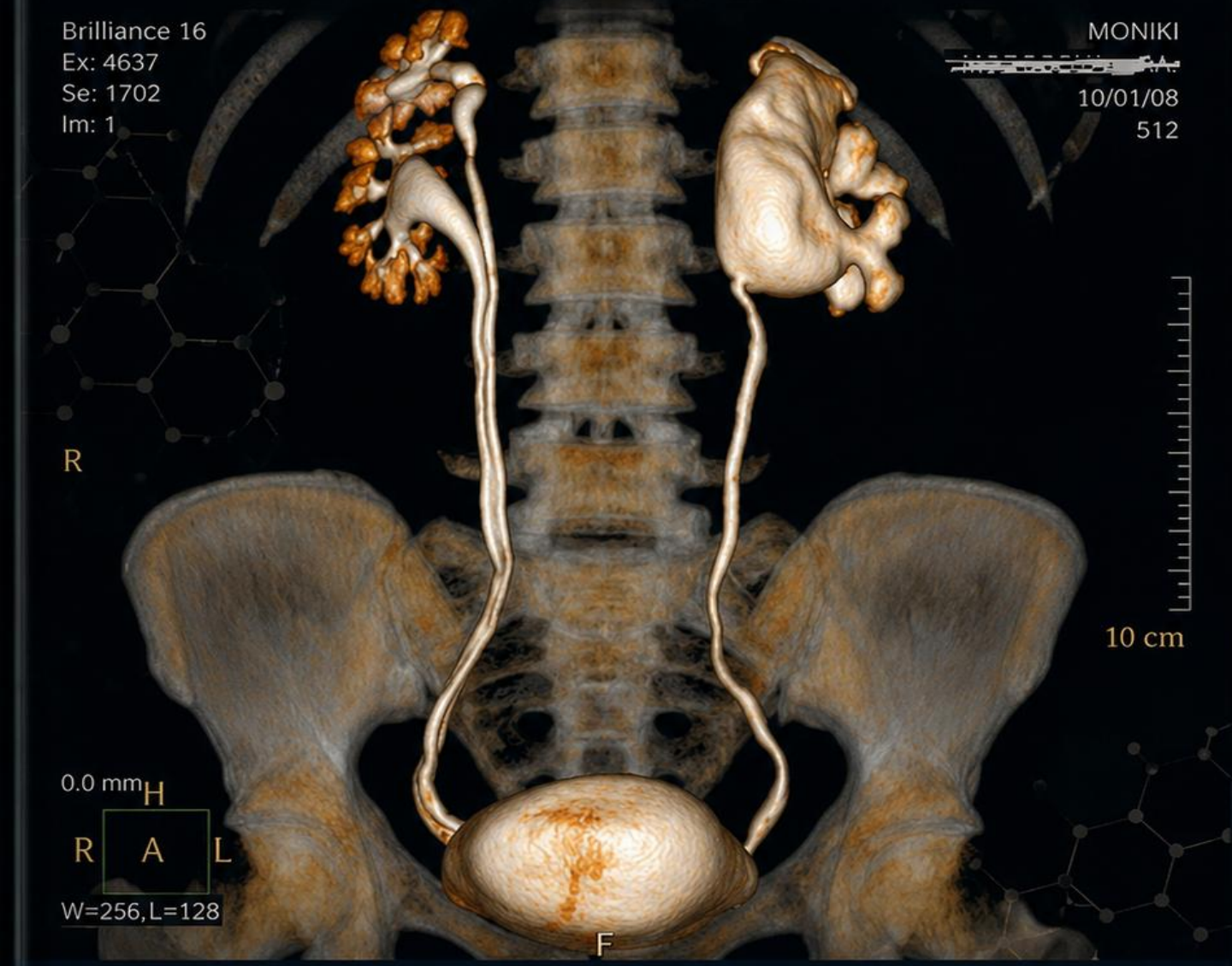
A. Akhmatov

PEDIATRIC NEPHROLOGY

STUDY GUIDE



EVIDENCE-BASED APPROACH

CHILD-CENTERED CARE

CLINICAL PRACTICE ESSENTIALS

FOR STUDENTS AND HEALTHCARE PROFESSIONALS

CARING FOR KIDNEYS. PROTECTING FUTURES.

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MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN
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PEDIATRIC NEPHROLOGY

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LIST OF ABBREVIATIONS

AB – antibiotics
A/P – arterial pressure
ADH – antidiuretic hormone
AST - alanine aminotransferase
ALT - aspartate aminotransferase
ATP – adenosine triphosphate
BM – basement membrane
KF – kidney failure
KRT – kidney replacement therapy
GD – hemodialysis
GU – hyperuricemia
GUU - hyperuricosuria
HUS – hemolytic uremic syndrome
GGFRT – hypoxanthine – guanine – phosphoribosyl transferase
GFR – glomerular filtration rate
DMN - dysmetabolic nephropathy
DFAT – diphenylamine test
Ig – immunoglobulin
XO – xanthine oxidase
LN – lupus nephritis
Hb – hemoglobin
OKC - oxalate - calcium crystalluria
PN – pyelonephritis
MEDA – minor external developmental anomalies
RCI – renal-cortical index
TRP – tubular reabsorption of phosphorus
UO – urinary organs
CrRF – chronic renal failure
CKD – chronic kidney disease
UTI – Urinary tract infection
ARF – acute renal failure
ARVI – acute respiratory viral infection
FA – phosphoethanolamine
IU – International Unit
EA – ethanolamine
EKD – exudative catarrhal diathesis (Allergic diathesis)
ESR – erythrocyte sedimentation rate
SGA – supraglomerular apparatus

INTRODUCTION

Among the diseases encountered in the daily work of a pediatrician, a doctor who deals with the laws of development of the child's body and the protection of its health, diseases of the urinary organs occupy a significant place. 5-6% of patients treated in children's hospitals are patients with kidney diseases. Considering that in adults with chronic renal failure, the disease often begins in early childhood, the urgency of this problem becomes even more apparent.

The kidney is not only an organ for excreting urine, but its function in ensuring the constancy of the internal environment of the body (homeostasis) is of particular importance. Pediatric nephrology studies the importance of kidney function in ensuring the constancy of the internal environment of the body at all stages of childhood, the significance of its disorders for the growing organism as a result of its diseases, their diagnosis, treatment and prevention methods. In recent years, pediatricians-nephrologists have noted that kidney diseases are becoming more common in children, and their clinical course is changing. Pediatric nephrology studies the importance of kidney function in ensuring the constancy of the internal environment of the body at all stages of childhood, the significance of its disorders for the growing organism as a result of its diseases, their diagnosis, treatment and prevention methods.

Large-scale epidemiological studies show that 30-50 out of 1000 examined children have kidney diseases. At the same time, the prevalence of kidney diseases is not the same among the population living in different geographical regions. In particular, Central Asia, including the region of Uzbekistan, is known as an endemic foci of kidney stones. Studies conducted over the past 30 years show that this region is endemic not only for the disease of stone formation, but also for the development of a much more common form of oxalate-calcium nephropathy.

Currently, glomerulonephritis and pyelonephritis in children have been sufficiently studied. However, with the development of nephrology, it has become clear that kidney diseases are very diverse in terms of their causes. In particular, hereditary nephritis, tubulopathies, diseases caused by metabolic disorders, and secondary nephropathies arising in connection with other diseases have begun to be distinguished from the above-mentioned group of diseases , which makes it possible to consistently carry out their treatment.

This textbook covers the specific features of kidney function in children, kidney diseases, urinary tract infections, pyelonephritis, hereditary kidney diseases, dysmetabolic nephropathies, acute and chronic renal failure, their diagnosis, treatment, prevention, and dispensary control in simple, student-friendly language.

The textbook entitled “Pediatric Nephrology” is intended for master’s degree residents of medical higher education institutions studying the subject “Emergency Conditions in Pediatric Nephrology.” It serves as one of the important resources prepared in English to facilitate their thorough mastery of both theoretical knowledge and practical skills.

The manual has been written in accordance with the curriculum requirements of the Ministry of Higher Education, Science and Innovations of the Republic of Uzbekistan.

CHAPTER I
PHYSIOLOGY OF RENAL FUNCTION AND ITS SPECIFIC FEATURES IN CHILDREN
PHYSIOLOGY OF KIDNEY FUNCTION

At present, the function of the kidneys and their significance for the body have been sufficiently studied, and in this regard, it is necessary to clearly distinguish two concepts from each other: one is the function of the kidneys themselves, and the other is the processes that ensure this function. The function of the kidneys consists of: - maintaining the constancy of the amount of osmotically active substances in the blood and other body fluids (osmoregulation), - maintaining the constancy of the volume of blood and extracellular fluids (volume regulation), - maintaining the balance of acidic and alkaline substances in the body, maintaining the balance of the ionic composition of the blood, removing foreign substances and end products of nitrogen metabolism from the body, removing excess organic substances in the body (amino acids, glucose, etc.); participating in the metabolism of proteins, fats and carbohydrates; ensuring the normalization of blood pressure, blood formation, and the exchange of biological substances (renin, vitamin D metabolism, etc.) (Fig. 1).

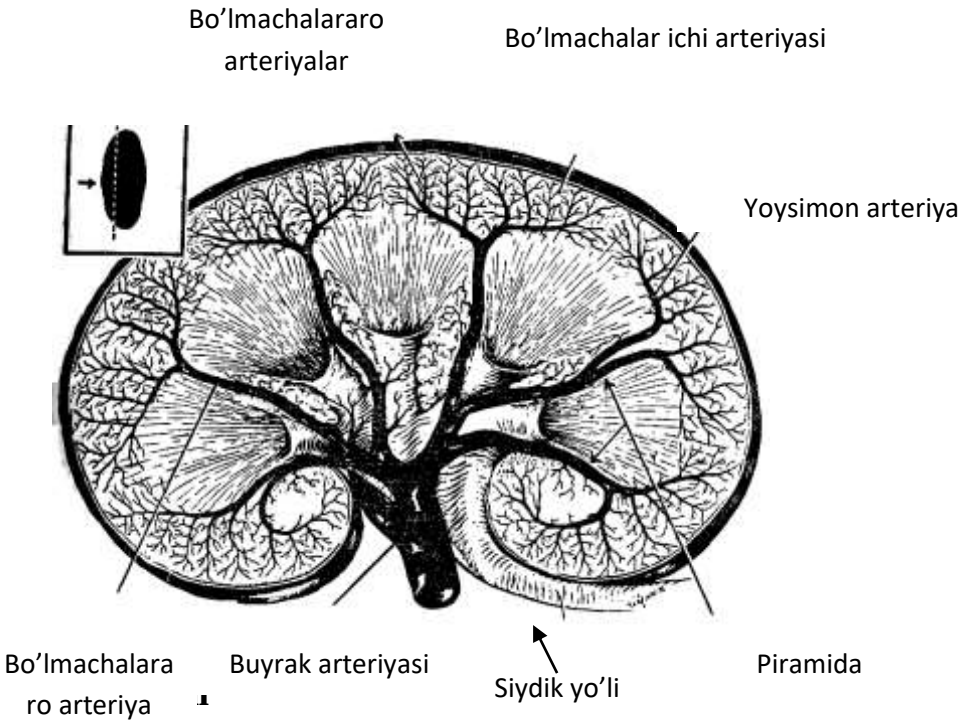


Figure 1. Human kidney inside arteries distribution (G.Galabov according to cited, 1976).

Kidney counting passed variety activity nephron in the wrappers ultrafiltration, in tubes reabsorption and secretion, kidney in tissues some synthesis of substances on account of done increases. Dehydration of the body or on the contrary liquid separation broken, swollen appearance to be, blood pressure exceed departure etc. often kidney activity and in the kidney fashion exchange from the violation evidence gives.

Filtration in the renal tubules. The filtration activity of the kidneys begins as early as 3 months of pregnancy (DeryuginaL.A., Chekhonaskaya ML, 2005). Therefore, a certain amount of urine is always present in the urinary bladders of infants. Urine excretion in the renal tubules occurs as a result of fluid filtration through the basal membrane by diffusion due to the fact that the diameter of the efferent arteriole is 3 times smaller than the arteriole entering it, and due to the presence of hydrostatic pressure in the blood vessels of the tubules . Therefore, the filtration rate mainly depends on the hydrostatic pressure in the blood vessels forming the tubules . When compared to the body surface of an adult (1.73 m^2), 1200 ml of blood flows through the kidneys every minute, which in total is 20-25% of the amount of blood pumped by the heart during this time. Thus, the renal artery is a fairly large artery, and since it starts directly from the aorta, the blood pressure in it is also relatively high. 20-25% of the blood volume passing through the kidneys passes through the vessels of the renal cortex, where the renal tubules are located. 6-8% of the blood flows through the outer part of the renal medulla, less than 1% through the inner part. Approximately 19% of the blood serum flowing through the kidneys is filtered every minute. Filtration is not an active process, but occurs due to the difference in hydrostatic pressure in the capillaries of the tubules (45-52 mm Hg) and in the interstitial space (8-15 mm Hg). In this sense, the total oncotic pressure of the blood serum (18-26 mm Hg) is also important. Therefore, when blood pressure decreases, difficulty arises in the excretion of urine, and when pressure in the urinary tubules increases, filtration also slows down. In infants and young children, due to the anatomical immaturity of the nephrons, filtration is much slower than in adults. The osmolarity of the first urine secreted by the nephron is equal to that of blood serum. Substances with a large molecular weight cannot pass through the small pores in the basement membrane during filtration. The molecular weight of albumin is 69,000, the diameter of the molecule is 3.55 nm, and less than 1% of it is filtered. In an adult, the amount of ultrafiltrate secreted by both kidneys is 120–130 ml per minute relative to the body surface area (1.73 m^2). In newborns, it is 4 times less.

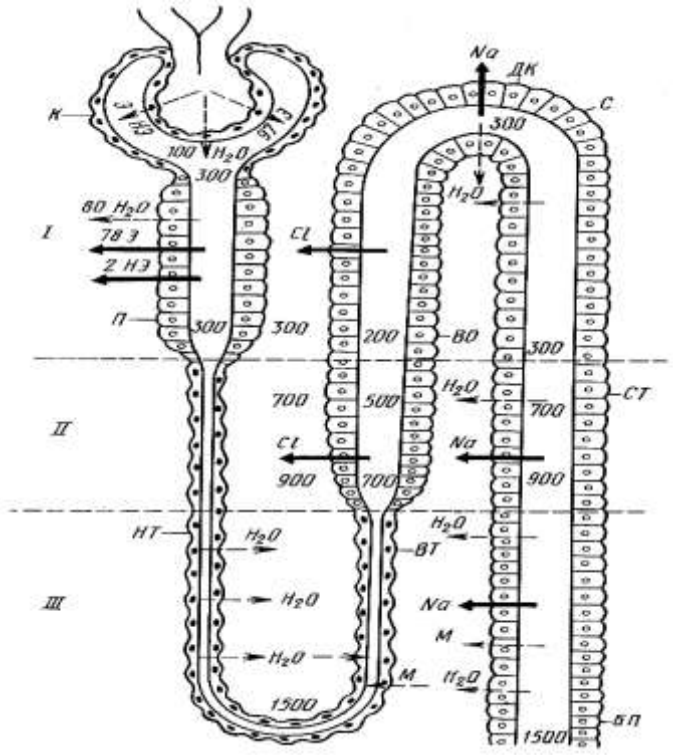
Electrolytes The filtration rate depends on the extent to which they are bound to proteins in the blood. For example, 40% of Ca^{++} is bound to albumin,

53% of which is excreted in the initial urine in an ionic state. Serum proteins are negatively charged (-), so they bind some cations.

The filtration process in the nephron tubules stops when the arterial blood pressure drops to 50 mm Hg. The filtration process is controlled by factors within and outside the kidney. Recent studies have shown that each nephron also has the property of self-regulation. This function is carried out by the supraglomerular apparatus (SGA) of the nephron, and the pressure on the afferent arteriole wall, and the macula densa in the tubules, which senses the amount of sodium chloride in the initial urine, affects the blood circulation and reabsorption processes in the nephron tubules.

The kidney's function is to osmotically thicken (concentrate) and dilute urine.

All structures of the kidney - ureters, blood vessels, connective tissue - participate in the activity of the kidney to maintain a constant level of biologically active substances in the blood. Depending on the state of water balance in the body, the kidneys can secrete urine with a low specific gravity (hypotonic) or concentrated (osmotically concentrated, hypertonic) (Figure 2).



2 - rasm. *In the nephron urine thickening (concentration) process (NatochinYu.V., 1983): I – Kidney bark part II – external kernel Article III – internal kernel substance; G - glomerulus (kidney balls); P – tube proximal Part 1; VT – Henle of the ring up rising thin part; VO – Henle of the ring up rising tide part; DK – bent distal tube; S – connector part; ST – collector tube; E – electrolyte; NE – nonelectrolyte substances.*

Urine thickening process essence from that What is a nephron? in their wrappers filtered primary 2/3 of urine kidney of the tubes in the initial (proximal) part again absorbed (reabsorption). The remaining blood serum with one kind comparison to the weight has (isoosmotic) part and urine tubes through kidney kernel in part located, Henle of the ring down directed thin to the part will pass and later 180 ^{0} corner harvest as bent, down descending to the part parallel located and up looking at directed to the part will pass, that is again kidney bark to the part looking at Urine of the tubes up directed in part water not absorbed, but his/her cells sodium and chlorides without water again sucking, surrounding additional in tissues high osmotic pressure harvest does. As a result harvest was osmotic difference water for conductive Genly of the ring down directed in part of liquid surroundings additive to the tissues the passage facilitates. Osmotic in terms of active was substances and of tubes in the void still, tube along passed yellow urine concentrated, its comparison weight exceed Goes. Genly of the ring when it reaches its peak urine size one how many once decreasing, its comparison weight to that towards sharp increases. Genly of the ring up ascending part and of the tube in the last (distal) part water again It does n't work , but sodium and chlorides absorption, above as said without water continue Urine concentration process in the pituitary gland working removable antidiuretic with the participation of hormone (ADG) managed The organism when dehydrated ADG working The release is powerful . and his/her impact under urine in the tubes of water again The absorption is powerful . and on the contrary to be possible. But nephron tubes to the cells ADG of impact power of the environment ion to the composition also garden It will be. This effects, such as hypercalcemia if observed or potassium deficiency decreasing departure possible. Urine osmotic to the process of thickening (concentration) one row pharmacological of substances impact to be able from the opportunity nephrological in clinics used (urine driver drugs and (bq). Kidney kernel in part in the body water from the norm more when (hyperhydration condition) of urine osmotic Density 500 mosmol/kg N2O to until decreases and on the contrary when (dehydration) condition) osmotic pressure of urine density 1400 mosmol/kgN_2O to until increases. Due to the process of osmotic thickening, the osmotic pressure of urine entering the renal pelvis is 1400 - 1450 mosmol/kg N_2O. In ensuring sufficient osmotic pressure of urine, osmotically active substances such as glucose, protein, urea, sodium are of great importance. Urea has a greater effect on the inner layer of the medulla, and sodium on its outer layer. It is in the inner layer of the medulla that the effect of antidiuretic hormone is also manifested, ensuring the reabsorption of fluid and urea from the collecting ducts.

Reabsorption and secretion processes in the renal tubules

The function of the proximal part of the renal tubules, which begins at the renal corpuscles, is to reabsorb the filtrate formed in the tubules, and in this part, 60% of water and sodium are reabsorbed into the blood.

The reabsorption of water and sodium ions partially causes the reabsorption of amino acids and glucose, that is, it occurs by secondary active absorption. This process also causes the partial passage of urea and other uncharged compounds from the lumen of the tubules into the tissues. In the proximal renal tubules, the reabsorption of water and chemical compounds occurs not only through the epithelial membranes, but also partially through the interstitial spaces. 1/3 of the reabsorbed sodium is actively absorbed, and 2/3 is diffused into the tissues connecting the cells. The process of active transport of substances occurs at the expense of a certain amount of energy, which is generated by the metabolism of ATP in the cells.

Kidney incretory function

A number of biologically active substances are produced in the kidney tissue, which have both local and broad-spectrum effects on the body. In particular, the supraglomerular system of the kidney secretes renin, which participates in the activity of the angiotensin system, which is involved in the tension of blood vessels, the filtration process in the renal tubules, and the control of the total volume of blood in the vessels.

Prostaglandins E and F, which are processed in the kidneys and enter the blood, increase blood circulation and sodium excretion in the kidneys. The kidneys also play an active role in ensuring blood clotting, and the urokinase produced here is considered an agent of the blood clotting system.

The kidneys produce a hormone (erythropoietin) that stimulates erythropoiesis. In particular, erythrogen produced in the kidneys is activated by hepatic erythropoietinogens and is released into the blood as erythropoietin.

It is clear that the normal functioning of the kidney is ensured by the coordinated work of all its systems (intercellular tissue, nephron, blood and lymphatic vessels). Therefore, any disruption of kidney function can lead to a disruption of the internal environment of the entire organism.

Maintaining acid-base balance in the body

The importance of the kidneys

In the process of constant metabolism in the human body, products that predispose to the development of acidosis naturally appear. However, the blood environment (pN) is maintained at a constant level due to the existing systems that provide acid-base balance, pH in cells is 7.0, pH in blood serum is 7.4, that is, it is at a level suitable for ensuring the activity of the majority of enzymes. Otherwise, the activity of some enzymes changes, leading to metabolic disorders. In particular, in an acidotic environment (pH - 6.8), the activity of cathepsin in cells increases, and, in turn, the activity of the enzymes of the oxidation-reduction system decreases. As a result, the cell may die. In a healthy

organism, this balance is corrected with the participation of special buffer systems. There are four types of buffer systems in the body: 1) hemoglobin - oxyhemoglobin, 2) carbonic acid - hydrocarbonate, 3) blood proteins, 4) phosphates. Of these, the hemoglobin-oxyhemoglobin buffer system is the most powerful, with its capacity being 9 times greater than that of the hydrocarbon system and 4 times greater than that of plasma proteins.

Under physiological conditions, the kidneys participate in several ways to maintain acid-base balance in the body.

1. *Filtration in the renal medulla.*
2. *Acidogenesis is the formation and release (secretion) of hydrogen ions.*
3. *Ammoniogenesis is the formation and secretion of ammonia in the cells of the renal tubules.*
4. *Reabsorption of bicarbonates and excretion of most of them.*
5. *Excretion of weak organic acids.*
6. *All of the above processes can only be fully realized if the filtration rate in the renal tubules is within the norm. A decrease in filtration rate to 30 ml/min can lead to the development of metabolic acidosis.*

The blood, are excreted into the urine by active secretion in the renal tubules - hydrogen ions in the urine can be up to 1000 mEq higher than in the blood serum, and in this regard, the pH of the urine can drop to 4.5 (Ginesinsky AG, 1963). Hydrogen ions that have passed into the lumen of the renal tubules are incorporated into the phosphate buffer system in the urine. The total amount of free and bound hydrogen ions is usually **determined as an indicator of titratable acidity.** Healthy kidneys secrete 30 - 40 mEq / l of titratable acids per day.

In the renal tubules, ammonia is formed during the metabolism of amino acids and is secreted into the urine. The kidneys participate in maintaining the balance of hydrocarbons in the body through filtration and reabsorption, 4000 - 5000 mEq / l of bicarbonate is filtered through the renal medulla during the day and night, and 80 - 85% of it is reabsorbed in the proximal tubules. The reabsorption of hydrocarbonate anions in the proximal tubules is 2 - 2.5 times faster than in the distal part. All processes in the renal tubules are interconnected, and the rate of reabsorption of hydrocarbonates also depends on a number of factors, such as the amount of carbon dioxide, hydrogen ions and potassium in the blood. From the above, it is clear that depending on which part of the nephron system is damaged, acid-base imbalance can develop into glomerular acidosis, renal tubular acidosis, and hyperchloremic acidosis.

CHAPTER II
THE MAIN SYMPTOMS AND EXAMINATION METHODS OF KIDNEY DISEASE IN CHILDREN.

Analysis of clinical signs (symptoms) and their complexes (syndrome) observed in a sick child serves to assess the type of disease, its course, severity, and kidney function, and on this basis to determine the nature and extent of treatment.

Usually, in addition to the symptoms that are generally characteristic of kidney diseases and develop due to a violation of the body's ability to maintain internal balance - pallor, weakness, loss of appetite (intoxication), three main sets of symptoms are observed (changes in the amount and composition of urine, edema, hypertension). Their occurrence depends on the type, form and course of the disease.

General properties of urine

For qualitative analysis of urine, morning, single-time urine is usually examined. Certain tests require certain periods and conditions. For example, to determine alimentary glucosuria, urine is examined in the morning before and after meals. To assess orthostatic proteinuria, urine is examined in the morning before waking up and after half an hour of movement after standing. A three-cup test is used to diagnose certain diseases (cystitis, urethritis): the first part of the urine is collected in the first cup, the middle part in the second, and the last part in the third and is examined separately. To determine the amount of chemicals in the urine (uric acid, urates, calcium, etc.), urine is collected for 6, 12 hours, or often 24 hours. The daily amount of urine can usually vary in healthy children depending on their diet, the amount of fluid consumed, and the season. In particular, the daily urine output is highest in winter and spring, and lowest in summer: in summer, the daily urine output is reduced by 37.5% compared to winter and spring. This is due to fluid loss through the skin and respiratory tract. At the same time, there are quantitative changes in the composition of urine in summer: in particular, the content of urates, oxalates and inorganic phosphates increases. This should be taken into account when monitoring a patient with nephropathy caused by metabolic disorders (dysmetabolic nephropathy).

, the process of urine excretion has a diurnal (day-night) variation (rhythm): during the day-night (day) period, the amplitude of urine excretion varies from + 38.3% (from 19:00 to 24:00) to - 44.7% (from 15:00 to 19:00) of the average amount every three hours · The maximum diuresis occurs between 15:00 and 18:00 (0.26 ml/min). Accordingly, there are quantitative changes in the composition of urine, for example, the calcium content in it changes from + 44.6% (from 24:00 to 9:00) to - 32.3% (from 18:00 to 24:00) compared to the average

amount every 3 hours of the day, ammonia from + 37.4% (from 12:00 to 22:00) to - 24.1% (from 24:00 to 9:00), the content of titratable acids from + 49.2% (from 15:00 to 24:00) to - 37.3% (from 6:00 to 9:00) , etc. (Table 1).

The lowest diuresis during the day and night occurs from 15:00 to 18:00 (-44.7%), at this time the largest amount of phosphorus (+29.6%), ammonia (+37.4%), sodium (+56.5%), amino nitrogens (+26%) is excreted with urine. These features of kidney function must be taken into account in medical practice.

Urinary syndrome

Pathological changes in urine of varying degrees and proportions are observed in various kidney diseases. Urinalysis allows you to diagnose the disease, assess its severity, and assess the effectiveness of the treatment.

