

Pediatrics: textbook

Про книгу

The national textbook was prepared in accordance with the academic program in pediatrics for pre-graduate training of specialists who major in dentistry. The given materials cover modern aspects of etiopathogenesis, diagnostics, treatment, and prophylaxis of the most common somatic and infectious childhood diseases as well as physiology and pathology of young children. It is recommended for students of dentistry faculties with the English-language form of training of higher medical education institutions of the 4th level of accreditation.

Pediatrics

Edited by Professor **T.O. KRYUCHKO**,
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SECOND EDITION, REVISED

Approved by the Ministry of Education and Science of Ukraine as a textbook for students of stomatological faculties of higher education establishments — medical universities, institutes and academies

Published in accordance with the Order of the Ministry of Health of Ukraine No. 502 as of 22 June 2010 as a national textbook for students of higher education establishments — medical universities, institutes and academies

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PREFACE

Pediatrics is the science that is continuously developing, and under the present conditions it requires constant search for the new methods of prophylaxis, diagnostics, and treatment of childhood diseases. Among others, the important tasks of the healthcare system include building the adequate level of medical aid delivery to children, which mainly depends on the qualification of regular personnel. The specialist involved in the delivery of medical aid to children must have a perfect sense of social responsibility, meet the child's needs for health and professional help from birth till teenage years. Children are extremely sensitive, with vulnerable psyche and special response to numerous factors, and therefore training of doctors with such qualities as sensibility, feeling of love and respect to the patient, high standards of professionalism, and constant need for self-improvement is very important.

Pediatrics is a standard educational subject, which is studied in all higher medical education institutions for the purpose of professional training of specialists, including those who major in dentistry. The national textbook was created for the purpose of providing future dental professionals with educational materials that contain up-to-date information on childhood physiology and pathology in the volume provided by the training program.

Chapter 4

CARDIOVASCULAR SYSTEM DISEASES IN CHILDREN. HEMORRHAGIC DISEASES IN CHILDREN

CARDIOVASCULAR SYSTEM DISEASES IN CHILDREN

Anatomy and Physiology of the Cardiovascular System in Children

The main fetal blood circulation is placental circulation in which processes of both enrichment of the blood with oxygen and release of CO₂ occur. There is one circle with fetal blood circulation pathway (ductus venosus, ductus arteriosus); tissues receive mixed blood (arterial-venous).

Blood circulation of the newborn. After the child is born and makes his/her first breath the lungs spread and fill with blood; the ductus venosus, foramen ovale and the remains of the umbilical vessels close and gradually become obliterated. The large and small circles of blood circulation begin to function in the newborn. Usually by the end of the 6th week of life the ductus arteriosus closes, by 2—3 months — the ductus venosus closes, by 6—7 months — the foramen ovale of the atrial septum closes.

The heart of the newborn takes a significant space of the chest; it is spherical and is located higher than in older children. The major axis of the heart is nearly horizontal due to the high position of the diaphragm.

In Table 34 you can see the description of the borders of relative cardiac dullness in children of different age groups.

Table 34

Borders of Relative Cardiac Dullness in Children of All Ages

Age, years	The Upper Border of the Heart	The Right Border of the Heart	The Left Border of the Heart
Younger than 2	The second rib	2 cm outward from lin. sternalis dextra	2 cm outward from lin. medioclavicularis sin.
2—6	The second intercostal space	1 cm outward from lin. sternalis dextra	1 cm outward from lin. medioclavicularis sin.
7—12	The top edge of the third rib	0.5 cm outward from lin. sternalis dextra	0.5 cm outward from lin. medioclavicularis sin.
Older than 12	The third rib or third intercostal space	On lin. sternalis dextra	On the line or 0.5 cm from lin. medioclavicularis sin.

Age characteristics of hemodynamic parameters. The most labile hemodynamic parameter is heart rate, which changes as the child grows and depends on external and internal factors (heart disease, endocrine disorders, anemia, etc.). Crying, restlessness, increased body temperature, movements can cause increased heart rate in children (Table 35).

