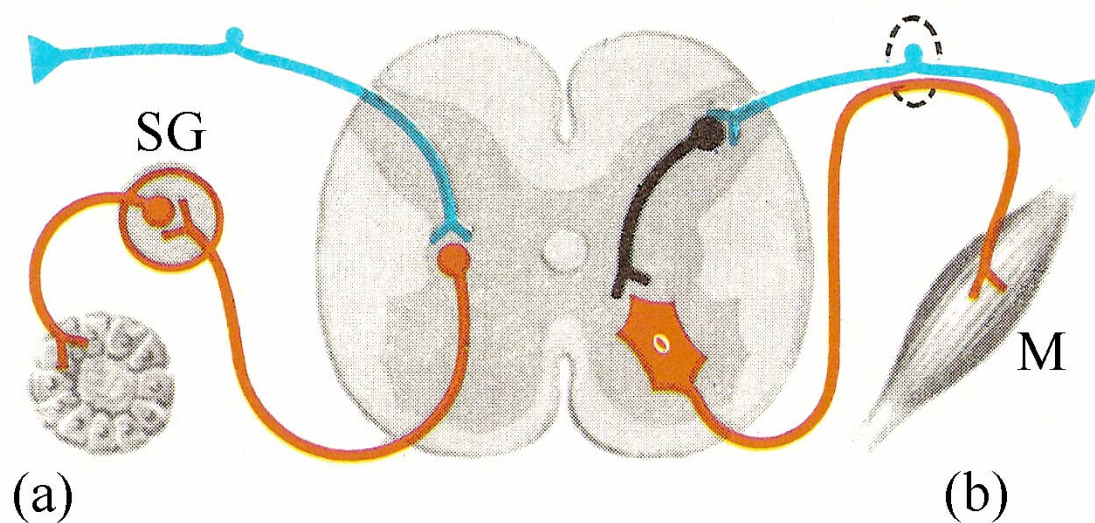


Figure 7.13. — The structure of different types of muscle and their physiological cross section (by Pokrovskiy V. M., Korotko G. F., 2000)
 (a) Sartorius muscle; (b) Biceps brachii; (c) Gastrocnemius muscle

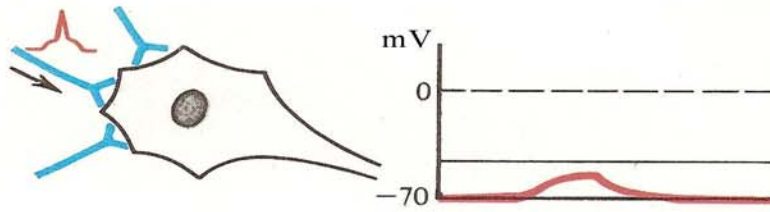
Unite 8

GENERAL AND PARTICULAR PHYSIOLOGY OF CENTRAL NERVOUS SYSTEM

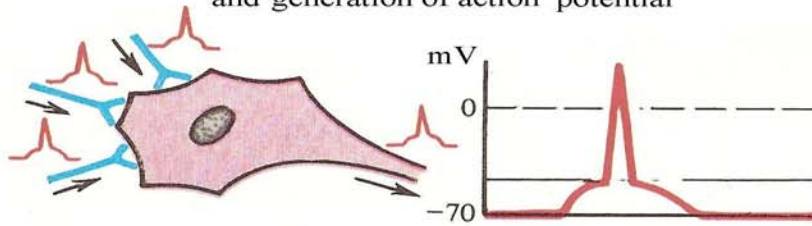


Space summation
(the irritant signals come simultaneously)

(a) Transmission of excitation from one axon
(decreasing of membrane potential)

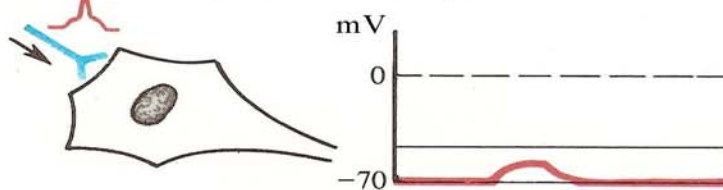


(b) Transmission of excitation from three axons
and generation of action potential

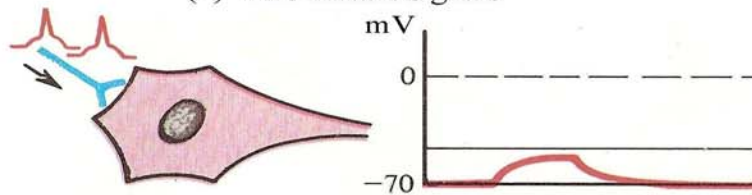


Time summation
(the irritant signals come successively)

(a) One irritant signal



(b) Two irritant signals



(c) Three irritant signals
and generation of action potential

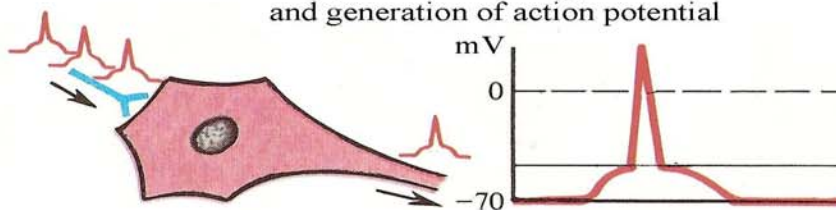


Figure 8.1. — Space and time summation in the nerve system
(by Korobkov A. V., Chesnokova S. A., 1986)

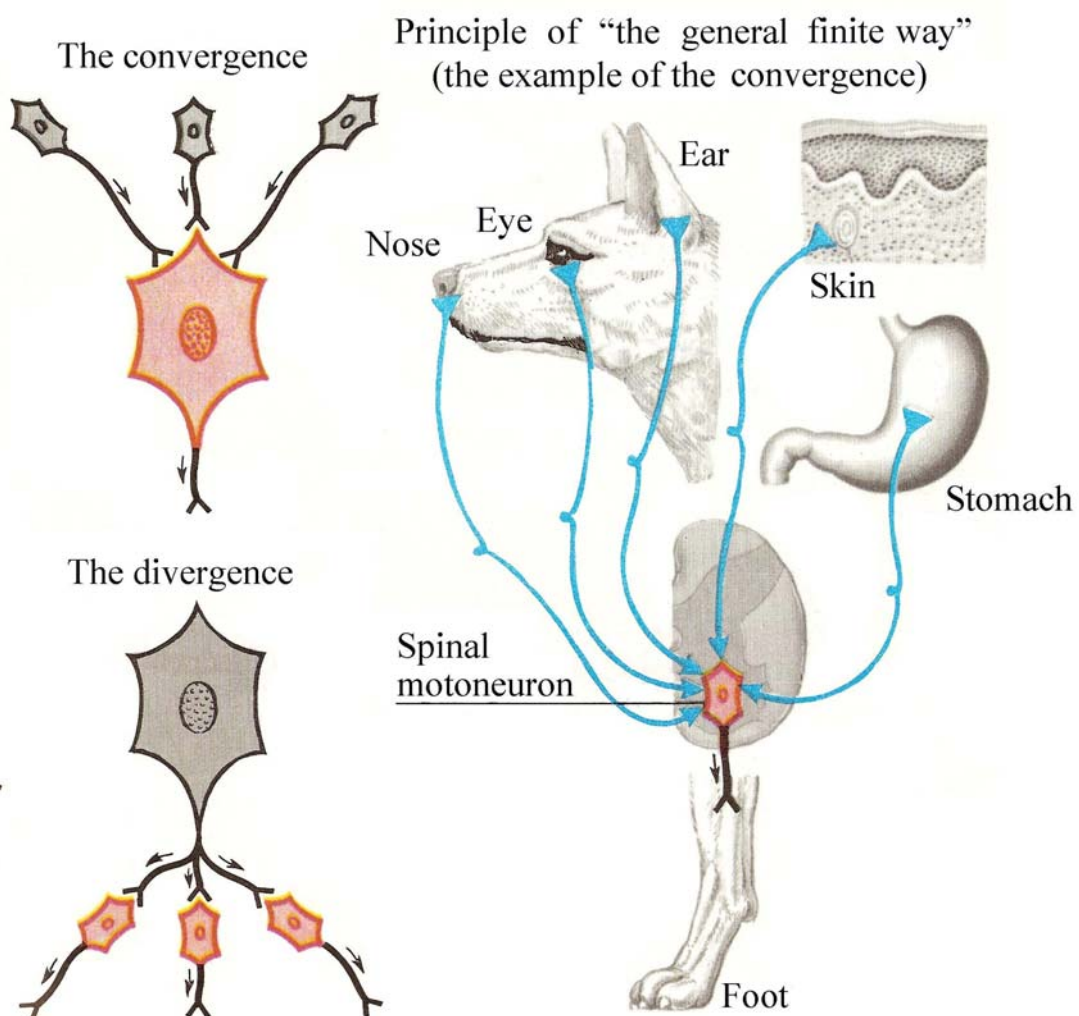


Figure 8.2. — Types of spreading of excitation in nerve system
(by Korobkov A. V., Chesnokova S. A., 1986)

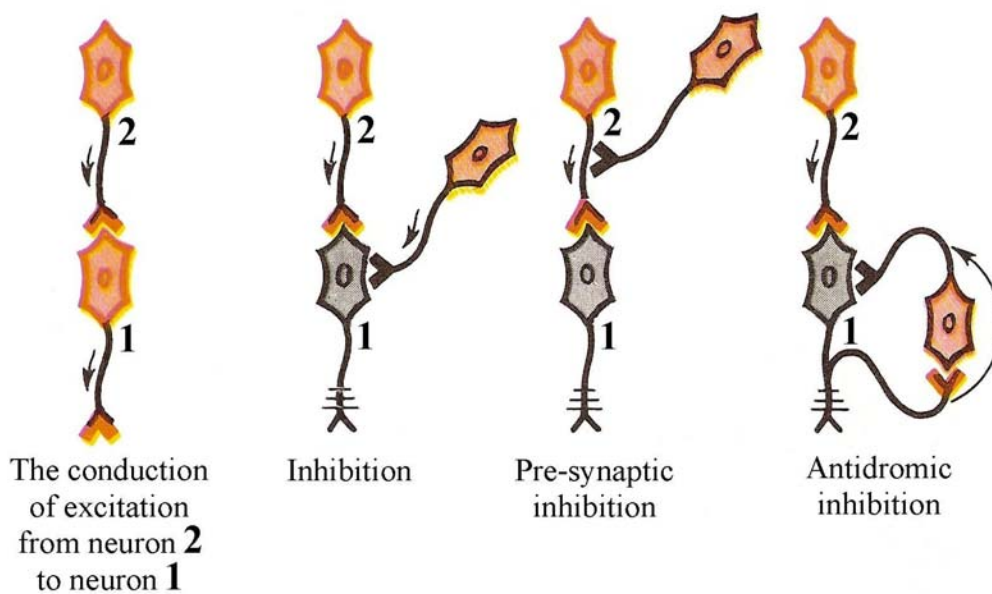


Figure 8.3. — Types of inhibition in the central nervous system
(by Korobkov A. V., Chesnokova S. A., 1986)

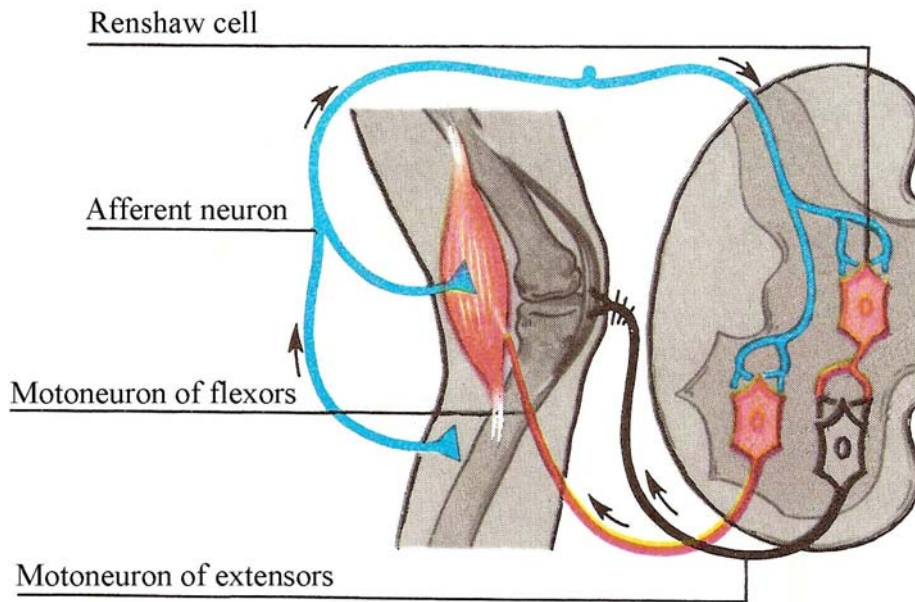


Figure 8.4. — Reciprocal inhibition (by Sherrington, 1897)

Reciprocal inhibition: the same afferents which stimulate one group of cells, through intercalary neurons generate inhibition of other group of cells. The example: excitation of an afferent simultaneously causes excitation of the motoneuron of one muscle group (flexors) and reciprocal inhibition of the motoneuron of antagonist muscles (extensors). It happens because afferents branch in spinal cord, one of branches stimulate motoneurons of flexors, other form inhibition synapses on motoneurons of extensors (through the Renshaw cell).

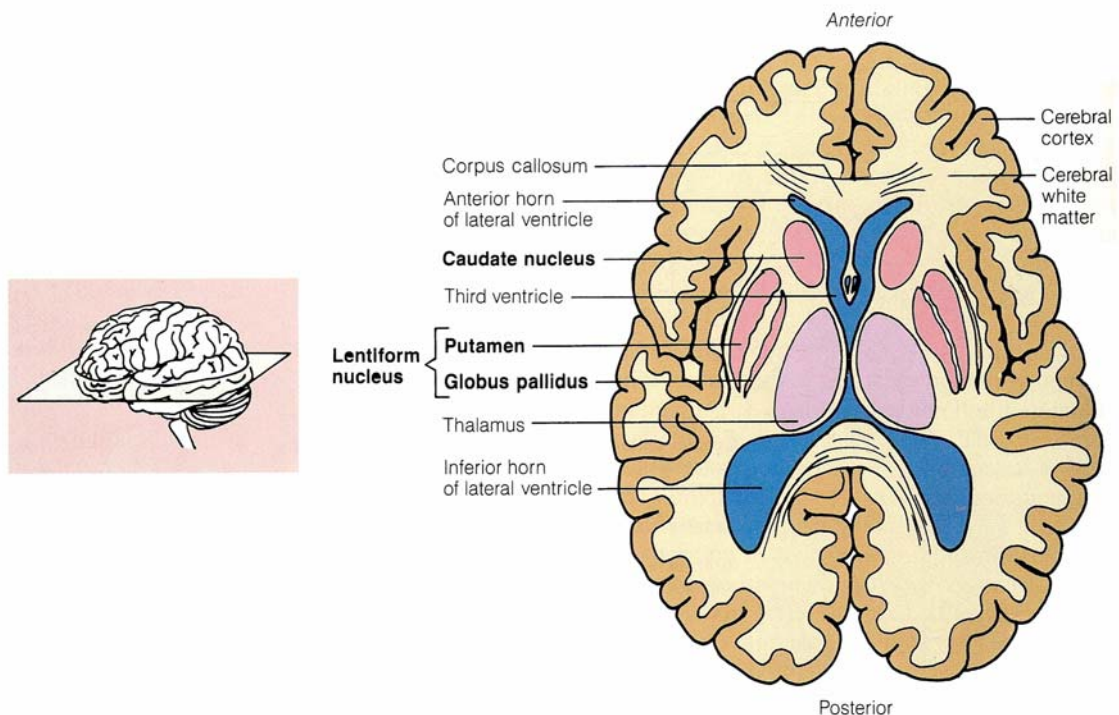


Figure 8.5. — A transverse section of the cerebrum and diencephalon showing the relationship of the basal nuclei to the thalamus and the lateral and third ventricles (by Elaine N. Marieb, 1989)