Erythrocyturia **is** a pathological sign, which can be detected when examining the urine sediment under a microscope, more than 3 erythrocytes per field of view, when quantitatively counting erythrocytes, it is possible to determine the release of erythrocytes of more than 1000 per ml according to the Nechiporenko method, 1000000 per day according to the Kakovsky-Addis method, and more than 1000 per minute according to the Amburger method.

of erythrocyturia : 1) blood clotting disorders, i.e. causes not directly related to the kidneys; 2) kidney diseases; 3) urinary tract diseases.

In patients with erythrocyturia due to the causes of the first group (blood coagulation disorders), it is important to find other symptoms of the underlying disease - bleeding, slow blood clotting , skin rashes. In such cases, special tests (blood clotting rate, fibrinolysis, platelet count) allow to establish the diagnosis of the disease. In diseases of the urinary tract, the combination of erythrocyturia with leukocyturia, back pain, bacteriuria is more common in secondary pyelonephritis and requires X-ray examinations to identify diseases such as congenital malformations of the urinary tract, urolithiasis. Along with the above symptoms, dysuric (painful urination) symptoms are observed in inflammation of the lower urinary tract (cystitis, urethritis). Microhematuria (hematuria - urine mixed with blood), which can increase from time to time, is also observed in hereditary nephritis, tubulopathies . In acute glomerulonephritis, hematuria is accompanied by edema, oliguria, cylindruria, proteinuria, and increased blood pressure, which facilitates the diagnosis. The detection of altered, destroyed erythrocytes in the urine sediment is a characteristic sign of glomerulonephritis. However, there are a number of nephropathies associated with metabolic disorders (urate, oxalate nephropathies, cystinuria, etc.), in which micro- and sometimes macrohematuria can occur without external symptoms observed in kidney diseases such as edema and hypertension. In such cases, to determine the cause of erythrocyturia, it is necessary to carefully study the patient's family

history and use special biochemical tests. In patients with glomerulonephritis, hematuria is caused by the destruction of the basal membrane of the renal corpuscles and increased permeability of the blood vessel walls.

Proteinuria is a relatively common urinary symptom. A healthy child excretes 30–60 mg of protein in their urine daily, mainly low molecular weight albumin, an amount that cannot be detected by conventional testing methods using simple methods.

Pathological proteinuria is observed when the permeability of the renal tubules increases (glomerular proteinuria), when protein reabsorption from the urinary tubules is impaired (tubular proteinuria), or when both processes are impaired (mixed proteinuria). Determining the ratio of proteinuria to other signs is important for diagnosis. In particular, proteinuria of more than 3 grams per day, leading to hypoproteinemia, is characteristic of glomerulonephritis with nephrotic syndrome. The combination of proteinuria with edema, hypertension, oliguria is observed in acute glomerulonephritis, accompanied by abdominal pain in urolithiasis and pyelonephritis, proteinuria and hypertension are observed in kidney tumors, in defects in the development of renal blood vessels. The combination of impaired skeletal bone formation and proteinuria is a characteristic feature of tubulopathies. Proteinuria and erythrocyturia are also found in hereditary nephropathies (hereditary nephritis, dysmetabolic nephropathies).

Proteinuria is the excretion of protein in the urine, an important criterion for the diagnosis of kidney and urinary tract diseases. This indicator also serves as a criterion for assessing the effectiveness of kidney disease treatment and determining the prognosis of the disease. In most healthy people, the amount of protein in the urine is 0.033 g/l, which is 50 mg per day. However, in 10-15% of healthy people, it can be 0.150 g/day (Emmanuel, 2007).

There are three levels of proteinuria in kidney disease:

- Mild proteinuria - from 300 mg to 1.0 grams per day, observed in inflammatory diseases of the urinary tract and kidneys, tubulopathies, urolithiasis, and chronic interstitial nephritis.

- Moderate proteinuria - from 1.0 to 3.0 grams of protein per day is excreted in the urine and is observed in primary and secondary glomerulonephritis, in the proteinuric stage of amyloidosis, and in hepatorenal syndrome.

- Severe, or nephrotic, proteinuria – more than 3.0 grams per day – is seen in nephrotic syndrome.

The procedure for determining daily proteinuria is as follows: For example, if a patient has 0.66 g/l of protein in his urine and his daily urine output is 500 ml, then the daily proteinuria is 0.33 g/day, or 330 mg. If for some reason

it is not possible to accurately calculate the daily urine volume, there is a method for determining the amount of proteinuria by calculating the ratio of the amount of protein in the urine to the creatinine (U_{cr}) in the morning urine volume (U_{pr}/U_{cr}) (SchawabS.J. et. al, 1987; Ruggenenti. etal., 1998). In this method, calculations are made in mg/dl and correspond to the daily amount of protein in grams. For example, if the patient's morning urine protein content is 150 mg/dl and creatinine is 50 mg/dl, then the daily proteinuria (150 : 50) is 3 g/24 hours. However, since the SI system of measurements is used in our medical practice, it is important to correctly convert them to mg/dl. For example, let's say the patient has U_{cr} – 4.4 mmol/l, U_{pr} – 3.2 g/l. So U_{cr} – 4.4 mmol/l = 4400 μmol = (4400 : 88.4) mg/dl = 50 mg/dl; U_{pr} = 3.2 g/l = (3.2 x 100) = 320 mg/dl. Thus, U_{pr}/U_{cr} = 320 : 50 = 6.4 g/ 24 hours.

The high efficiency of this method of determining daily proteinuria has been confirmed in practice (AM Yesayan, 2004). In children with glomerulonephritis , **leukocyturia** is observed in the first days and in 40-50% of cases. Aseptic, abacterial leukocyturia is a characteristic sign of glomerulonephritis, in each field of view of the microscope up to 30-35 leukocytes can be observed, and they are mainly due to lymphocytes (up to 50%) and eosinophils (up to 20%).

Casts can be observed in up to 100% of cases of glomerulonephritis. They are formed in the distal part of the nephrons during abacterial inflammation and are excreted in the urine. The basis of the casts is protein in structure, containing various inclusions - formed elements of blood, epithelial cells, fat, detritus. According to their nature, the casts can be cellular (erythrocytic, leukocyte, epithelial), granular, hyaline and waxy. The appearance of granular and waxy casts in the urine sediment indicates a severe course of the disease.

Edema syndrome

The connection of edema in the body with kidney disease was substantiated by R. Bright in 1827. Edema is the main symptom of glomerulonephritis, and depending on the form of the disease, it can be widespread (anasarca) from a slight drooping of the face to all cavities in the body (abdominal cavity, pleura, pericardial cavities). Usually, edema begins in areas rich in soft subcutaneous tissue - the face, around the eyes, and then spreads to the body and arms and legs. Therefore, edema is first noticeable on the face, especially after waking up in the morning. The origin of edema is complex, and the main causes can be a decrease in fluid filtration in the renal corpuscles, sodium and fluid retention in the body, hyperaldosteronism, and increased vascular permeability, which leads to fluid leakage from the blood vessels into the surrounding tissues. This is caused by an increased sensitivity of the distal

parts of the nephrons to antidiuretic hormone or a sharp increase in the production of this hormone in the body.

In glomerulonephritis, edema can develop nephrotic or nephritic. **Nephrotic edema** is characterized by widespread edema throughout the body, severe proteinuria (more than 3 grams per day), hypoproteinemia, and hypercholesterolemia. Hypoproteinemia leads to a decrease in the oncotic pressure of the blood, which passes from the blood vessels to the peripheral tissues, leading to a decrease in the volume of circulating blood (hypovolemia). In glomerulonephritis, increased vascular permeability creates conditions for fluid to pass into the tissues. A decrease in the volume of circulating blood stimulates receptors on the walls of blood vessels and increases the activity of other factors that ensure the constancy of the volume of fluid in the vessels. In particular, the secretion of antidiuretic hormone increases, and the secretion of aldosterone from the adrenal cortex increases (secondary hyperaldosteronism). These compensatory changes aimed at maintaining blood volume have a secondary effect, namely, reduced urine output, which leads to fluid and sodium retention in the body, and further edema. When nephrotic edema occurs in a young child with allergic symptoms and hematuria, the presence of allergic, renal, and cardiovascular diseases in his family tree is determined. If nephrotic syndrome is observed in children with impaired development of the SAA and proteinuria persists for a long time (more than a year), its prognosis may be poor.

In the nephritic form of edema, increased vascular permeability, increased hydrostatic pressure, and the limiting effect of antidiuretic hormone on salt and fluid excretion are more important, and a decrease in oncotic pressure is less important. This type of edema can be observed even with proteinuria.

The swelling is mainly located on the face, waist and lower leg. In such patients, the McClure-Aldrich test is accelerated. Usually, in a healthy child, a blister formed when 0.1 ml of saline is injected into the skin is absorbed within 45 minutes.

of hypertensive syndrome in childhood in any case requires an initial analysis of the condition of the kidneys. There is the following method for assessing blood pressure in healthy children: up to 1 year of age, systolic blood pressure (mm Hg) = 80 + n, where n is the number of months. After one year of age = 90 +2n, n is the number of years. The minimum blood pressure +is ½ systolic blood pressure of 5 mm Hg (MS Ignatova, 1989).

In cases where arterial hypertension is combined with various syndromes, the following diagnostic algorithm is used (Figure 3). The arterial blood pressure indicator depends to some extent on the child's physical development and age, as well as on the geographical environment and climate. Hypertensive syndrome

can be observed in various diseases (Fe o chromocytoma, vegetative dystonia, as a complication of hereditary lipoproteinemias, diabetic nephrosclerosis, etc.), but the observation of this syndrome in children first of all requires "nephrological vigilance" (Ya.Yu. Illek, MR Nuritdinov, 1993; MS Ignatova, 1989).

Hypertension is usually observed when the amount of vasoconstrictive products belonging to the group of biogenic amines in the blood increases and the renin - angiotensin system is activated (Figure 3). Hypertensive syndrome in children is more often observed in the hematuric form of diffuse glomerulonephritis, mixed type, chronic renal failure, congenital anomalies of the development of renal blood vessels, dysplasia, hypoplasia of renal tissue, and unilateral renal aplasia.

Hypertension is observed in 45% of children with acute glomerulonephritis during the first 1-2 weeks. In this case, diastolic pressure increases mainly. The increase in blood pressure in acute glomerulonephritis is associated with a violation of the blood pressure-regulating function of the kidneys. The renin-angiotensin-aldosterone system plays a central role in maintaining salt-water metabolism and arterial blood pressure in the body, and it regulates the activity of the kidneys, adrenal glands, arterioles and central nervous system. The nervous system provides interaction. The main substance in this system, renin, is produced in the supraglomerular area of the kidneys, 90% of which is produced. Decreased blood flow in the kidneys, activation of the sympathetic-adrenal system, and an increase in the sodium content in the lumen of the distal tubules of the nephron increase the production of renin . Renin, in turn, affects angiotensin in the liver, causing it to be converted to angiotensin-I, and then to angiotensin-II. Angiotensin-II causes narrowing of the arteriole blood vessels. In addition, it increases the release of aldosterone, indirectly causing sodium and water retention in the body (Esayan AM, 2002; Karabaeva AJ, et al., 2006).

The degree of hypertension in glomerulonephritis may vary depending on the form, activity level, and course of the disease. In chronic glomerulonephritis, the increase in blood pressure is initially temporary, but as the disease progresses, it becomes a permanent sign. In glomerulonephritis, blood pressure normalization occurs, first systolic, then diastolic pressure decreases.

Acute renal failure, regardless of the nature of the cause, is accompanied by an increase in blood pressure and sometimes leads to hypertensive encephalopathy (Illek Ya.Yu., Nuriddinov MR, 1993).

A persistent increase in blood pressure is also observed in diseases of the renal blood vessels (anomaly, stenosis, aneurysm), kidney tumors, hormonally

active tumors of the adrenal gland (pheochromocytoma). Hypertension is not characteristic of diseases such as hereditary nephritis, tubulopathy, pyelonephritis, but is a constant sign of the period of development of renal failure.

In the hematuric form of GN, arterial blood pressure is 15-20 mm Hg higher than normal for the first 10-14 days. However, in the nephrotic form of GN, blood pressure in most cases does not change. However, in the mixed form with nephrotic edema, a sharp increase in blood pressure and its prolonged persistence is a characteristic feature.

General blood test

Peripheral blood parameters change significantly in various kidney diseases, allowing us to judge not only the presence of kidney disease, but also the degree of disease activity, the state of kidney function. Often, signs characteristic of the inflammatory process associated with infectious agents are observed in the blood - leukocytosis, acceleration of the erythrocyte sedimentation rate. In glomerulo- and pyelonephritis, normochromic anemia is sometimes observed. The number of small-sized erythrocytes, reticulocytes may decrease. However, their strong changes occur only in the period of advanced chronic renal failure. In this case, severe normochromic anemia is also a characteristic sign. At the same time, a decrease in the total amount of protein in the blood is an indicator of its quality, in particular, in glomerulonephritis, a decrease in the amount of albumin αand βan increase in globulins (dysproteinemia) are observed, and the level of cholesterol may increase. In the early stages of glomerulonephritis and pyelonephritis, no more pronounced changes are observed in the peripheral blood, but during the period of impaired renal function, specific changes occur - anemia, azotemia, metabolic acidosis.

The color of urine - depending on the amount of urochromes A and B, uroblin, uroblinogen and bq, which are its components, can range from light yellow (straw color) to brownish yellow. In the first days of a baby's life, his urine is colorless, and later it becomes turbid and yellow, since during the first week after birth, a large amount of uric acid is excreted with urine. When urine is stored for a long time, its color darkens as a result of oxidation of chromogens.

When the amount of protein in the blood decreases, when taking diuretics, in the polyuria stage of renal failure, in chronic glomerulonephritis, urine becomes discolored. This is especially noticeable in diabetes insipidus. On the contrary, when fluid is lost through sweating, when insufficient fluid is consumed, urine becomes thicker and its color becomes more saturated.

The red color of urine may be due to the presence of red blood cells in it. For example, in glomerulonephritis, urolithiasis, and tumors of the urinary tract, urine may be stained a reddish color reminiscent of meat broth.

The color of urine also changes under the influence of some drugs: when a patient takes methylene blue, his urine is blue, when he drinks 5-NOK - orange, if it contains indican, it can be brown. The color of urine also changes in diatheses, urates stain urine brown, phosphates - milky white. In infectious hepatitis and other jaundices caused by damage to liver tissue, the color of urine increases.

Bacteriuria

The most important criterion for determining the presence of microbial inflammation is the level of bacteria in the urine - bacteriuria. A bacterial count of 100,000 or more per ml of urine indicates the presence of microbial inflammation in the kidneys or urinary tract.

Bacteriuria is diagnosed by microscopic examination of the urine sediment - bacterioscopy, culture of urine in a special microbiological medium (bacteriological methods), and in nephrology clinics, some tests with semi-quantitative value (nitrite test). The advantages of the bacteriological method are that it allows you to determine the type of microbe in the urine , its quantity and sensitivity to drugs. Quantitative examination of the urine sediment is widely used in the clinical setting to identify latent diseases. Quantitative examination of the urine sediment to detect **latent erythrocyturia** is used in combination with a prednisolone challenge test as a criterion for detecting chronic glomerulonephritis and excluding acute glomerulonephritis: for this purpose, the person being examined is recommended to take 15 mg of prednisolone in the evening, and the next day at 6:00 am ، after emptying the bladder, the patient takes another 15 mg of prednisolone. The child's urine is collected over the next 9 hours and the number of red blood cells in it is determined. The test is considered positive if the child excretes 8,000 red blood cells per minute or if the red blood cell count is 100% higher than before the test.

The prednisolone challenge test is also used to detect occult leukocyturia: in the morning, the patient collects urine in a container for one hour, and then 30 mg of prednisolone in 10 ml of saline is injected into his vein for 3-5 minutes. After that, hourly urine is collected for three hours in a separate container, and the number of erythrocytes, leukocytes and cylinders in the urine in each of the four containers is determined. This test takes into account three things: the number of leukocytes, the rate of their increase after the test, and the presence of "active" leukocytes in the urine sediment. In cases of inflammatory processes in the kidneys, the number of leukocytes in one hour can reach 400,000 or more.

Quantitative determination of blood cells in urine methods

Microscopic examination of the urine sediment allows direct observation of erythrocytes, leukocytes, casts, and salts. When examining the urine sediment of a healthy child, 1–2 erythrocytes and up to 5 leukocytes can be found in each field of view of the microscope. However, the presence of more than 2–3 erythrocytes in the field of view indicates erythrocyturia, and the presence of more than 5–7 leukocytes indicates leukocyturia. Various crystals can be detected in the urine sediment in varying quantities (Fig. 3).

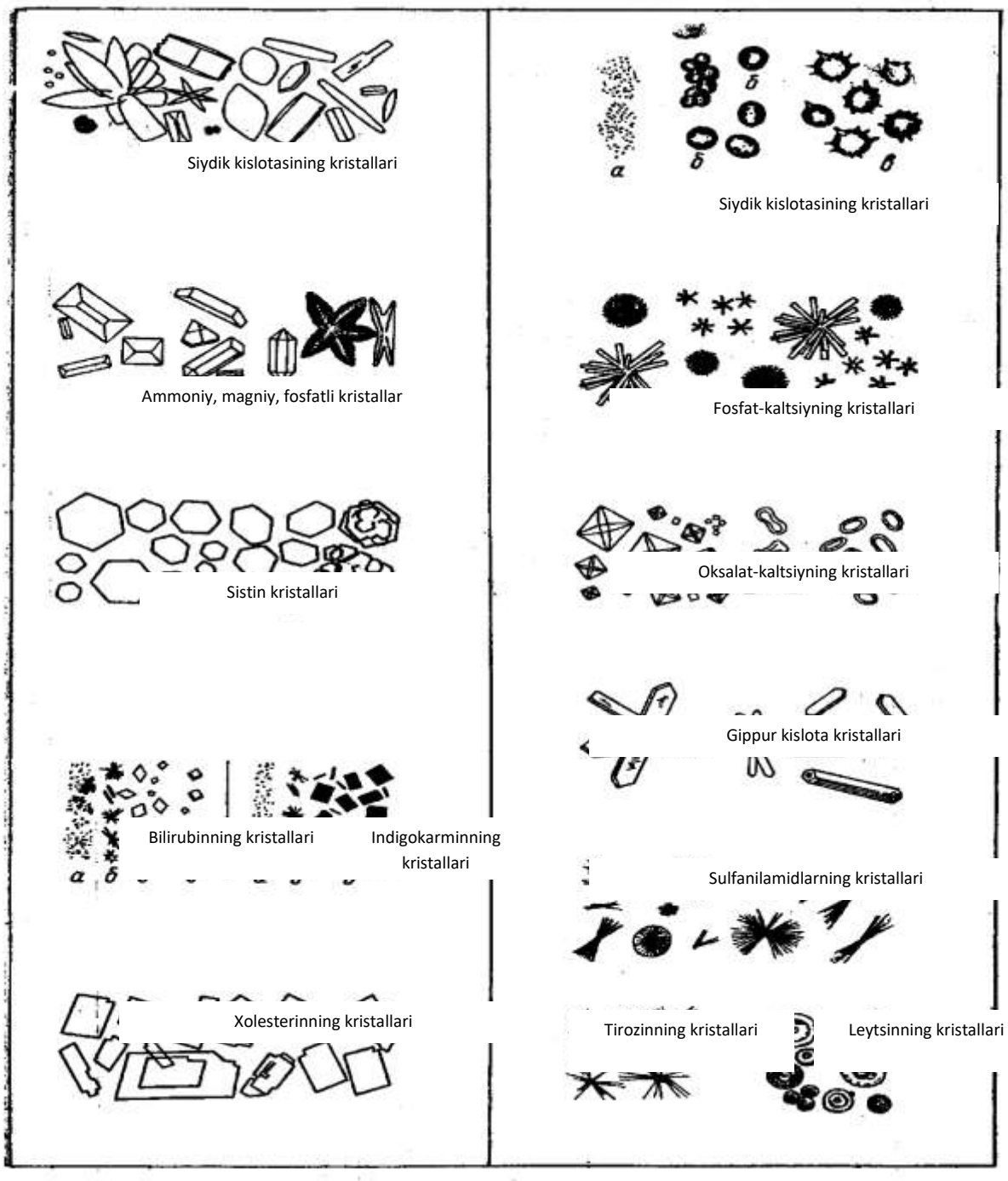


Figure 3. *Urine sediment (G. Majdrakov, N. Popov, 1976)*

Even in the urine of healthy children, small amounts of leukocytes can be isolated - up to 2,000,000 leukocytes per day, up to 1,000,000 erythrocytes, and sometimes up to 100,000 cylinders. There are 4 different methods for determining the amount of blood cells in the urine: **1) Kakovsky - Addis method -** determination of the amount of blood cells separated in 24-hour urine; **2)** Determination of the amount separated in one minute or one hour - **Ambourgeois method** ; **3)** Determination of the amount of blood cells separated in one ml of urine - **Nechiporenko method; 4)** Determination of the amount in uncentrifuged urine (Table 1).

Table 1 .

The highest content of leukocytes and erythrocytes in the urine of healthy children (EA Yureva, VV Dlin, 2002)

Inspection methods	Term	Leukocytes	Erythrocytes
Kakovsky – Addis method	24 hours a day	2,000,000	1,000,000
Ambourgeois method	In 1 minute	2000	750
	In 1 hour	120,000	45,000
Nechiporenko method	In 1 ml of urine	2000	1000
Examination of uncentrifuged urine (AB Kanatbaeva, 1971)	24 hours a day	2500 000	1500,000
	In 1 minute	2500	1000
	In 1 hour	150,000	60,000
	1 ml ^{3} – in	10	3

Given that these methods are widely used in hospitals and outpatient clinics, we will specifically highlight their correct implementation.

Kakovsky – Addis method. There are various versions (modifications) of this method. In particular, according to the method recommended by Addis in 1925, fluid intake on the day of examination was sharply limited, and 12-hour urine was collected at night, and blood cells were multiplied by 2 and counted for 24 hours. This method is not used in its original form for children. If urine is stored in a cool place, in acidic conditions, and its relative density is not less than 1012, the blood cells in it are completely preserved for 24 hours. In order for the relative density of urine to be at this level, it is not necessary to limit fluid intake. The currently widely used version of this method is as follows: the child being examined maintains his usual daily routine, diet, fluid is not limited. He urinates in the morning and is discarded. A time is set (for example, 7:00 a.m.) , this is

the time when urine collection begins. All urine is collected and the last one is taken at 7:00 the next morning · 1-2 thymol crystals (10 mg/l) are added to the urine and stored in a refrigerator. The entire urine or 100 ml of it can be sent to the laboratory after thorough mixing. Before testing, the urine medium and specific gravity are determined. If the specific gravity of urine is less than 1012 or the medium is alkaline, blood cells are partially destroyed during the day. In such cases, it is more expedient to use the Ambourgeois or Nechiporenko method. The number of blood cells is counted in the usual way in a Fuchs-Rosenthal chamber. If the number of blood cells in the Fuchs-Rosenthal chamber is designated as N, and the 24-hour urine volume is V, the number of blood cells in the daily urine (x) is determined as follows: X = (100 x VxN) : 3.2

Ambourg method – urine collected for a short period of time (usually 3 hours) is examined. This method is convenient for any conditions (hospital, clinic) and is not inferior in accuracy to a daily urine test. Usually , urine is collected from 7:00 to 10:00 in the morning without any special preparation . The calculation of the amount of blood formed elements is calculated according to the above formula: X = (100 x VxN) : 3.2, where V is the volume of urine for 3 hours, N is the number of blood cells in the volume of the Fuchs-Rosenthal chamber. To determine the amount per minute, the number obtained for the volume of three hours is divided by 180 – (number of minutes) or by 3 to determine the volume per hour.

Nechiporenko method – proposed in 1961, the number of blood cells is calculated in 1 ml of urine. All the methods mentioned are associated with centrifuging urine and checking its sedimentation. However, when centrifuging, not all blood cells in the urine, but 1/3 - ½ of them, fall into the sediment (EA Yureva, VV Dlin, 2002). Therefore, in recent years, the method of **examining urine without centrifugation** has become more widespread. The procedure for collecting and examining urine is carried out in the same way as in the Kakovsky - Addis, Amburghe methods, only the urine is placed directly into the counting chamber without centrifugation and the number of blood cells is determined according to the formula X = (1000 x VxN): 3.2. By dividing the resulting number by hours and minutes, the hourly and minute amounts of blood cells in the urine are determined.

Three-cup test . The three-cup test is used to determine whether hematuria and leukocyturia are related to the kidneys (renal) or to the post-renal system (postrenal). For this, the patient collects the first, middle, and last portions of urine into three separate containers when urinating. If hematuria and leukocyturia are detected in the urine in the first and second containers, the pathological process is considered to be located in the urethra or bladder, while

if hematuria and leukocyturia are detected in the urine in the last container or in all containers, the pathological process is considered to be located in the kidneys or in the renal calyces, or in the upper urinary tract.

Zimnisky test . This test allows you to assess the kidney's ability to excrete urine, osmotically dilute and concentrate urine. The test is performed during the child's usual diet and lifestyle, without any special measures.

The subject's urine is collected every three hours in separate containers throughout the night. The amount of urine in each container is determined for the day (from 6:00 to 18:00) and the evening (from 18:00 to 6:00) hours. In healthy people , the amount of urine should be 65-75% of the fluid consumed, and daytime diuresis should be greater than evening diuresis. If evening diuresis is equal to or greater than daytime diuresis (nocturia), this is a sign of impaired renal filtration. In addition, the specific gravity of urine also changes during the day. The relative density of primary urine is equal to the relative density of blood serum (1.010), the relative density of excreted urine can be from 1.001 to 1.040. If the relative density of urine is around 1.007-1.015, it is considered hyposthenuria, and if it is around 1.010-1.012, it is considered isosthenuria. If sugar or protein is excreted in the urine, a correction should be made to the relative density: each percent of sugar in the urine increases its relative density by 0.004, and each 3% of protein by 0.001.

pH of urine depends mainly on the amount of free hydrogen ions. In infants, the pH is 5.4–5.9, in infants 6.9–7.8. The pH value depends on the nature of the diet and is mainly in the range of 4.5–6.5. The pH should be determined in freshly collected urine. If more protein is consumed with food, the urine environment shifts to the acidic side, and if more plant products are consumed, to the alkaline side. This is taken into account in the practice of dietary treatment of kidney diseases. When more phosphates are excreted with urine, the urine becomes alkaline. During starvation, prolonged diarrhea, diabetes mellitus, and acidosis developed as a result of metabolic disorders, the urine environment shifts to the acidic side.