Table 35

Heart Rate in Children of All Ages
(Maidannyk V.G., 2009)

Child's Age	Heart Rate, bpm	Child's Age	Heart Rate, bpm
Newborn	140—160	5 years	100
6 months	130—135	8 years	90
1 year	120—125	10 years	80—85
2 years	110—115	12 years and older	70—75
3 years	105—110		

Non-rheumatic Carditis

Non-rheumatic carditis (ICD-10: I40) is an inflammatory disease of the heart which has non-rheumatic and non-coronary nature. The disease arises under the action of various infectious agents, it is characterized by inflammatory infiltration of the myocardium with fibrosis, necrosis and/or degeneration of myocytes. The inflammatory process may involve cardiomyocytes, as well as the interstitial tissue, vessels, conductive system of the heart and pericardium.

Epidemiology. Numerous research findings and epidemiological data show an increase in non-coronary diseases of the myocardium, whose incidence is around 7—9 % of all diseases of the cardiovascular system. In Ukraine inflammatory myocardial lesions are detected in 2.3—9.0 % patients with heart diseases.

Etiology — see Fig. 28.

Children aged from 1 day to 19 years, who present with rapidly developing dilatation of the left ventricle and its dysfunction, show viral genome in 68 % of cases: enterovirus is found in 30 % of cases, adenovirus — in 58 %, herpesvirus — 8 %, cytomegalovirus — 4 %.

Chapter 4

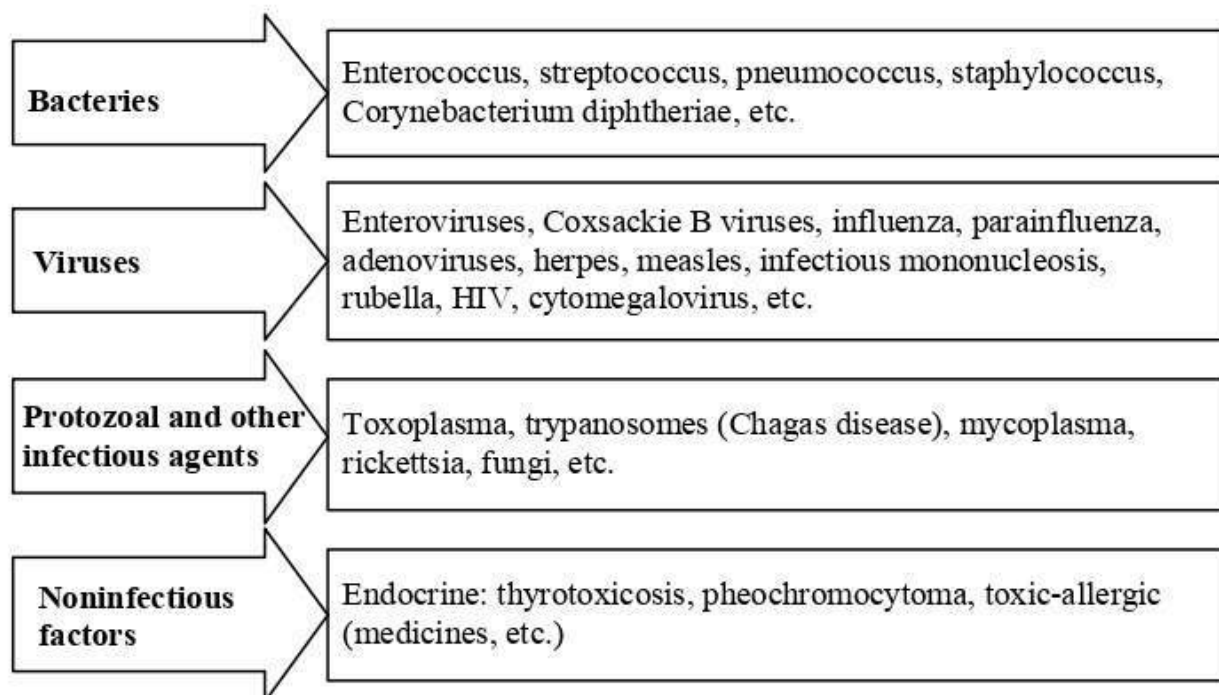


Fig. 28. Etiological factors of non-rheumatic carditis in children

Classification — see Table 36.