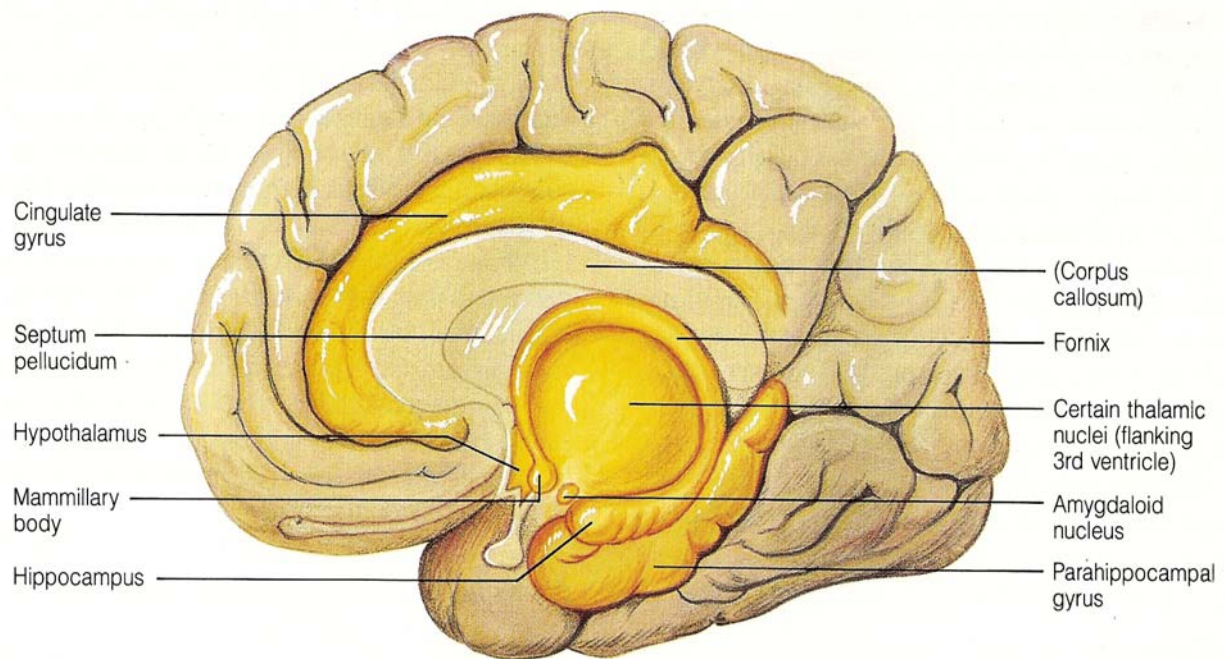


Figure 8.6. — Medial view of the brain, showing some of the structures that constitute the limbic system, the emotional-visceral brain (by Elaine N. Marieb, 1989)

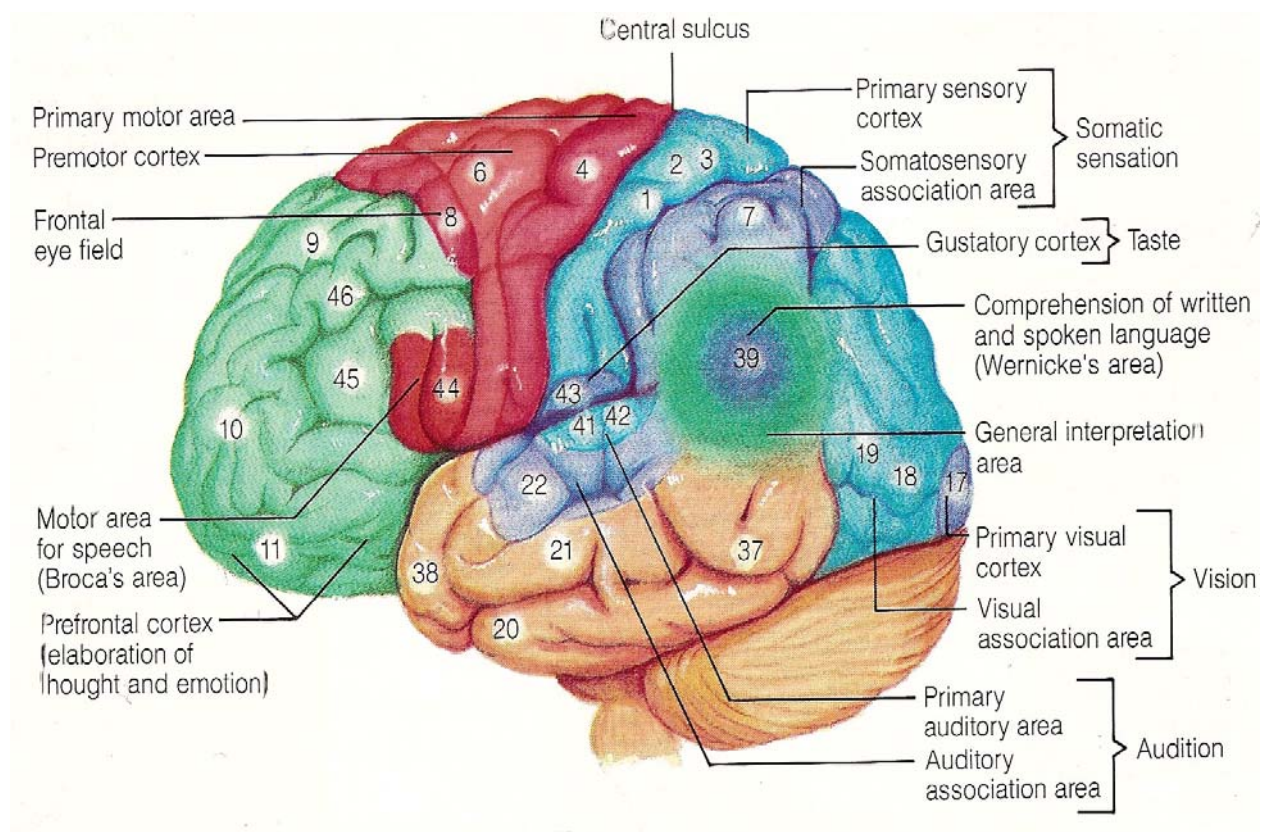


Figure 8.7. — Functional areas of the left cerebral cortex (by Elaine N. Marieb, 1989)

The olfactory area, which is deep to the temporal lobe on the medial hemispheric surface, is not identified. Numbers indicate regions plotted by the Brodman system.

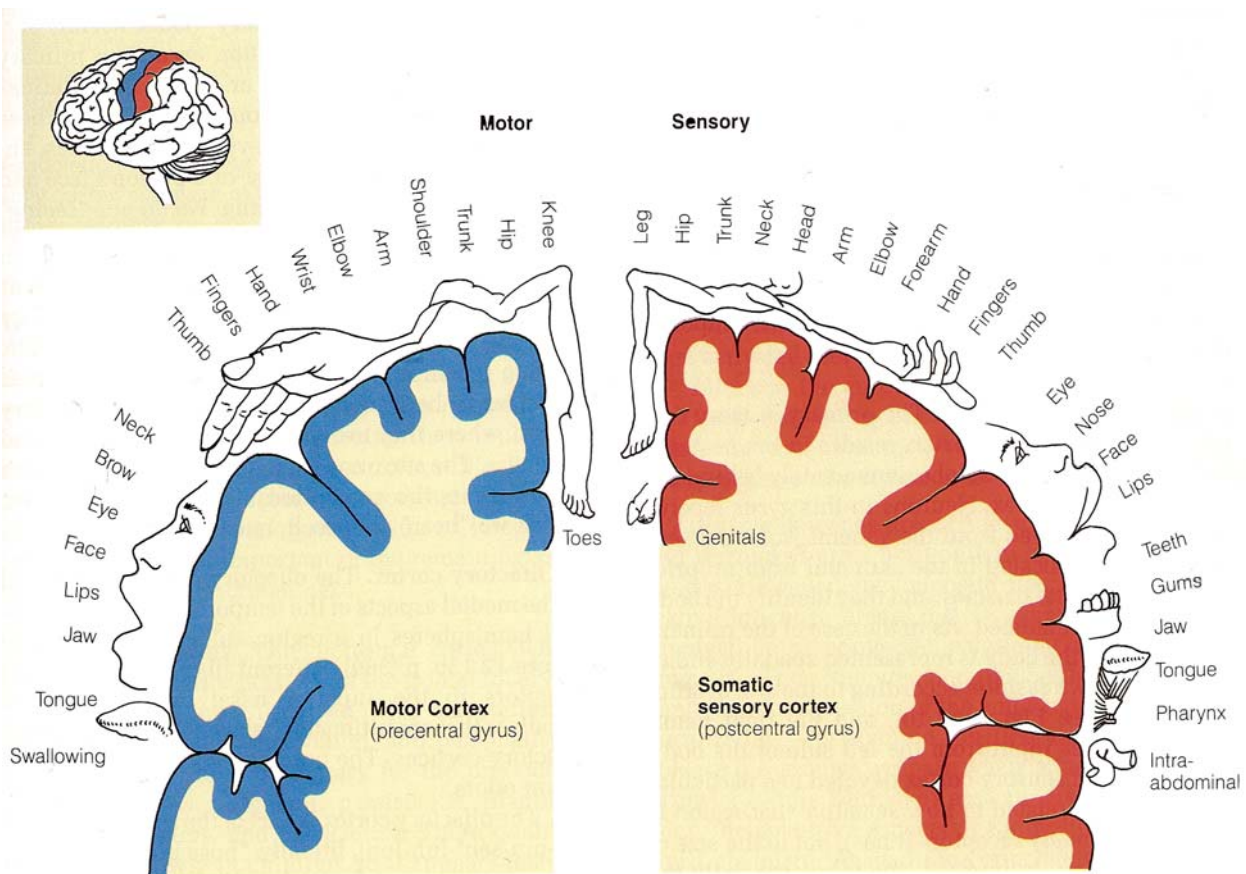


Figure 8.8. — Sensory and motor areas of the cerebral cortex
(by Elaine N. Marieb, 1989)

The relative amount of cortical tissue devoted to each function is indicated by the amount of the gyrus occupied by the body area diagrams. The primary motor cortex is represented on the left, and the somatic sensory cortex on the right.

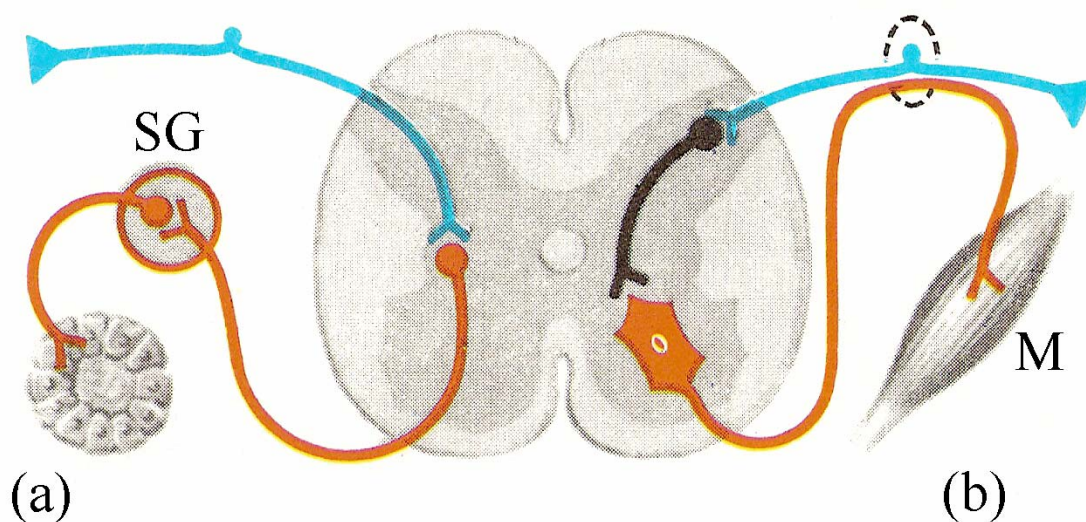


Figure 8.9. — Reflex arch
(by Korobkov A. V., Chesnokova S. A., 1986)

- (a) — reflex arch of the vegetative reflex
- (b) — reflex arch of the somatic reflex
- SG — sympathetic ganglion; M — muscle

Table 8.1. — Differences of sympathetic department of vegetative nervous system from parasympathetic

Principle of difference	Sympathetic department	Parasympathetic departments
The localization of nerve centers in the brain	Thoracal-lumbar department of the spinal cord	Midbrain, medulla, sacral department of spinal cord
Location of vegetative ganglions	Vertebral and prevertebral ganglions	Intramural plexuses
The excreted mediator	The main mediator is noradrenalin	The main mediator is acetylcholine
Influence on the function of organs	Ensures the power supply of an organism (influences on the redistribution of blood flow, increases the heart rate, increases metabolism, increases the level of glucose in blood)	Corrects changes in the organism induced by sympathetic nervous system (restores and maintains homeostasis)

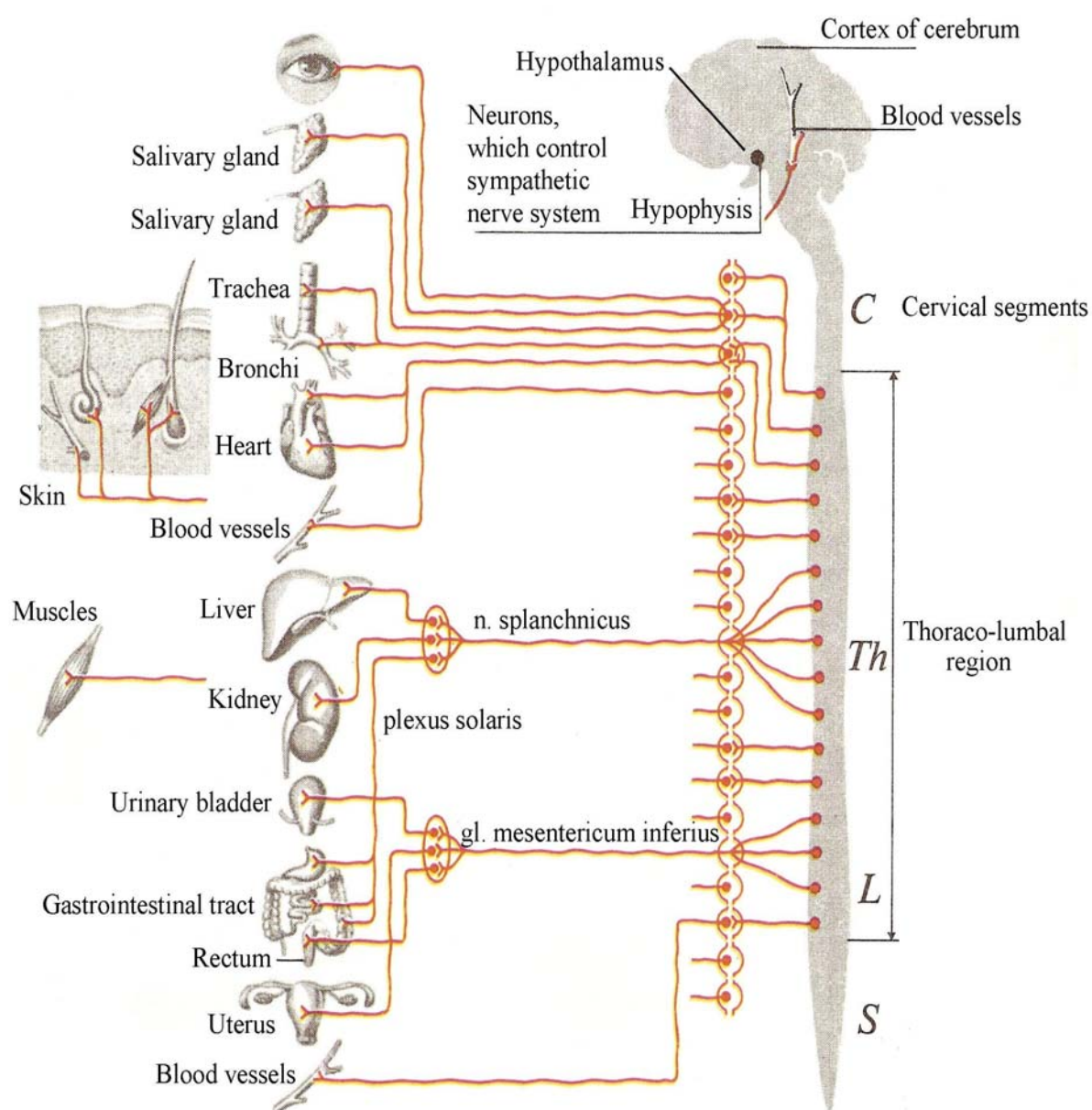


Figure 8.10. — General plan of the structure of sympathetic nerve system (by Korobkov A. V., Chesnokova S. A., 1986)