The urine environment changes in all diseases accompanied by changes in breathing, heart function, vomiting, diarrhea. The urine environment is also observed in hereditary changes in the functioning of the renal tubules.

Radiological examination of the urinary organs check

X-ray examination of the urinary organs usually begins with a general radiography without contrast media. For this, the child is given a cleansing enema in the evening and in the morning, and a diet is prescribed for 1-2 days, which limits black bread, milk and fruits. This method allows you to get an idea of the location, shape and size of the kidneys. Normally, the surface of the

kidneys is smooth, the upper pole corresponds to the XI thoracic vertebra, and the lower pole to the IV lumbar vertebra. The left kidney is located ½ -I vertebra higher than the right kidney.

An increase in kidney size is observed in polycystic kidney disease, hydronephrosis, and glomerulonephritis, and unilateral enlargement is observed in hydronephrosis and tumor diseases.

Small size of the kidneys is observed in congenital hypoplasia, and their narrowing due to chronic inflammatory processes in the kidneys is observed in nephrosclerosis.

In addition, carbonate and oxalate stones in the kidneys and urinary tract, as well as the accumulation of calcium salts in the kidney tissue (nephrocalcinosis) can also be detected in this way. For more detailed information, it is recommended to perform excretory urography.

Excretory urography is widely used in uro-nephrology clinics for the following reasons:

- leukocyturia observed in children up to one year of age;
- changes in urine lasting more than 3 months;
- unexplained pain in the abdomen and lower back;
- urinary incontinence;
- Inconspicuous abdominal injuries that cause hematuria;
- Conditions such as kidney and urinary tract stones or birth defects can cause such a need.

Preparing the patient for the examination . 2 days before the examination, the consumption of black bread, milk, and raw fruits is strictly limited.

The patient is given a cleansing enema the night before the examination and the morning of the same day (2 hours before the examination). It is recommended that the patient urinate until the bladder is completely empty before the contrast medium is administered. The sensitivity to the contrast medium used is determined the day before the examination. For this purpose, 65% hypac, 60% verografin, 76% urografin, 50% triiodothyronine solutions are used. The contrast medium, heated to 37 ^{0} C, is injected intravenously for 1-2 minutes at a rate of 1.5-2 ml per kg of weight for children under 3 years of age, and 1 ml/kg for older children (after 8 years of age). After that, X-rays are taken after 3-5, 10-12, 15 and 30 minutes. If necessary (i.e. if delayed images are required), this can be repeated after 1, 2, or 4 hours. Usually, the first X-ray is taken with the child standing, then with the child lying on his back. In cases where it is not possible to inject the contrast agent directly into a vein, the contrast agent can also be injected subcutaneously, intramuscularly, or through the colon, but in these cases the quality of the X-rays will be lower.

Excretory urograms allow you to get an idea of the structure, size, and location of the kidneys. In addition, X-ray planimetric studies are of particular importance. One of the widely used forms of excretory urography is the infusion method, which is recommended for examining children under one year of age and patients with impaired renal excretory and concentrating function. When using the infusion method, the contrast medium is diluted in a 5% glucose

solution to 35% and dripped into a vein for 5-30 minutes . For children under one year of age, 5 ml of a 35% solution per kg of weight is recommended, 2-3 ml/kg for 3-5 years of age, and 1-1.5 ml/kg for 7-14 years of age. X-rays are taken 1, 10, and 20 minutes after the start of the solution injection. In patients with impaired renal excretory function, delayed X-rays are also taken. Usually, after the start of contrast medium administration, the urinary excretory systems (renal pelvis, urethra) are distinguished within 3-5 minutes, and the bladder within 5 minutes. Usually, the excretion of contrast medium from both kidneys is observed equally.

When it is necessary to assess kidney developmental defects, blood vessel development, and the state of blood circulation in the kidney, and in the case of suspected tumors, renal angiography is performed in specially organized conditions and in specialized centers.

Less harmful triatomic iodine-binding agents (hypak, urografin, urotrast) are recommended for examination. The substance to be injected is diluted in a 5% glucose solution to 35%. This, in turn, allows the contrast agent to be injected in a much larger amount than usual and is excreted from the body relatively quickly. For intravenous administration, the contrast agent is poured into a device and injected into a vein in the forearm (sometimes in the head for children under 1 year old) at a rate of 120-150 drops per minute for 5-7 minutes up to 3 years of age, and after 3 years of age at a rate of 100 drops per minute for 7-10 minutes (in cases of a sharp decrease in kidney function, the number of drops per minute is reduced to 60-80 and can last 20-30 minutes). X-ray imaging of the kidneys and urinary tract is performed 10, 15, 20 minutes after the start of the contrast agent injection into the vein. The last time is performed with the patient in an upright position.

Drip infusion excretory urography does not require special patient preparation, but 6 hours before the examination, feeding is stopped and the child is given 200.0 ml of the next meal as soon as the contrast medium is dripped. The air bubble formed during feeding expands the stomach and intestines, displacing them from the kidney area, and the quality of the X-rays improves.

There are two methods for evaluating the obtained excretory urograms: 1) visual assessment of the anatomical and physiological characteristics of the kidneys and urinary tract depending on the child's age, and 2) special measurements of the renal and calyx areas and mathematical analysis of their size and proportions (x-ray planimetry).

When evaluating excretory urograms, attention is first paid to the number, size and location of the kidneys. Usually, in a newborn, the upper border of the kidney corresponds to the X thoracic vertebra, and the lower one to the greater pelvis . In older children, the upper border reaches the XI thoracic vertebra, and the lower border reaches the IV lumbar vertebra. Usually (in 60% of cases), the left kidney is located about one vertebra higher than the right, and the displacement of both kidneys when the child is in a supine position does not exceed the height of one lumbar vertebra. If the displacement is greater than this, the kidneys can be considered to have moved down (nephroptosis).

Measuring the length and width of the kidney on an X-ray is not difficult. The following data on the size and weight of the kidney in children are available in the literature (Table 2).

After the age of five, the length of the kidney is calculated according to the following formula: X= 0.379 x U + 6.65 cm; X is the length of the kidney, U is the child's age. If the figure obtained as a result of the examination differs from this indicator by 20% or more, this indicates an increase or decrease in the size of the kidney: congenital immaturity of the kidney (hypoplasia) or its failure to bend due to disease (nephrosclerosis) leads to its decrease, while hydronephrosis, polycystic disease, double kidney, and tumors lead to its increase.

Table 2 .

Kidney size and weight in children

(AV Mazurin, IM Voronsov, 1985)

Age	Weight, g	Length, cm	Width, cm	Thickness, cm	Area, cm^2
Baby	11 - 12	4.2	2.2	1.8	-
5 months	22.6 - 23.6	5.5	3.1	1.9	-
1 year old	36 - 37	7.0	3.7	2.6	19.0
5 years old	55 - 56	7.9	4.26	2.76	32 - 34
11 years old	82 - 84	9.8	5.15	3.3	41.3
15 years old	115 - 120	10.7	5.3	3.5	48.7

There are other methods for determining the length of the kidneys. In particular, it has been established that there is a reliable relationship between a person's height and the length of the kidneys. A similar relationship is known between the total height of the $L_1 – L_2$ vertebrae and the length of the kidneys (Hodson's index , MS Ignatova, P. Grossman, 1986). Accordingly, there are also diagrams for measuring the length of the kidneys (Figures 1, 2).

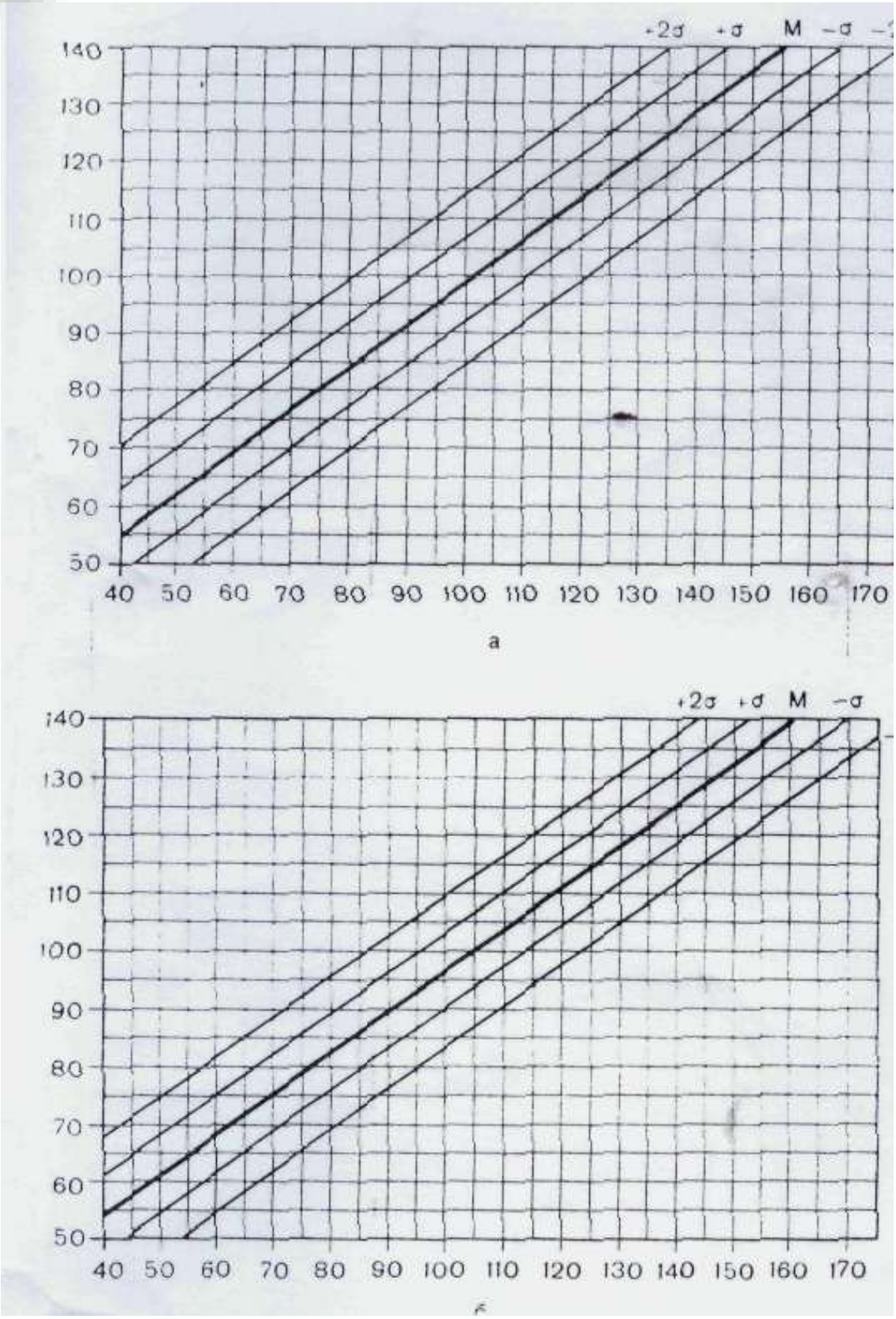


Figure 1. Diagram of determining *the size of the kidneys for a given age of a child based on the size of the* $L_I - L_{IV}$ *segment of the spine. a) left kidney; b) right kidney: Length of the* $L_I - L_{IV}$ *segment along the abscissa, mm; Length of the kidney along the ordinate, mm (* MS Ignatova, P. Grossman, 1986 *).*

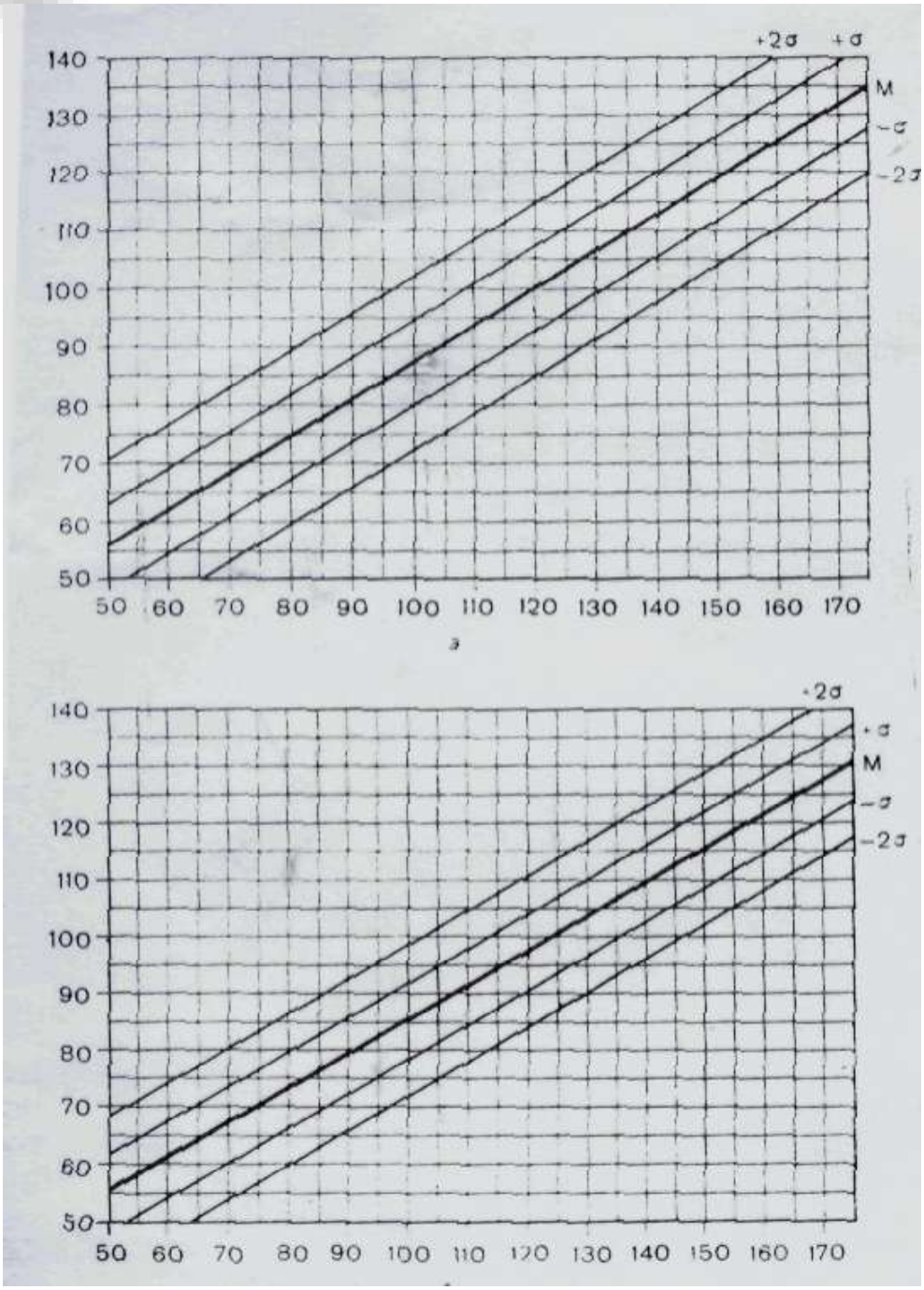


Figure 2. *Diagram of determining the size of the kidneys for a given age of a child in relation to his height. a) left kidney; b) right kidney: On the abscissa – height, cm; On the ordinate – length of the kidney, mm (* MS Ignatova, P. Grossman, 1986 *).*

In healthy children, the difference in length between the two kidneys does not exceed 0.7-1.0 cm. In cases of unilateral kidney enlargement or reduction, this difference may be significant. When assessing the anatomical condition of the kidney based on nephro-urograms, it is necessary to take into account that it may have a different structure even in healthy children : the cups belonging to the urinary collecting system are connected to the urinary bladder by the ureter

(neck). In children under one year of age, the calyx is mainly located directly inside the kidney, and in later years, depending on the completion of the formation of the kidney, it can have different shapes: a small calyx is located completely inside the kidney tissue (intrarenal type), or a smaller part of the calyx is outside the tissue, in the area of the kidney's hilum (mixed type), and finally, the renal calyx is not triangular, but round, and most of it is not directly covered by the kidney tissue (extrarenal type). In diseases, narrowing, stretching, and expansion of the neck of the calyx are observed, often in the inflammatory process (pyelonephritis). At the same time, if an obstacle to the outflow of urine occurs (stone, tumor, sclerosis), expansion of the calyx is observed (pyeloectasia). Congenital and secondary narrowing and expansion of the urinary tract from the calyx to the bladder can occur. Special measurements are performed to assess the condition of the renal tissue and the function of the urinary tract. In particular, the surface area of the calyces is determined as follows (Figure 3):

$$\text{Surface} - \frac{ABxBCxKM}{1/2(AB+BC)}$$

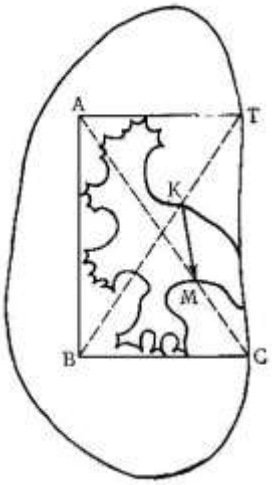


Figure 3. *Determination of the surface of the renal pelvis - calyx (explanation in the text, Derzhavin VM, Kazanskaya IV, 1973)*

Here AV is the length of the cup system; VS is its width (width); KM is the height of the cup.

of the bowl (KM), two diagonals are drawn according to the bowl-cup system (AS,VT), and the points where the diagonals intersect with the boundaries of the bowl are considered its upper and lower points (KM).

When there is difficulty in the outflow of urine and pressure in the calyx increases (urostasis), its expansion is observed. If the calyx is located inside the kidney, its expansion is less noticeable, but in cases located outside the kidney, it can average 2 cm $^{2.}$ In healthy children, the calyx-calyx area depends on its location relative to the kidney (Table 3):

Table 3 .

Calyx-calyceal system area in children

(AI Derzhavin, IV Kazanskaya, 1973)

Type of cup	Calyx-coccyx area by age (cm ^{2})				
	Up to 3 years old	3 – 5 years old	5 – 7 years old	7 – 11 years old	11 – 15 years old
Inside the kidney	2.3	2.5	3.16	3.7	4.2
Mix	3.5	3.4	4.1	4.8	5.5
Outside the kidney	3.75	4.4	4.9	5.5	6.4

Excretory urography allows to assess parenchymal changes in addition to the urinary collecting and excretory system. For this purpose, the ratio of the area of the calyceal system to the area of the kidney is determined. The area of the kidney is calculated according to the following formula:

3.14 x Half the length of the kidney (1/2) x Half the width of the kidney (1/2).

This ratio is constant for children of all ages and depends on the type of location of the urine collection container (Table 4).

Table 4 .

Renal Cortical Ratio (RCR)

(AI Derzhavin, IV Kazanskaya, 1973)

Renal pelvis, type of location	Ratio indicator
Inside the kidney	0.091
Mix	0.118
Outside the kidney	0.155

Changes in the renal parenchyma (inflammation, fibrosis, etc.) or enlargement or reduction of the renal pelvis system lead to changes in this indicator: in particular, in pyelonephritic changes, the indicator increases, and in glomerulonephritis and polycystic kidney disease, it decreases.

Another unique feature **of excretory urography with drip infusion is that the entire urinary tract from the pelvis to the bladder is visible. However, when examining the contrast medium without drip infusion, such a situation should be interpreted as a sign of decreased contractile function (hypotonia) of the urinary tract.**

In this sense, it is important to calculate the contractility of the calyx-calyceal system in the excretory urogram. For this, the surface of the calyx-calyceal system is calculated during the period of filling the calyx with contrast (10th minute) and its contraction period, and their ratio is determined (Table 5).

Table 5 .

Hello . in children cup – bowl system contraction indicator (%)

(A . I . Derzhavin , I . V . Kazanskaya , 1973)

Age (years)	Cup placement type		
	Inside the kidney	Mix	Outside the kidney
5 – 7	21	20	25
7 – 11	22	23	24
11 – 15	22	25	26

Hypotonia of the calyceal system, i.e., a decrease in the contractility index, is observed in the initial period of primary pyelonephritis, when its expansion (dilation) develops due to secondary obstructive pyelonephritis. In the initial period of obstructive pyelonephritis, on the contrary, hypermotor dysfunction is observed.

Ultrasound examination of the kidneys is currently the most common method, which is painless, without complications, and most importantly, allows you to get a clear idea of the size, shape, to some extent anatomical structure, location, and relationship of the kidneys to neighboring organs, and therefore is recommended for any changes in SAA.

Renal angiography is used to study the vascular system of the kidneys . This method allows obtaining arterio-, nephro-, venograms and excretory urograms, which gives a clear picture of the arteries, veins, abdominal aorta, kidney tissue, their size, shape, etc. However, this is a very complex examination method and can only be performed in specially equipped centers in cases of suspected persistent hypertension, vascular malformations or tumors.

Retrograde pyelography is a complex, non-physiological method used in cases where excretory urography does not provide sufficient information, but full information about the urinary tract, renal pelvis, and calyces is required. It is less commonly used in children.

Cystography - an X-ray examination of the bladder by filling it with a contrast medium. It is performed in cases of physical injuries, reflux, and suspected developmental defects.

about the topography and function of the kidneys can be obtained using **radionuclide renography**.

Methods of testing kidney function and evaluating their results

In nephrology, along with clinical observations, urine output, urine analysis, play a key role. Over a certain period of time (daily, night, daytime, hourly, minute), the urine output (diuresis) may change in various diseases, increasing (polyuria) or decreasing (oliguria), or even completely stopping (anuria). The urine output of a healthy child after 1 year is determined as follows: **600 + 100 (n – 1)** , where 600 is the amount of urine excreted by a one-year-old child during the day and night, n is the child's age. A decrease in the amount of urine output up to 30% of the amount for the child's age is considered oliguria, a complete cessation or a decrease of more than 10% of the normal amount is considered anuria, and vice versa, an increase of 1.5–2 times is considered polyuria. Usually, 2/3 of the total amount of urine is excreted during the day. A greater amount of urine excreted at night than during the day (nocturia) indicates a violation of the urinary excretion rhythm and is observed in glomerulonephritis, pyelonephritis, and congenital and hereditary kidney diseases.

An increase in urine volume is observed in healthy children with excessive fluid intake, in sick children during the period of edema, in diabetes mellitus and diabetes insipidus, in the recovery period of acute renal failure, in the initial stages of chronic renal failure. Polyuria leads to a decrease in the relative density of urine (1012 - 1010) (hypostenuria). Severe extrarenal fluid loss (profuse sweating, vomiting, diarrhea), acute glomerulonephritis, leads to oliguria. In oliguria in acute glomerulonephritis, an increase in the relative density of urine to 1024 - 1030 and above is observed. The relative density of urine in healthy children up to 6 months is 1002 - 1004, at 3 - 5 years old - 1010 - 1020, at 10 - 12 years old - 1011 - 1025. Usually, the urine of a healthy child is straw-colored, yellowish. Its color depends on the ability of the kidneys to thicken (concentrate) urine. When this ability of the kidneys decreases, the urine becomes colorless, and when it reaches the level of hypostenuria, the urine becomes colorless like water. The color of urine also depends on the quality of food and some medications. For example, if a child eats a lot of beets or takes amidopyrine, the urine turns red, from furazalidone, furagin - yellow, from trichopol, essential - dark brown. The presence of blood in the urine gives it a color similar to meat broth, the release of methemoglobin makes it black, bilirubin turns brown. The increased excretion of urates with urine makes it brown, a brick color, phosphates - milky white, etc. Freshly collected urine usually does not foam, it can foam strongly only if it contains protein and sugar. The urine of a healthy child is clear, its turbidity is observed when leukocytes, erythrocytes, salts, epithelial cells are excreted in large quantities with urine.

Kidney function assessment

To assess kidney function, its ability to excrete urine, concentrate and dilute urine, and the amount of a number of substances in the blood and urine, the norm of which is directly related to kidney function, are taken into account.

The Zimnisky test is widely used to assess the excretory, concentrating (concentrating) activity of the kidneys, and the rhythm of urine excretion. For this, urine is collected in 8 containers every three hours during the day and night, while maintaining the usual eating and drinking regimen appropriate for the child's age. Usually, in healthy children, the relative density of urine in each container differs by at least 0.010. In cases of impaired renal tubular thickening activity, the relative density of urine decreases. In cases where this activity is latent, special load tests can be used (Folgard test). In early children, collecting urine every 3 hours can be difficult. In this case, urine can be collected freely (when the child wants to urinate for 24 hours) and evaluated in the same manner **(Reiselman method).**

To conduct the Folgard test , the child is limited to dry food (bread, eggs, oil) for 36 hours, without giving liquid food and water, and after 12 hours, urine is collected every 2 hours for 24 hours. The volume and relative density of urine in each container are determined. In a healthy child, the relative density of urine increases from 1022 to 1032 and higher. If the kidney's thickening function is impaired, the relative density of urine remains low. On the contrary, the dilution ability of the kidneys should also be studied. For this, the child's weight is measured on a scale, then water is given in an amount of 20 ml/kg, and urine is collected for 4 hours (every 30 minutes for the first 2 hours, every hour for the next 2 hours), then the patient's weight is measured again . A healthy child excretes 75% of the fluid he has taken in over 4 hours, the relative density of urine decreases to 1001-1003, and the child's weight remains unchanged. When the excretory function of the kidneys changes, fluid is retained in the body, its relative density changes little, and the patient's weight increases due to the retained fluid.

Since the biochemical composition of blood and urine depends on the ability of the kidneys to purify the blood from metabolic products, the determination of a number of nitrogenous substances is accepted in nephrology practice (urea, creatinine, uric acid, amino acids, residual nitrogen). Violation of the kidney's cleansing function leads to the accumulation of residual nitrogen in the blood - azotemia. Typically, in the blood serum of children under 14 years of age, 14.6 - 29.3 mmol / l of residual nitrogen is determined, of which urea is 2.5 - 6.8, amino nitrogen is 3.21 - 15.0, uric acid is 0.14 - 0.33 mmol / l, and creatinine is 0.044 - 0.088 mmol / l, that is, about 50% of the total residual nitrogen is urea, and 15% is amino acids. The amount of nitrogenous substances in the blood can sometimes increase even with normal kidney function (in cases of vomiting, diarrhea, excessive consumption of nitrogen-rich foods). The determination of some substances can sometimes provide more accurate information than the total amount of nitrogen (urea, creatinine). In particular, the determination of the blood clearance rate (clearance) is widely used in nephrology. Clearance (clearance rate) is the number of ml of blood that the body clears of a substance through the kidneys per minute and is determined as follows (S): S= (U x V): R, where U is the amount of the substance being examined in the urine, V is the diuresis per minute, and R is the amount of this substance in the blood serum.