Table 36

Classification of Non-rheumatic Carditis in Children

Etiology	Clinical Presentation	Prevalence	Course	Pathogenesis	Anatomical Features
<ul style="list-style-type: none"> — Viral — Bacterial — Allergic — Parasitic — Protozoan 	<ul style="list-style-type: none"> — With rhythm disturbances — With pain syndrome — With heart failure — With thromboembolism of the lung artery — Oligosymptomatic 	<ul style="list-style-type: none"> — Focal — Diffuse 	<ul style="list-style-type: none"> — Acute — Subacute — Chronic 	<ul style="list-style-type: none"> — Primary infection — Infectious-allergic — Toxic-allergic 	<ul style="list-style-type: none"> — Parenchymal — Interstitial

Pathogenesis — see Fig. 29.

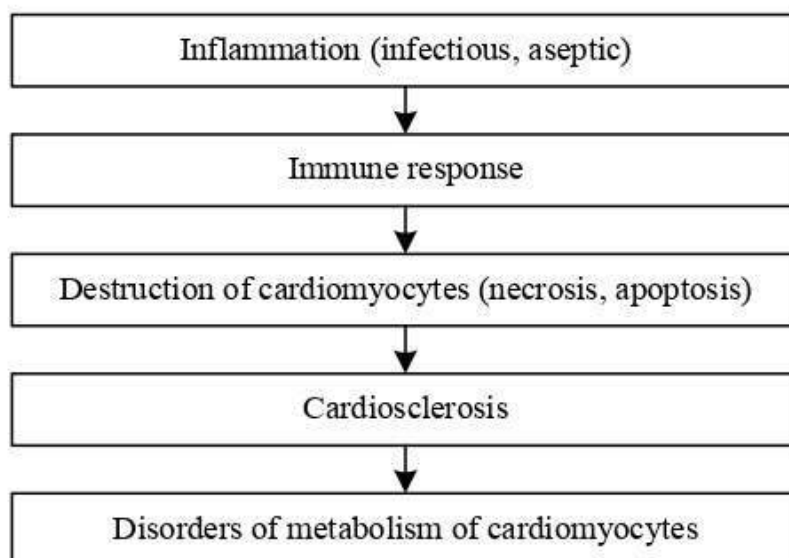


Fig. 29. Pathogenesis of non-rheumatic carditis in children

Clinical manifestations — see Table 37.

Table 37

**Clinical, Laboratory, and Instrumental Manifestations
of Non-rheumatic Carditis in Children**

Clinical Data
Malaise, unexplained muscle weakness, hypodynamia
Pain in the heart, sometimes intense, palpitations, interruptions, breathlessness
In some cases mild joint pain
Body temperature is often low-grade or normal
Blood pressure often decreases
Arterial hypertension is practically not found
Tachycardia
Expansion of the boundaries of relative dullness of the heart
Diffused and non-resistant apex beat
Weakened I heart sound
Systolic murmur at the apex of the heart
Symptoms of either left ventricular heart failure or total heart failure
Important, but not permanent signs of myocarditis are disturbances of heart rhythm (tachycardia, rarely bradycardia, ectopic arrhythmia) and conductivity, gallop rhythm
Paleness of the skin, cyanosis of the nasolabial triangle

Chapter 4

Diagnostics (Table 38). The criteria for clinical diagnosis of myocarditis have been proposed by the New York Heart Association (NYHA) (Table 39). The diagnosis is considered confirmed in the presence of a previous infection in combination with one major and two minor criteria.

Table 38

Laboratory and Instrumental Data for Non-rheumatic Carditis

Laboratory Data
ESR acceleration, neutrophilic leukocytosis, increase of C-reactive protein, sialic acids and seromucoid
Data of Instrumental Examinations
X-ray: cardiothoracic ratio increase
Electrocardiogram (ECG): reduction of the voltage of QRS complexes during the first 2—3 weeks of the disease; further — signs of disturbances of repolarization processes, overload of the left ventricle, perhaps — AV block, rhythm disturbances
Ultrasonography: dilation of the left ventricular cavity, rarely of the left atrium and right ventricle, decrease in the indexes of pumping and contractile function of the myocardium

Table 39

Diagnostic Criteria for Non-rheumatic Carditis (NYHA)

<i>Major criteria</i>	Pathological ECG changes (signs of ischemia, hypertrophy of the heart chambers)
	Cardiomegaly — an increase in the overall size of the heart or cavities (usually the left ventricle)
	Congestive heart failure or cardiogenic shock
	Increase of blood cardiac-specific enzymes (lactate dehydrogenase, malate dehydrogenase, creatine phosphokinase) and cardiac-specific proteins (troponin T, I, etc.)
<i>Minor criteria</i>	Tachycardia
	Weakening of the I heart sound
	Gallop rhythm