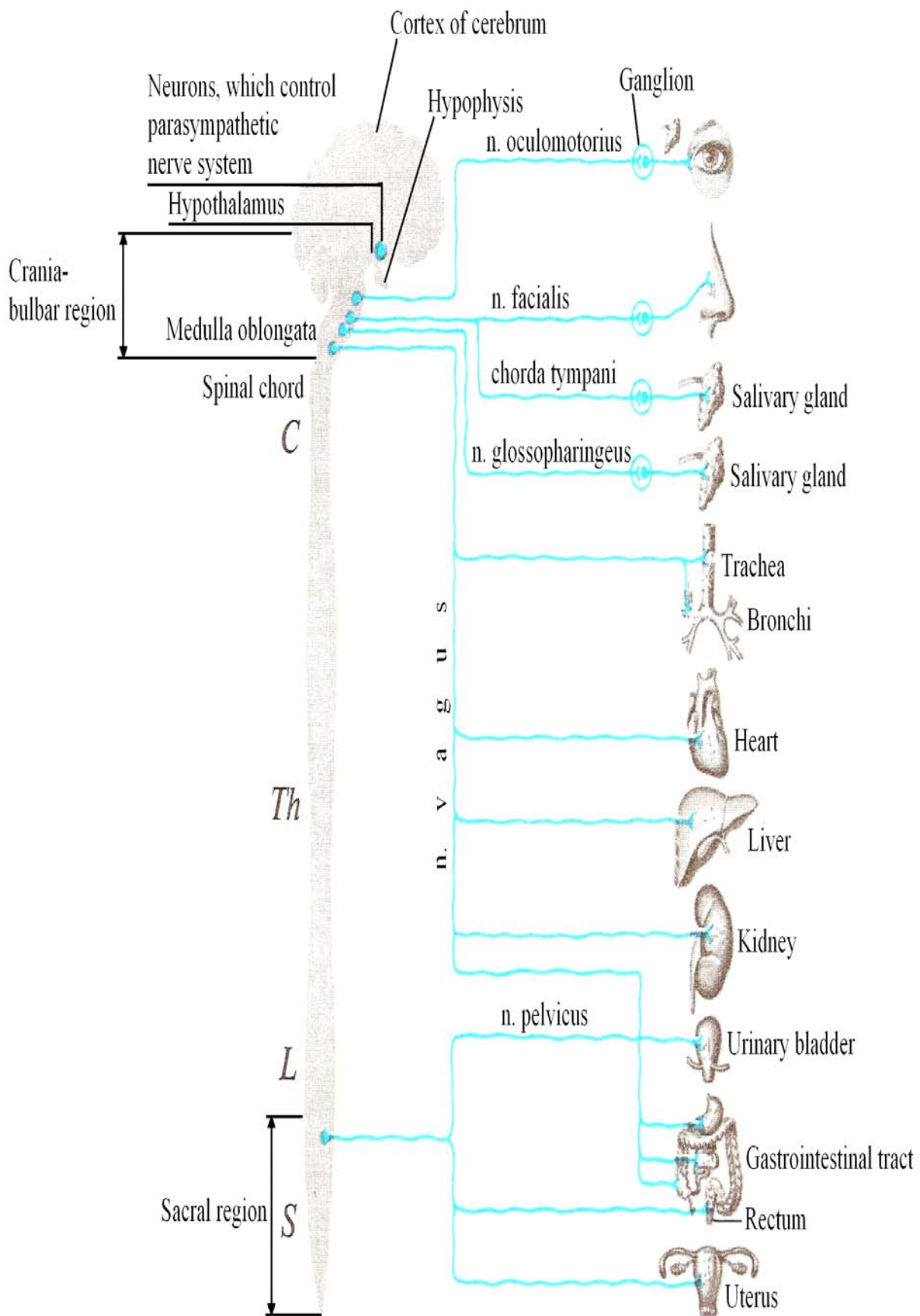
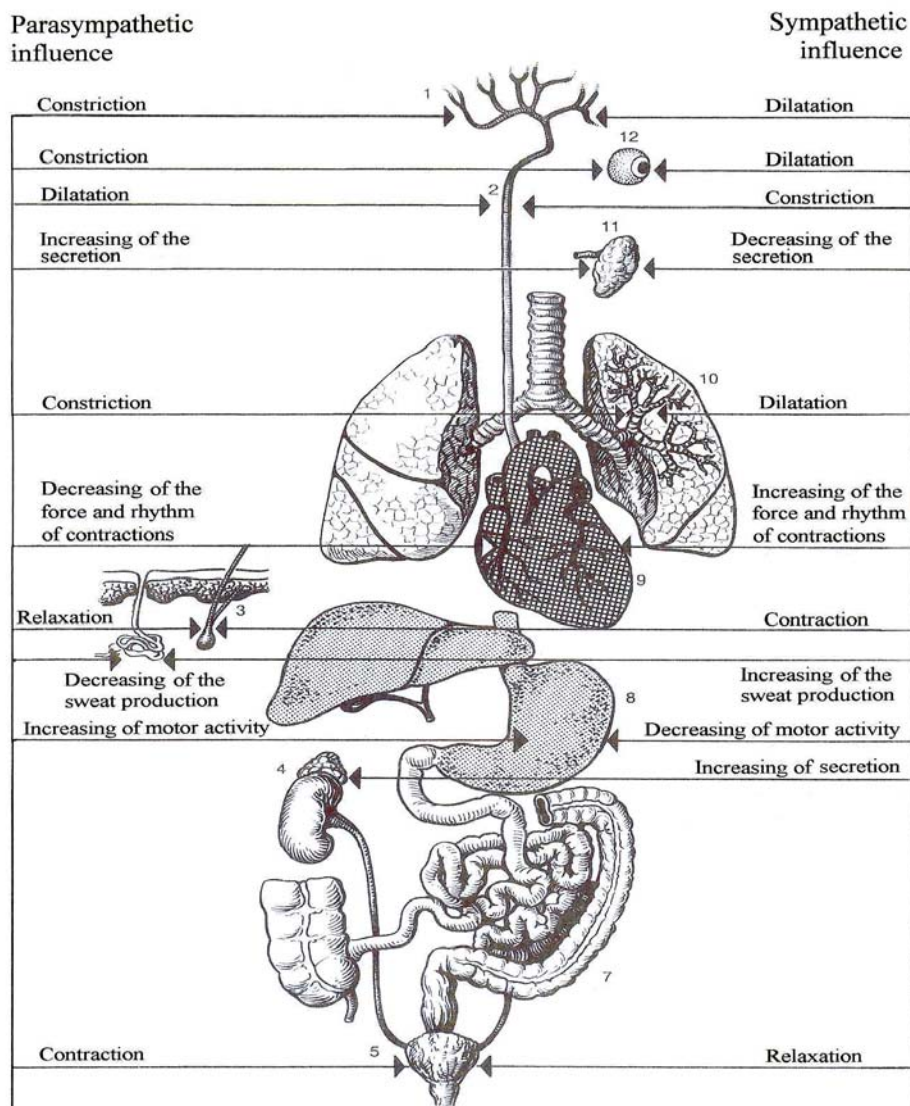
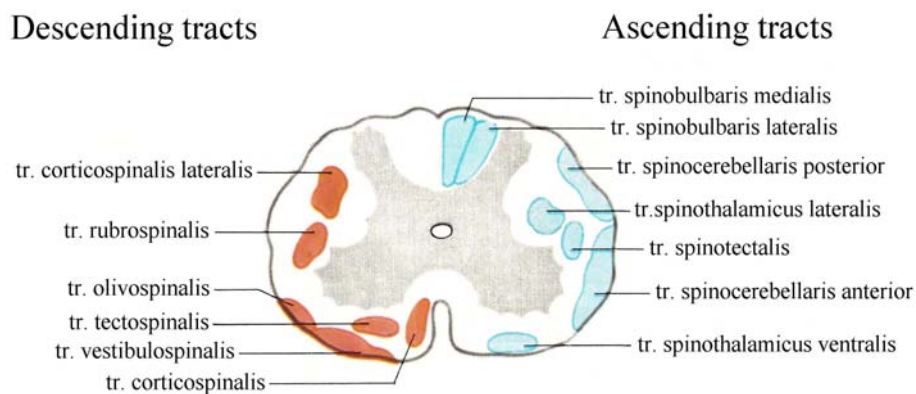


Figure 8.11. — General plan of the structure of parasympathetic nerve system
(by Korobkov A. V., Chesnokova S. A., 1986)



1 – vessels of brain, 2 – periphery vessels, 3 – hair muscle, 4 – suprarenal gland, 5 – urinary bladder, 7 – intestines, 8 – stomach, 9 – heart, 10 – bronchi, 11 – salivary glands, 12 – pupil

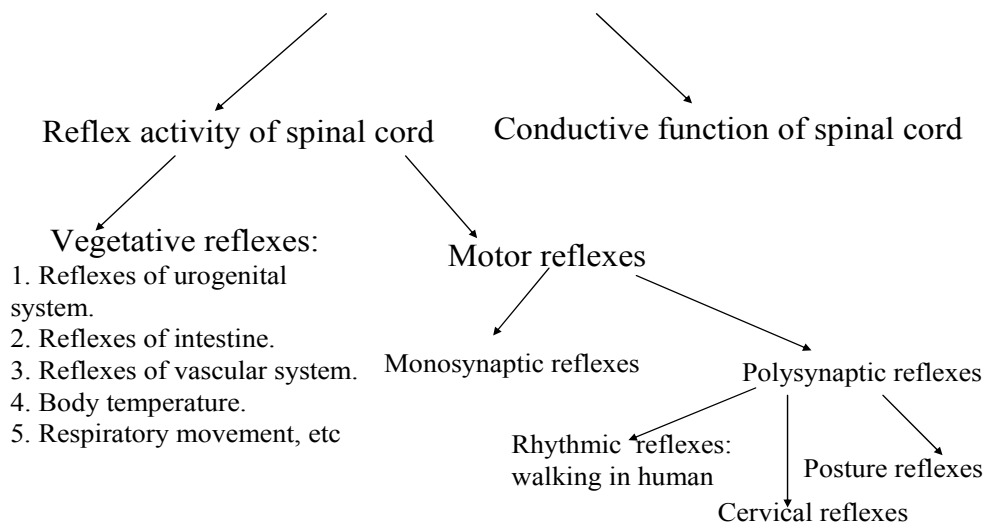
Figure 8.12. — Changing of functions of different organs at irritation of sympathetic and parasympathetic nerve fibrils, which innervate them



**Figure 8.13. — Conductive pathways of spinal cord
(by Korobkov A. V., Chesnokova S. A., 1986)**

Picture shows the cross- section of spinal chord.

Functions of spinal cord



Scheme 8.1. — Functions of spinal cord

Table 8.2. — The basic conductive pathways

Conductive tracts	Columns of spinal cord	Physiological importance
I. ascending (sensory) tracts		
1. Tract of Goll	posterior	Touch sensibility, sense of body position and passive movements, sense of vibration
2. Wedge-shaped fascicle of Burdach	-//-	-//-
3. Dorsolateral tract	lateral	Tracts of pain and temperature sensitivity
4. Dorsal spinocerebellar tract of Flexig	-//-	Impulses from muscle proprioceptors, receptors of ligaments and tendons
5. Ventral spinocerebellar tract of Govers	-//-	-//-
6. Dorsal spinothalamic tract	-//-	Pain and temperature sensitivity
7. Spinotectal tract	-//-	Sensory tract of visual motor reflexes and pain sensitivity
8. Ventral spinothalamic tract	front	Tactile sensitivity
II. Descending (motor) tracts		
1. Lateral corticospinal tract (pyramidal)	lateral	Impulses to skeletal muscles Arbitrary movements.
2. Rubrospinal tract	-//-	Impulses supporting the tone of skeletal muscles.
3. Dorsal vestibulospinal tract	-//-	Impulses providing pose keeping and body equilibrium.
4. Olivospinal tract	-//-	Unknown function.
5. Reticulospinal tract	front	Impulses support the tone of skeletal muscles, regulate condition of spinal vegetative centers and sensitivity of muscle spindles.
6. Ventral vestibulospinal tract	-//-	Impulses providing pose keeping and body equilibrium.
7. Tectospinal tract	-//-	Impulses provide optic and acoustic reflexes.
8. Ventral corticospinal tract (pyramidal)	front	Impulses to skeletal muscles Arbitrary movements.

Table 8.3. — Function characteristic of cranial nerves

Name of a cranial nerve	Effect of action	Function
XII n. hypoglossus	motor	Innervates muscles of tongue.
XI n. accessorius	motor	Innervates muscles of neck.
X n. vagus	mixed	Afferent information goes from internal organs. Innervates muscles of gullet and larynx, internal organs, heart.
IX n. glossopharyngeus	mixed	Innervates muscles of gullet.
VIII n. vestibulocochlearis	sensory	Afferent information goes from vestibular and ear apparatus.
VII n. facialis	mixed	Afferent information goes from gustatory receptors of tongue. Innervates mimic muscles.
VI n. abducens	motor	Innervates eye muscles.
V n. trigeminus	mixed	Afferent information goes from mucous membrane of nose, teeth, tongue. Innervates masticatory muscles and muscle stressing tympanic membrane.
IV n. trochlearis	motor	Innervates eye muscles.
III n. oculomotorius	motor	Innervates eye muscles.
II n. opticus	sensory	Innervates retina.
I n. olfactorii	sensory	Innervates mucous membrane of nose

Table 8.4. — Characteristics of cerebellar deficiency

Type of disorder	Characteristics
Asthenia	Increased fatigability of muscles, decreased force of muscle contraction.
Astasia	Impossibility to stay quiet (constant tremor of head)
Dystonia	Involuntary increase or decrease of muscle tone
Tremor	Small by amplitude fluctuation movement arising synchronously in different parts of the body
Dysmetria	Disturbances of evenness movements
Hypermetria Hypometria	Patient carries the hand by an object which is tried to take. Patient does not carry the hand to an object which is tried to take.
Astasia	Disturbances of movement coordination.
Dysarthria	Disturbance of speech.

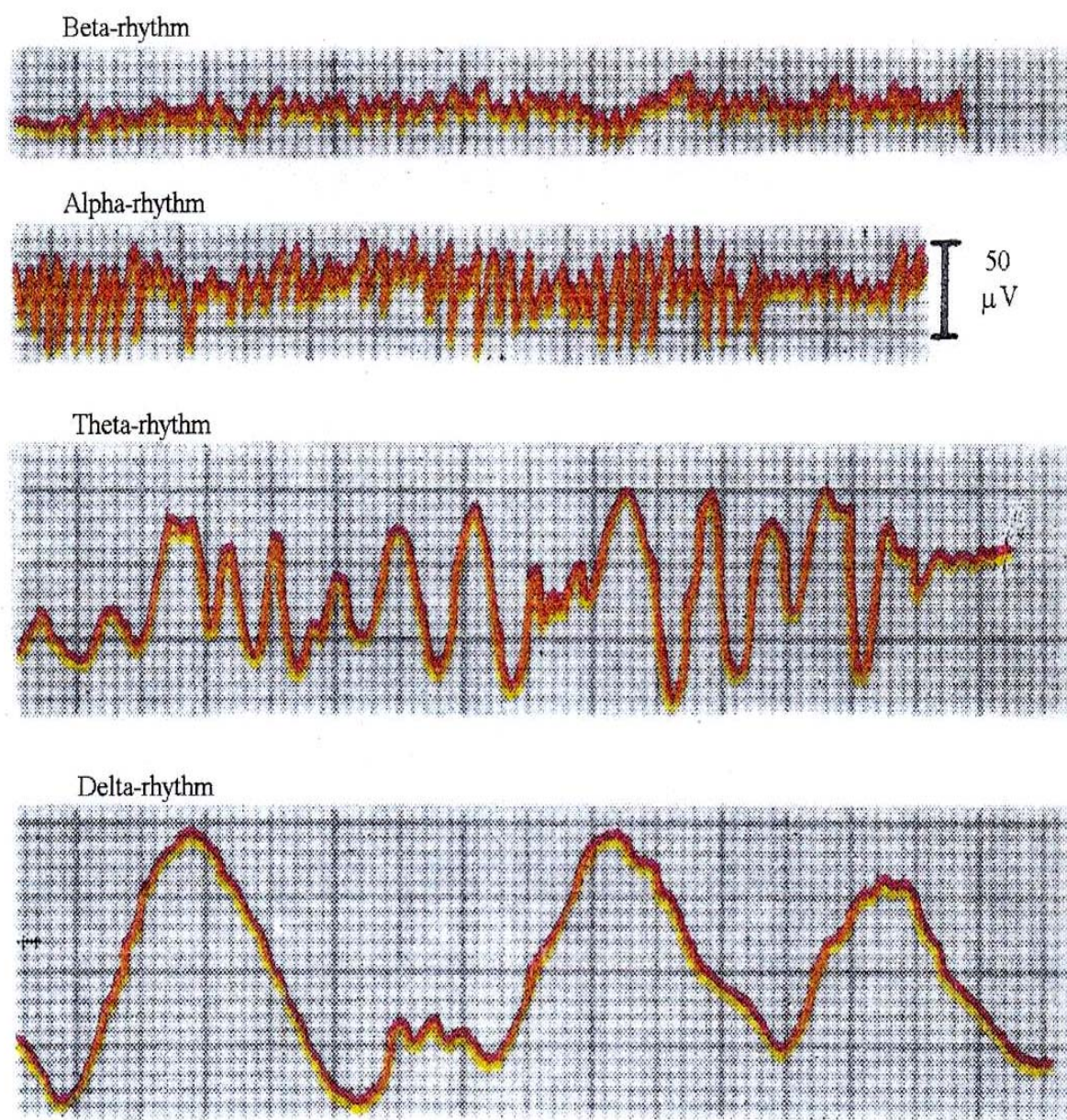


Figure 8.14. —The rhythms of electroencephalography
(by Korobkov A. V., Chesnokova S. A., 1986)