Only the clearance of substances that are filtered in the renal corpuscles and not reabsorbed in the tubules provides complete information about purification (insulin, mannitol, sodium thiosulfate). Creatinine also gives a result close to these substances and is therefore widely used in nephrology practice. Urine is also filtered, but is partially reabsorbed in the renal tubules. For comparative purposes, creatinine clearance is usually taken relative to the average body surface area of adults (1.73 m ^{2}). For this, the body surface area (S) of the child being examined must also be determined: According to **the Dubois formula** , this is: S = 167.2 x $\sqrt{танавазни\,(кг)хбуйи(см)}$ **body weight (kg) x height (cm).** There is also a special nomogram for determining the body surface area based on the child's height and weight (Figure 4).

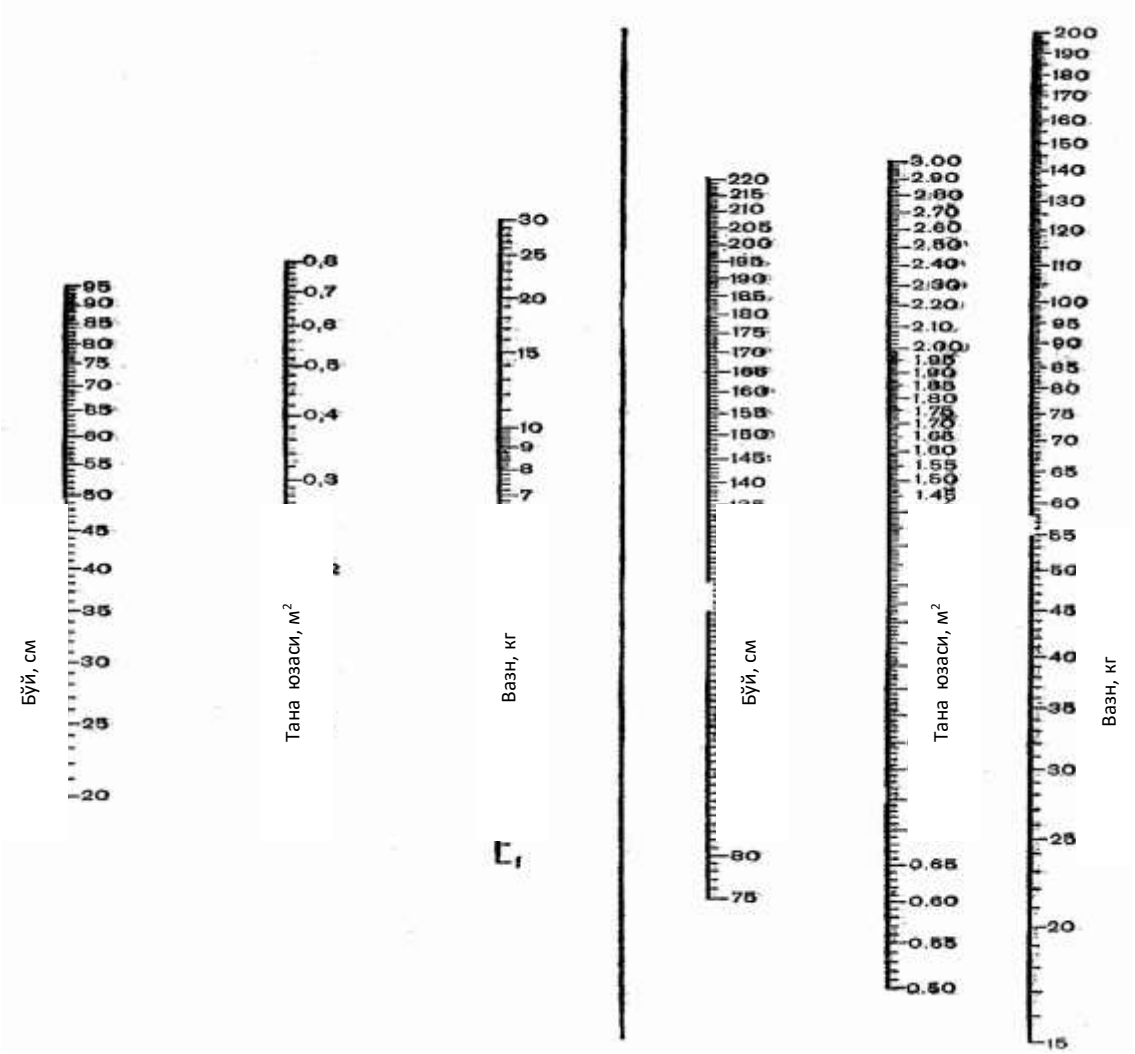


Figure 4. *Nomogram for determining body surface area based on height and weight of a child* (according to I. D. Crawford , M. E. Terry , G. M. Rourke , **1979**) . The point where the straight line connecting the height and weight indicators intersects the *central scale indicates the body surface area* .

To determine the endogenous creatinine clearance, taking into account the small changes in the amount of creatinine in the blood throughout the day, the following procedure is performed: at 8:00 in the morning , blood is taken from a vein to determine creatinine, and urine is collected from 7:00 to 21:00 and from 21:00 to

7:00 . This allows you to determine the night and daytime clearance separately. In a healthy child, the clearance is 80 to 120 ml/min x 1.73 m ^{2}. Calculation:

$$S = \frac{KcxV}{Kk} \text{ x } \frac{1{,}73}{bolaning\tan auzasi}$$

Here: S is the cleaning index,

V – one-minute diuresis,

Ks – creatinine in urine,

Kq is an indicator of serum creatinine.

In cases where collecting the patient's urine is difficult, endogenous creatinine clearance can be calculated from the amount of creatinine in the blood using the Schwartz (Y. Schwartz) * formula:

$$C_{cr}(\text{ml / min / 1.73 m}^2) = \frac{0{,}0484xboyi(см)}{Scr(ммоль/л)}$$

S_{cr} – the amount of creatinine in the blood. For children older than one year, a coefficient of 0.0616 is taken.

K – calculation coefficient

0.33 – for premature babies under 2 years of age;

0.45 – for children born at term up to 2 years of age;

0.55 – 2 – for boys aged 14 and girls over 14;

0.55 – for boys over 14 years old;

L – height (cm);

C_{cr} – blood creatinine level (μmol/l in serum)

Example: A healthy boy aged 14 years and 6 months, height 165 cm, C_{cr} – 80 μmol/l;

$$GFR = \frac{KxL}{Скрx0{,}0113} = \frac{0{,}7x165}{80x0{,}0113} = 127.7 \text{ ml/min/ 1.73 m2}$$

The rate of water reabsorption in the renal tubules is determined by the difference between the amount of fluid filtered through the renal corpuscles in one minute and the amount of urine excreted in the same period.

The calculation is carried out according to the following formula:

$R_{H2O} = \frac{Скр - V}{Скр}$ x 100, where Skr is creatinine clearance, V is the one-minute diuresis rate.

In healthy children, the rate of water reabsorption (R) from the renal tubules is 98–99%. In addition, testing the ability of the renal tubules to secrete substances also allows for a partial assessment of their function. For this purpose, substances such as paraaminohippurate (PAG) and phenolsulfophthalein (phenolrot) are used.

kidney diseases are diverse in their causes, course, and consequences, patients often have common complaints such as fatigue, malaise, headache, back and abdominal pain, pallor, and drooping eyelids, which make the nosological diagnosis of the disease quite difficult.

In particular, glomerulonephritis begins 1-3 weeks after a severe cold or an infectious streptococcal disease, and the patient also has a characteristic appearance: he is pale, his face and sometimes all his limbs are swollen, urine output changes sharply, decreases, and becomes reddish. Pyelonephritis is characterized by more fever, back and abdominal pain, protein in the urine, and the excretion of disease-causing bacteria.

However, there are a number of kidney diseases (hereditary nephritis, tubulopathies, damage to the kidneys by toxic products of metabolism, etc.), the visible external symptoms of which are absent or appear very late. Thus, it must be admitted that due to the diversity of the causes of kidney diseases and the relative relativity of external symptoms, their diagnosis based only on clinical signs and even a general urine analysis is likely to lead to errors. For this reason, modern nephrology requires the conduct of multi-stage special examinations. The improvement of nephrological examination methods makes it possible to diagnose latent, alternating, rare and complex diseases.

Periodic attacks of severe pain in the abdomen and lower back - kidney pain attacks - are a typical symptom of urinary tract and kidney stone disease.

Urinalysis is important in these diseases. For example, the presence of protein and broken red blood cells (erythrocytes) in the urine is a sign of glomerulonephritis, mainly leukocytes and bacteriuria - a sign of pyelonephritis, and the presence of unchanged erythrocytes is more often observed in inflammation of the urinary tract and kidney stones. However, kidney diseases accompanied by similar changes in the urine are not the only ones - such changes in the urine are also likely to be observed in kidney diseases caused by polycystic disease, congenital malformations of the urinary tract and kidneys, hereditary nephritis, tubulopathies, metabolic disorders (cystinuria, uraturia, etc.). Thus, kidney diseases and their causes are very diverse, and it is impossible to definitively determine them not only on the basis of a clinical examination of the patient, but also, often, on the basis of a general urine analysis. Therefore, a special program is needed for the diagnosis of kidney diseases. This program is structured in several stages and places special emphasis on the patient's genealogical analysis (study of the family tree). Assessing the type of diseases present in the patient's relatives and parents allows us to identify not only hereditary diseases, but even a hereditary predisposition to kidney disease.

the clinical signs of the disease, biochemical and genealogical analysis, hereditary diseases can be easily distinguished from glomerulonephritis.

Pyelonephritis is a secondary disease that occurs mainly in children with metabolic disorders, congenital defects of the development of the urinary organs and immunologically imperfect. A thorough study of the genealogy of patients shows that in families with hereditary nephritis, interstitial nephritis leading to chronic renal failure in adulthood, diseases of the organs of vision and hearing, tubulopathies with rickets-like changes in the bones of the skeleton and secondary pyelonephritis, sometimes stone disease, occur. In this regard, the comparative diagnosis of multifactorial nephropathies resulting from metabolic disorders is fraught with certain difficulties. In this regard, the following special examination program has a great advantage (Table 6).

Table 6

Phased examination program for nephrology patients

(“ Order of the Minister of Health of the Republic of Uzbekistan No. 671 “On measures to improve nephrology and hemodialysis care for the population of the Republic of Uzbekistan” . 2018).

Inspection methods	Importance for diagnosis
I. Genealogical analysis (family tree analysis)	1. Identification of hereditary nephropathies 2. Determining susceptibility to kidney disease 3. Determining the direction of metabolic testing, taking into account diseases of the kidneys and other organs present in the offspring.
II . Biochemical examination	1 – screening indicators: a) test for the determination of peraminoaciduria using ninhydrin. b) systinnianiklaschunyod - azidlinima. c) Benedict's test for the detection of sugars. g) Fehling's test for the detection of phenylalanine. d) Sulkovich test for calcium determination. e) nitrite test to detect bacteriuria. Stage 2 – quantitative biochemical tests: a) daily (24h) excretion of oxalates in urine. b) calcium c) phosphorus g) protein d) uric acid e) amino acids j) indicators of ammonio-acidogenesis.
III . Clinical screening.	1. The patient's age at the time of initial onset of symptoms. 2. Presence of extrarenal (outside the kidneys) signs. 3. Changes in urine and its stagnation. 4. The origin of the disease depends on other diseases. 5. Hemogram, proteinogram, anti-streptococcal antibodies in the blood.

	6. Kidney function status.
IV. X-ray and ultrasound examinations.	1. Identification of hereditary and teratogenic birth defects of the kidneys and urinary tract. 2. Determining the presence of a stone. 3. To determine the presence of inflammation and destructive changes. 4. Radiography and radioplanimetry
V. Immunological and morphological examination methods	1. Identifying immune and autoimmune processes. 2. Morphological diagnosis.

The analysis of the patient's family tree has long been widely paid attention to. The achievements of modern medicine allow its use in even wider practice. Observations show that in the conditions of a nephrology clinic, based on the analysis of the family tree, it is possible to identify not only hereditary nephritis in particular, but also the possibility of determining the existing hereditary predisposition to kidney diseases.

In the conditions of modern nephrological clinics, the combination of genealogical analysis and biochemical methods shows the specificity of the composition of diseases occurring among the patient's relatives in metabolic disorders : in hyperoxaluria, kidney stones, chronic nephritis, pyelonephritis, hypertension, gastrointestinal diseases are observed, while in families with metabolic disorders causing uraturia, obesity, gout, spondylosis, tumor diseases are more often observed, and in cystinuria, stomach ulcers and gastritis, cholepathies are more often observed. This makes it possible to predict and investigate certain metabolic disorders based on genealogical analysis. Unlike the above-mentioned multifactorial kidney diseases associated with metabolism, hereditary nephritis is characterized by kidney diseases in the family tree, hearing impairment in older children and adults. Screening tests and quantitative biochemical tests make it possible to diagnose metabolic (dysmetabolic) nephropathies.

Clinical screening is an independent field, which includes the timing of the onset of the disease, renal (volume, color, composition of urine) and extrarenal signs (edema, increased blood pressure), and the relationship to other diseases, which is important for practice. For example, in early childhood, hereditary nephritis, dysmet a bolic nephropathies are more likely to manifest as changes in the urine. However, unlike glomerulonephritis, these changes can be observed not 2-3 weeks after the onset of the disease, but in the first days, and extrarenal signs and edema are not observed. For a complete diagnosis of the disease, to

identify congenital and hereditary defects, ultrasound and excretory urography are useful. In very complex cases, the method of morphological examination of kidney tissue obtained by biopsy is also used in specialized centers.

To screen large groups of healthy children for early detection of kidney disease, it is sufficient to conduct part of the above-mentioned screening program - genetic analysis , the first (screening) stage of the biochemical testing system, and clinical screening. The first stage of the biochemical testing included in the mentioned program is also notable for its ease of implementation, low cost, and sufficient information. Conducting a full program of testing for a definitive diagnosis of the disease requires specially trained personnel and an organizational laboratory environment.

Chapter III
PAST GLOMERULONEPHRITIS

Glomerulonephritis is an infectious-allergic inflammatory process of both kidneys, which is based on the defeat of the renal corpuscles. At the same time, in this disease, all organs and types of metabolism are involved in the pathological process. Glomerulonephritis can be observed in children of all ages, including the nephrotic form, up to 1 year old. In children, it is mainly observed after 5–7 years of age. The occurrence of glomerulonephritis depends on factors such as climatic conditions, season, hereditary predisposition, and epidemic spread of infectious diseases.

According to our observations, glomerulonephritis is 1.5-3.0% of patients admitted to hospitals for treatment, and its prevalence among the population under 14 years of age is 14.8 ± 1.22 per 1000 children. Among various diseases of the urinary system in children, it occupies a significant place (13.7-32.3%). When this indicator is analyzed by age and gender of the child, it is found that glomerulonephritis among various nephropathies up to 3 years of age is 12.2%, in children 8-14 years old it is 21.7%, in girls 14.0%, and in boys 29.7%.

The development of the disease also depends on the seasons, mainly in winter and autumn, which corresponds to the period when acute respiratory diseases are widespread. Glomerulonephritis, which developed in connection with the streptococcal infectious factor, is distinguished from secondary glomerulonephritis, hereditary nephritis, which is observed for other reasons (collagenosis, hemorrhagic vasculitis, etc.), and is called primary glomerulonephritis. According to the classification of primary glomerulonephritis, proposed by GN Speransky and colleagues in 1966 and repeatedly discussed and improved over the next 30 years (NA Korovina et al., 1990), acute, chronic and rapidly worsening hematuric, nephrotic and mixed forms of the disease are distinguished (Table 7).

Table 7

Classification of glomerulonephritis in children

(E.K.Petrosyan, S.S.Paunova et al., 2015)

Shapely	Course and clinical variant	Morphological variant	Pathogenetic mechanism	Stages	Kidney function status
Primary Secondary	Sharp : - separate urinary syndrome	Diffuse proliferative endocapillary GN Minimal changes	Immunocomplex GN C 3-nephropathy	Active Incomplete convalescence	OBE stages [1,2]

	- with nephritic syndrome - With nephrotic syndrome			Convalescence	
	Subacute: - Rapidly worsening	Extracapillary (crescent) GN Necrotizing GN	Immunocomplex GN S3-nephropathy S1q-nephropathy Immunocompromised GN Anti – GBM * nephritis	Period of onset of the disease Period of exacerbation of clinical signs	Stages of OBE
				Transition to a chronic form	SBE stages [3]
	Chronic: - Recurrent - Persistent - Increasingly heavy - with separate urinary syndrome - with nephritic syndrome - With nephrotic syndrome (including with hematuria and AG)	Minimal changes Focal-segmental glomerulosclerosis Mesangioproliferative GN Membranoproliferatve GN- densified deposits disease -Membranous nephropathy Necrotizing GN Fibrillary-immunotactoid GN Other morphological forms of chronic GN	Immunocomplex GN IgA-nephropathy IgM-nephropathy S3-nephropathy S1q-nephropathy Immunocompromised GN Anti – GBM nephritis	Recurrence Incomplete convalescence or complete clinical and laboratory remission	OBE, SBE stages

* GBM - glomerular basement membrane

acute glomerulonephritis according to the International Classification of Diseases (ICD-10) is:

N00 – Acute nephritic syndrome (with minimal lesions)

N00.0 – Acute nephritic syndrome with mild glomerular damage.

N00.1 – Acute nephritic syndrome with focal and segmental glomerular lesions (focal and segmental: hyalinosis, sclerosis. Focal glomerulonephritis)

N00.2 – Acute nephritic syndrome, diffuse membranous glomerulonephritis

N00.3 – Acute nephritic syndrome, diffuse mesangial proliferative glomerulonephritis

N00.4 – Acute nephritic syndrome , diffuse endocapillary proliferative glomerulonephritis

N00.5 – Acute nephritic syndrome, diffuse mesangiocapillary glomerulonephritis (Membranous-proliferative glomerulonephritis, types 1 and 3 or unspecified (NAC))

N00.6 – Acute nephritic syndrome, dark sediment disease (Membranous-proliferative glomerulonephritis, type 2)

N00.7 – Acute nephritic syndrome, diffuse sickle-cell glomerulonephritis (Extracapillary glomerulonephritis)

N00.8 – Acute nephritic syndrome with other changes in renal function Proliferative glomerulonephritis
nephritic syndrome , unspecified .

Acute glomerulonephritis - acute onset 1-3 weeks after exposure to the causative agent, with pronounced clinical signs characteristic of the onset, exacerbation and relapse periods. Usually, it ends in 80-90% of cases, with the patient's clinical and biochemical recovery from the disease within 3-4 months. The onset and exacerbation period of the disease usually lasts 2-4 weeks, the relapse (recovery) period lasts 2-3 months. Sometimes the recovery period lasts up to a year.

Clinical classification of glomerulonephritis:

1. Acute poststreptococcal glomerulonephritis
- cyclical, accompanied by the development of regeneration
- protracted and chronic

2. Acute nephritic syndrome developing in systemic diseases (lupus nephritis, nephritis in Schönlein-Genoch disease and other vasculitides)

3. IgA nephropathy

4. Acute glomerulonephritis

5. Membranoproliferative glomerulonephritis

Stages:

• Period of exacerbation of clinical and laboratory manifestations
• Period of relapse of symptoms
• Without complications
• With complications (hypertensive crisis, acute cerebral circulatory insufficiency, OBES, acute left ventricular heart failure)

Acute progressive glomerulonephritis is clinically similar to the mixed form of the disease, and is distinguished by a severe course of the disease at the very beginning and persistent impairment of kidney function.

The hematuric form of glomerulonephritis is characterized by the presence of gross hematuria along with the general symptoms of the disease, which is characterized by its persistence. It is mainly observed in children older than 5 years.

The nephrotic form of the disease is observed mainly (70%) in patients before school age, and its characteristic symptoms are severe dehydration, proteinuria and hypoproteinemia, and hypercholesterolemia.

The mixed form of the disease is more often observed after 10-12 years of age and is morphologically characterized by proliferative-membranous, proliferative-fibroplastic changes in the tubules, damage to the renal tubules and connective tissue. In this case, all the syndromes that occur in glomerulonephritis (excessive, hypertension, hematuria) develop together. Proteinuria is nonselective (i.e., the excretion of high molecular weight proteins through the urine), which is a sign of a severe course of the disease. When referring to the acute course of hematuric glomerulonephritis in these terms, it refers to acute glomerulonephritis, as described in other sources, chronic glomerulonephritis - to undulating, protracted, latent forms, and the nephrotic form - to the form of the disease called "pure", "idiopathic", "primary" nephrotic syndrome.

The classification of this disease also involves distinguishing between periods of activity (active, inactive). At the same time, it is of practical importance to determine not only the period of the disease, but also its level of activity (MS Ignatova, Yu.E. Veltishev, 1973) (Table 8).

Table 8

Glomerulonephritis activity level criteria

(MS Ignatova, Yu.E. Velti sh ev, 1973)

Activity level	Signs
III (strong)	Signs directly related to kidney function (renal) and beyond (extrarenal), severe changes in metabolism.
II (average)	Changes directly related to kidney function and relative changes in metabolism.
I (low)	Changes in urine are detected by special examination methods, and metabolism is slightly altered.
0 (inactive)	Clinical-laboratory remission.

To assess kidney function, filtration in the renal tubules, reabsorption in the tubules, and the kidney's ability to concentrate urine are determined.

In glomerulonephritis, the filtration process is initially impaired, which is assessed by determining the clearance of endogenous creatinine. The function of the renal tubules is assessed by determining the content of carbohydrates, amino acids, phosphates, ammonia in the urine, and finally, its specific gravity, more precisely, the osmolarity of blood and urine.

If the filtering and thickening ability of the kidneys is normal, then renal function is considered to be preserved. If the filtration of urine in the kidneys occurs equally during the day and at night or more at night (nocturia), then **the first degree of violation of its norm (rhythm) is considered to be BFB** $_{I}$ (since in a healthy child 2/3 of the daily urine is excreted during the day, and only 1/3

at night). **In BFB II** , a decrease in endogenous creatinine clearance up to 50% compared to the normal norm and a violation of the night-day filtration rhythm, a tendency to increase the amount of uric acid and residual nitrogen in the blood, and a change in ammonio-acidogenic activity are observed. Patients develop iso-, hypostenuria, anemia and hypertension during the exacerbation of the disease. This is a relatively balanced period of impaired renal function, which in the future passes into chronic renal failure **(BFB III)** . Now patients develop persistent hypertension, anemia, recurrent hypo- and isosthenuria, hyperazotemia , acid-base imbalance, and electrolyte imbalance.

Etiology **.** Primary acute glomerulonephritis always develops 1-3 weeks after an infectious disease. Such diseases are most often caused by streptococci (angina, exacerbation of chronic tonsillitis, jaundice), which is confirmed by the appearance of antibodies to streptococcal antigens in the blood of patients.

The importance of streptococci in the development of glomerulonephritis has also been confirmed by experiments on animals. It should be noted that in practice, glomerulonephritis is more often observed in connection with respiratory viral diseases (influenza) (52%), after a child has eaten a cold or after prophylactic vaccinations. These factors can be the direct cause of glomerulonephritis or act by activating latent streptococcal foci in the body. Usually, a cold in the body also has a similar effect, that is, it leads to the activation of latent streptococcal foci (23%).

When smears were taken from the throat of patients and bacteriologically examined, serological types of streptococci 12, 4, 18, 25, which have a structure close to the kidney tissue (i.e. nephritogenic), were isolated in 70.8% of patients at the onset of the disease. At the same time, the high titers of immune cells against streptococcal components (antistreptolysin – O , antihyaluronidase, antistreptokinase) in the blood of patients also confirm the above idea.

Pathogenesis. The development of acute glomerulonephritis is complex, it can be caused by damage to the renal corpuscles under the influence of the complex "antigen + antibodies" or be of a primary autoimmune nature. Complexes of "antigen + antibodies" in the blood are formed only when the body's sensitivity is increased and the number of antibodies in the blood increases sharply, and they settle in the renal corpuscles. This is due to the fact that the antigens included in the composition of group A streptococcal species are structurally similar to the basement membrane of the renal corpuscles, and they have a nephritogenic property. These antigens enter the blood in diseases caused by a number of streptococci and cause the formation of antibodies against themselves as foreign bodies. As evidence that glomerulonephritis develops precisely as a result of sensitization to streptococcal infection, there is evidence that it manifests itself after 1-3 weeks, which is necessary for a change in sensitivity after the onset of streptococcal disease, and that streptococci are not found directly in the kidneys and urine. At the same time, the increased content of immune cells against antigens included in the composition of streptococci, such as antistreptolysin-O, antistreptokinase, antistreptolysin, in the blood of patients, as well as diffuse inflammation of both kidneys at once, indicate that

this disease occurs as an allergic process. Under the influence of the complex "antigen + antibodies", the endothelium of all blood vessels in the body is damaged. Since this process is especially strong in the basal membrane of the renal corpuscles, clinical syndromes characteristic of glomerulonephritis (edema, hypertension, changes in urine) occur. In this process, the products formed as a result of the combination of antigen and immune cells are foreign to the body, and they now have the properties of autoantigen (foreign to the body's own tissues). Therefore, the formation of immune cells against these also continues, and the process takes on a chain nature. Such a process tends to last a long time and requires certain treatment to break the autoimmune chain. Sometimes such an autoimmune process has a primary nature, and the "antigen + antibody" complex can be formed not in the blood, but directly in the kidneys. It is believed that such a situation can occur under the influence of M-protein (nephritogenic) streptococci, which are structurally close to the kidney tissues and basement membrane. In these cases, severe, irreversible damage to the kidneys occurs (bad-quality nephritis). The life and activity of the whole organism, each organ and cell are directly related to the state of the cell membrane. The structure of membranes, their structural changes are the basis of a number of congenital, hereditary and acquired diseases. As is known, temporary changes in the composition of membranes are always observed when various infectious, toxic and other agents affect the body. However, the course of the disease, in particular, the transition to a chronic course, and its complications directly depend on the degree of membrane damage and its duration. Damage to membranes in the body is mainly associated with the oxidation of lipids that make up the membrane, the effect of phospholipases on them, and immunological effects that may occur on the surface of the membranes. The destruction of cell membranes (membranolysis) is associated with, in particular, phospholipase, lysophosphotides and some intermediate products that are formed as a result of the free oxidation of lipids. Lysolecithin, which is formed in this process, is an important factor leading to the thinning of membranes and an increase in their permeability. Under physiological conditions, the formation of lysophosphatides occurs under the influence of endogenous phospholipases, and the rate of this process reflects the degree of renewal of phospholipids in biological membranes. The intensification of this process leads to an increased accumulation of lysophosphatides and their detrimental effect on the membrane. The level of activity of this process is directly related to the cytoplasmic phospholipases A and C in the body. Normally, there are 2 types of phospholipases in the kidney: specific and general. **Specific phospholipases** are present in the walls of blood vessels, are activated by bradykinin, angiotensin, and increase the formation of prostaglandins in the body. On the contrary, **general phospholipases** are the same in all kidney tissue, their activity increases under the influence of hypoxia, but do not affect the formation of prostaglandins. The activity of phospholipases increases in vitamin E deficiency, hypoxia, under the influence of substances such as thyroxine, bradykinin, deoxycorticosterone, and, conversely, decreases under the influence

of vitamin E, steroid hormones, quinoline derivatives, and aspirin. In various kidney diseases, changes in the composition of phospholipids entering the membrane of kidney cells have been identified, and this process can be influenced in the treatment of the disease in several ways:

1. Antioxidant - the use of pharmacological substances with anti-oxidative properties;

2. Use of substances that inhibit the activity of endogenous phospholipases and drugs rich in them to replace unsaturated fatty acids and phospholipids lost as a result of severe oxidation.