Treatment. Hospitalization, bed rest in the acute period; its duration depends on the severity of carditis: in moderate forms — from 3 to 5 weeks, in severe — 8 weeks or more. The diet enriched with potassium (raisins, dried apricots, nuts, bananas, baked potatoes, buckwheat). In the acute phase the

foods that cause stimulation of the cardiovascular system should be eliminated: coffee, strong tea, fried food, etc. It is recommended to limit salt and fluid in heart failure.

Anti-inflammatory therapy is carried out for 3—5 weeks, depending on the course of the disease. The main NSAIDs and their dosage in the treatment of non-rheumatic carditis:

- diclofenac 2.5—3 mg/kg/day in 3—4 doses;
- ibuprofen 30—40 mg/kg/day in 3—4 doses;
- indomethacin 2.5—3 mg/kg/day in 3—4 doses.

In a severe course of carditis accompanied by cardiomegaly, heart failure, pericarditis it is necessary to prescribe prednisolone, 0.8—1.5 mg/kg per day for 2—3 weeks. In a protracted course of carditis quinolines should be prescribed (chloroquine or hydroxychloroquine in the dosage of 5—8 mg/kg/ day) for 6—9 months because of the risk of autoallergic reactions (Fig. 30).

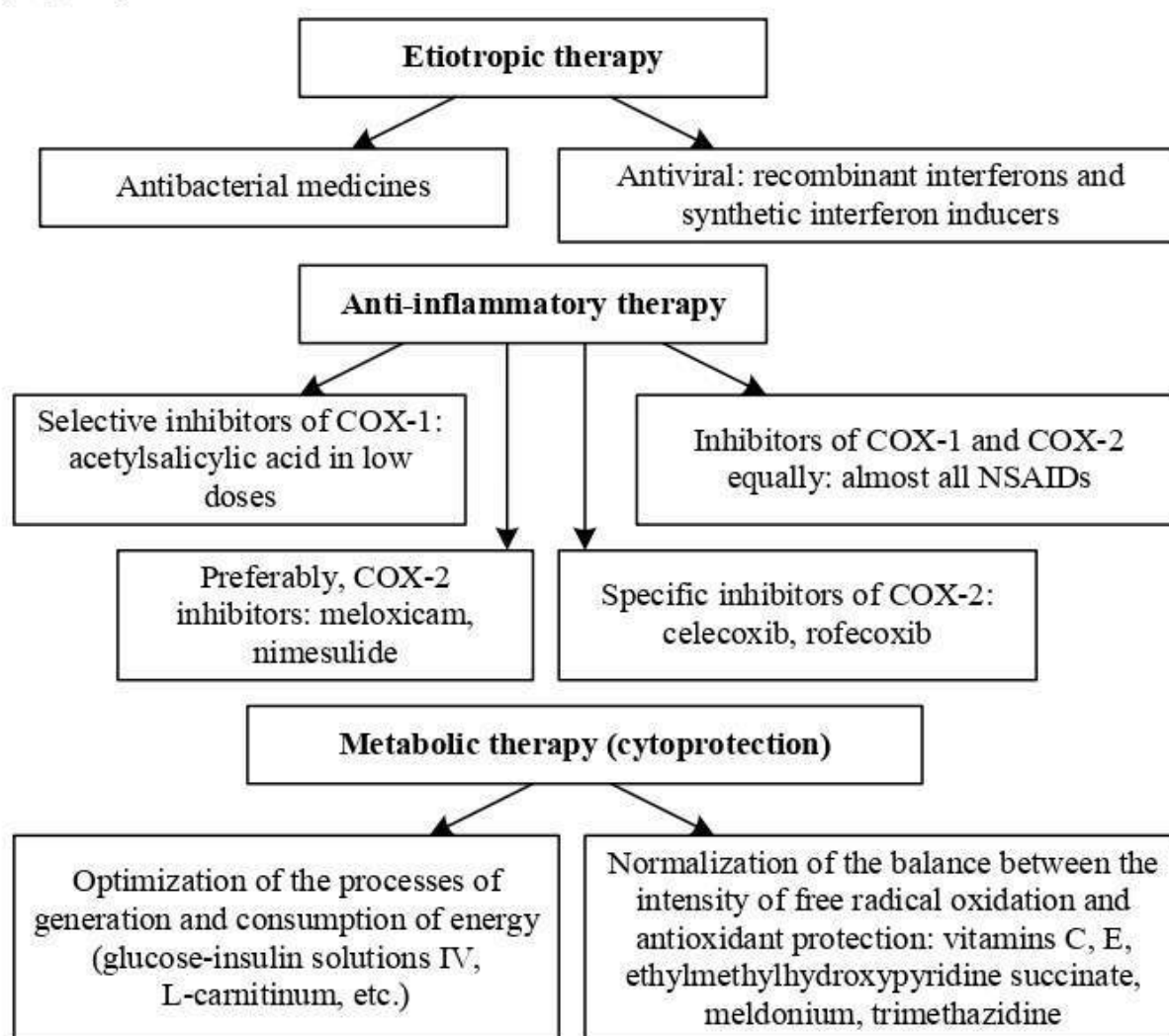


Fig. 30. The principles of medical treatment of non-rheumatic carditis in children

Chapter 4

Metabolic therapy for myocarditis concerns cytoprotection and optimization of cardiomyocyte metabolism under conditions of myocardium inflammation. Cardonat, trimetazidine and L-carnitine are administered. The average duration is 4 weeks. Therapy should be combined