Table 8.5. — Characteristic of electroencephalogram rhythms

Type of rhythm	Amplitude, μV	Frequency, Hertz	Conditions at which the type of rhythms is registered
α - Alpha	50	8–13	At condition of mental and physical rest with closed eyes
β - Beta	25	14–30	At condition of mental and physical activity
θ - Theta	100–150	4–7	During falling asleep, at condition of moderate hypoxia and narcosis
Δ - Delta	250–300	3–5	At loss of consciousness, during deep sleep, hypoxia and narcosis, at damage of cerebral cortex

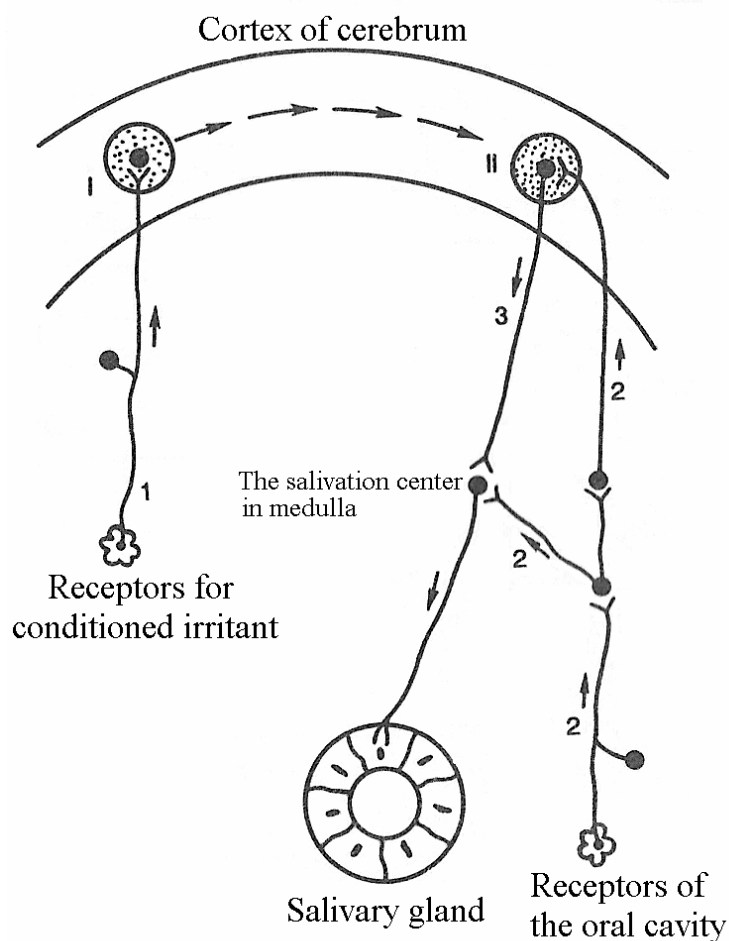


Figure 8.15. — Formation of conditioned reflex
(by Pokrovskiy V. M., Korotko G. F., 2000)

I 3 the focus of excitation in cortex of cerebrum, which is formed at the action of conditioned irritant;

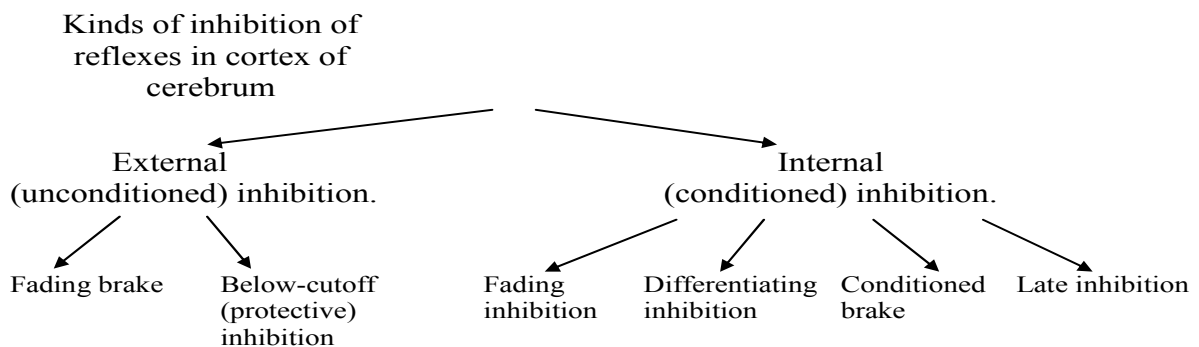
II — the focus of excitation in cortex of cerebrum, which is formed at the action of unconditioned irritant;

1 — afferent nerve ways of conditioned signal;

2 — afferent nerve ways of unconditioned signal;

3 — efferent nerve ways.

At multiple repetition of action of conditioned and unconditioned irritants time connection between the focuses of excitation in the cortex of cerebrum is formed.



Scheme 8.2. — Kinds of inhibition of reflexes in cortex of cerebrum

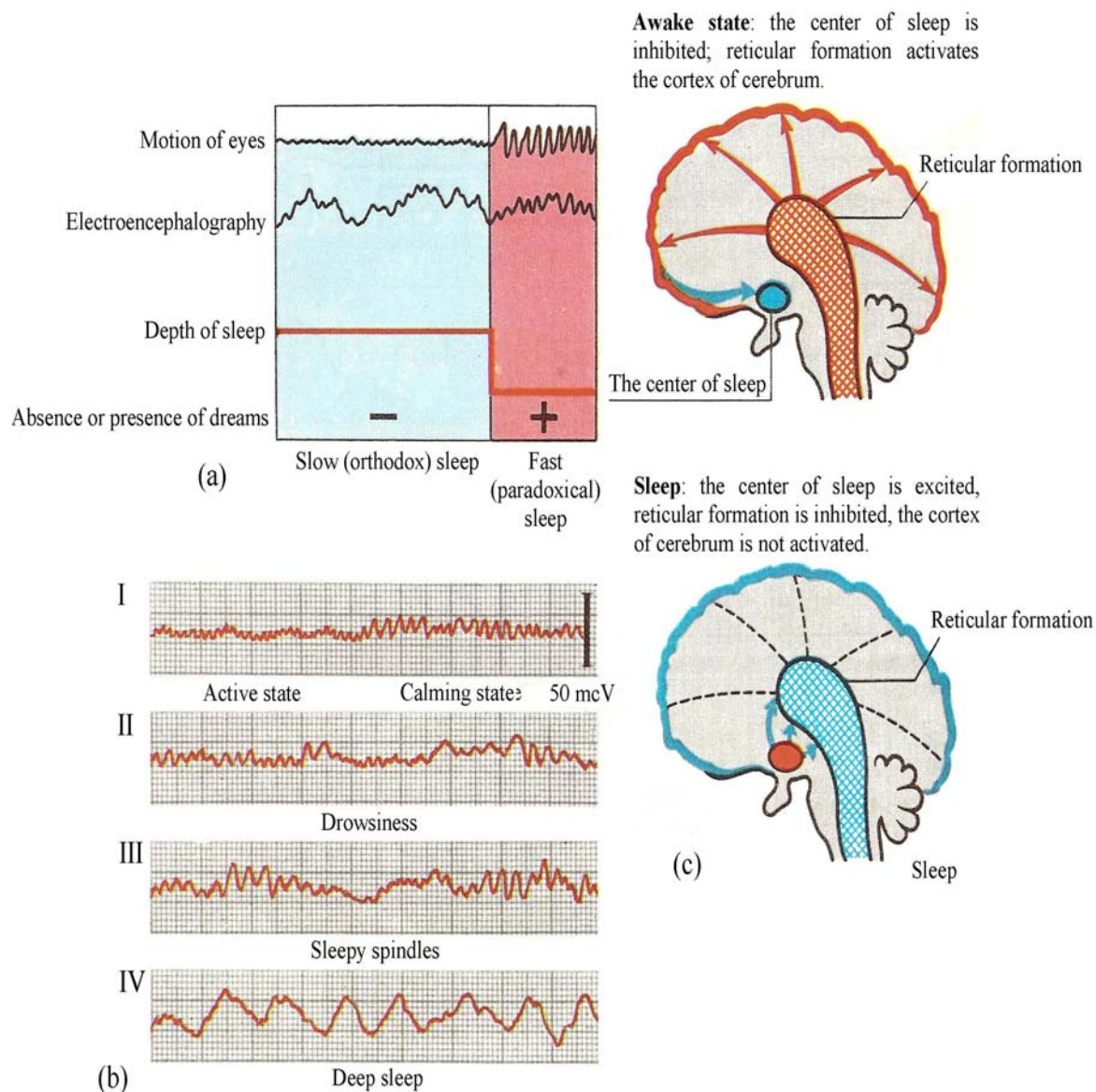
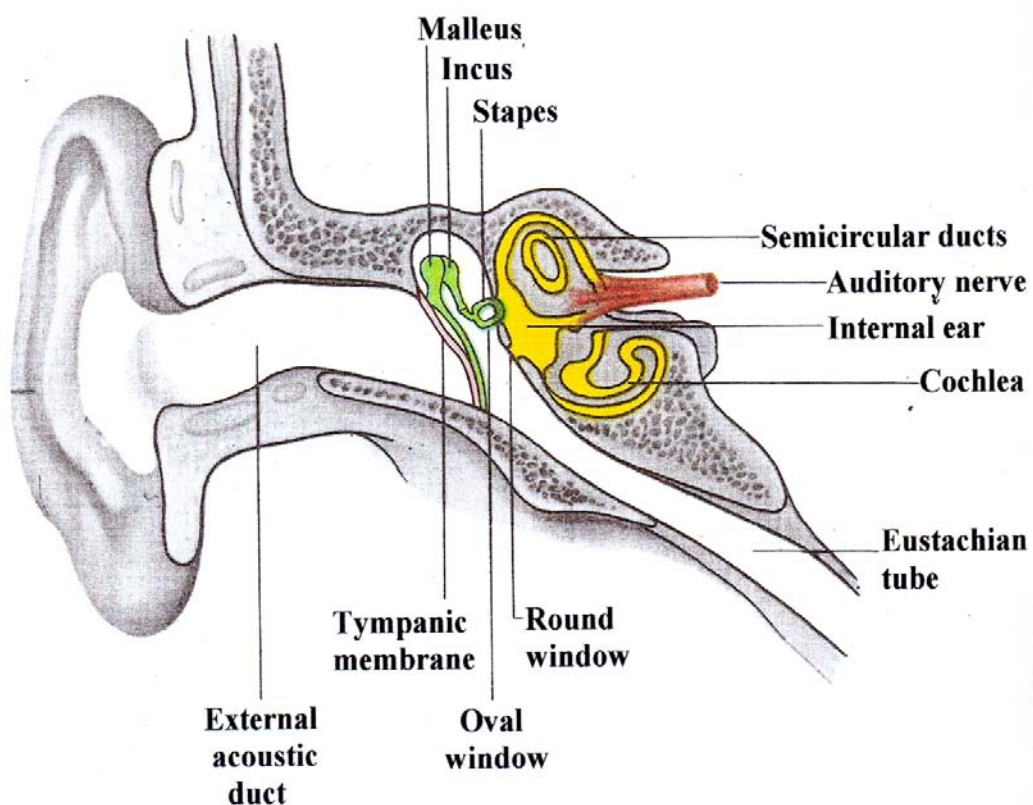


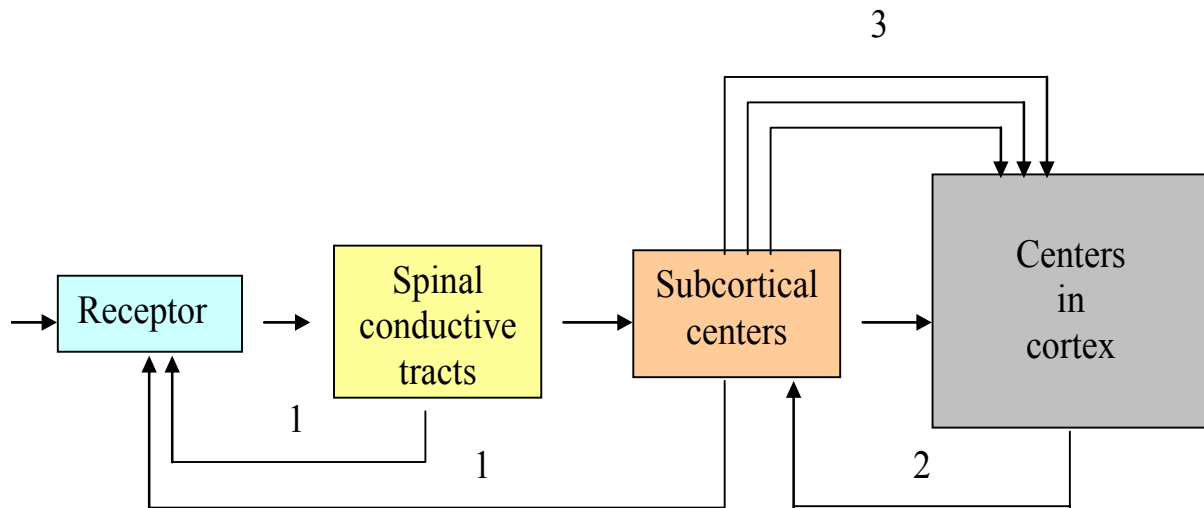
Figure 8.15. — Phases of sleep
(by Korobkov A. V., Chesnokova S. A., 1986)

- (a) — Fast and slow sleep.
- (b) — Phases of falling asleep (I–IV).
- (c) — Functional correlation of brain structures at state of sleep and awake state.

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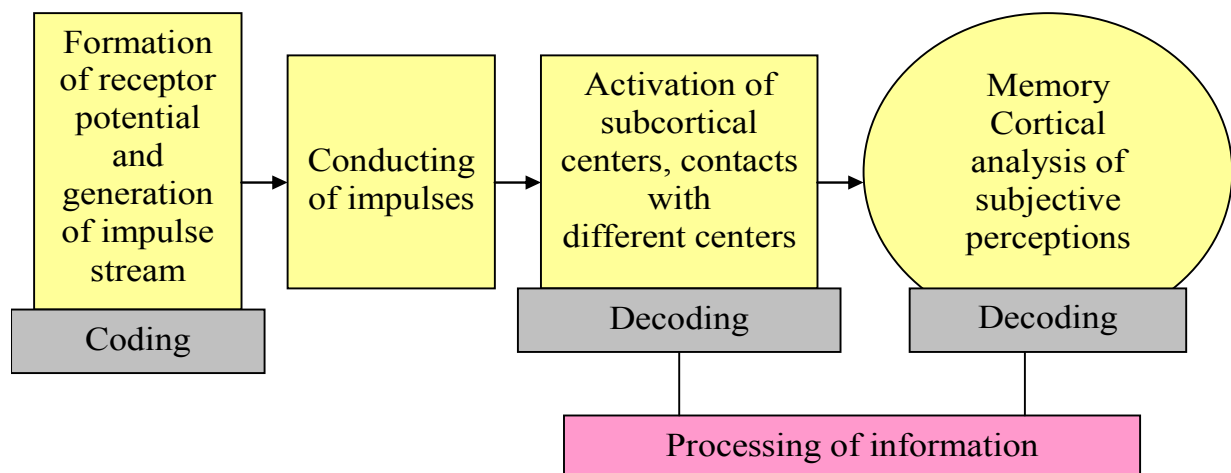
PHYSIOLOGY OF SENSORY SYSTEMS





Scheme 9.1. — The general principle of structure and functioning of sensory systems (by Korobkov A. V., Chesnokova S. A., 1986)

1 — Sympathetic system regulating the level of receptor excitation
 2 — Cortex 3 — Reticular formation



Scheme 9.2. — Stages of analyzer functioning (by Korobkov A. V., Chesnokova S. A., 1986)