Clinical picture. In most cases, the clinical symptoms of glomerulonephritis appear 1-3 weeks after the child has had an infectious disease, when the child becomes ill, the child becomes pale, the face and eyelids swell, the urine becomes orange or reddish - similar to meat stew. The onset of the disease lasts 5-10 days, the swelling is initially observed only on the patient's face and eyelids, it is stronger in the morning, when the child wakes up, and later fluid can accumulate in the body cavities (abdomen, pleura, pericardium). Although a relative increase in body temperature is sometimes observed in the first days of the disease, an increase in temperature is not a characteristic feature of this disease. On the contrary, it should be remembered that in patients with glomerulonephritis, even mild cases can occur without an increase in body temperature. At the beginning of the disease, the child may experience headaches, back pain, and sometimes pain during urination, nausea and vomiting. At the beginning of the disease, the patient's arterial blood pressure rises by 15-25 mm Hg compared to the norm for his age, and often returns to normal within 1-2 weeks under the influence of the treatment. However, if the disease develops in connection with the child's exposure to cold, physical injury, or preventive vaccinations, the latent period (onset period) may be reduced to 3-5 days. The period when all the symptoms of the disease are fully manifested - the acute period - lasts 2-4 weeks in cases where the disease proceeds without complications. At the beginning of the disease, renal eclampsia is sometimes observed - the patient may have anuria, hypertension, and therefore severe headache, vomiting, convulsions, and even loss of consciousness. During this period, an acute disruption of the homeostatic function of the kidneys is possible, which is associated not with a sclerotic process, but with an acute inflammatory process and is transient in nature. Therefore, this condition is called **"acute renal failure in the acute period of glomerulonephritis** . "

The general clinical picture of glomerulonephritis consists of several syndromes, and the form of the disease is distinguished depending on the degree of development of these syndromes and their combination. The main symptoms of glomerulonephritis are edema, urinary and hypertension syndromes, which are observed in the initial period of the disease in combination with the symptoms of general intoxication of the body described above.

Glomerulonephritis is a disease of the entire body, not just the kidneys, and the nervous system is not left out. In particular, the patient's extreme anxiety or

fatigue, headaches, and vegetative changes are symptoms caused by mild irritation of brain cells and short-term compression of cerebral blood vessels.

Urinary syndrome is present in all forms of glomerulonephritis, but manifests itself to varying degrees.

Oliguria - occurs the earliest, manifests itself simultaneously with back pain and headache, hypertension. As a result of a decrease in the number of actively functioning nephrons in the patient's kidneys, the destruction of their blood vessel endothelium due to inflammation, the formation of small thrombi in these vessels, fluid filtration slows down, daily diuresis decreases to 20-50% of the usual volume, dropping to 80-100 ml per day. This urine has a high specific gravity (1030-1040). Oliguria is considered to be the excretion of less than 200 ml of urine per m ^{2} of the patient's body surface per day, anuria is less than 50 ml, and polyuria is more than 1500 ml. Sometimes short-term anuria may occur. This is also caused by water reabsorption in the distal part of the renal tubules ("antidiuresis"). When the patient lies still for 4-5 days, blood circulation in the kidneys improves, urine filtration is restored, and these changes disappear.

Hematuria is an important sign of glomerulonephritis, and the presence of more than 3 erythrocytes in each field of view of a microscope in freshly collected (unpreserved) urine, or more than 1000 erythrocytes per ml of urine when examined by the Necheporenko method, or more than 1000 erythrocytes per minute of urine separated by the Ambourghe method, or more than 1 million erythrocytes in one night-day urine according to Kakovsky-Addis. The presence of up to 40 erythrocytes in the field of view of a microscope does not cause a change in the color of the urine (microhematuria). Microhematuria is observed in 100% of patients with glomerulonephritis. When the urine contains more erythrocytes than this, its color becomes noticeable (macrohematuria). At the onset of the disease, 80% of patients have macrohematuria, and its level varies throughout the course of the disease. Most often, hematuria decreases from 3-4 weeks of the disease and completely disappears within 2 months. Sometimes residual hematuria can persist for 6 months or more. Glomerulonephritis is accompanied by the appearance of altered, fragmented erythrocytes in the urine, which are formed due to their diapedesis through the walls of the capillaries of the renal pelvis. Hematuria can occur in its pure form, but is more often combined with proteinuria, leukocyturia, and cylindruria. **Proteinuria** is defined as the excretion of more than the usual 60-100 mg of protein per day in the urine, and in kidney diseases the amount of protein excreted daily is 1-2-3 grams or more. Proteinuria is considered to be severe proteinuria when the daily loss of protein in the urine is 3 grams or more and occurs in patients with nephrotic syndrome. Proteinuria occurs mainly as a result of changes in the permeability of the capillary blood vessels - podocytes, basement membrane and endotheliocytes - in patients with glomerulonephritis. It is known that in a healthy organism, both the basement membrane and podocytes and protein molecules have a negative charge, and accordingly, protein molecules are pushed away from the blood vessel wall according to a certain law of physics. In pathological conditions, the membrane and podocytes lose their negative

charge and become positively charged, which creates conditions for increased filtration of proteins. The limitation of protein reabsorption in the renal tubules is another factor in proteinuria. For acute primary glomerulonephritis, selective, that is, excretion of uroproteins with a molecular weight of up to 1,000,000 (mainly albumin) with urine is characteristic, and a large amount of high molecular weight (globulins) (nonselective proteinuria) occurs in acute exacerbations and mixed forms of the disease . Glomerulonephritis with nonselective proteinuria is severe, often treatment with steroid hormones does not give sufficient results. For primary acute glomerulonephritis, selectivity is characteristic, and proteinuria completely disappears during the period when the patient is clinically well.

Leukocyturia - occurs in 40-50% of patients with acute glomerulonephritis, and up to 20-30 leukocytes can be present in each field of view. In glomerulonephritis, leukocyturia is mainly due to lymphocytes (up to 50%) and eosinophils (20%). In contrast, in pyelonephritis, neutrophils are mainly observed. Long-term (more than 1-2 weeks) leukocyturia is not a characteristic sign of acute glomerulonephritis. In such cases, pyelonephritis and inflammatory diseases of the urinary tract may be present. In patients with chronic glomerulonephritis, the presence of intermittently active pyelonephritis is detected in 10-15% of patients.

Cylindruria - in acute glomerulonephritis, it is often observed in 30-70% of patients, in the amount of 8-12 per field of view. They are uroprotein in composition and have a cylindrical shape. Under the influence of the inflammatory process, they are formed in the distal part of the renal tubules, contain various inclusions, and accordingly, cellular (erythrocytic, leukocyte, epithelial), granular, hyaline, waxy types are distinguished.

Edema syndrome is a prominent and early developing symptom of glomerulonephritis, the degree of which can range from morning puffiness of the eyelids and face (nephritic edema) to complete edema of the whole body, even accumulation of water in all body cavities (anasarca) (nephrotic edema). In glomerulonephritis, sometimes even in cases where it is not clearly visible, the McClure-Aldrich test (the period of absorption of 0.1 ml of physiological fluid injected into the skin) shows a pronounced increase in the degree of hydrophilicity of the tissues, that is, the formed bubble is absorbed within 40-50 minutes, if the hydrophilicity of the tissue increases. The causes of edema in glomerulonephritis are complex (Fig. 2), it develops due to increased vascular permeability, hypoproteinemia, secondary hyperaldosteronism.

In the nephritic form of glomerulonephritis, edema is observed on the face, eyelids, and body. In this case, there is no pronounced proteinuria or hypoproteinemia. In this case, edema develops due to a greater change in hydrostatic pressure and increased vascular permeability.

In the nephrotic form of the disease, severe hypoproteinemia, hyperproteinuria, and decreased oncotic pressure are added to the above, edema spreads widely, and fluid accumulates in the abdominal, pleural, and pericardial

cavities. During the patient's recovery period, edema gradually disappears over 2 - 3 - 4 weeks as urine output is restored.

Damage to the cardiovascular system is manifested mainly by an increase in arterial blood pressure. Arterial blood pressure at the onset and peak of the disease rises by 15-20 mm Hg (2 - 2.6 kPa) above the patient's age-appropriate indicator and remains there for 1-2 weeks. This period coincides with the period when the patient's condition improves, edema and proteinuria begin to decrease. It is known that under physiological conditions the kidneys participate in the process of blood pressure control by ensuring the constancy of the balance of sodium and water metabolism (homeostasis).

The kidney's participation in this process is carried out through the renin-angiotensin-aldosterone system, which is associated with the coordinated activity of the arterioles, adrenal glands, and central nervous system. 90% of the renin substance, which is the initial link in the renin-angiotensin-aldosterone system, is formed in the epithelioid cells of the supraglomerular zone of the kidneys. In glomerulonephritis, as a result of impaired blood circulation in the kidneys, ischemia, and an increase in the sodium content in the tubules, renin is produced in excess of the norm and interacts with the angiotensin substance contained in the 2-globulin in the liver α, converting it into angiotensin I (hypertensin I) . Angiotensin I is a polypeptide containing 10 different amino acids, which, under the influence of enzymes in the blood serum, lungs and other tissues, is converted into an 8-amino acid polypeptide - angiotensin II (hypertensin II), which has a strong vasoconstrictor effect. At the same time, angiotensin increases the formation of aldosterone and indirectly causes sodium and fluid retention in the body. This, in turn, affects blood pressure by increasing the volume of blood circulating in the blood vessels and activating the sympathetic-adrenaline system.

Hypertension can be of varying degrees, depending on the form and course of glomerulonephritis. In the nephrotic form of the disease, short-term hypertension (20-30 mm Hg or 2.6-3.9 kPa) is observed, and sometimes in this form, blood pressure may not change at all.

In the hematuric form, such hypertension is often observed in 70% of patients for 1–2 weeks. However, a persistent increase in systolic blood pressure from 120 to 160 mm Hg (14.9–20.3 kPa) and diastolic blood pressure from 90 to 120 mm Hg (11.9–14.9 kPa) is observed in the mixed form of glomerulonephritis.

In addition, during the period of glomerulonephritis, a relative attenuation of its sounds can be heard directly from the heart, sometimes systolic murmurs of varying degrees. These signs, as observed in all organs in glomerulonephritis, occur as a result of edema of the heart muscle, a decrease in its contractility, sometimes as a result of fluid accumulation in the pericardial cavity. These changes are manifested on the ECG by signs such as a relative decrease in R, R and T waves, slowing of atrioventricular conduction and an expansion of the R – Q interval, which improve the patient's general condition and pass along with edema and other signs. However, sometimes, due to high hypertension, severe hydro-ionic changes, signs of acute circulatory disorders may occur in the early

stages of glomerulonephritis. In this case, the patient may experience shortness of breath, blueness of the limbs and face (cyanosis), rapidly increasing edema, a sharp enlargement of the liver, and the appearance of numerous small and medium-sized wet rales in the lungs. In some cases, when the disease begins with such symptoms, it may be difficult to distinguish glomerulonephritis from heart disease itself in the early stages.

The form of the disease is determined by the degree to which the above syndromes have developed.

In the active period of glomerulonephritis, there is a decrease in endogenous creatinine clearance by 50% or more compared to the normal rate (1.3 - 2 ml / sec). During this period, the blood purification function of the kidneys is also impaired, and the amount of residual nitrogen and urea in the blood increases (normal rate is 6.0-8.0 mmol / l). During oligo- and anuria, metabolic acidosis, hyperkalemia are observed. Hypo- and dysproteinemia develop in the blood. In nephrotic syndrome, hypercholesterolemia, hyperlipidemia and dyslipidemia are observed. In the blood of patients, there is a 2-5-fold increase in the number of immune cells against antigenic products of streptococci, such as streptolysin, streptokinase, hyaluronidase, deoxyribonuclease, +and a decrease in the amount of complements due to their use in the process of "antigen-antigen immune cells" and their localization in the kidneys.

Peripheral blood analysis may show varying degrees of anemia, since in this case the production of hemopoietins in the kidneys, which enhance hemopoiesis, is reduced. In addition, metabolic disorders, metabolic acidosis lead to a shortened life span of erythrocytes. The number of leukocytes and eosinophils in the blood increases, and the erythrocyte sedimentation rate accelerates to 30-40 mm / h.

Comparative diagnosis. In cases where the history of the disease is detailed and all the symptoms are fully manifested at the time of onset, the diagnosis of glomerulonephritis does not cause difficulties . However, in practice, this is less common, there are a number of diseases (congenital, hereditary, secondary nephropathies, etc.) that are accompanied by changes in the urine, without external symptoms, the complex of which is no less common than glomerulonephritis.

Acute glomerulonephritis should first be differentiated from kidney damage caused by infectious diseases, drug-induced nephropathy, hereditary nephritis, urolithiasis, dysmetabolic nephropathies, renal tuberculosis, and secondary glomerulopathies (systemic lupus erythematosus, hemorrhagic vasculitis , etc.).

It is extremely important to differentiate glomerulonephritis from congenital and hereditary kidney diseases. There are a number of drugs (corticosteroids, cytostatics) that are relatively widely used in the treatment of glomerulonephritis, which are not only useless in other diseases (kidney malformations, hereditary nephritis, amyloidosis) but can also be harmful.

Unlike glomerulonephritis, hereditary nephritis does not have external signs of the kidneys (swelling, increased blood pressure). Hematuria is detected by chance, when examined for some disease, and is permanent. There is no 2-3

week interval between the initial disease, which is characteristic of glomerulonephritis. Changes in urine are detected by chance mainly at an early age - due to urolithiasis, URVK, when examining for admission to kindergarten and school. Glomerulonephritis is rare in children of early age (especially the hematuria form). For the diagnosis of hereditary nephritis, an analysis of the patient's family tree is of great importance, which reveals the presence of male individuals in the family with chronic diseases, chronic renal failure, and deafness that began after 12-20 years of age.

Sometimes, during the acute period of acute bronchitis, sepsis, acute respiratory viral diseases, as a result of the accumulation of toxic and allergic products in the body and their harmful effect on the renal blood vessels, proteinuria (up to 200-300 mg), microhematuria, and sometimes cylindruria can be observed. In this case, urine changes are observed only during the acute period of the infectious disease, and disappear without a trace with the recovery of the underlying disease, unless they are a manifestation of a previously latent disease in the kidneys (hereditary nephritis, tubulopathy, developmental defects).

In this case, these changes can be considered as infectious-toxic damage to the kidneys. However, sometimes these changes are persistent and may indicate the development of infectious inflammation in the child (interstitial nephritis or pyelonephritis).

Acute glomerulonephritis should be differentiated from pyelonephritis, a common disease in children. The main symptoms of glomerulonephritis - edema, hypertension - are not characteristic of primary pyelonephritis. However, pyelonephritis, which in most cases develops secondary in children with tubulopathy , developmental defects of the urinary organs, and metabolic disorders, can be accompanied by hematuria and general symptoms (pallor, headache, loss of appetite). In this case, the hematuric form of glomerulonephritis is distinguished by the presence of unchanged erythrocyte membranes, predominantly observed and stable, mainly granular forms of leukocyturia, and the excretion of more than 100,000 microbes per ml of urine. Excretory urography and ultrasound examination can confirm the diagnosis by showing infiltrative changes mainly in the renal parenchyma in glomerulonephritis, and changes in the calyces and renal pelvis in pyelonephritis.

It can also sometimes be difficult to distinguish acute glomerulonephritis from the activated period of chronic glomerulonephritis. This difficulty arises when the presence of chronic glomerulonephritis has not been previously detected. In this case, a thorough study of the patient's family tree, life and medical history is required. Unlike acute glomerulonephritis, activation of chronic glomerulonephritis develops not 2-3 weeks after the intercurrent illness, but during it or after 2-3 days. In this case, even if it is determined that the patient had acute glomerulonephritis several years ago, in this case it is possible to think about the latent chronic course of the disease.

Urolithiasis - also accompanied by hematuria. Erythrocytes are not changed, because they are not formed by diapedesis, but by damage to small blood vessels

by stones and crystals of oxalate, cystine, etc. In this case, unlike glomerulonephritis, there is constant pain in the lower back, sometimes attacks of renal colic in the lower back, pain radiating to the groin, external genitalia and groin, dysuria. Since stone disease often occurs with secondary pyelonephritis, an increase in body temperature, leukocyturia may be observed. The definitive diagnosis of the disease is made on the basis of ultrasound and X-ray urological examinations.

Renal tuberculosis is also accompanied by symptoms of general intoxication of the body, subfebrile body temperature, aseptic leukocyturia and hematuria. The diagnosis of the disease is based on the presence of such patients in the family, the presence of tuberculosis foci in other internal organs of the patient - lungs, lymph nodes, bones, etc., the results of the tuberculin test, and the results of urine testing for tuberculosis mycobacteria.

The diagnosis of secondary kidney damage in systemic lupus erythematosus and other systemic connective tissue diseases, hemorrhagic vasculitis requires taking into account changes in the skin, joints, and other organs of the underlying disease (Liskina GA, 2003).

Hemorrhagic vasculitis (Scheinlein-Genoax disease) is often accompanied by hematuria, and there is also a form of this disease that is mainly accompanied by kidney disease (capillarotoxic nephritis). In these cases, the presence of other symptoms of hemorrhagic vasculitis (arthralgia, abdominal pain, symmetrical small rashes on the limbs and body) during the onset of the disease is important for the diagnosis.

There are a number of hereditary and secondary metabolic disorders (cystinuria, hyperoxaluria, uraturia, impaired tryptophan metabolism, etc.), which are characterized by the accumulation of nephrotoxic products in the body, which, during their excretion with urine, damage the kidneys and urinary tract, leading to hematuria. In these cases, hematuria, unlike glomerulonephritis, is observed mainly in early childhood (most often in children under 5 years of age). Such changes are most often detected accidentally due to colds, acute respiratory infections and other diseases. Urine changes do not occur after 2-3 weeks, which is typical for glomerulonephritis, but during the course of the underlying disease. In addition to the above, family history and appropriate biochemical tests are of decisive importance in their diagnosis. This problem is discussed in a separate section.

When conducting a comparative diagnosis of glomerulonephritis, it should be borne in mind that it can occur in combination with a number of other diseases. In particular, chronic glomerulonephritis in 20-30% of cases is accompanied by a microbial inflammatory process of the kidneys and urinary tract. In this case, clinical signs of glomerulonephritis and pyelonephritis can be observed simultaneously, persistent leukocyturia and bacteriuria can be detected. Leukocyturia observed in patients with acute glomerulonephritis for 1-2 weeks of the disease is transient and occurs without bacteriuria. X-ray contrast and bacteriological examinations allow to establish the diagnosis of the disease. The addition of pyelonephritis often occurs 1-2 years after the onset of

glomerulonephritis. In this case, neutrophilic leukocyturia, hypoisosthenuria appear in the urine. In turn, pyelonephritis can cause activation of glomerulonephritis.

Glomerulonephritis also has a certain specificity when it occurs in children with metabolic disorders (uraturia, hyperoxaluria, etc.). In this case, the appearance of unchanged erythrocytes in the urine is accompanied by tubulo-interstitial changes in glomerulonephritis, so the ability of the kidneys to concentrate urine is impaired in the early stages of glomerulonephritis. As a result, hypo-, isosthenuria are observed with reduced filtration (oligouria). This is not typical for glomerulonephritis. However, such a situation may occur if tubulo-interstitial nephritis is also present.

In addition, glomerulonephritis in children with developmental defects of the kidneys and urinary tract is often severe, mixed, and prone to chronic course. In a child with dysembryogenesis, glomerulonephritis and pyelonephritis may also coexist, which creates certain difficulties in their treatment. In all cases, careful comparative diagnosis of the disease increases the effectiveness of treatment.

Indications for hospitalization:

urgent hospitalization - severity of the patient's condition (oliguria, azotemia, very high hypertension, edema); complicated acute respiratory distress syndrome (hypertensive crisis, acute cerebral circulatory insufficiency, acute renal or heart failure);

planned hospitalization – uncomplicated acute respiratory distress syndrome, in order to clarify the diagnosis in the case of a prolonged course of the disease.

List of basic diagnostic procedures:

• Complete blood count, hematocrit
• Quantitative determination of SRO
• Determination of creatinine, urea, uric acid
• Determination of the ball filtration rate using the Schwarz formula.
• Determination of total protein and its fractions
• Determination of ALT, AST, cholesterol, bilirubin, total lipids
• Determination of potassium, sodium, chlorides, iron, calcium, magnesium, phosphorus
• ASL-O, streptokinase determination
• Coagulogram (prothrombin time, fibrinogen, thrombin time, activated partial prothrombin time (APTT))
• Serum fibrinolytic activity
• Determination of acid-base balance
• Detection of markers of hepatitis A, B, C, D viruses using IFA
• Detection of intrauterine and zoonotic infections using IFA
• Detection of anti-DNA autoantibodies, antinuclear autoantibodies, antineutrophil cytoplasmic and perinuclear antibodies, and anti-GBM antibodies using IFA.
• Determination of S1q, S3, S4 fractions of complements using IFA
• General urine analysis, determination of daily proteinuria
• Urine protein electrophoresis (determination of proteinuria selectivity)

• Determination of protein/creatinine ratio.
• Zimnisky test
• Abdominal organs UTT
• Renal vascular Dopplerometry
• Fundus examination

List of additional diagnostic procedures:

• Immunological testing methods: ANA, ANCA, anti-DNA double helix antibodies S3, S4, S50, cryoglobulins, anti-cardiolipin antibodies, anti-Streptolysin-O antibodies, anti-GBM antibodies
• Biopsy of kidneys, skin, subcutaneous fat layer, muscles, rectal mucosa
• Detection of markers of hepatitis A, B, C, D viruses using IFA
• Chest X-ray
• ECG, EchoCG
• Determination of Bence-Jones proteins in blood and urine
• Detection of HBV-DNA and HCV-RNA by polymerase chain reaction (PCR)
• Coagulogram 2 (soluble fibrin monomer complexes (RFMC), ethanol test, antithrombin III, platelet function)
• Detection of immunoglobulins A, M, G, E using IFA
• CT, MRI
• Throat swab
• Infectionist, otolaryngologist consultations
• Bacteriological examination of urine.

List of diagnostic procedures performed prior to planned hospitalization:

• Complete blood count
• Determination of the amount of renin in urine
• Blood urea nitrogen
• Blood electrolyte levels
• Total protein
• Transaminases
• Thymol test
• Bilirubin
• Kidneys UTT
• Throat swab

Laboratory test results:

Urine is colored coffee, tea or "meatballs" (hematuria); there is a slight proteinuria - from 1-3 g/milk to 3 g/milk. Up to. In the urine sediment - altered erythrocytes, erythrocyte cylinders are detected. There are slight immunopathological processes: ESR 20-30 mm/s, increased antistreptococcal AT titer (antistreptolysin-O, antistreptokinase, antihyaluronidase), hypocomplementemia due to the SZ-component and a decrease in total cryoglobulin. A decrease in CFT, an increase in the concentration of creatinine in the blood (azotemia) are detected. Nonspecific indicators of inflammation: an increase in the concentration of SRO, fibrinogen, a decrease in the amount of

total protein, albumins; mild anemia (due to hydremia) is observed. The presence of a low level of complement S3 even after 6-8 weeks of the active period of the nephritic syndrome is consistent with MPGN nephritis, which in turn is a direct indication for kidney biopsy. Biopsy should be performed on the basis of strict indications: for the purpose of comparative diagnosis with chronic glomerulonephritis, including systemic diseases of the connective tissue, rapidly developing glomerulonephritis and atypical course of the disease; the absence of positive changes in the course of the disease for 1 week, a sharp decline in kidney function. The following morphological changes are characteristic of OGN:

- Diffuse proliferative endocapillary glomerulonephritis picture
- Infiltration of renal tubules with neutrophils and monocytes
- Electron-hardened deposits of immune complexes
- Detection of extracapillary proliferation in some renal tubules
- In the capillaries and mesangium, IgG, SZ complement component, less often - S1q and S4, IgA, IgM (TQYu).

9th adv.

Nephritis Comparative diagnosis of the syndrome

(MS Ignatova, Yu.E. Velti sh ev, 19 85) **:**

Characters	Acute nephritic syndrome	Chronic nephritis syndrome	Urinary tract infection (Acute hemorrhagic cystitis)	Nephrotic syndrome
Onset of the disease	Acute, infection-related (most often with streptococci), ARVI	Sharp, or slow,	Acute, associated with cold hardening	Slowly slowly
Tumors	Not so strong, more on the periphery	Usually severe at the onset of the disease, may recur	No	Widely spread, until anasarca
Arterial hypertension	Unstable hypertension	Less severe, slowly progressive	No	Hypotension and hypertension too to be possible
Dysuria	No	No	+++	No
Intoxication	++	+	No	Not suitable

Hematuria	Glomerular in nature	Not so strong, constant	Not glomerular in nature	Not suitable
Proteinuria	Less than 3g/milk	Less than 2g/milk	minimum	More than 3g/ milk
Leukocyturia	Not suitable	Not suitable	+++	Not suitable
Hyperazotemia	Usually at the beginning of the disease, transient	It gets worse as the disease progresses.		Rarely, transient during the period of disease activity

Treatment.

The goal of treatment is:

− Removal from active period
− Elimination of azotemia
−Eliminate oliguria, edema, and convulsions
− Normalization of arterial blood pressure
− Reduce/eliminate proteinuria, hematuria
− Diagnostic verification.

Treatment tactics:

Non-drug treatment: Bed rest in the first days, then ward and general regimens. Diet No. 7 (7a, 7b): restriction of salt and fluid intake (the volume of fluid is determined by the amount of diuresis the previous day + 300 ml), the amount of calories and vitamins is maintained. If edema is present and increases, salt intake is reduced to 0.2-0.3 g/day, proteins to 0.5-0.6 g/kg of body weight (mainly due to animal proteins).