with ATP, Espalipon, riboxin. The second direction of the cytoprotective therapy for myocarditis is normalization of the balance between free radical oxidation and antioxidant protection. It is recommended to use natural antioxidants (vitamins C, E) or xenobiotics which inactivate free radicals.

Prognosis. Complete recovery for the majority of patients during the first year of the disease is common. However, besides recovery, carditis consequences are possible: dilated cardiomyopathy, myocardiosclerosis, sudden death.

Acute Rheumatic Fever

Acute rheumatic fever (ICD-10: I00-I02) is a systemic inflammatory disease of the connective tissue, mainly affecting the cardiovascular system (rheumatic heart disease), joints (migratory polyarthritis), nervous system (chorea), skin (annular erythema, rheumatic nodules) and other organs with toxico-immunological mechanism of development in children with genetic predisposition, on the infection background with group A beta-hemolytic streptococcus.

Epidemiology. Rheumatic diseases rank third among other types of pathology in adults. The morbidity of children with acute rheumatic fever in Ukraine is 0.04 per 1000 children.

Etiology. The only etiological pathogen of ARF is group A beta-hemolytic streptococcus.

Pathogenesis — see Fig. 31.

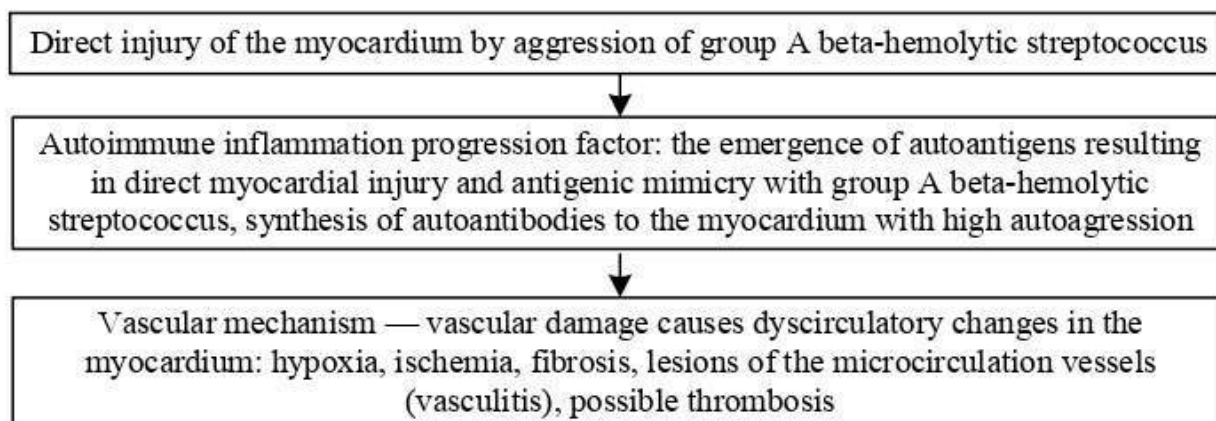


Fig. 31. Pathogenesis of acute rheumatic fever

Classification of rheumatic fever (Table 40) was offered by the Institute of Rheumatology (2001) and modified by the Ukrainian Association of Rheumatologists (2004).