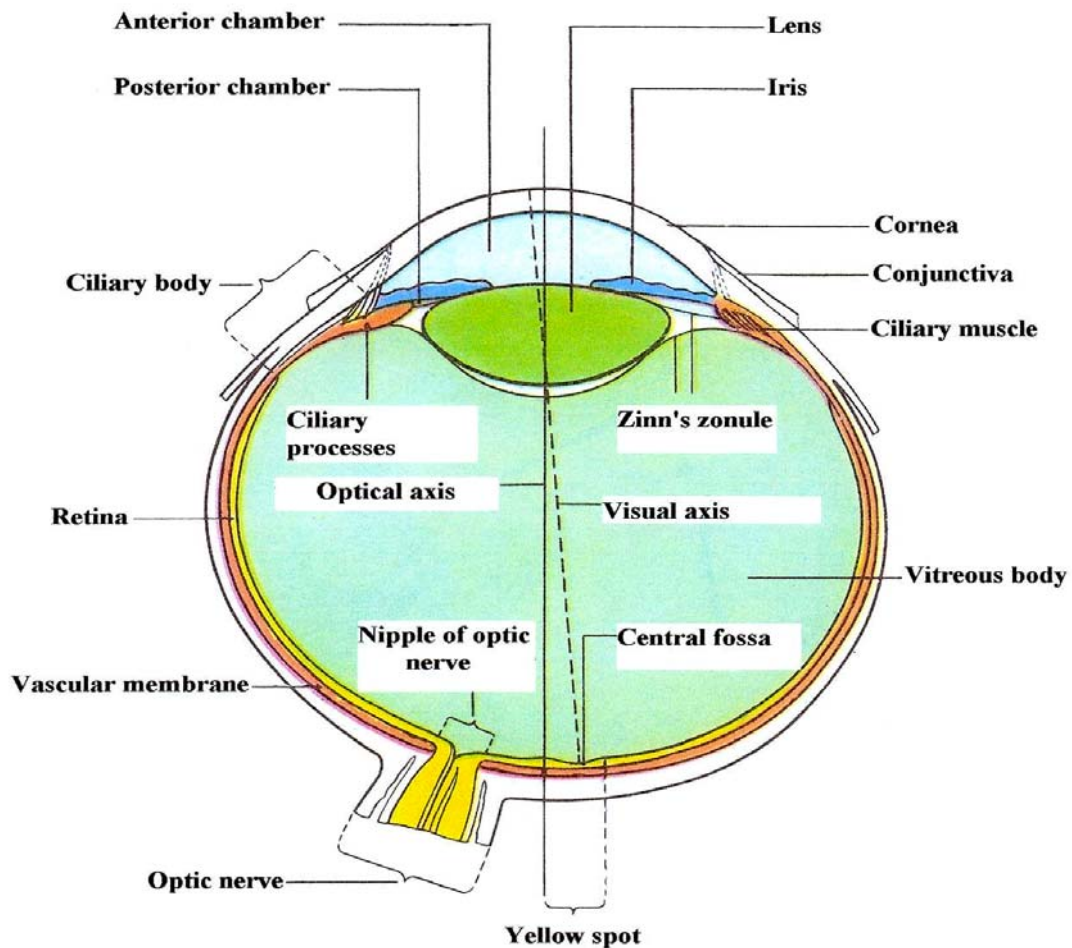


Figure 9.1. — Structure of the human eye
(by Korobkov A. V., Chesnokova S. A., 1986)

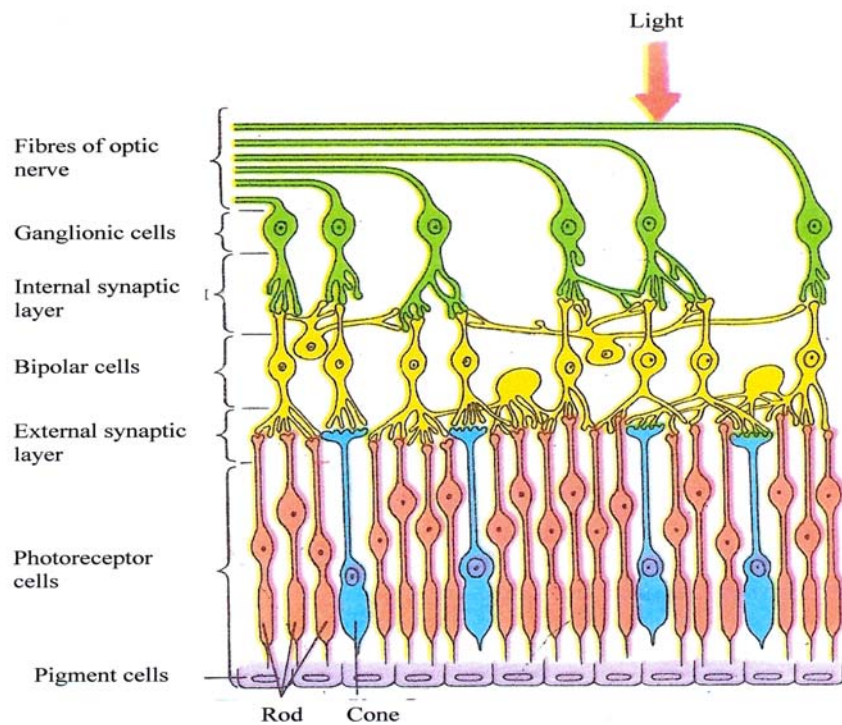
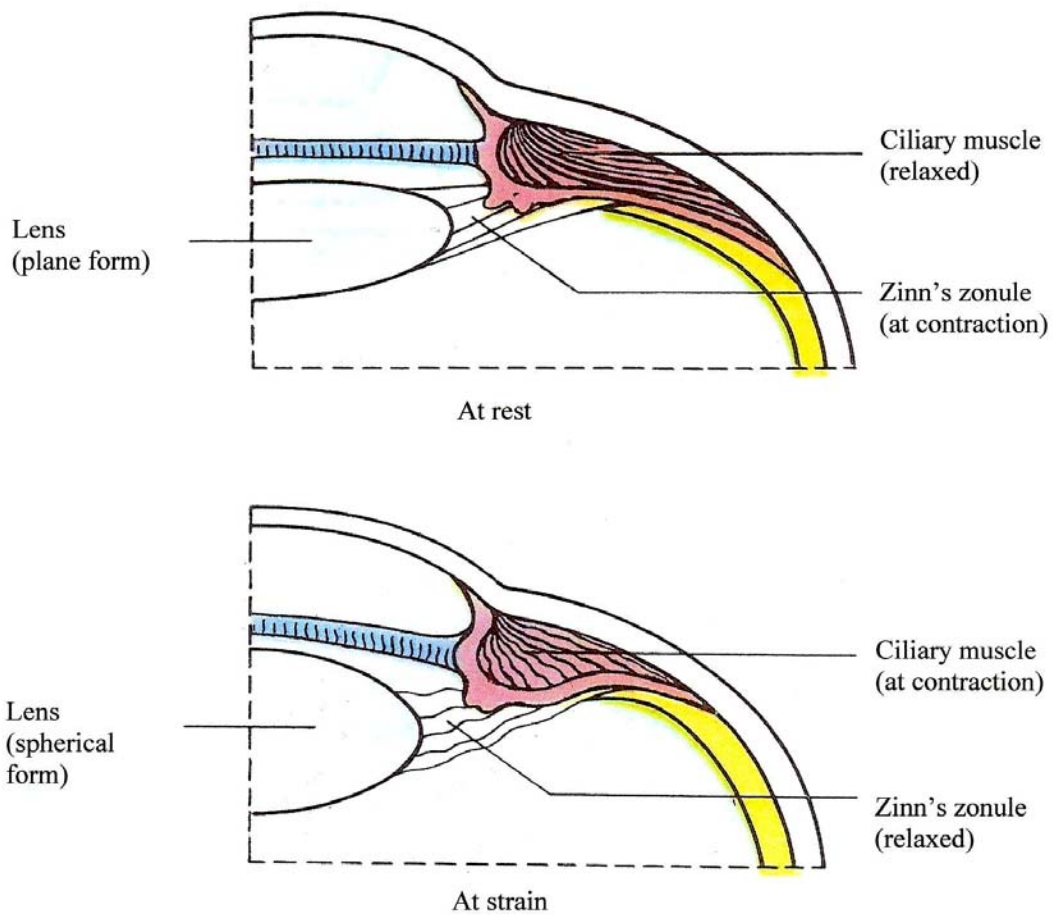
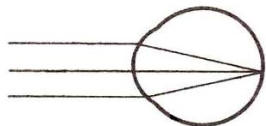


Figure 9.2. — The structure of the retina
(by Korobkov A. V., Chesnokova S. A., 1986)

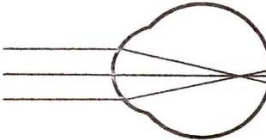


**Figure 9.3. — The mechanism of the accommodation of an eye
(by Korobkov A. V., Chesnokova S. A., 1986)**

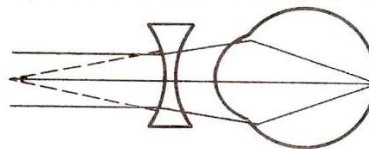
Refraction in the normal eye



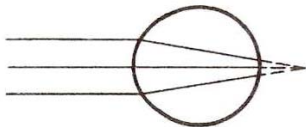
Refraction at myopia



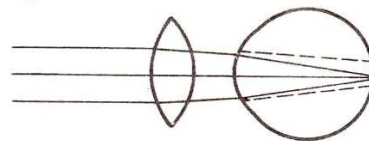
Optic correction of myopia



Refraction at hypermetropia



Optic correction of hypermetropia



**Figure 9.4. — Refraction in the normal eye, at myopia and hypermetropia.
Optic correction of myopia and hypermetropia
(by Pokrovskiy V.M., Korotko G.F., 2000)**

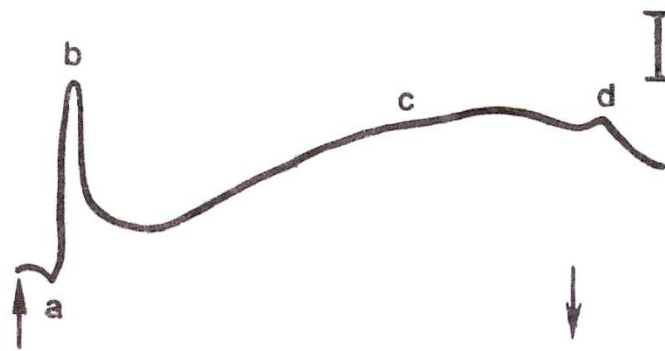


Figure 9.5. — Electretinogram
(by Pokrovskiy V. M., Korotko G. F., 2000)

The summary electrical response of the retina of the eye to the action of light is called electretinogram (ERG). ERG distinguishes several typical waves. Wave a reflects excitation of internal segments of photoreceptors and horizontal cells. The wave b appears from the activation of glial cells of the retina by ions of potassium released at excitation of bipolar and amacrine neurons. Wave c reflects activation of cells of pigment epithelium, and wave d - horizontal cells.

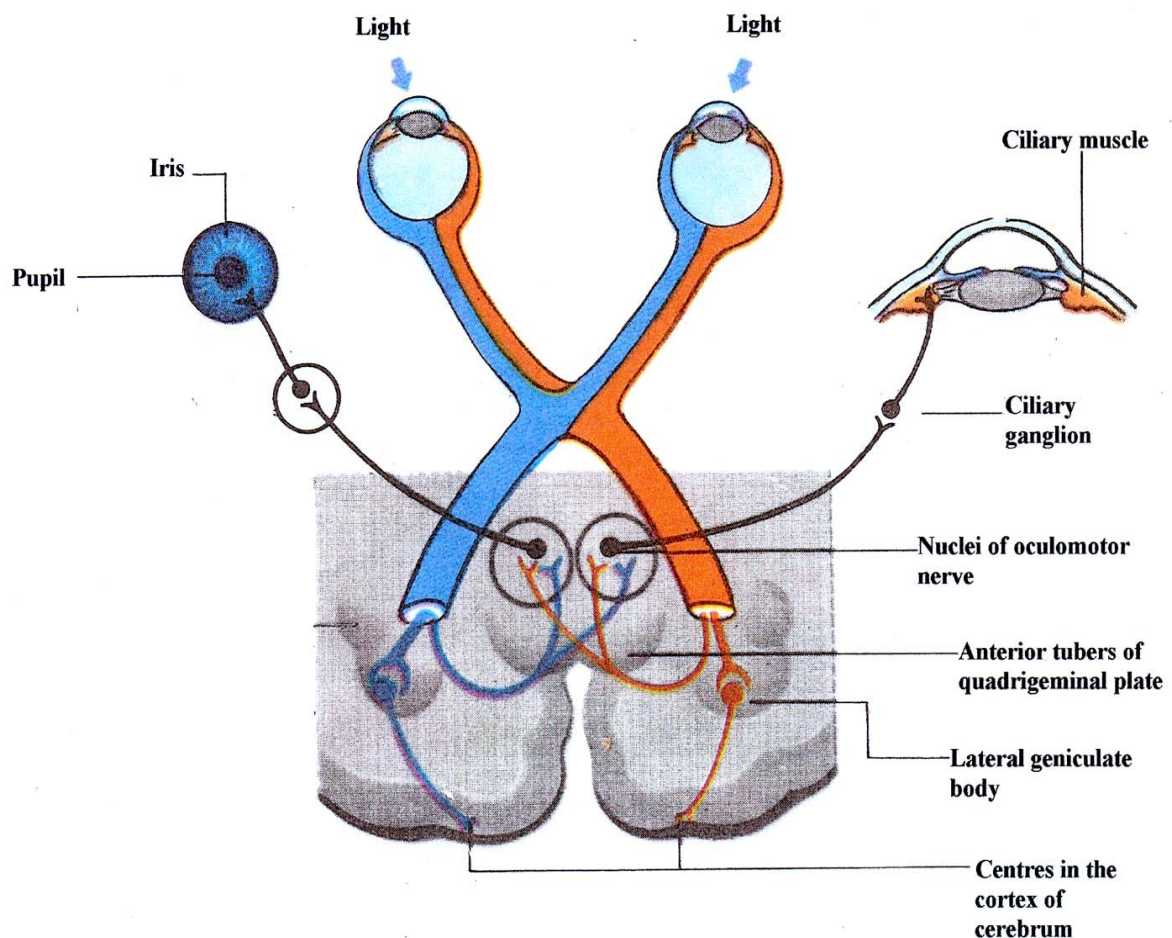


Figure 9.6. — The connection of optic ways with the process of control of the pupil's diameter and accommodation process
(by Korobkov A. V., Chesnokova S. A., 1986)

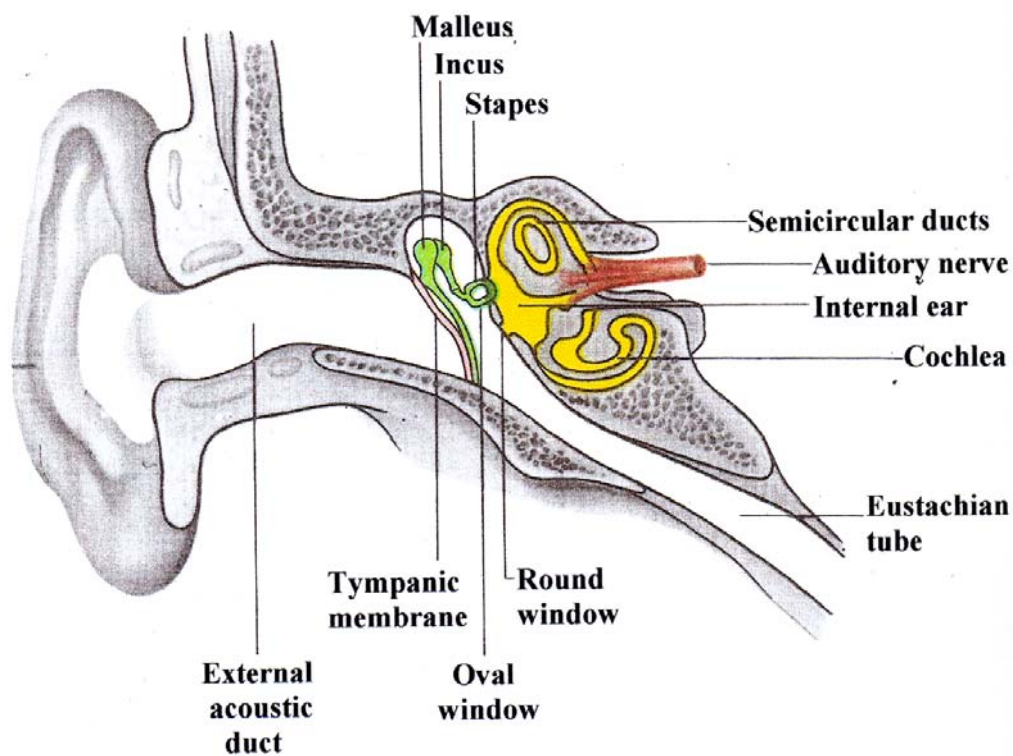


Figure 9.7. — Structure of auditory analyzer
(by Korobkov A.V., Chesnokova S.A., 1986)