Drug treatment:

1. To improve microcirculation in the kidneys, antiplatelet agents are used - dipyridamole tablets 25 mg, 75 mg/day; pentoxifylline 100 mg/day.

2. For antihypertensive and nephroprotective purposes, angiotensin-converting enzyme inhibitors - fosinopril 20 mg/day, enalapril 20 mg/day, ramipril 10 mg/day in tablets; calcium channel blockers - amlodipine 10 mg/day, nifedipine in tablets 10 and 20 mg, 40 mg/day; beta-adrenoreceptor blockers - bisoprolol 10 mg/day, alpha- and beta-adrenoreceptor blockers carvedilol 25 mg/day, angiotensin II receptor antagonists (losartan 100 mg/day, telmisartan 80 mg/day in tablets and bq).

3. In case of edema, hyperhydration and related complications, loop diuretics (furosemide 2-3 mg/kg, hydrochlorothiazide (50-100 mg/day in tablets) are used. If ineffective, ultrafiltration is used.

4. Antibacterial agents are used in cases of foci of infection or acute infectious process to eliminate the focus of infection and eradicate the pathogen. In poststreptococcal acute respiratory viral infection (swallow taken from the

throat, when the antistreptococcal AT titer is increased) - penicillin G 1.0 million TB is prescribed 6 times / day for 10 days).

5. Amoxicillin, clavulanic acid in tablets 250-500 mg 2 times a day for 7-10 days, cefaclor (powder for suspension preparation 125 mg/5 ml) 250-500 mg/ml 2 times a day for 7-10 days are prescribed as the drugs of choice for sinusitis and pneumonia.

6. If an allergy to β-lactam antibiotics is detected, macrolide antibiotics are prescribed: azithromycin 500 mg once a day for 5 days, spiramycin tablets 1.5 million IU and 3.0 million IU, 6 million IU per day for 7 days.

7. In case of severe azotemia and hyperkalemia, hemodialysis is performed.

Preventive measures:

− prevention of viral, bacterial, fungal infections
− prevention of electrolyte imbalance
− prevention of eclampsia, cardiovascular insufficiency, DVT syndrome

Further observations:

At the outpatient stage after hospitalization: adherence to the regimen (protection from cold, stress, physical exertion); adherence to the diet; sanitation of chronic foci of infection, antihypertensive treatment.

Dispensary observation for 5 years (first year - quarterly measurement of AQB, complete blood count, urinalysis, determination of serum creatinine and calculation of KFT according to the Schwarz formula).

If extrarenal symptoms (arterial hypertension, edema), severe urinary syndrome persist for 2 months or more, it is necessary to perform kidney biopsy examinations, since in such cases there is a high probability of identifying dangerous morphological variants of GN requiring immunosuppressive therapy.

CHAPTER IV
CHRONIC GLOMERULONEPHRITIS

Chronic glomerulonephritis - is characterized by a continuous, recurrent or chronic course, lasting more than one year. There is no clear difference between the causative agents of acute and chronic glomerulonephritis, it seems that the internal - immunological, biochemical, constitutional characteristics of the organism and hereditary predisposition are more important.

Chronic glomerulonephritis is a bilateral immune-allergic generalized inflammation of the kidneys, which is a disease that often recurs and lasts a long time, with progressive deterioration of kidney function and damage to other organs in the body. Sometimes chronic glomerulonephritis occurs without obvious symptoms (latent), leading to sclerosis of the glomeruli and chronic renal failure and uremia. Currently, 50% of patients requiring hemodialysis and transplantation have chronic renal failure due to chronic glomerulonephritis.

Etiology and pathogenesis. The causes of chronic glomerulonephritis are still not fully understood. As in nephritis in general, the association of streptococcus, viruses and bacteria is important in its genesis. In 80-90% of such patients, there is some chronic focus of infection, and in most cases, glomerulonephritis is also activated under the influence of their exacerbation.

The development of chronic glomerulonephritis can be caused by a child's eating cold food, repeated administration of serum, use of nephrotoxic drugs, and sometimes exposure to chemicals. In some cases, acute glomerulonephritis (10-15%) develops into a chronic course if it is diagnosed late, treated poorly, the diet is grossly violated, and medical supervision is not organized during the recovery period. However, the course of acute and chronic glomerulonephritis often varies depending on the presence or absence of a family predisposition, the level of maturity of the immunological system and the kidney tissue. In most cases, its course does not show signs of acuteness, and the disease progresses to a chronic course at the very beginning. In such cases, it is impossible to associate the onset of the disease with any infectious disease. Thus, chronic glomerulonephritis can be considered a group of glomerulopathies that develop from various causes, leading to sclerosis of the glomeruli, renal tubules, and connective tissue, and chronic renal failure.

any case, it has been established that chronic glomerulonephritis develops due to the formation of immune complexes against the kidney's own tissue (autoaggression), since immune complexes against kidney tissue can be found in patients already in the early stages of the disease. In addition to the large number of immune complexes against the basement membranes of the glomeruli in such patients, a decrease in complement in the blood, hypercoagulability, and the deposition of complement and immunoglobulin (immunoglobulin and S_3) complexes on the basement membrane confirm the autoimmune nature of the disease. These phenomena also indicate the causes of the development of sclerosis in the kidney and its dysfunction.

Clinical picture. Chronic glomerulonephritis does not have specific, unique clinical signs. Its symptoms are diverse and not always noticeable. During the

period of its activation due to a viral or bacterial disease, the syndromes observed in acute glomerulonephritis are clearly manifested, corresponding to the form of the disease. Sometimes the disease can be clinically latent, occurring only with pathological changes in the urine. But in both cases, the course becomes more severe, kidney function decreases, and after 2-20 years the patient develops symptoms of chronic renal failure, leading to a terminal condition.

Chronic glomerulonephritis is divided into hematuric, nephrotic, and mixed forms, and one form can transition into another as the disease progresses.

The hematuric form of the disease begins mainly in school age and proceeds with nephritic syndrome. The main symptom is constant hematuria of varying degrees. In such patients, edema is minimal or absent, proteinuria is less than 1 g per day, and arterial blood pressure rises periodically. Kidney function is maintained for a long time, the specific gravity of urine, as well as the amount of residual nitrogen and urea in the blood are within the norm, and during the activation of the disease it can increase for a short time. However, as a result of progressive sclerosis of the kidneys, their entire homeostatic function is disrupted, all signs of chronic renal failure appear (acidosis, hypo- or hyperkalemia, hyperazotemia, anemia, hypertension, etc.). This period may vary depending on the patient's hereditary and constitutional characteristics, the degree of morphological maturity of the kidneys, etc.

Chronic glomerulonephritis can sometimes be so insidious that it is only discovered when the patient is diagnosed with kidney failure.

Of the disease **The nephrotic form** is more common in children of early age, often in their family history there is a predisposition to ECD, bronchial asthma and other allergic diseases, kidney diseases. The disease manifests itself with the gradual development of all the symptoms observed in acute glomerulonephritis over several days after exposure to a causative agent. The characteristic clinical and biochemical features of this syndrome are widespread edema, anasarca, severe proteinuria (65-160 and more) and hypoproteinemia (up to 40-50 g / l), hypercholesterolemia (6-12 mmol / l). In the urine sediment, there may be an absolute lack of erythrocytes and leukocytes, the presence of cylinders, and blood pressure may also be normal. During the active period of the disease, oliguria (up to 50-100 ml); high specific gravity of urine (1030-1040), hyperaminoaciduria (excretion of amino acids in large quantities with urine due to limited reabsorption) are observed. The content of nitrogenous substances in the blood may increase slightly during the period of activity, but is generally maintained at a normal level for a long time . It is important that these changes are recurrent and tend to be chronic, with a wave-like course, and clinical and biochemical indicators do not differ quantitatively from the nephrotic form of acute glomerulonephritis.

There can be various reasons for the development of nephrotic syndrome. For this reason, it is customary to distinguish two groups - primary and secondary nephrotic syndrome. After R. Bright described the disease of the kidneys with edema, in which the blood serum acquires a milky color (nephrosis) in 1827, the concept of nephrotic syndrome has changed several times. In particular, this

syndrome was considered not a kidney disease, but a disorder of protein and fat metabolism, and in 1939-1940 it was considered mainly a consequence of fat metabolism. However, modern electron microscopic studies have confirmed that the basis of this disease is damage to the basement membrane of the capillary vessels that enter the renal corpuscles, which are mainly allergic and autoimmune in nature.

However, it would be a mistake to associate nephrotic syndrome only with glomerulonephritis, since it can sometimes be observed in tuberculosis, osteomyelitis, amyloidosis, lead, mercury poisoning, and a number of other cases. Usually, when we talk about primary nephrotic syndrome, we mean nephrosis observed in congenital nephrotic syndrome, glomerulonephritis, and amyloidosis. Secondary nephrotic syndrome, on the other hand, has certain and specific features of its development in a number of cases, such as polycystic disease, collagenosis, metabolic disorders (diabetes mellitus, lipoidosis, glycogenosis), poisoning with drugs, salts of heavy metals.

The nephrotic form of glomerulonephritis can develop immunologically in two ways: 1) the deposition of antigens and antibodies (antibodies) in the blood on the walls of the blood vessels of the renal corpuscles, 2) kidney disease as a result of immunological activation of the capillary basement membranes and the deposition of antibodies in the blood. This type of immunological process occurs mainly at an age when the kidney tissues are not fully mature, and the body is not immunologically ready to neutralize the antibodies appearing in the blood at a sufficient speed. Therefore, in experimental conditions, nephrotic syndrome develops mainly in young animals, and in humans in children aged 1-7 years. It is also known from the literature that nephrotic syndrome is prevalent in children under 3 years of age in 1.8%, in children aged 3-5 years in 7-8: 100,000, and after 8 years of age in 1: 100,000.

Most scientists believe that the development of nephrotic syndrome is influenced by toxicosis of pregnancy, congenital defects in the development of kidney tissue, birth injuries (asphyxia, brain injury), infectious diseases of the fetus, immunological characteristics appropriate to the child's age, and allergic diseases.

One of the main causes of the development of nephrotic edema, hypoproteinemia, is the immunologically induced increase in the permeability of the basement membrane of the capillaries and, as a result, the loss of protein in the urine, which is the main reason, but it cannot be called the only reason. Because the decrease in protein in the blood is also to some extent due to a decrease in protein production by the liver, long-term dietary nutrition, and finally, impaired protein absorption from the intestines. Hypo- and dysproteinemia are mainly αcaused by the loss of albumins with small molecular sizes in the urine and, conversely, the retention of γ_2 -globulins in the blood. The increase in 2-globulins in the blood corresponds to the immunologically active period of the disease. In children with nephrosis, the total amount of fats in the blood increases by 2-4 times. The development of hyperlipidemia in combination with hypoproteinemia and proteinuria can serve as a basis for considering it as one of

the means of preventing a sharp decrease in colloid-osmotic pressure in the blood. Hyper- and dyslipidemia observed in nephrotic nephrosis are also of such clinical significance that such changes are not observed in tumors associated with the liver or heart.

Thus, in the development of nephrotic edema, changes in the structure of the renal tubules, loss of protein in the urine, a decrease in the osmotic-colloid pressure of the blood, increased vascular permeability, hypovolemia , hyperaldosteronism, etc. play an important role. In primary lipoid-nephrotic syndrome, signs of damage to the renal tubules are observed - transient glucosuria, acidosis, aminoaciduria, hyperkalemia. The resulting hypokalemia, in turn, leads to a decrease in the filtration process in the renal tubules, impaired urine concentration function of the tubules. These factors, in turn, further intensify degenerative changes in the tubular cells, their spaces become clogged, and urine passage becomes difficult.

Clinical picture. Nephrotic syndrome develops gradually. Patients and their parents first notice pallor and swelling that increases day by day. The sick child becomes lethargic, capricious, and loses his appetite. The swelling is initially noticeable on the child's face and eyelids in the morning, his face may become round and slightly fat. However, within a few days, the swelling increases, the patient's eyes can hardly open, and the swelling spreads to the body and legs. Fluid can also accumulate in the abdomen and chest cavities. As a result of ascites, the patient's breathing becomes difficult due to the movement of the diaphragm and the restriction of lung movement by hydrothorax. As the swelling progresses, the patient may complain of dry mouth, nausea, and sometimes diarrhea. Due to increased permeability of the intestinal walls, the patient's stool contains a large amount of protein, which is what some call "protein" diarrhea. The patient's complete loss of appetite, proteinuria, and protein diarrhea, although not noticeable due to metabolic disorders and edema, lead to severe weight loss.

The onset of edema is marked by a sharp decrease in urine output, its daily volume may be 100 ml or less **.** Urine is reddish, rich in protein, and its specific gravity is high (1030 - 1045 and more). Daily protein loss with urine is 5 - 10 - 20 g and more, of which 70% is albumin. In the urine sediment, the epithelium of the urinary tract, various cylinders are detected in large quantities. Hematuria and leukocyturia are characteristic of primary lipoid-nephrotic syndrome. The content of creatinine, urea and residual total nitrogen in the blood does not change significantly. The clearance of endogenous creatinine may be normal, decreased or, conversely, increased.

There are no significant changes in the activity of the heart and blood vessels, a short-term increase in blood pressure may often remain unchanged, the boundaries of the heart and its rate do not change with age, sometimes a systolic murmur appears on three sides of the heart and the heart sounds become slightly muffled.

peripheral blood, mild hypochromic anemia may be detected. The number and formula of leukocytes change only when other intermediate infectious diseases are added, that is, leukostasis is observed.

ESR is within 30-60 mm/h throughout the disease. In patients with nephrotic syndrome, protein metabolism disorders are noticeable, hypoproteinemia (up to 30-40 g/l), hypoalbuminemia (up to 15-29%), and a decrease in α_2- globulins by up to 13% are observed. At the same time, hyperlipidemia , hypercholesterolemia, and a decrease in potassium and calcium are also observed. Often, due to the swelling of the liver, it can protrude 4-6 cm below the costal margin, and the spleen 1-2 cm below. Nephrotic syndrome is characterized by recurrent and severe exacerbations, which often occur in connection with angina, angina pectoris, and sometimes without any apparent cause. Despite the fact that nephrotic syndrome is a chronic and relapsing disease, in most cases its outcome is favorable, which is achieved mainly due to the use of corticosteroid hormones for treatment. With each exacerbation of the disease, symptoms of renal dysfunction appear, which worsen with each passing year and eventually end in chronic renal failure. During this period, its extrarenal symptoms (anemia, uremic symptoms, persistent hypertension) appear.

Mixed glomerulonephritis is characterized by a combination and strong manifestation of all the syndromes observed. In patients, along with severe nonselective proteinuria and other symptoms of nephrotic syndrome, hematuria (0.5-0.8 ±7 and more per day), leukocyturia (0.2 x 10^7) are observed. If pyelonephritis also occurs, leukocyturia intensifies, bacteriuria appears. Arterial blood pressure increases to 90-60 mm. cm. and more, and it is persistently elevated, and signs of left heart failure and acute circulatory failure may develop.

In a patient with a mixed form of chronic glomerulonephritis, chronic renal failure can develop in a relatively short period of time, as evidenced by a decrease in ammonio-acidogenesis, an increase in blood urea nitrogen to 8.33 mmol/l, creatinine to more than 0.1 mmol/l, and a decrease in glomerular filtration rate to 25% of the patient's previous value over the past 6 months.

Differential diagnosis depends on the stage of the disease. If the patient has a history of acute glomerulonephritis, there is no problem. However, for the diagnosis of latent glomerulonephritis, chronic glomerulonephritis, renal tuberculosis, hereditary nephritis, tubulopathies, collagenoses, renal malformations, and dysmetabolic nephropathies should be considered.

comparative diagnosis is the differentiation of secondary glomerulonephritis, hematuria is more common in hemorrhagic vasculitis. In this case, the presence of other signs of hemorrhagic vasculitis is of great importance. Rapidly developing, severe nephropathy with hematuria and hypertension is also observed in a systemic disease of the connective tissue - nodular periarteritis . Glomerulonephritis with nephrotic syndrome can be relatively easily distinguished from the characteristic symptoms, and in particular from the diffuse edema observed in patients with cirrhosis of the liver. In this regard, the diagnosis of renal amyloidosis is also important. Usually amyloidosis develops as a complication of long-term purulent diseases

(tuberculosis, osteomyelitis), rheumatoid arthritis, tumors. The so-called periodic disease - the presence of other nephrotic individuals in the family, their chronic renal failure, the development of renal amyloidosis is a characteristic feature of this disease, which is characterized by abdominal pain and fever attacks. Amyloidosis is characterized by widespread persistent edema, enlargement of the liver and spleen, and impaired absorption of nutrients from the intestines.

The most reliable way to differentiate between nephritis and amyloidosis is to determine whether amyloid material is morphologically present in the kidney tissue or intestinal mucosa or gum tissue (Tareeva IE, Mukhin NA, 1983).

Secondary nephrotic nephritis is also observed in systemic lupus erythematosus. Systemic lupus erythematosus is characterized by simultaneous involvement of several organs. This disease is accompanied by erythema resembling a butterfly on the cheeks, polyserositis, pneumonitis, and a prolonged increase in body temperature. However, sometimes these systemic symptoms are less developed, and the symptoms of glomerulonephritis are more pronounced. This is called the “nephritic” mask of systemic lupus erythematosus. The detection of LE cells and antibodies to DNA is important for diagnosis.

Mixed glomerulonephritis with nephrotic syndrome and hypertension should be differentiated from secondary systemic glomerulonephritis, primarily lupus glomerulonephritis . It should be remembered that sometimes this type of glomerulonephritis develops in cases where there is dysplasia of the renal tissue and congenital kidney defects.

Thus, the comparative diagnosis of glomerulonephritis is important for determining the treatment of the disease, which varies significantly depending on the causes of its occurrence. For example, acute interstitial glomerulonephritis, which developed under the influence of drugs, requires the cessation of taking antibiotics , the appointment of prednisolone in moderate doses for a relatively short period (2-3 weeks) , nephropathies caused by metabolic disorders require a special diet depending on the metabolic period, lupus nephritis requires strong immunosuppressive therapy, and vice versa, renal amyloidosis requires the avoidance of corticosteroids and cytostatics in treatment, etc. In short, the more complete the comparative diagnosis of the disease, the better the outcome of the treatment.

Moderately acute glomerulonephritis is a very severe form of the disease, leading to persistent renal failure within a few weeks or months. Fortunately, this form of the disease occurs less often (1-2%). The etiology of the disease is largely similar to acute glomerulonephritis. Often, 2-4 weeks ago, it is determined that there were cases of angina, influenza, exacerbation of chronic tonsillitis, prophylactic vaccination, exposure to cold water. However, the disease can manifest itself in a shorter period (4-6 days) and is almost always accompanied by nephrotic syndrome. Arterial blood pressure is high, which quickly leads to cardiac dysfunction. The specific gravity of urine, which is high in the first days, quickly decreases, hypo-, isosthenuria develops. Severe hypoproteinemia, hyperazotemia, hypercholesterolemia are detected in the

blood. The patient's condition worsens rapidly and can last from 1.5 months to 2 years. The patient's death occurs due to azotemic uremia, heart failure, and secondary diseases (septicemia, sepsis).

Complications of glomerulonephritis

In some cases, glomerulonephritis can lead to conditions that require emergency medical care. The most common complication of acute glomerulonephritis is acute renal failure in the acute phase of glomerulonephritis, which occurs in the first days of the disease and is usually benign.

"Acute period of renal failure" is a renal (kidney) form of acute renal failure, which differs from other forms in the absence of pronounced stages characteristic of this condition (initial, oliguria, recovery), a relatively mild and rapid complete recovery of homeostasis. Acute period of renal failure in glomerulonephritis occurs as a result of impaired blood circulation in the kidneys, ischemia, a sharp decrease in filtration in the glomeruli. Symptoms of renal failure often do not progress due to the main symptoms of the disease (edema, oliguria, hypertension). A decrease in diuresis to 30% of the usual volume should be considered a clear sign of acute period of renal failure. At this time, the patient's general condition worsens - hyperazotemia and signs of azotemic intoxication are observed - drowsiness, headache, weakness. Appetite worsens, vomiting, diarrhea are observed. Electrolyte metabolism disorders - hyperkalemia, cardiac dysfunction - pose a risk of spontaneous cardiac arrest. Therefore, regular ECG monitoring is necessary . Timely pathogenetic treatment allows you to restore the urinary function of the kidneys and eliminate sharp changes in homeostasis. If, despite pathogenetic treatment, the urea level in the blood is 40 - 50 mmol / l, potassium is more than 6.5 mmol / l for 3 - 5 days, and the patient's weight is increasing by 5 - 7% per day with persistent acidosis, it is recommended to immediately begin hemodialysis or plasmapheresis.

Hypertensive encephalopathy - can be observed in patients with a rapid and sharp increase in blood pressure, even in the absence of severe edema and azotemia. In glomerulonephritis, arterial blood pressure in some patients rises sharply to high values for 1-2 days (or even within a few hours), and as a result, signs of encephalopathy develop: the child becomes very restless, his vision darkens, vision deteriorates, seizures are observed. The patient has a more pronounced increase in systolic blood pressure, heart sounds become louder, tachycardia appears.

In cases where the general edema typical of glomerulonephritis develops very rapidly in a short period of time, pulmonary edema may occur. This phenomenon is caused by increased capillary permeability, a decrease in the oncotic and osmotic pressure of the blood, which leads to fluid leakage into the lung tissue and alveoli. Along with the symptoms typical of glomerulonephritis, the patient also experiences cough, shortness of breath, and bluish lips and face in a short time, forcing him to take a semi-sitting position and become restless. In severe cases, the patient foams (sometimes reddish) from the mouth. Moist rales of various volumes are heard in the chest.

Basics of glomerulonephritis treatment.

The effectiveness of the treatment of all complications of acute glomerulonephritis is directly related to their early detection, and in this case, the prognosis of the disease is not negatively affected.

Treatment of glomerulonephritis is a complex problem and must be organized taking into account the causes of the disease, the form and degree of activity of the disease, and kidney function.

The patient needs comprehensive treatment, which includes the following: 1. (Symptomatic) treatment measures aimed at influencing certain symptoms of the disease present in the patient (daily regimen, diet, blood pressure-lowering - hypotensive, diuretic drugs , etc.). 2. Antibacterial (etiological) effect. 3. (Pathogenetic) treatment aimed at moderating immunobiological inflammatory processes in the body, disorders in the blood coagulation system.

In the acute course of glomerulonephritis, treatment is carried out in a hospital setting. The patient is recommended to lie still for 2-3 weeks until the symptoms of the disease subside. Then, gradually, they are allowed to sit up and walk around the room. Lying still in a warm place accelerates the dilation of blood vessels, a decrease in blood pressure, increased blood circulation in the kidneys, and increased urine output. If the patient does not have such symptoms, the strictly limited regimen is also reduced to 1-2 weeks.

The diet is prescribed taking into account the functioning of the kidneys, the degree of spread of edema, and the state of blood pressure. In the early days of the disease, when urine output is sharply reduced, and edema is strongly developed, a sparing diet (especially in terms of protein and table salt) is used. Dietary treatment is an important tool that has a general effect on the patient's body. Dietary food should be selected taking into account the age of the child, the causes of the disease, the type of disease and the stage at which it is, as well as the course of the disease. The main goal of dietary treatment is to restore the disturbed balance between the enzymes present in the body and the chemical compounds of food. This means aiming to bring the physical and chemical composition of food as close as possible to the characteristics of the metabolic processes taking place in the patient's body.

A special place in pediatrics should be given to diet. Because it is necessary to take into account the possibility of metabolic disorders in diseases, the needs of the growing child, and the age-specific characteristics. In some children's diseases, in particular, congenital metabolic disorders, diet is the only way to treat them. In these diseases, only dietary treatment can prevent the child from becoming mentally retarded and disabled for life, and ensure his proper physical and mental development. Diet is also an important part of treatment for other diseases, because without following the diet, treatment with drugs alone will have little effect or will not be useful at all.

The role of diet in diseases of the urinary system in children is currently being considered in a broader sense. For example, according to modern concepts, “dietary foods prescribed for kidney disease” cannot be used for all kidney diseases. After all, dietary foods closely depend on the causes of the disease, its

duration, stage, degree and nature of kidney dysfunction, and general changes in the body . If table salt is consumed very little in kidney diseases accompanied by edema, then there is no need to sharply limit salt in types that do not cause edema and do not increase blood pressure.

Considering the above situations, the basic principles of dietary treatment for kidney diseases can be stated as follows:

- prescribing protein, salts, and fluids, taking into account the nature and duration of the disease;
- prescribing dietary food based on the kidney's ability to tolerate it or not;
- Diet is prescribed to prevent kidney strain, protect it, and modify its function. Diet is especially important when certain types of kidney function are normal or have decreased.

The following general dietary requirements are imposed on patients with glomerulonephritis:

- During the acute phase of the disease, the protein content in food should be low;
- During the acute phase of the disease, salt intake is severely restricted, and then for a long time, salt intake is reduced (no more than 2–4 g per day);
- How much fluid you need to drink is determined based on the amount of urine you have the previous day.

In the early stages of acute glomerulonephritis, a diet low in animal protein and salt is recommended. This will improve kidney function, reduce swelling, lower blood pressure, and significantly reduce inflammation.

In the acute period of glomerulonephritis and in the acute form of its chronic form, if the body has clearly manifested edema and blood pressure is elevated, it is advisable to start the treatment with a fruit-sugar diet. Because it is difficult for a child to give only fruit or only sugar. That is why they are mixed. Sugar is given at the rate of 10-12 g per 1 kg of the child's body weight. It is even more advisable to use navvot instead of sugar. If the patient is given apples, it should be taken into account that approximately 300 g of apples contain 40 g of carbohydrates. On diet days, carrots, fruit puree, jam can be given. Usually, 1/3 of the total sweets given per day are given sugar, and the remaining 1/3 can be given fruit puree and jam. It will not hurt to give one or two cups of tea. If the need for such a diet arises again, it can be used again after 5-7 days. It is recommended to give 300 g of fruit per day to children aged 4-7, 500 g to children aged 7-11, and 800 g to children aged 11-14 . Fruits such as apples, grapes, melons, watermelons and other melon products are recommended.

After the fruit-sugar diet, a milk-vegetable diet can be used. In this type of diet, protein should be 50-55% of the norm necessary for the child's age (1.0-1.5 g/kg of body weight). For breakfast, lunch and dinner, various vegetables, potatoes, cereals and pasta dishes are served. You can prepare cereal soups with milk, vegetables and fruits. Salt should not be added to these dishes. It is

recommended to drink a liquid equivalent to the amount of urine excreted during the day.