Table 40

Acute Rheumatic Fever Classification

Clinical Manifestations		Degree of Activity	Stages of Circulatory Disorders	NYHA Functional Class
Main	Additional			
— Carditis — Arthritis — Chorea — Annular erythema — Rheumatic nodules	— Fever — Arthralgia — Abdominal syndrome — Serositis	III — high II — moderate I — minimal	0, I, IIA, IIB, III	0, I, II, III, IV

Clinical manifestations — see Table 41.

Table 41

Peculiarities of Acute Rheumatic Fever in Children

Decreased severity of the disease; catastrophic variants of the disease have virtually disappeared
Predominance of primary morbidity in older children (12—18 years)
Decreased exudative inflammation due to a moderate degree and minimum activity that leads to reduction of pancarditis, polyserositis and visceritis
In childhood nervous system involvement occurs in the form of small chorea
During recurrent attacks of ARF, carditis is most common
Among children extracardiac manifestations of the disease in the form of polyarthritis, annular erythema, rheumatic nodules are more common than among adults
There are new clinical forms of ARF such as autoimmune neuropsychiatric disorders associated with streptococcal infection
Children show a more prominent effect from timely rational therapy than adults

Diagnostics. We use the diagnostic criteria for rheumatic fever (Kisel—Jones) supplemented by A.I. Nesterov, reviewed by the American Heart Association (1992), and modified by the Association of Rheumatology (2003).

Chapter 4

The presence of two major or one major and two minor criteria indicate a high probability of ARF (Table 42).

Table 42

Diagnostic Criteria of Acute Rheumatic Fever

Major Criteria	Minor Criteria
Carditis	Arthralgia
Polyarthrititis	Fever
Annular erythema	Increased level of the acute phase reagents (ESR, CRP)
Chorea	Prolongation of the PR interval on ECG
Subcutaneous rheumatic nodules	Symptoms of mitral and/or aortic regurgitation with Doppler echocardiography
	Data confirming GAS infection: beta-positive streptococcal culture, taken from the oropharynx, or rapid determination of streptococcal antigen positive test; elevated titers of antistreptococcal antibodies (antistreptolysin O, anti-DNA-polymerase B) twofold or more

Treatment. First stage — hospitalization. The regimen in the acute phase is bed rest (on average 3—6 weeks). Activity is increased gradually. Diet with a high content of protein, vitamins, restriction of salt and carbohydrates.

The second stage of treatment involves staying in hospital for further treatment to achieve full remission within 30—45 days.

The third stage — clinical observation, follow-up observation, and *preventive treatment*.

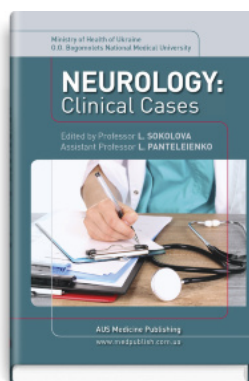
Medical therapy is aimed at two main links of the pathogenesis — the infectious agent and immunopathological inflammatory reaction (Table 43).

Table 43

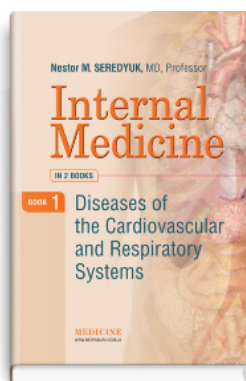
Acute Rheumatic Fever Treatment

Therapy	Name and Dose of the Drug	Indications
<i>Antibacterial for primary treatment and prevention</i>	Penicillin G 20000—50000 IU/kg/day, 4 times a day, during 10—14 days, intramuscularly	The first attack of ARF and its recurrences

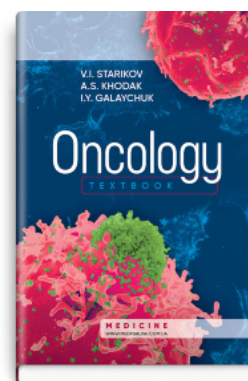
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