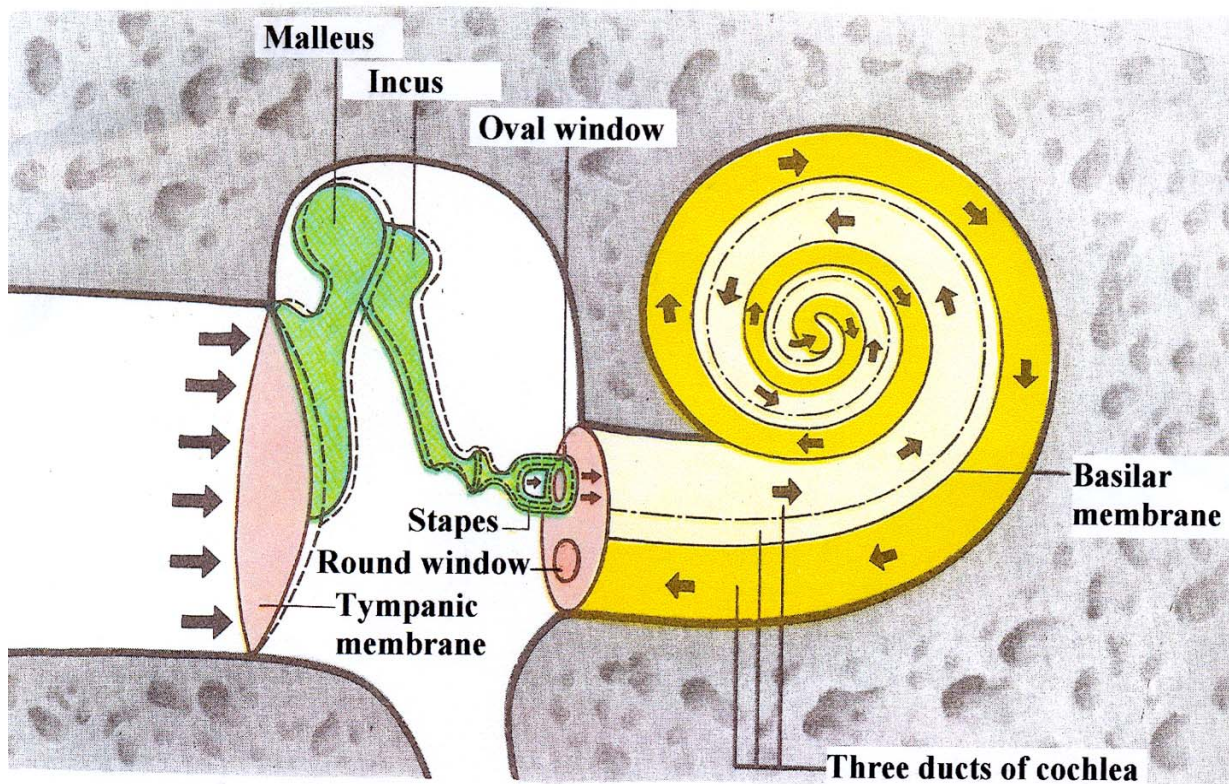


Figure 9.8. — Structure of middle and internal ear
(by Korobkov A.V., Chesnokova S.A., 1986)

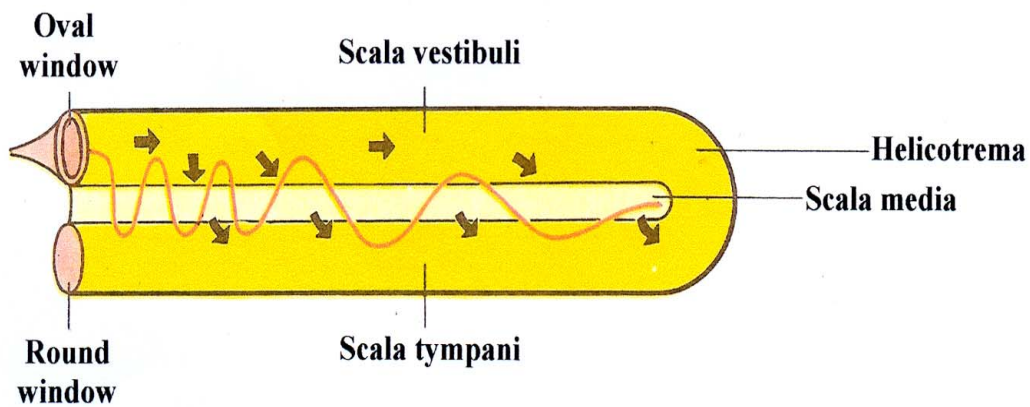


Figure 9.9. — Spreading of sound vibrations in cochlea
(by Korobkov A. V., Chesnokova S. A., 1986)

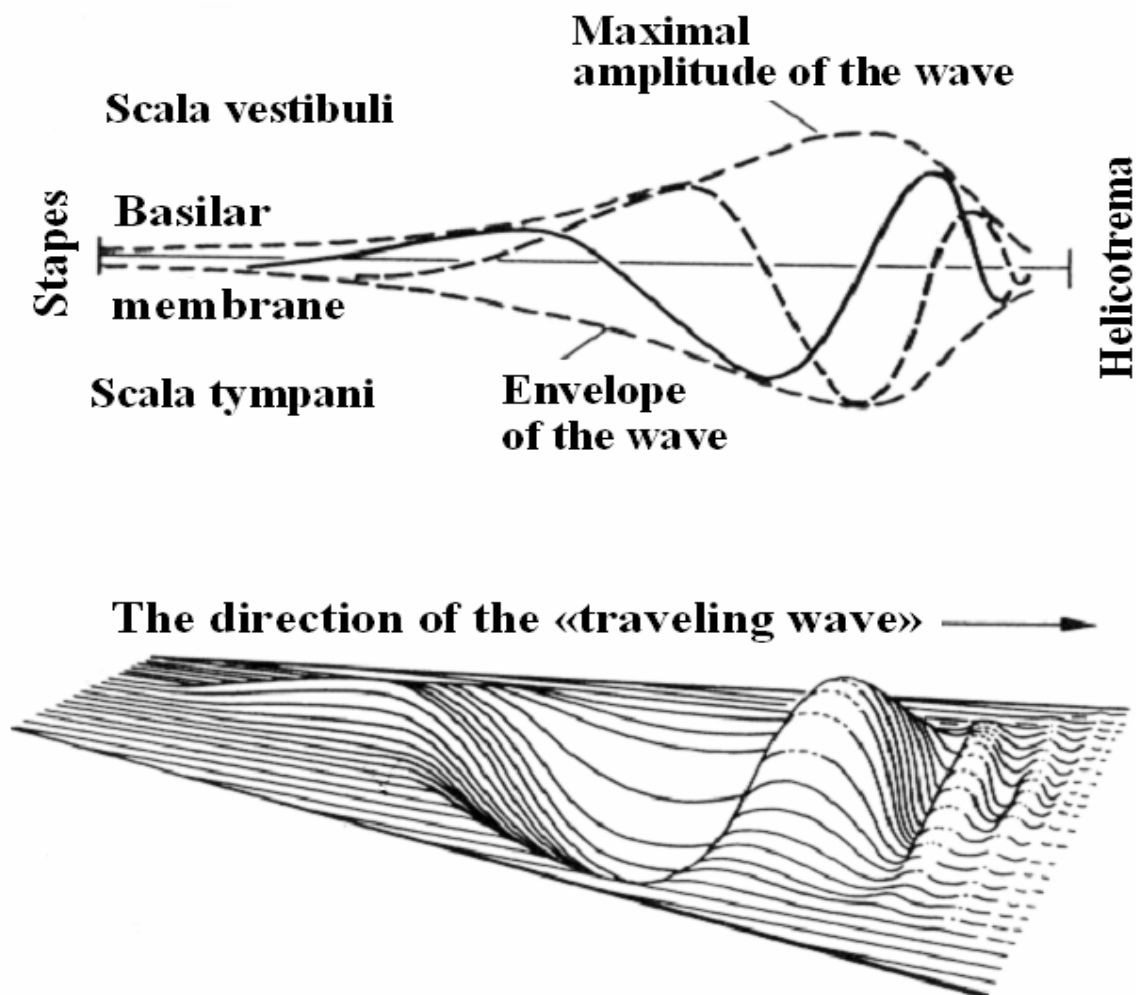


Figure 9.10. — The theory of a «traveling wave»

The basilar membrane is mostly rigid at the base of cochlea, i.e. at its narrowest point. Towards the apex its rigidity decreases. At vibration of the membrane wave «runs» from its base to the apex. High-frequency vibrations pass by the basilar membrane in short distance, and long low-frequency waves pass more far.

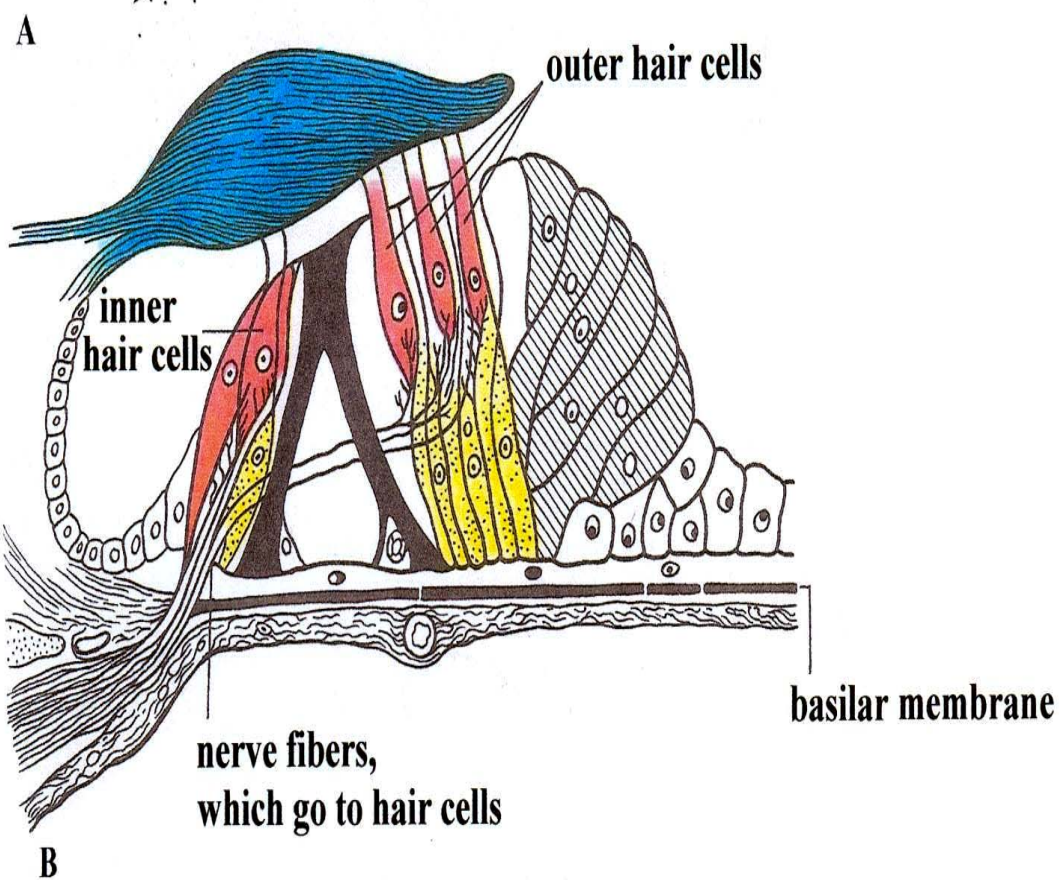
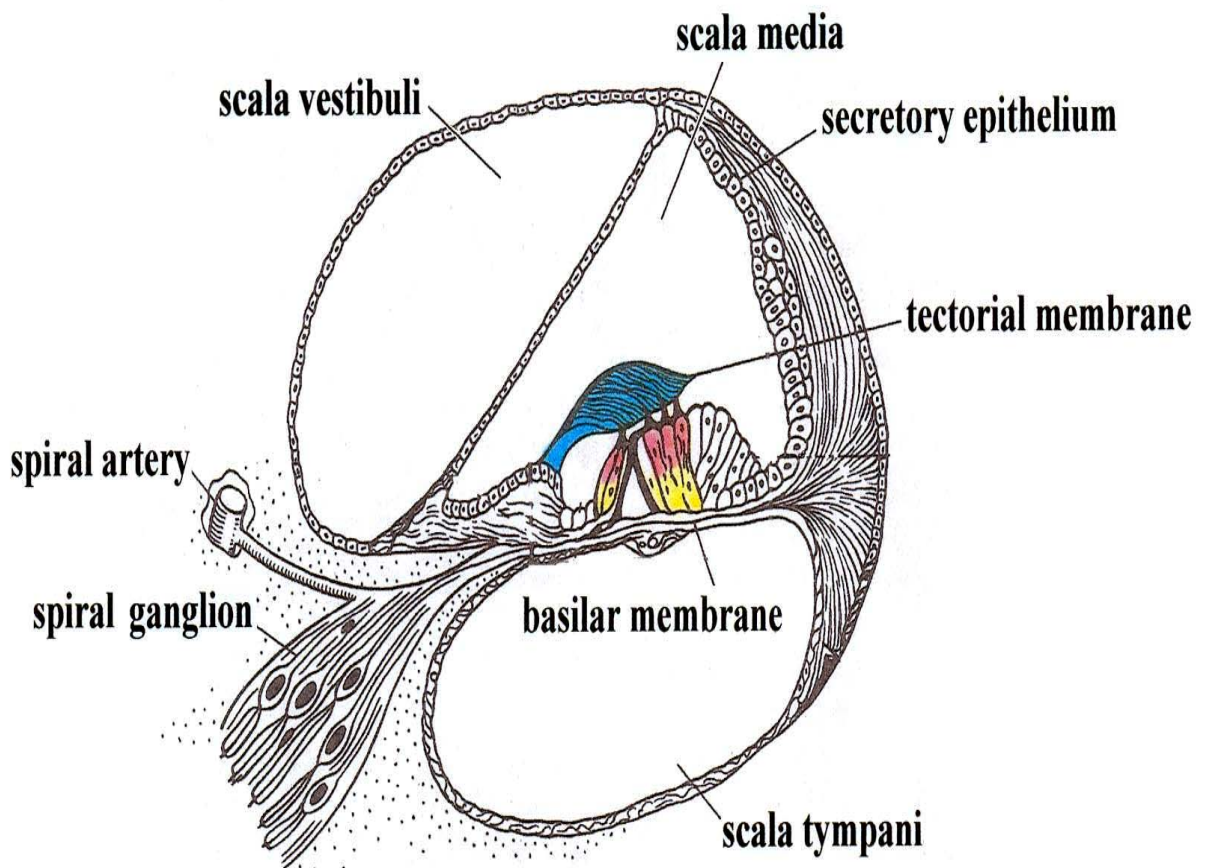
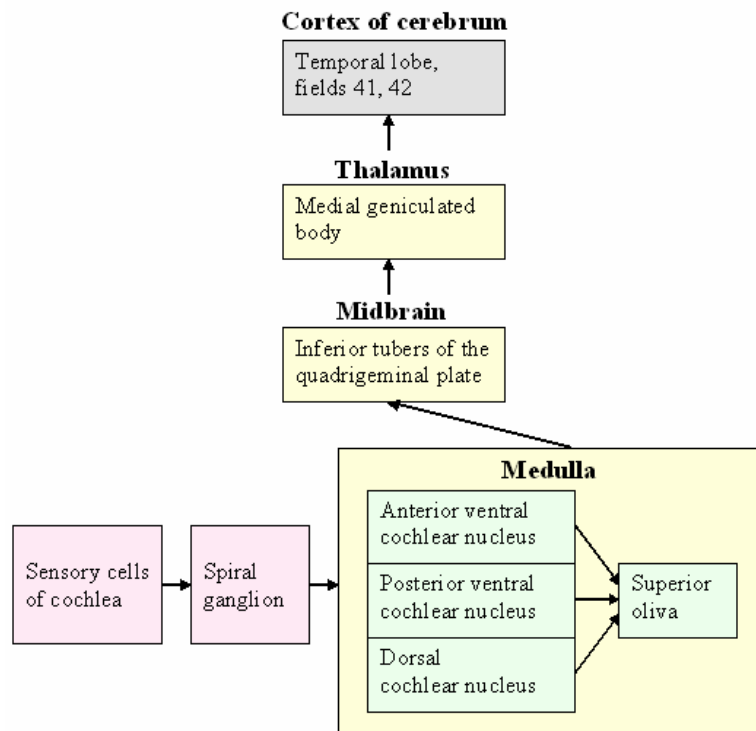


Figure 9.11. — Cross-section of the cochlea (A) and the structure of spiral (Corti's) organ (B) (by Pokrovskiy V. M., Korotko G. F., 2000)



Scheme 9.3. — Scheme of auditory analyzer (by Vlasova A., 1980)

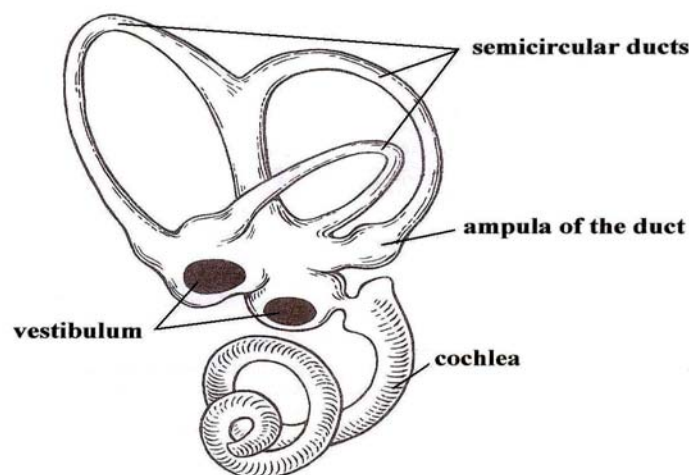


Figure 9.12. — The structure of the labyrinth of the temporal bone (by Pokrovskiy V. M., Korotko G. F., 2000)