In mild acute glomerulonephritis, if the child's general condition is not bad, the amount of urine excreted is also appropriate for his age. In such cases, it is possible to immediately switch to a milk-vegetable diet. Milk-yogurt, eggs, cream, butter, vegetable oils, cereals, potatoes, vegetables, fruits-cheeses, fruit juices, sugar, jam, marmalade, unsalted bread can be given.

For such patients, food is prepared by boiling and mashing. Salt is not added to it. Fruit juice can be added to improve the taste a little.

It is recommended to serve porridge, pasta, eggs, sweet tea or milk tea **for breakfast .**

Lunch can be as follows:

The first one is liquid foods with milk, fruit, cereals, soup with potatoes, and vegetables;

The second is served with boiled or steamed potatoes, mashed potatoes or vegetables, stuffed cabbage, fried dumplings made from cereals or potatoes, wet fruits - chevals, vegetables, pasta dishes. It is also possible to pour a little oil, cream or fruit juice on top of the prepared dishes; the third is served with drinks made from fruits, compote, jelly, lemon or custard.

Dinner: fruit - cheval, fruit juices, yogurt, potato or apple pancakes, boiled potatoes, cereal pancakes, rice, pasta, porridge, white flour bread.

In cases where the amount of urine excreted during the day or night is low, it is advisable not to give fruit juices, otherwise the potassium content may increase. Later, depending on the increase in the amount of urine, fruit juices can be given gradually. During the period of excessive urine excretion (polyuria), fruit juices should be given on time, so that the potassium content in the blood is not allowed to decrease. Considering that during the period of starvation, the metabolism is seriously disrupted and the proteins in the body are rapidly broken down, it is necessary to provide the body with sufficient calories, even if the kidney function is significantly impaired. Therefore, the above-mentioned low-protein, low-calorie diet foods should be given every 7-10 days, for a maximum of 1-2 days.

When increasing the variety of diet foods, it is necessary to give foods such as shirchoy, shirkhurda, shirguruch, boiled meat, dumplings made with vegetables. These foods should be given gradually, that is, for 3-4 months. As urine output increases, potassium-rich foods such as pickles, raisins, tomatoes, pumpkin, quince, grapes, cherries, pomegranates, lemons, potatoes, beets, carrots, green peas, buckwheat groats, and oatmeal can also be used in food.

Children do not like salt-free food, it quickly “stomachs up”. Therefore, patients taking hormones can be given 1 g of salt in their food starting from 3-4 weeks, and children prone to high blood pressure after 5-6 weeks. Gradually, wheat bread is introduced, then meat, fish, cheese, and finally, the amount of salt is increased from 1 g to 4 g. Even after the patient is discharged from the hospital, the amount of salt in the diet at home for 1-2 years should not exceed 2-4 g per day. From the period of remission to 6 months, patients should not be given

mushroom soups, smoked, salted and allergenic products (strawberries, citrus fruits, etc.).

Children with chronic glomerulonephritis, if their kidney function is significantly impaired, should be careful about their diet for a long time. First of all, it is necessary to regulate protein, water and salt metabolism. The diet is prescribed depending on the symptoms of the disease and the degree of renal dysfunction.

If chronic glomerulonephritis worsens, edema develops, and blood pressure increases even though kidney function is not impaired, it is recommended to give milk-based, green foods with limited protein (up to 50-55% of the age-appropriate norm), just like in acute glomerulonephritis. However, it is important to ensure that the calories are sufficient.

If chronic glomerulonephritis is accompanied by mild changes in urine, a diet with a protein intake appropriate for the age group but with a salt intake of up to 18 mmol/L can be given. If there is no benefit from the treatment, it is better to use foods with a lower protein intake.

chronic glomerulonephritis, when the activity is significantly reduced, and even if there is a small amount of protein in the urine, it is possible to increase the variety of foods only after 1-2 months. You can gradually switch to eating foods that are relatively high in protein and sodium (oven-baked wheat bread, boiled meat, cottage cheese, fish).

After 4 months of the disease activity decreasing, the child can be given foods appropriate to his age. However, even then it is necessary to correctly determine the ratio of animal fat and vegetable oil (60 and 40%). To meet these requirements, in addition to the foods used during the exacerbation of the disease, the child is also given meat, fish, and cottage cheese. At this time, the first meal should be prepared from greens and vegetables without meat, and dishes made from meat and fish should be eaten in the first half of the day. This is the same period if these foods are boiled. Potatoes can also be fried.

It is advisable for patients not to drink meat and fish soups, eat fried meat, smoked foods, spicy and salty foods until 6 months after the disease has subsided. The intended goal will be achieved if 1-2 g of salt is added to the patient's food in 2-3 months, 3-5 g in 4 months, and 5-6 g from 6 months onwards.

Antibiotics. Given the etiological importance of infectious agents (especially streptococci) in the etiology of nephritis, antibiotics are recommended for 2-3 weeks. Penicillin group drugs (penicillin, ampicillin, oxacillin) are more appropriate. Erythromycin, oleandomycin can also be used. The use of nephrotoxic antibiotics (streptomycin, kanamycin) is not recommended. If the patient has a chronic focus of infection, continuous treatment with antibiotics should be continued for up to 2 months. During the acute period of the disease, antihistamines (dimedrol, pipolfen, suprastin, tavegil, zaditen) are prescribed for 1.5-2 months, alternating every 5-7 days. If the patient is lethargic and prone to sleepiness, then other antihistamines can be used instead of starting treatment with dimedrol and pipolfen. In addition to the above, in all forms of the disease, vitamins (especially ascorbic acid, rutin), calcium chloride are prescribed to

improve metabolism. Thus, the treatment of all acute forms of glomerulonephritis begins with a specific regimen, diet, the appointment of antibiotics and antihistamines, vitamins.

Since hypercoagulation is often observed in the hematuria form of glomerulonephritis, heparin 100 U/kg and antiplatelet agents (dipyridamole) are recommended.

-inflammatory drugs - aspirin, indomethacin (indocid, methindol), ibuprofen or voltaren - are prescribed. It is advisable to prescribe them when the patient's condition has significantly improved, swelling has decreased, and blood pressure has stabilized. This group of patients is prescribed aminoquinol, delagil, 5-6 mg/kg per day, plaquenil 4-5 mg/kg for 3-6 months, which have the property of suppressing the inflammatory process to a certain extent and reducing sclerosis of the kidneys.

The opinions of specialists regarding the use of glucocorticoid therapy in the form of glomerulonephritis with hematuria differ. In particular, LPGavryushova and HM (1990) consider such treatment to be inexpedient, taking into account the risk of a number of complications. However, in cases of prolonged hematuria (6 months or more), the beneficial effect of short-term (1.5-2 months) administration of prednisolone in moderate doses (1 mg/kg) is known from nephrological practice (MSIgnatova, 1978).

In the nephrotic form of glomerulonephritis, glucocorticoids are widely used, since they have not only anti-inflammatory, but also antihistamine, immunosuppressive properties. In the treatment of nephrotic nephritis, prednisolone is prescribed at a dose of 2-2.5 mg per kg of the patient's weight for 4-6 weeks. At the same time, anti-hypercoagulant and anti-aggregation agents (heparin, curantil), vasodilators (euphyllin, theophylline and bq) are used. If clinical remission is not observed within 4-6 weeks, this is a sign of hormonal resistance of the disease, which requires continuation of treatment with cytostatic (immunological processes suppressive) drugs.

A four-component regimen has been recommended for the treatment of severe glomerulonephritis with refractory nephrotic edema (Kinkite – Smith, 1972): - glucocorticoids (2 – 2.5 mg/kg), immunosuppressants (0.2 – 0.3 mg/kg), heparin (100 – 300 IU), and dipyridamole (10 mg/kg).

Given the importance of allergies in the development of glomerulonephritis, it is recommended to use Zaditen (0.025 mg/kg) and Intal (0.1 g 4 times a day for children aged 2–12 years) in the years following antihistamines.

Treatment of mixed glomerulonephritis is a more complex problem, and it is necessary to take into account which symptoms are predominant (hypertension, nephrotic edema, etc.). Treatment with prednisolone alone in this form of glomerulonephritis is often ineffective, and therefore it is recommended to switch to a four-component treatment early. In cases where the patient has developed renal failure, it is not advisable to use immunosuppressants , often corticosteroids.

of glomerulonephritis , in which cases even a four-component treatment with the usual amount of drugs may not help the patient. In such cases, treatment with

extremely high doses of corticosteroids (pulse therapy) is recommended (methylprednisolone is prescribed up to 1000 mg / g for 3-7 days). In such cases, it is also advisable to resort to plasmapheresis, hemosorption methods.

To counteract the varying degrees of edema and decreased urine output (oliguria) observed in glomerulonephritis, it is recommended to use diuretics (Lasix, Verasphine , Hypothiazide, Mannitol, Mannitol).

About the scope of action and method of use of diuretics. Substances that increase the excretion of water and salts from the body through the kidneys are called diuretics.

In order to use diuretics appropriately, it is necessary to have sufficient knowledge about water-salt metabolism in the body, the process of urine excretion, and the effects of diuretics on it. Therefore, we will briefly review some of the issues, albeit in part.

In particular, the main system that ensures the functioning of the kidney is the nephron, which is composed of a bundle of capillary blood vessels, its sheath and tubules. On the walls of the arterioles that bring blood to the renal tubules, where they enter the tubules, there is a supraglomerular apparatus (SGA), which in certain cases has the property of releasing renin into the blood. The tubules of the nephron consist of 4 parts (Fig. 4):

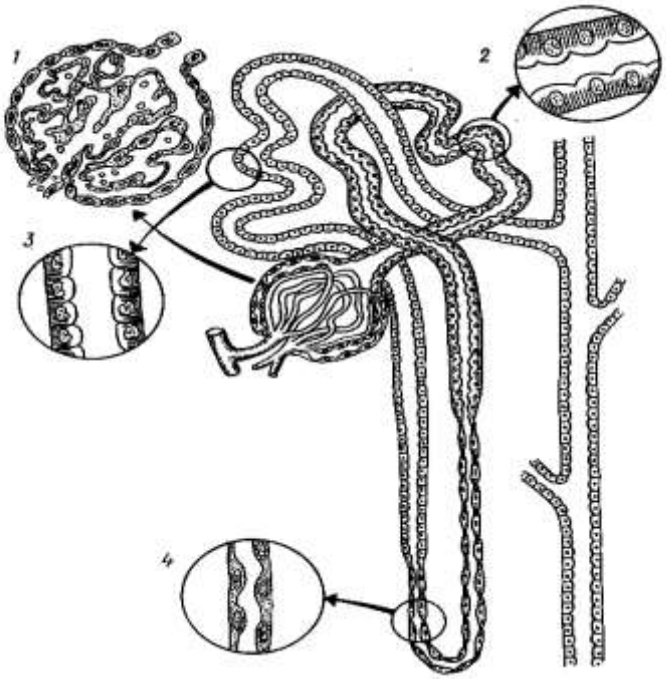


Figure 4. *Structure of the nephron: 1- Glomerulus; 2- Proximal part of the ureter; 3- Distal part of the tubule; 4- Thin part of the loop of Henle* (MS Ignatova, Yu.E. Velti sh ev, 1973)

In the convoluted part of the distal tubules is the maculadenza, which is associated with the YA and has receptor properties that sense changes in the chemical composition of urine. In the renal medulla, there are cells that participate in the formation of prostaglandins . The formation of urine is the result of a complex process of filtration in the renal medulla, reabsorption in the tubules and secretion. The filtration process in the renal medulla depends on the

hydrostatic pressure in the capillaries (70 mm Hg), the oncotic pressure of the protein in the blood serum (30 mm Hg), and the hydrostatic pressure of the ultrafiltrate passing through the space between the medulla and its shell (Shumlyansky-Bauman shell) (20 mm Hg). Filtration can occur only if the blood pressure in the capillaries of the renal medulla is higher than the fluid pressure in the renal corpuscles and the total oncotic pressure. This difference, commonly referred to as the "useful filtration pressure", is equal to 20 mm of air pressure .

The daily volume of primary urine filtered by the tubules is 150–200 liters, of which 96–98% is reabsorbed in the tubules, and finally 1–1.5 liters of urine is excreted. The osmotically active components of primary urine are close to those of blood serum (glucose, urea, creatinine, uric acid) and differ mainly in the amount of anions that do not pass through the membranes. The substances filtered by primary urine are reabsorbed in the renal tubules. In particular, with primary urine secreting 500–1000 gNaCl per day, only 5–10 gNaCl is excreted in the urine.

With the participation of enzymes located in the epithelial cells of the proximal part of the renal tubules, amino acids, glucose, protein, phosphates, chlorides, vitamins, trace elements and water, as well as 2/3 of NaCl, are reabsorbed. However, urine in the proximal tubules is still maintained in an isoosmotic state with respect to blood serum. In the distal part of the renal tubules, water, sodium reabsorption, potassium excretion (secretion) occur under the influence of antidiuretic hormone - ADG (processed in the posterior pituitary gland), aldosterone (adrenal gland hormone) and biologically active substances produced in the kidneys (renin, prostaglandins).

Water-salt metabolism in the body depends on the external balance, their internal distribution and directions in the body. **The average water balance consists of the amount of fluid entering the body** (drinking - 1300 ml, with food - 1000 ml, as a result of metabolism - 200 ml) and being excreted from it (diuresis - 1400 ml, through the skin and lungs - 1000 ml, with feces - 100 ml). On average, 60% of the body weight of a healthy man, and 50% of the weight of a woman - consists of water. 2/3 of the total water is located outside the cells.

The extracellular environment consists of the aqueous environment inside the blood vessels (4–5% of body weight is blood serum) and the interstitial environment (interstitial fluid, which makes up 15% of body weight), with intercellular fluid making up 1–2%.

The osmotic pressure of blood is the sum of the pressures of osmotically active substances in the blood and is 285–295 mol/l. Approximately 50% of the plasma osmotic pressure is due to Na and 30% to Cl. An increase in plasma osmotic pressure causes an increase in the production of ADG and a feeling of thirst. The osmotic pressure of plasma, which is formed due to high-molecular substances such as albumin, globulin, fibrinogen, is called colloid osmotic pressure and is equal to 25 mm Hg in healthy people. The total amount of fluid in the body and its distribution in the internal environments of the body are constant and are related to the amount of electrolytes and proteins in the fluids inside and outside the cells. The distribution of water in the body between the

internal and external environments of blood vessels, according to Starling's theory, occurs in connection with the colloid-osmotic pressure of blood serum and tissue fluid, the hydrostatic pressure in the capillaries, and the permeability of the capillary walls.

For example, oncotic pressure is aimed at retaining fluid in the blood vessels, while hydrostatic pressure helps it to flow out of the blood vessels. In the last arterial capillaries, the hydrostatic pressure (34 mm Hg) is higher than the oncotic pressure (20–25 mm Hg), and due to this difference, ultrafiltration from the blood vessels into the interstitium is observed. At the beginning of the venous capillaries, the hydrostatic pressure is lower than the oncotic pressure, and therefore the fluid tends to move from the tissues into the vessels. At the same time, metabolic waste products also pass into the venous blood.

Electrolytes are important in maintaining the osmotic constancy of the internal environment of the body (osmotic homeostasis). Sodium is the most important cation that provides the osmotic pressure of the extracellular fluid environment. An increase in the concentration of Na in the extracellular fluid leads to the outflow of water from the cell, and vice versa, a decrease in its amount leads to the movement of surrounding water into the cell. The main cation of the intracellular fluid environment is potassium (97% of the total potassium) , and proteins are mainly creatinine, phosphorus, carbohydrates, proteins, and are present in a partially ionized form.

In the interstitial space and plasma, K^+ is in an ionized form. The concentration of osmotically active substances in the intracellular and interstitial space depends on the work of the Na / K pump. As a result of this pump, K freely passes through the cell membrane into its interior, while sodium is expelled into the surrounding environment. Due to the Na / K pump, a certain amount of water is released on both sides of the cell membrane.

A Na:K gradient is formed, which in turn is influenced by Sa ions. Like potassium, magnesium is mainly an intracellular cation and participates in various enzymatic processes. Phosphate and sulfate are also more intracellular. Chlorine is more associated with Na and is stored in the extracellular fluid. Hydrocarbons play an important role in the buffer system of the extracellular fluid.

In maintaining the water-salt balance in the body, the kidney's activity is manifested in the form of filtration in its membranes, reabsorption of fluid and various substances from the tubules, and secretion (secretion). Since the interior of the cell has a negative charge, and sodium has a positive charge, it enters the cell based on the gradient difference. When passing through the membrane, a certain amount of energy is required to force the electrochemical gradient and pass sodium through the basement membrane. This energy, which is generated during oxidation and glycolysis, is accumulated as ATP, and as the Na – K – ATPase enzyme, it participates in the passage of sodium from the cell into the blood and the entry of K into the cell. "Permeases" (protein-binding carriers) participate in this process. The synthesis of permeases is enhanced by the action of aldosterone. Aldosterone is a hormone of the adrenal cortex, which begins to

be released more when the volume of fluid in the extracellular environment decreases and the sodium content in the plasma decreases. This response can also be observed when the potassium content increases. Under the influence of aldosterone, Na reabsorption in the renal tubules increases sharply, and as a result, urine output decreases. If aldosterone is produced in excess of the norm , most of it is discharged with the participation of liver enzymes. Since aldosterone ensures the reabsorption of sodium, in cases where its production in the adrenal glands is impaired, sodium loss with urine increases sharply. In this sense, the importance of antidiuretic hormone (ADH) is particularly great, and its production in the hypothalamus increases when the total volume of blood circulating in the blood vessels decreases and the osmotic pressure of blood plasma and intracellular fluid increases. The effects of ADG occur in the distal tubules and collecting ducts of the nephron.

When the total volume and osmolarity of intracellular and extracellular fluid decreases, urine output also decreases, and conversely, when its volume and osmolarity increase, urine output also increases.

The control of water-salt metabolism and urine output in the body is a very complex process, in which catecholamines also participate. Catecholamines directly affect the supraglomerular (SGA) apparatus, increasing the release of angiotensin and aldosterone. In addition to increasing the release of aldosterone, angiotensin also directly affects the reabsorption of sodium in the distal tubules.

Thyroid and triiodothyronine have a direct effect on the kidneys - they increase blood circulation in the kidneys, increase fluid filtration in the tubules, i.e. urine excretion, and also increase the excretion of Na and K with urine. Insulin increases the reabsorption of sodium and water in the renal tubules, i.e. has an antidiuretic effect. On the contrary, glucagon increases the excretion of sodium, potassium, chlorine and water by the kidneys. Under the influence of testosterone, urine and sodium excretion decrease. Anabolic steroids also increase filtration in the renal tubules. Estrogens, on the other hand, slow down the excretion of water and sodium. Progesterone has an opposite effect on aldosterone. Prostaglandins of groups A and E change the ratio of blood circulation in the cortex and medulla of the kidney, leading to increased natriuresis and diuresis. The kallikrein-kinin system has an opposite effect on the renin-angiotensin system in terms of its effect on the state of blood vessels and the regulation of sodium balance. Serotonin to some extent reduces blood circulation, filtration, and diuresis in the kidneys. The direct effect of histamine on the kidneys leads to a decrease in the reabsorption of water and sodium from the renal tubules. Diuretics differ from each other in terms of their effect on the above-mentioned stages of urine formation, which means that these properties of drugs must be taken into account for their successful use in medical practice.

Classification of diuretic drugs:

1. Supernatremia-causing agents: furosemide, bumetanide, ethacrynic acid.
2. Carbonic anhydrase inhibitors: acetazolamide (diacarb, diamox).
3. Thiazide and non-thiazide sulfonamides: dichlorothiazide, cyclomethiazide, chlorthalidone, clopamide.

4. Potassium-sparing agents: triamterene, amiloride, spironolactone.
5. Osmotic agents: mannitol, urea.
6. Xanthine derivatives: euphylline, theophylline, diaphylline.
7. Uricosuric agents: inacrinone, thienyl acid.

Diuretics with a variety of effects are used to treat edema caused by kidney disease (Table 10).

Table 10 .

Use of diuretics in kidney disease
order (MS Ignatova, Yu.E. Velti sh ev, 1973)

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Urine drivers	Type of tumor			
	Nephritic	Nephrotic form		
		Hormone sensitive	Hormone dependence	Hormone-resistant
Prednisolone	------	++ 2mg/kg (at least 2-3 weeks)	+	------
Hypothiazide	+ 3 – 5 days	+	+	++
Lasix	+ if a quick effect is required, 1 – 3 days	+	+	++
Urethritis	+ 1 – 2 days if a quick effect is required	+	++	+
Aldactone	-------	++ (3-5 days before or at the same time as the hormones are	++	++ days in combination with osmotic agents and heparin)

		administered)		
Osmotic agents	------	++ daily or every other day with aldactone	++	++
Heparin	-------	+	+	+

Note: ++ must be used; + can be used; --- does not apply.

When using diuretics, their action characteristics, speed, duration of action, and eventual occurrence

The measures listed in the table serve to restore metabolic balance in the body, restore sensitivity to diuretics. One of the life-threatening complications of uncontrolled use of diuretics is a violation of potassium balance in the body, which requires early detection and correction (Table 11).

Table 11.

Clinical signs of hypo and hyperkalemia.

(MS Ignatova, Yu.E. Velti sh ev, 1973)

Hypokalemia	Hyperkalemia
Modesty The same thing Abdominal distension (intestinal paresis may be observed) Decreased reflexes Enlargement of the heart borders Decreased arterial (more diastolic) blood pressure, tachycardia Ectopic arrhythmia, increased sensitivity to cardiac glycosides ECG : shortening of the QT interval and T wave, increase in the systolic index and appearance of a U wave.	Modesty A feeling of "ants crawling" on the body -paresthesia in the arms, legs, mouth area, pain in the muscles of the tongue, arms, legs. Bradycardia and ventricular fibrillation may occur. ECG: T wave is high and sharp, R wave is shallow

Basics of treatment with urinary drivers.

1. Avoid excessive urine output in the short term. Achieving an average weight loss of 0.5–1 kg per day based on urine output is not considered dangerous.
2. Prescribe the appropriate amount of diuretic medication for each patient and administer them in the order they are intended.
3. Prescribing potassium-sparing diuretics to patients at risk of developing secondary hyperaldosteronism.
4. Take breaks from time to time while performing the urinary catheterization procedure.
5. Ensuring a water-salt intake regime.

When selecting and prescribing diuretics to a patient, the doctor must take into account several factors: the symptoms of the disease that caused the diuretic treatment, secondary diseases that coexist with the main disease and may affect the absorption of the drug into the blood, its distribution in the body, and its binding to proteins, the degree of need for diuretic treatment, and the presence of conditions that may cause complications of such treatment.

The main criteria for determining the diuretic and its dosage are the underlying disease, its development process, and the characteristics of its course.

Among the sodium-expelling agents, furosemide is an anthranilic acid derivative, and bumetanide is an anthranilic acid isomer. These drugs have the strongest sodium-expelling properties compared to all other diuretics. These drugs are absorbed from the gastrointestinal tract strongly - 70 - 75%. They show their effect after 10 - 15 minutes when administered intravenously, and after 50 - 60 minutes when taken orally. The half-life of furosemide is 35 minutes, and it acts for 6 - 8 hours. The effect of furosemide is manifested mainly in the ascending part of the loop of Henle, located in the medulla of the nephron, and is partially due to an increase in K, Ca, Mg and phosphates and the retention of urates in the body. 95% of furosemide entering the body binds to serum proteins and is excreted from the body mainly through the kidneys - by filtration through the renal tubules and tubules . It has a diuretic and sodium-excreting effect in proportion to the amount of furosemide taken.

Furosemide is available in tablets of 0.005, 0.02, 0.04, 0.08 and 0.5 g (5, 20, 40, 80 and 500 mg) and in ampoules of 1% of 1, 2, 5, 10 and 25 ml (lasix, 20 mg). Usually, children are prescribed 1-3 mg per kg of body weight.

Bumetanide has a similar potency to furosemide, but its effect is much stronger - in terms of diuretic effect, 1 mg of bumetanide is equivalent to 40 mg of furosemide. The duration of action of bumetanide is shorter than that of furosemide. Tablets 0.001 g. and ampoules 0.025% 2 ml.

E **tacrine acid (uregit)** - easily absorbed into the blood from the gastrointestinal tract and binds to proteins. 1/3 of ethacrynic acid absorbed into the blood is excreted by the liver, 2/3 by the proximal tubules of the kidneys. Its diuretic effect begins after 20-40 minutes, reaches a peak in 2-4 hours and lasts 4-8 hours. The diuretic effect of uregit is accompanied by a significant loss of sodium, chlorides, and partly potassium and hydrogen ions. Children are given

0.025 (25 mg). The dose can be increased to 0.1-0.2 grams. Release form tablets 0.05 g.

Acetazolamide (fonurite, diacarb, dilamox) is an inhibitor of the carbonic anhydrase enzyme and is well absorbed from the intestines into the blood.

It is excreted from the body through the renal tubules. It accumulates in the renal cortex in 2-3 times more than in the blood. When acetazolamide is administered in an amount of 5-20 mg/kg, its diuretic effect begins after 1 hour, reaches a peak after 4-6 hours and lasts for 8-12 hours. The effect of acetazolamide occurs in the proximal tubules, where water reabsorption is reduced by 50%, and SO_2 reabsorption is reduced by 80%. The excretion of hydrogen ions also decreases, and the excretion of sodium, bicarbonates, potassium, calcium and phosphates, in turn, increases. The sulfonamide group, which is part of acetazolamide, binds to zinc, which is part of the carbonic anhydrase enzyme. This causes the release of hydrogen ions in the tubular lumen, disruption of its exchange with sodium ions, and, as a result, the excretion of sodium in large quantities with urine. In this case, the reabsorption of bicarbonate in the tubules also slows down, and the urine enters an alkaline environment, which leads to a decrease in the excretion of ammonium with urine. After 1-2 days after stopping the intake of acetazolamide, the alkaline composition of the blood is restored, it is prescribed for 3-5 days with breaks of 2-3 days. Its diuretic effect is not very strong. As the level of bicarbonate in the blood decreases, its effect on the treatment decreases, and then its sodium and diuretic effect disappears. Release form: powder and tablets 0.25 g.

Thiazide (hydrochlorothiazide **, cyclomethiazide, polythiazide) and thiazide-free (chlorthalidone - hygroton, ipadamide, brinaldix)** sulfonamides are widely used diuretics. Thiazide sulfonamides are rapidly absorbed through the intestines and excreted through the renal tubules. Under their diuretic effect, the excretion of sodium, potassium, chlorine, bicarbonates increases and the excretion of calcium and uric acid decreases. The effect of thiazide diuretics is achieved by reducing sodium reabsorption in the ascending limb of the loop of Henle and reducing the activity of phosphodiesterase.