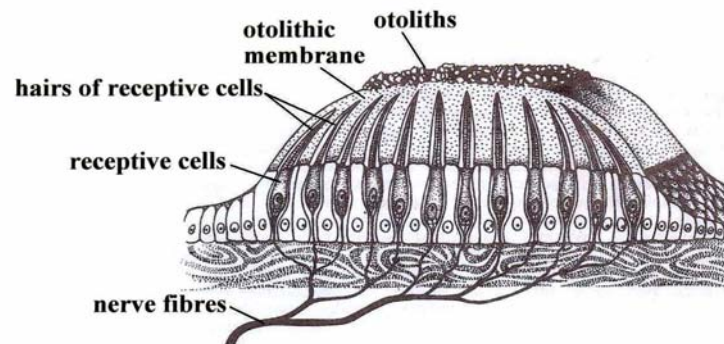


Figure 9.13. — The structure of the otolith apparatus (by Pokrovskiy V. M., Korotko G. F., 2000)

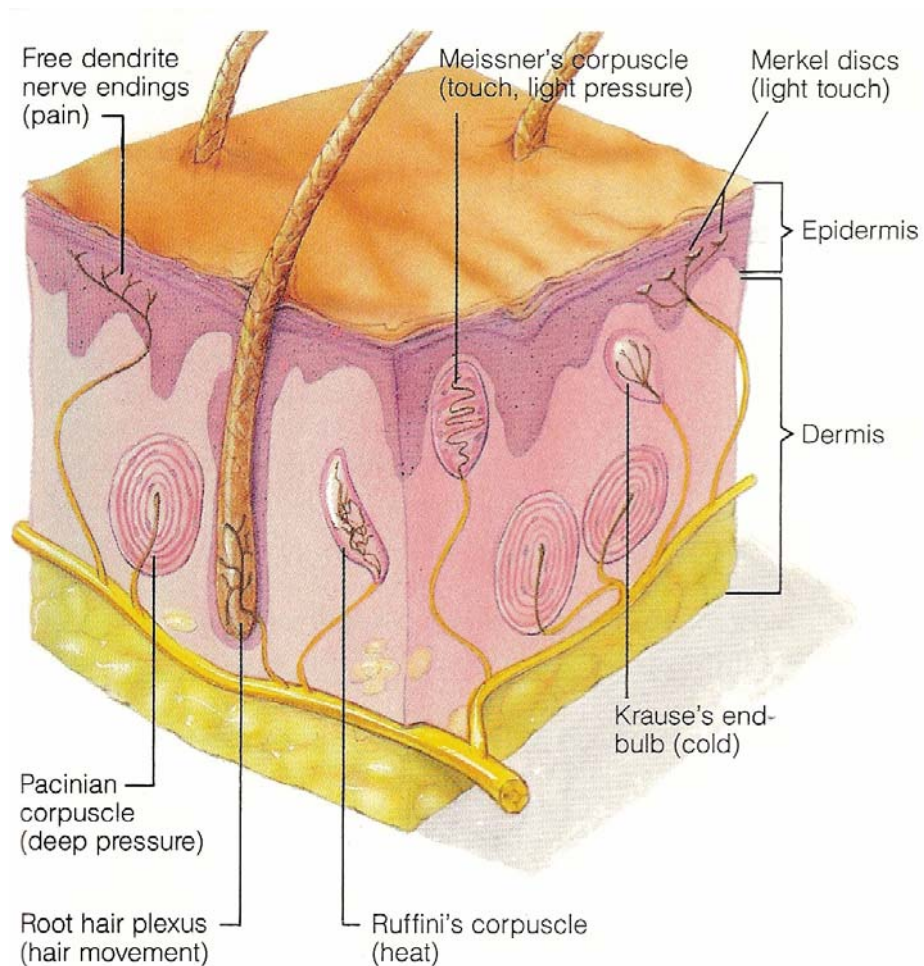


Figure 9.14. — Afferent and efferent connections of the vestibular apparatus
 (by Korobkov A. V., Chesnokova S. A., 1986)

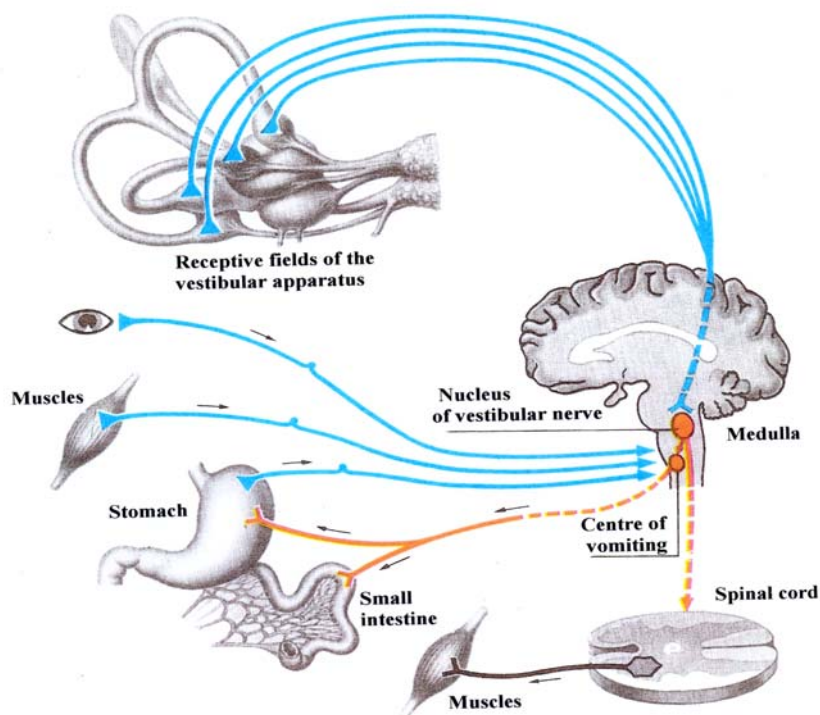
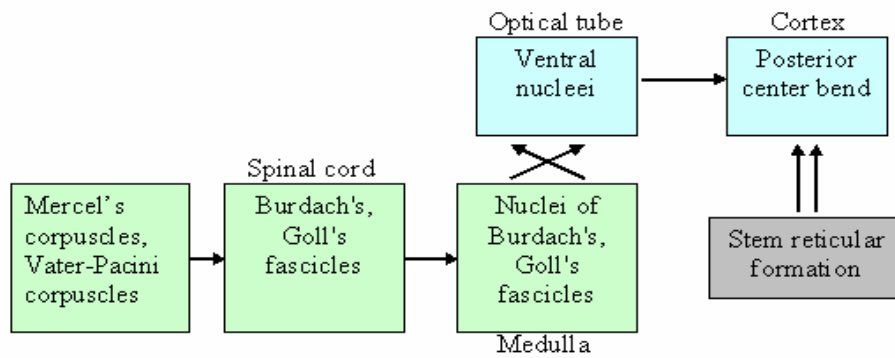
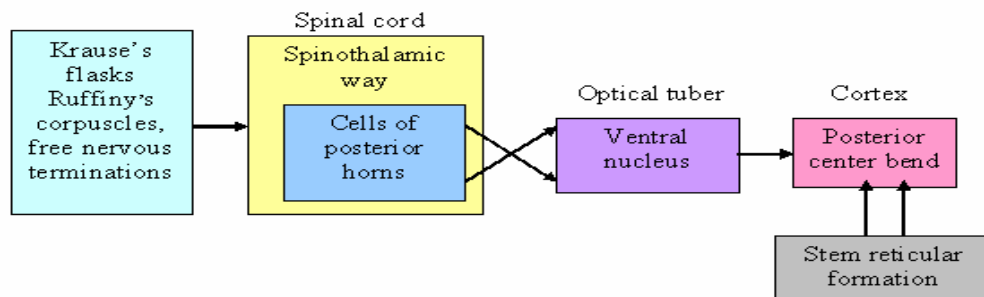


Figure 9.15. — Skin receptors (by Elaine N. Marieb, 1989)



Scheme 9.4. — Conductive tracts of tactile sensitivity (by Vlasova A., 1981)



Scheme 9.5. — Conductive tracts of temperature and pain sensitivity (by Vlasova A., 1981)

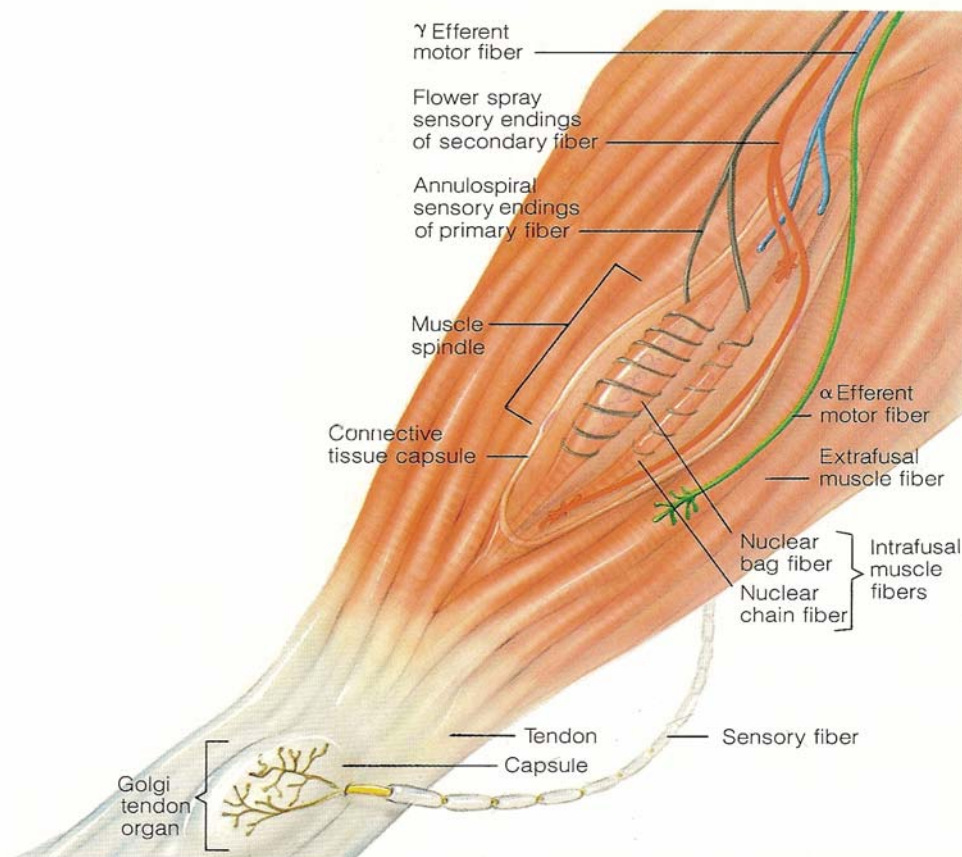


Figure 9.15. — The muscle spindle and Golgi tendon receptor (by Elaine N. Marieb, 1989)

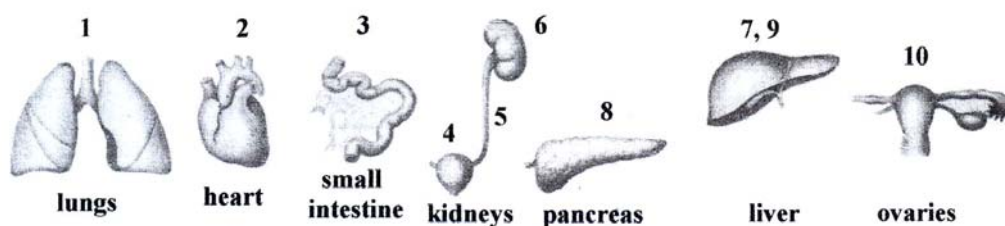
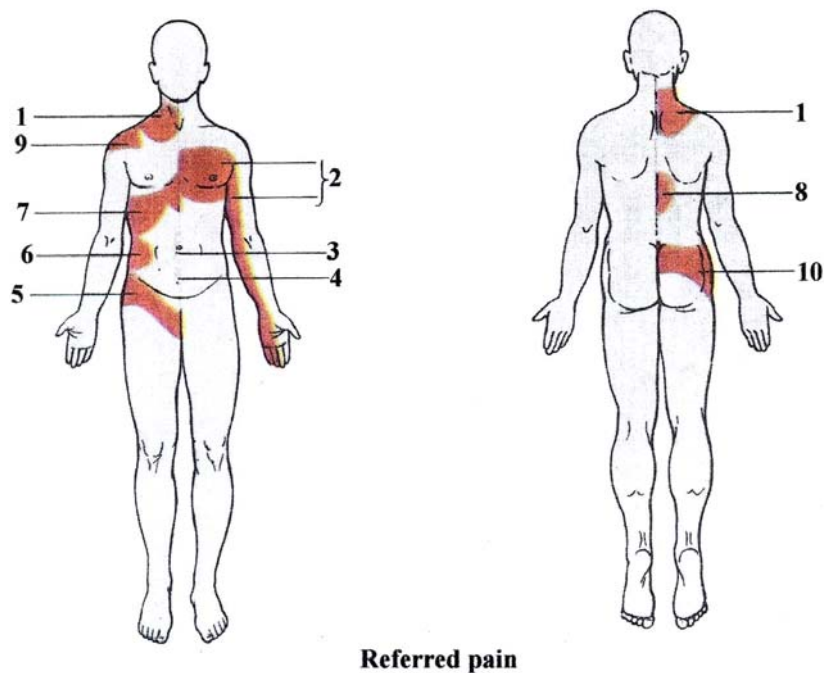


Figure 9.16. — Pain zones on the surface of skin, which appear at the affection of internal organs (by Korobkov A. V., Chesnokova S. A., 1986)

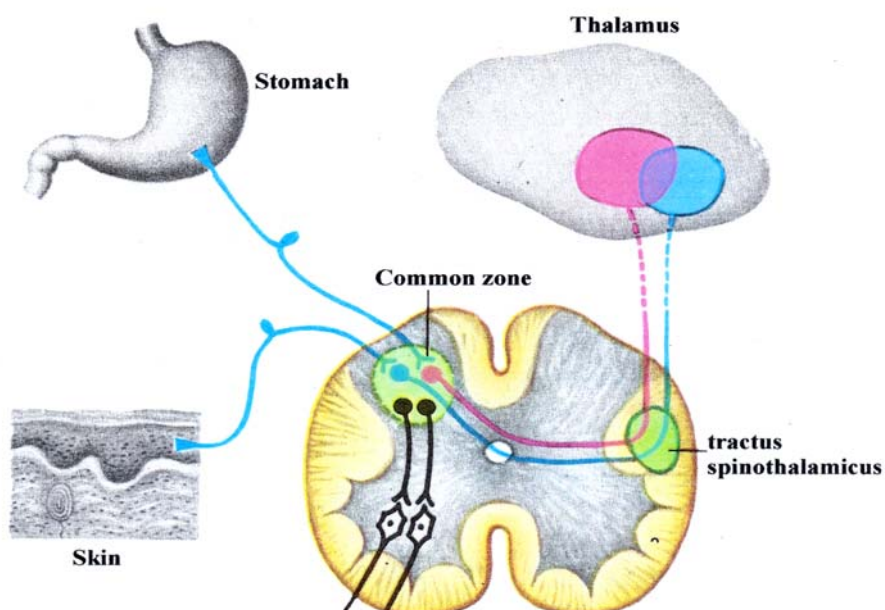


Figure 9.17. — Supposed mechanism of the origin of referred pain (by Korobkov A. V., Chesnokova S. A., 1986)

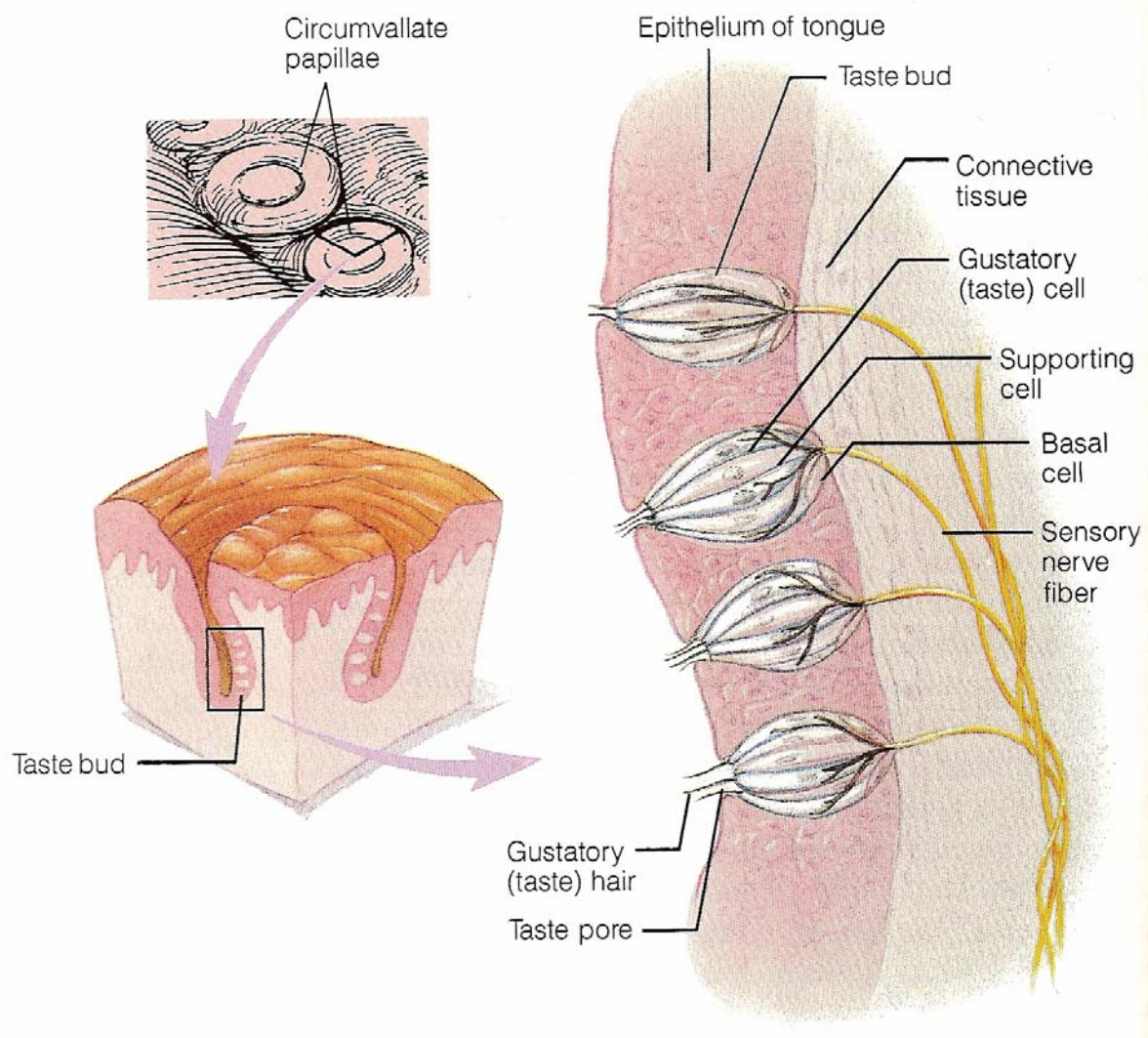


Figure 9.18. — Structure of taste buds (by Elaine N. Marieb, 1989)

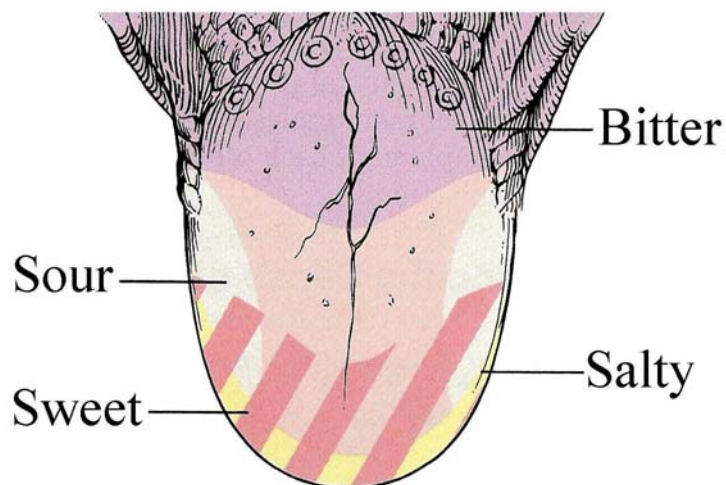


Figure 9.19. — Relative patterns of taste sensitivity on the tongue dorsum (by Elaine N. Marieb, 1989)

Since the area most sensitive to sweet overlaps the sour and salty areas, the sweet area is indicated by stripes.

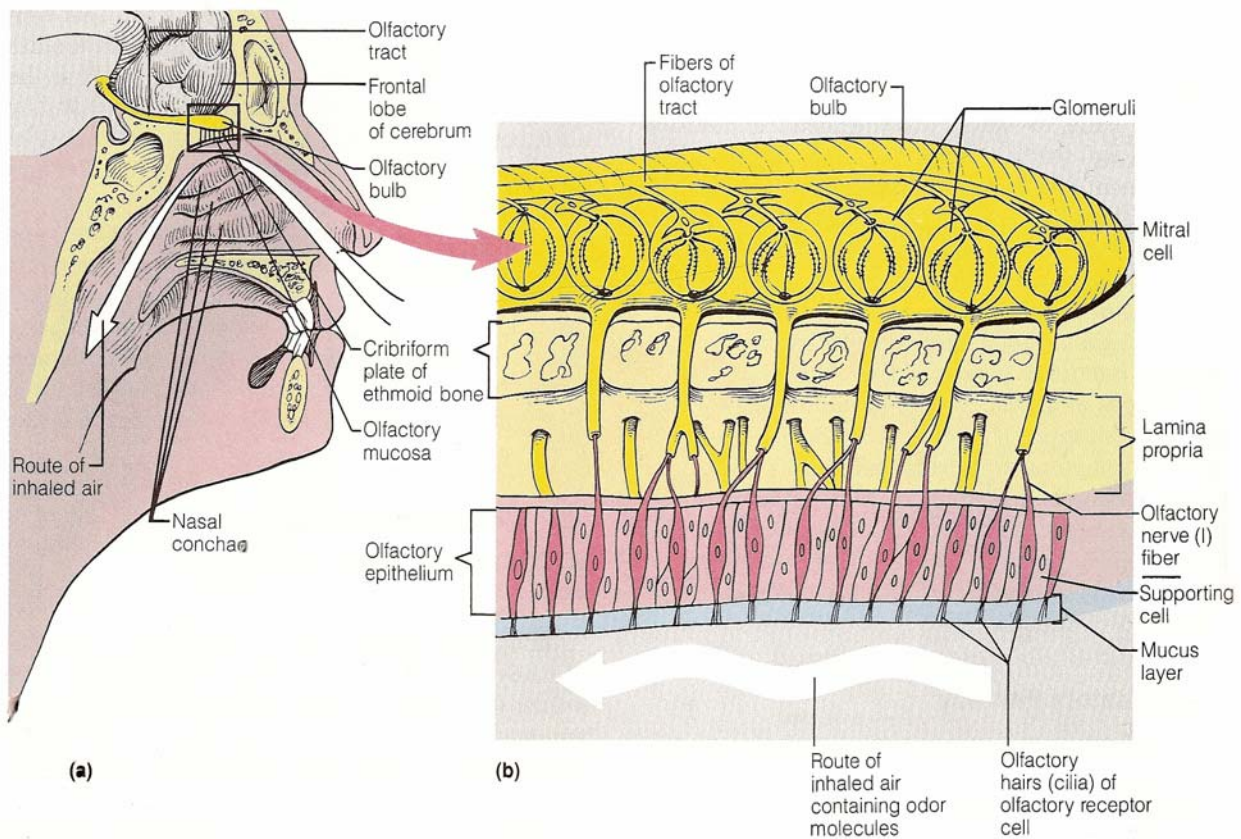


Figure 9.20. — Olfactory receptors (by Elaine N. Marieb, 1989)

(a) Site of olfactory epithelium in the superior aspect of the nasal cavity.

(b) Enlarged view illustrating the cellular composition of the olfactory epithelium and the course of the fibers of the olfactory nerve (I) through the ethmoid bone to synapse in the overlying olfactory bulb. The glomeruli and mitral cells (output cells) within the olfactory bulb are also shown.

BASIC PHYSIOLOGIC CONSTANT

Constant of blood system	
Amount of blood in adults (6–8% of body weight)	4,5–6 l
Hematocrit (m)	0,44–0,46
(f)	0,41–0,43
Blood: deposited	45–50 %
circulating	50–55 %
Volume of blood plasma	approx. 3 l
Structure of blood plasma:	
Water	90–92%
Solid residual	8–10%
General protein	65–80 g/l
Albumins	45 g/l
Globulins	20–35 g/l
Fibrinogen	3 g/l
Residual nitrogen	14,3–28,5 millimole/l
Glucose (whole blood)	3,30–5,55 millimole/l
(plasma)	3,88–6,10 millimole/l
Triglycerides	0,40–1,81 millimole/l
Inorganic substances	0,9 %
Viscosity of blood in adults	5
Relative density	1,050–1,060
pH of arterial bloods:	7,40
venous	7,35
pH borders compatible with life	7,0–7,8
Amount of erythrocytes: (m)	4,5–5,0×10 ¹² /l (tera per litre)
(f)	3,8–4,5×10 ¹² /l (tera per litre)
Amount of hemoglobin (m)	130–160 g/l
(f)	115–145 g/l
Color parameter: adults	0,8–1,0
Osmotic resistance of erythrocytes: Min	0,46–0,48% solution of NaCl
Max	0,32–0,34% solution of NaCl
Erythrocyte sedimentation rate (m)	1–10 mm / hr
(f)	2–15 mm / hr
Neonatal	1–2 mm / hr
Leucocytes: amount in adults	4–9×10 ⁹ /l (giga per litre)
in newborns	15–20×10 ⁹ /l (giga per litre)
The leukocytic formula (%): Neutrophils:	
Myelocytes	0
Metamyelocytes	0–1
stab neutrophil	1–5
segmentonuclear	45–70
Eosinocytes	1–5
Basophils	0–1
Lymphocytes	20–40
Monocytes	2–10
Index of regeneration (shift to the left)	0,05–0,1
Amount of thrombocytes	180–320×10 ⁹ /l (giga per litre)
Blood coagulation time (by Lee-White)	5–7 min
Constant of cardiovascular system	
heart rate: in adults	60–80 / min
in neonatals	135–140 /min
Systolic volume of blood	65–70 ml

Minute volume of blood: at rest	4,5–5 l
at physical work	Up to 30 l
Time of cardiac cycle	0.75–1,0 sec
Arterial pressure: Max (systolic)	110–125 mm Hg
Min (diastolic)	60–85 mm Hg
Constant of respiratory system	
Respiration rate: in adults	12–18 / minute
in neonatal	40–55 / minute
Excursion of thorax: (m)	7–10 cm
(f)	5–8 cm
Interrelation of duration inspiration-expiration	1:1.2
Respiratory volume	0,3–0,9 l
Reserve volume of inspiration	1,5–2,0 l
Reserve volume of expiration	1,0–1,5 l
Vital capacity of lung	3,5–5,0 l
Residual volume	1,0–1,5 l
Functional residual capacity	2,5 l
Capacity of inspiration	2,0 l
Dead space	140–170 ml
Coefficient of lung ventilation	1/7
Minute volume of respiration: at rest	Up to 7 l
at physical activity	Up to 120 l/minute
Alveolar ventilation	4,2–5,6 l/minute
Maximal ventilation lung	120–170 l/minute
pO ₂ in alveolar air	110 mm Hg
pCO ₂ in alveolar air	40 mm Hg
pO ₂ in arterial blood	100 mm Hg
pCO ₂ in arterial blood	39 mm Hg
pO ₂ in venous blood	40 mm Hg
pCO ₂ in venous blood	46 mm Hg
Volume of forced expiration	3 l
Oxygen capacity of blood	19 percent by volume
Ventilation-perfusion coefficient	0,8–0,9
Consumption of oxygen at rest	350 ml/min
Coefficient of use O ₂ at rest	40%
Constant of digestive system	
Saliva: amount of excreted saliva daily	1,5 l/day
pH	7,4–8,0
Gastric juice: daily volume	2,0–2,5 l
pH	1,5–1,8
Intestinal juice: pH juice of small intestines	5,05–7,07
Pancreas juice: daily volume	1,5–2,0 l
pH	7,8–8,4
Bile: daily volume	500–1500 ml
Constants of metabolism and energy	
Biological value of proteins:	
animal origin	70–95%
vegetable origin	60–65%
Daily need:	
Proteins	70–80 g (of them 30% are animals)
Fats	70–80 g (of them 75–80% are animals)
Carbohydrates	400–450 g

Water content in an organism	
Men	61%;
Women	51%; (compare 53,5%)
Neonatal	75%
Formation of water in an organism at oxidation:	
100 g of carbohydrates	55 ml
100 g of proteins	41 ml
100 fats	107 ml
Daily balance of water	near 2,5 l
Food value:	
1 g of fats	9,3 kcal (39,0 kilojoule)
1 g of carbohydrates	4,1 kcal (17,1 kilojoule)
1 g of proteins	4,1 kcal (17,1 kilojoule)
Respiratory coefficient at oxidation in an organism	
Carbohydrates	1
Fats	0,7
Proteins	0,8
The basic metabolism:	
Men	7117 kilojoule (1700 kcal) a day
Women	6410 kilojoule (1530 kcal) a day
Constants of thermoregulation	
temperature	
axilla	36,5–36,9°C
in oral cavity	36,4–37,2°C
rectum	36,8–37,6°C
daily temperature fluctuation	
Max	at 4–6 p.m.
Min	at 3–4 a.m.
hyperthermia	body temperature > 37°C
hypothermia	body temperature < 35°C
Constants of excretion	
Efficient filtration pressure	20 mm Hg
General filtration surface of glomuluses	1.5–2 m ²
Renal blood flow	of 1200 ml/minutes
Renal plasma flow	650 ml/minutes
Amount of initial urine a day	150–170 l
Amount of final urine a day	1,5 l
Relative density	1,012
Color	from amber — yellow to stramineous
Transparence	transparent
pH	5,0–7,0
Constants of sensory systems	
Frequency of sound fluctuations heard by the person	16–20000 Hz
Closest point of clear vision	10 cm
Acuity of vision (normal)	1,0 and more

LITERATURE

Basic

1. Физиология человека: учебник; под ред. В. М. Покровского и Г. Ф. Коротько. В 2 т. — М.: Медицина, 1997.
2. Физиология человека / Под ред. Р. Шмидта и Г. Тевса. — М.: Мир, 1996.
3. Физиология человека / Под ред. Г. И. Косицкого. — М.: Медицина, 1985. — 544 с.
4. Human physiology: series of lectures for overseas students in English medium = Физиология человека: конспективный курс лекций для иностранных студентов, обучающихся на английском языке / А. И. Киеня, Э. М. Заика, В. А. Мельник; под редакцией проф. Э. С. Питкевича; перевод на англ. яз. Р. А. Карпов, В. А. Мельник. В 2 ч. — Гомель: УО «Гомельский государственный медицинский университет», 2006.
5. Заика, Э. М. Физиология сердечно-сосудистой системы: конспективный курс лекций / Э. М. Заика. — Гомель: Гомельский государственный медицинский университет, 2005. — 53 с.
6. Киеня, А. И. Физиология жидких сред организма человека: учеб. пособие / А. И. Киеня. — Гомель, 2001. — 70 с.
7. Киеня, А. И. Руководство к практическим занятиям по нормальной физиологии / А. И. Киеня, Э. М. Заика; под ред. проф. А. И. Киени. — Гомель, 1999. — 115 с.
8. Руководство к практическим занятиям по физиологии / Под ред. чл.-корр. АМН СССР Г. И. Косицкого и проф. В. А. Полянцева. — М.: Медицина, 1988.
9. Практикум по нормальной физиологии / Под ред. проф. Н. А. Агаджаняна и проф. А. В. Коробкова. — М.: Высшая школа, 1983.
10. Атлас по нормальной физиологии / Под ред. проф. Н. А. Агаджаняна. — М.: Высшая школа, 1986.
11. Орлов, Р. М. Нормальная физиология: учебник / Р. М. Орлов, А. Д. Наздрачев. — М.: ГЭОТАР-Медиа, 2005. — 696 с.

Optional

1. Киеня, А. И. Здоровый человек. Основные показатели: справочник / А. И. Киеня, Ю. И. Бандажевский. — Мн.: «Экоперспектива». — 1997.
2. Киеня, А. И. Международная система единиц в медицине и биологии / А. И. Киеня, Ю. И. Бандажевский. — Гомель, 1995.
3. Textbook of medical physiology // C. Guyton. — 1986. — 1057 p.
4. Human anatomy and physiology // Alexander P., Spence-Elliott B. Masson.
5. Human physiology. The mechanisms of body function // Arthur J. Vander James H Sherman Dorothy S. Luciano. — 1986. — 715 p.
6. Lecture notes on human physiology // John J Bray, Patricia A. Cragg, Anthony D. C. Macknight, Roland G. Mills and Douglass W. Taylor.
7. Human anatomy and physiology // Elaine N. Marieb. — 1989. — 995 p.
8. Review of medical Physiology, International edition. — 2003. — 912 p.

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