When taken orally, their diuretic effect begins after 100 minutes, reaches a peak in 2-4 hours, and lasts for 8-12 hours. The fact that thiazide sulfonamides in nephrotic diabetes do not increase urine output, but rather decrease it, is important for comparative diagnosis in practical medicine. If the patient cannot take thiazides, thiazide-free sulfonamides can be prescribed, but they are weaker diuretics.

Hydrochlorothiazide (hypothiazide) - 2 mg/kg for children (3.5 mg/kg for children under 6 months).

It is available in tablet form in strengths of 0.025, 0.05 and 0.1 g.

Cyclomethiazide - for children 0.00025 - 0.0005 g (1/2 - 1 tab.) per day or 1 time in 2 - 3 days.

The release is in the form of tablets of 0.0005 g (0.5 mg).

Potassium-sparing diuretics:

Triamterene - 10-88% is absorbed into the blood from the gastrointestinal tract, 2/3 of which is metabolized in the body, 1/3 is excreted unchanged through the renal tubules. The metabolized derivatives of triamterene have diuretic properties. The diuretic effect of triamterene taken orally begins after 45-60 minutes, peaks in 4-6 hours and lasts for 8-12 hours.

The release is in the form of capsules of 0.05 g (50 mg).

Amiloride is absorbed from the gastrointestinal tract by up to 90%. It is excreted unchanged in the urine. After administration, its diuretic effect begins after 2 hours, reaches a peak in 6-10 hours and lasts for 12-24 hours. Its effect is achieved by limiting the reabsorption of sodium and chlorides in the distal and collecting tubules of the nephron, as well as the excretion of potassium and sodium ions.

It is available in tablet form in strengths of 0.0025 and 0.005 g (2.5 and 5 mg).

Spironolactone (veroshpiron, aldocton) is an aldosterone inhibitor, well absorbed from the gastrointestinal tract into the blood and 98% of it binds to serum proteins. It breaks down in the body, forming conrenone (a substance with diuretic properties) and concrenoate (a substance with antiarrhythmic properties). The half-life is up to 35 hours, and the diuretic effect peaks 2-5 days after administration and lasts 48-72 hours. Spironolactone is structurally similar to aldosterone, so it binds to cytoplasmic receptors with proteins specific for aldosterone binding, leading to sodium excretion and potassium retention. Diuretic properties are not strong.

It is available in tablets of 0.025, 0.05 (25 and 50 mg) and in ampoules of 0.2% 10 ml.

Diuretic osmotic agents – Mannitol, urea (carbamide) 0.5 – 1.5 g/kg (based on dry matter) have a strong diuretic effect when administered intravenously. After administration, its effect begins 15 – 20 min., reaches a peak in 1 – 1.5 hours and lasts 4 – 5 hours. Released in 30 g lyophilized flakes, 15% 200 and 400 ml and 20% 500 ml.

Xanthine derivatives (aminophylline, theophylline), pyrimidine (allation) and triazine derivatives (chlorazanil) also increase fluid filtration in the renal tubules. When xanthine derivatives are taken orally, their diuretic effect begins after 30–45 minutes, reaches a peak in 1.5–2 hours, and lasts for 4–6 hours. When administered intravenously, it begins after 2–5 minutes, reaches a peak in 20–30 minutes, and lasts for 2–4 hours. Currently, xanthine derivatives are less commonly used for this purpose, since they are weaker diuretics, but when used in combination with natriuretic drugs, their effect is significantly enhanced.

Hypotensive treatment . Hypotensive agents with various pharmacological properties are used to influence hypertension in patients with glomerulonephritis. A low-salt diet prescribed to patients in the first weeks of treatment also helps to lower blood pressure. When used in combination with such a diet, the effect of rauwolfia alkaloids (reserpine, raunatin) is also stronger. Alkaloids dilate renal blood vessels, increase blood circulation in them, and reduce arterial blood pressure. In cases of increased blood pressure in a patient,

reserpine is prescribed from the first day of treatment at a dose of 0.1 - 0.4 mg per day and is continued until blood pressure is completely normalized. In cases where the effect of reserpine is insufficient, it can be prescribed in combination with dibazole (intramuscularly or orally) or hypothiazide . If hypertension is resistant to such treatment, the antiadrenergic agent isobarine (octadine) can be used initially at 10-12 mg once a day, and if necessary, twice a day, with constant monitoring of blood pressure.

The following groups of antihypertensive drugs used in the treatment of glomerulonephritis are prescribed depending on the situation:

Group I includes central nervous system depressants: valerian, valerian root , bromine, derivatives of barbiturate acid. In small quantities, sodium etaminal, phenobarbital also belong to this group.

Group II includes rauwolfia derivatives, methyldopa (dopegit), and clonidine (hemiton).

Group III - mainly peripheral vasodilators (octadine - isobarine, β and α - adrenoblockers). Octadine has a strong hypotensive effect, limiting the conduction of excitation (impulse) in the postganglionic part of the sympathetic nerves. Therefore, octadine is used in cases where other hypotensive agents have not been effective.

When the patient has tachycardia, as well as in cases of heart rhythm disturbances, β -adrenoblockers are useful - analaprin, inderal, obzidan. These drugs reduce the pumping power of the heart and the heart rate. In nephrology practice, α -adrenoblockers (phentalamine, tropafen) are not recommended for the treatment of hypertension associated with glomerulonephritis.

Group IV antihypertensive agents include diuretics (furosemide, hypothiazide) and are widely used in nephrology practice, often in combination with other groups of antihypertensive drugs.

In recent years, more attention has been paid to angiotensin-converting enzyme inhibitors (enap, enam, captopril, and bq), angiotensin II receptor blockers (losartan, candesartan, irbesartan, and bq), and calcium channel blockers (nifedipine, verapamil) as agents with antiproteinuric, hypotensive, and renoprotective properties.

Antiadrenergic agents such as dopegite (aldomet), isobarine, and clonidine (hemiton) also have a strong hypotensive effect and are widely used in nephrology. Treatment with dopegite begins with 100–120 mg, which can be increased to 250 mg 1–2 times a day. Dopegite can be used alone or in combination with reserpine and hypothiazide.

In cases where tachycardia is also observed in a patient with high blood pressure, it can be moderated by administering 0.5–0.6 mg of obsidian or inderal per kg of the patient's weight.

often observed in glomerulonephritis, is quickly relieved by reserpine in combination with a salt-free diet. In the nephrotic form, the effect of reserpine is more rapid when used in combination with aldactone. Hypertension that is resistant to the use of all the above-mentioned agents gives every reason to

suspect congenital defects of the renal vascular structure or dysplasia of the renal tissue in the patient.

Glucocorticoid drugs are widely used in pediatric practice as active agents in the treatment of a number of diseases (rheumatism, nephritis, collagenoses, etc.). Glucocorticoid hormones are prescribed to patients with glomerulonephritis primarily due to their immunosuppressive, antihistamine, anti-inflammatory, and cell membrane-strengthening (membrane-protecting) properties.

There are various methods of treating glomerulonephritis with corticosteroids. Most often, they are prescribed in a dose of 1.5 - 2.0 - 2.5 mg / kg (or 40 - 50 mg per m ^{2} of body surface area) for 4 - 6 weeks, followed by a long-term periodic course of treatment aimed at maintaining the achieved result. With this treatment, when the patient's symptoms begin to subside, that is, after 4 - 6 weeks, the amount of hormone taken is gradually reduced, after 1.5 - 2 months it is reduced to ½ or 1/3 of the initial treatment dose and, first, a break of 1 day, then 2 days, etc., is transferred to a periodic course of treatment for 6 - 12 months - the patient takes the hormone 3 days a week, then a break of 4 days (if necessary, such treatment can last up to 2 years). The periodic treatment regimen can be slightly modified for patients prone to relapse, in which case the hormones are administered daily without a break for four days at a time. The usefulness of such corticosteroid therapy in the treatment of nephrotic nephritis is recognized by all modern nephrologists (Table 18).

Among glucocorticoids, the most widely used type in clinical practice is prednisolone, and when the need arises to use other types (triamcinolone, urbazone, dexamethasone), they are prescribed in an amount equivalent to the amount of prednisolone.

However, in nephritis with severe hematuria and hypertension, corticosteroids can lead to an exacerbation of these symptoms and a worsening of the patient's general condition. Therefore, treatment of glomerulonephritis with prolonged hematuria with a small dose (1 mg/kg) of prednisolone for no more than 1.5-2 months (MSIgnatova, 1973), taking into account the periodic activity of the adrenal glands , that is, in the first half of the day (7-10-13 ^{00}), often gives a positive result. In such cases, the effect of the treatment is often observed not during treatment, unlike the nephrotic form of glomerulonephritis, but later, after the use of hormones is stopped. However, before starting such treatment, it is necessary to make sure that this patient does not have a disease other than glomerulonephritis that causes hematuria (urolithiasis, developmental disorders, etc.).

In the most severe mixed form of glomerulonephritis, treatment with glucocorticoids often does not give a positive result, but can even worsen the patient's condition. Therefore, for the treatment of glomerulonephritis with hematuria, it is more appropriate to use 4-aminoquinoline drugs rather than glucocorticoids.

Observations of modern nephrology practice show that it is still advisable to prescribe glucocorticoid hormones only if it is certain that the patient has

nephrotic syndrome. Sometimes, during the period of clinical remission achieved with glucocorticoid therapy, the disease may relapse. In such cases, it is necessary to return to the original full dose of hormones and repeat the treatment. Treatment of the second and subsequent relapses of nephrotic syndrome with glucocorticoids in combination with cytostatics gives a more stable result, and the risk of complications from glucocorticoids is also somewhat reduced.

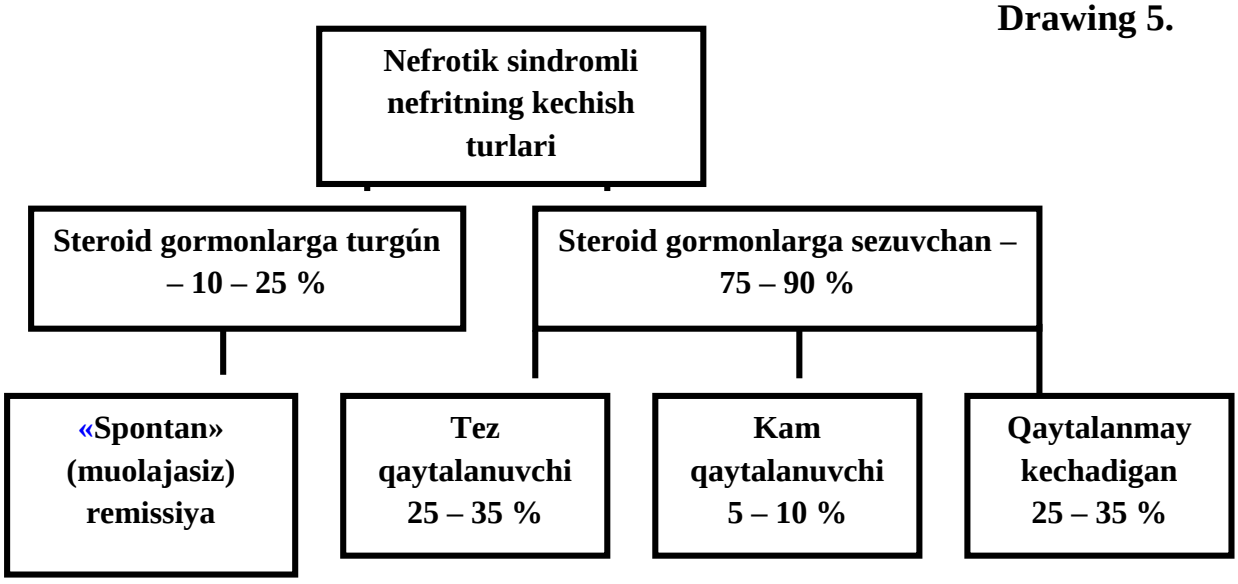
Complications of glucocorticoid treatment. During the treatment of glomerulonephritis with nephrotic syndrome, a number of changes are observed in fat, protein, carbohydrate, electrolyte metabolism, blood pressure, and blood morphological parameters under the influence of glucocorticosteroid hormones, which are not considered complications, since they are within the scope of action of these hormones. These conditions, called the “complex of symptoms of exogenous hypercorticism,” include the catabolic effect of prednisolone, increased glyco-lipogenesis, which leads to the accumulation of fat in certain parts of the body and an increase in the patient's weight (Cushing's syndrome). In such patients, dry skin, increased blood vessels in the palms of the hands and face, and hypertrichosis are also observed. Such changes do not pose a threat to the patient's life and do not require immediate discontinuation of hormones. Exogenous hypercortisolism develops in different degrees: I degree - the patient's face becomes full, appetite increases, weight increases, but not more than one-fifteenth of the initial weight; II degree - the patient's fat layer thickens significantly in the face, neck, shoulders, and abdomen, the face acquires a "moon-shaped" and rounded shape , small blood vessels are enlarged on the palms of the hands, and discolored lines of the skin appear, especially in the abdomen, and his weight increases by 1/14 - 1/12 of the initial weight; III degree - due to the thickening of the fat layer, the patient's waist hangs, the corners of his mouth droop, his eyes are narrowed from obesity, the blood vessels on the patient's face and palms are noticeably dilated, and his weight increases by 25 - 30% compared to the initial weight. During hormone therapy, complications that are dangerous to the patient's life can also develop - adrenal insufficiency, gastrointestinal ulcers, bleeding, steroid diabetes (hyperglycemia and glucosuria), arterial hypertension, osteoporosis, mental changes, exacerbation of focal infections, etc. - such manifestations of hypercorticism require discontinuation or sharp limitation of hormone therapy (Table 19). Since the effect of glucocorticoids on the body is multifaceted, their complications are also diverse. A study of complications that develop during the treatment of nephritis, mining diseases, collagenoses and other diseases with glucocorticoids shows that they are mainly divided into 2 categories: **The first group** is the most common complications, which include pathological obesity and trophic changes and rashes on the skin. **The second group is** more serious complications, which include hypertension, hyperglycemia and glucosuria, protein and mineral metabolism disorders, sepsis, and ulceration. When treated with large doses (1.5–2 mg/kg) of glucocorticoids, obesity begins after 2–3 weeks. The decrease in clinical signs of the disease also occurs during this period.

During the 2nd week of treatment with glucocorticosteroids, a shift in the peripheral blood is observed: leukocytosis develops in 70% of patients. Since leukocytosis is mainly due to mature leukocytes, a shift in the ratio of cells, that is, the leukocyte formula, to the “left” is not observed. At the same time, the number of erythrocytes and platelets in the blood increases, and the erythrocyte sedimentation rate decreases. Such changes in the blood indicate the positive effect of glucocorticoids. If leukocytosis is not accompanied by an increase in the number of erythrocytes and platelets in the blood, if the ESR accelerates, if a “shift to the left” occurs, this indicates a worsening of the patient's general condition, perhaps the emergence of activity in latent foci of infectious factors in the body , which requires reducing the amount of hormones and intensifying antibiotic treatment .

In 43% of patients treated with glucocorticoids, an increase in blood pressure is observed. This is due to the fact that the used corticosteroid drugs are not completely devoid of the mineralocorticoid effect, which leads to sodium retention in the body . Controlling this condition and combining glucocorticosteroid treatment with rauwolfia derivatives makes it possible to prevent the development of hypertension. As a result of the catabolic effect of glucocorticoids, azotemia may increase in the blood of patients. In patients treated with hormones for a long time, their weight increases, growth retardation, and osteoporosis of bones are also observed. In patients treated with glucocorticoids for three to six months, the hormone production activity of the adrenal glands decreases. Therefore, stopping such treatment and switching to periodic treatment should be carried out gradually. At this time, measures are taken to strengthen the function of the adrenal glands (glyceram, ascorbic acid).

Nephritis with nephrotic syndrome in children has a different course and requires appropriate treatment tactics. According to the results of the "standard treatment" in children - (60 mg/day per m $^{2\ \text{of body}}$ surface area or 2 mg/kg/day), this disease has the following manifestations (Figure 5).

Drawing 5.



Therefore, long-term treatment with glucocorticoids and cytostatic drugs can cause various serious complications. At the same time, international clinical observations have shown that prolonged treatment in the treatment of hormone-sensitive nephrotic syndrome does not affect the course of the disease. Accordingly, according to modern international standards, the treatment regimen with prednisolone when the nephrotic form of GN is first observed is as follows (Savenkova ND, 2005): 2 mg/kg for 4-6 weeks, then switch to an alternative course of 1.5 mg/kg/ 48 hours for 4-6 weeks and stop treatment for the next 2 weeks. Thus, according to the international standard , the duration of such treatment is 3 months. Treatment of nephrotic syndrome with steroid hormones in children and adolescents gives a positive effect in 95-99% of cases. There are the following types of nephrotic syndrome with minimal changes:

- rapidly recurring, meaning 2 or more recurrences within the first 6 months.
- corticosteroid-dependent form, that is, relapse during the period of reducing the amount of steroid hormones or in the first 2 weeks after discontinuation.
- corticosteroid-resistant form - ineffectiveness of treatment with full-dose prednisolone (2.5 mg/kg/day, or 60 mg/m2/day) for 4-6 weeks · Such cases account for 1-5%. In such cases and if there is a risk of complications with continued treatment with steroids, it is recommended to use cytostatics. More often, alkylating compounds are prescribed - chlorbutin (leukeran) 0.15-0.2 mg/kg/24 h. 8-10 weeks, cyclophosphamide 2.5 mg/kg/24 h. Given the risk of complications of cytostatics, it is necessary to monitor that their recommended dose per course does not exceed 10-11 mg/kg/course for chlorbutin, and 200-250 mg/kg/course for cyclophosphamide. If the disease relapses within 12 months of treatment, it is not recommended to re-administer cytostatics. In such cases, it is recommended to continue treatment with cyclosporine (Sandimmune - Neoral) or levamisole (Dekaris) (2.5 mg / kg / 48 hours) for 6-12 months .

Antithrombotic treatment. In glomerulonephritis, a direct-acting drug that reduces blood clotting (anticoagulant) is widely used. Heparin affects various stages of blood clotting, activates the fibrinolytic properties of blood, reduces thrombin formation, and blood cell adhesion. The need to prescribe heparin in the treatment of glomerulonephritis arises in the following cases: 1) with an increased tendency to blood clotting (hypercoagulability); 2) the appearance of signs of increased blood clotting in the blood vessels inside the kidneys - an increase in the products of fibrin breakdown in the blood, a decrease in the amount of fibrinogen and a simultaneous rapid decline in kidney function; 3) the appearance of signs of increased blood clotting inside the blood vessels; 4) widespread severe edema; 5) severe hyperlipidemia.

In antithrombotic therapy, heparin is administered at a dose of 100–200 U/kg of the patient's weight per day, administered subcutaneously 4 times a day in alternating doses. In more severe cases, the dose can be increased to 400–500 U/kg. Heparin is often administered subcutaneously, by electrophoresis into the renal area, or as a drop under the tongue.

necessary , heparin can be administered directly into a vein or intramuscularly. Heparin treatment is carried out under the control of a

coagulogram every 2-3 days, and if 4-5 hours after a single injection, the blood clotting time increases by 1.5-2 times compared to the previous one, the prescribed amount of heparin is considered sufficient. Excessive use of heparin can lead to blood loss and increased hematuria. Its long-term use leads to osteoporosis, especially when used together with hormones. The effect of heparin is enhanced by curantyl (dipyridamole), trental, aspirin, papaverine. On the contrary, if it is necessary to reduce the effect of heparin, a 1% solution of protamine sulfate is administered continuously or dripped into a vein, and within 15 minutes, each mg of it (i.e. 0.1 ml of a 1% solution) neutralizes 100B heparin.

Indirect-acting anticoagulants (neodicoumarin, phenylin, syncumar, and bq) are less commonly used in pediatric nephrology practice.

Antiplatelet agents are substances that reduce platelet aggregation and prevent blood clot formation. They also have the property of expanding blood vessels. Such drugs include curantyl, trental, prodectin (parmidine), nicotinic acid, papaverine, theonikol (complamine) and non-steroidal anti-inflammatory drugs (aspirin, butadione, voltaren and bq). The most widely used of these is curantyl, which is prescribed at a dose of 2-3 mg / kg of the patient's weight for 2-3 months.

Aminocholine derivatives were obtained in the 1930s and are widely used in the treatment of a number of chronic somatic diseases (rheumatoid arthritis, nephritis, amyloidosis, etc.). The effect of aminocholine drugs (delagil, chlorhexidine, plaquenil) is noticeable after 3-4 weeks, mainly after 6-12 months. These drugs are prescribed at a dose of 5-10 mg per kg of the patient's weight, and the treatment lasts for 6-12 months, sometimes up to 2 years. Aminocholine substances have anti-inflammatory, antisclerotic, immunosuppressive properties. They are contraindicated in active forms of glomerulonephritis with edema and hypertension, in cases of advanced renal failure, in cases where the patient has a duodenal ulcer and a family predisposition to it, in cases where the patient has anemia and leukopenia. Therefore, this group of drugs is mainly recommended for the treatment of chronic forms with changes in urine. When using them, constant monitoring should be carried out with a general blood test, hemostasis, blood pressure, ECG, and ophthalmological examination. Aminocholine group drugs are used in pediatric nephrology for the treatment of chronic glomerulonephritis with hematuria, chronic pyelonephritis, and hereditary nephritis.

Patients with glomerulonephritis often (70-90%) have one or more chronic foci of infectious agents (dental caries, sinusitis, chronic tonsillitis, etc.). After the acute symptoms of glomerulonephritis subside, after 3-4 weeks of treatment, it is necessary to achieve the most complete cure of dental caries, since such a tooth is a focus of β -hemolytic streptococcus. The same approach should be taken for any other chronic foci. Especially since chronic tonsillitis and glomerulonephritis are incompatible with each other, tonsillectomy is recommended for such patients during the period of remission. To prevent exacerbation of the disease under the influence of tonsillectomy, if the patient takes hormones periodically, they are transferred to daily 10 days before the

operation, the patient is prescribed a hypochloremic diet, antibiotics, and antihistamines. In this case, the incidence of exacerbation of glomerulonephritis after surgery does not exceed 3%. Tonsillectomy can be performed on a patient with glomerulonephritis 1.5–2 months after the start of treatment.

Cytostatic agents. In nephrological clinical conditions, it has been practically confirmed that it is advisable to use cytostatic drugs in cases where the nephrotic form of glomerulonephritis is frequently recurrent and hormone-resistant. For this purpose, cytostatic agents such as leukeran (chlorbutin) are prescribed at a dose of 0.15–0.2 mg/kg per day, and the full dose is taken for 6–8 weeks. After achieving clinical remission, the initial dose is reduced by half, and the treatment is continued for 6–10 months.

During treatment with cytostatics, periodic administration of prednisolone at a dose of 0.5 mg/kg significantly reduces their harmful effects and prevents complications. In this case, conditions are created for reducing the dose of cytostatics and completely eliminating hormones from the treatment. This is very favorable for patients who have experienced complications from hormone treatment. Active treatment with immunosuppressants should be carried out in a hospital setting under constant clinical supervision, blood tests (especially under the control of the number of leukocytes and platelets). If leukopenia is observed during treatment with cytostatics, the dose of cytostatics should be reduced, and if the number of leukocytes decreases to 3000, such treatment should be discontinued.

During treatment, latent foci of infection may be activated, and septic complications may develop. Therefore, such treatment also requires the appointment of antibiotics. The possibility of serious complications of cytostatics requires careful consideration of the need for them before using them.

cytostatic drugs, azathioprine in an amount of 2-4 mg/kg and cyclophosphamide in an amount of 1-3 mg/kg can also be used in nephrological practice in the above order. Bearing in mind that alkylating cytostatic compounds are potentially toxic agents, their dosage should be strictly controlled throughout the entire course of treatment, and the dose of leukeran should not exceed 10-11 mg/kg per course, and cyclophosphamide 200-250 mg/kg.

The use of cytostatics, which is not without risks, is justified in the following cases: 1) the lack of a positive result from treatment with 2-2.5 mg/kg of prednisolone for at least 4-6 weeks (clinical stability to hormones); 2) with good results from hormone treatment, repeated exacerbations of the disease when trying to reduce or stop the amount of hormone (the patient becomes hormone-dependent); 3) a mixed form of glomerulonephritis.

non-steroidal anti-inflammatory drugs - salicylic acid and its derivative acetylsalicylic acid (aspirin) - have been used in medical practice for more than 100 years. Substances in this group also have antipyretic, analgesic, and antiplatelet properties. This group of drugs has immunosuppressive, anticomplementary, and fibrinolytic activity. Their antipyretic and analgesic effects are noticeable within a few hours after administration, and their anti-inflammatory effect is noticeable within 2-4 weeks. These drugs are mainly

recommended for the treatment of glomerulonephritis without external symptoms, accompanied only by changes in the urine (proteinuria, hematuria, proteinuria - microhematuria), incomplete nephrotic syndrome (proteinuria and other nephrotic symptoms without edema), and the nephritic form of acute glomerulonephritis. Treatment with these medications can last 1-4 months.

When using these drugs, it is necessary to take into account the risk of developing ulcers in the gastrointestinal tract (aspirin, indomethacin, after meals, with milk), the risk of developing hemorrhagic syndrome with aspirin, the risk of increasing edema with indomethacin, ibuprofen, and the possibility of causing hypertension, and most of them can cause allergic reactions.

In order to influence and correct the various immunological changes observed in glomerulonephritis, instilling human interferon into the patient's nose twice a day for 2-3 weeks during the most active period of the disease reduces the risk of viral and bacterial infections.

To improve the function of T and V lymphocytes, the administration of 1-1.5 mg/kg of levamisole (decaris) for 3 days every week (for a total of 4-6 weeks) significantly accelerates remission . For this purpose, T-activin and thymosin also give good results.

In recent years, great attention has been paid to membrane-strengthening drugs in the treatment of glomerulonephritis. For this purpose, it is recommended to prescribe α -tocopherol (vitamin E) 1-3 mg/kg, dimephosphone 30-50 mg/kg per day for 14-21 days. In our clinic, the state of cell membranes in glomerulonephritis and other kidney diseases and methods of their stabilization have been studied for many years (A. Akhmatov, 1988, N. Karimova, 1989). Given the importance of membranolytic processes in the development of glomerulonephritis, in order to prevent its recurrence, it is useful to prescribe antioxidants to the child under observation for 2-3 weeks during the change of seasons, even in the presence of transient diseases (SARS and BK).

During the period when glomerulonephritis activity decreases and the amount of hormones begins to decrease, the patient is prescribed anabolic hormones, which accelerate the normalization of metabolism, enhance synthetic processes, and reduce azotemia. For this purpose, 0.2 - 0.5 mg of dianobol, methandrostenolone or nerabol per kg of the child's weight per day for 3 - 4 weeks can be recommended. Or 0.4 - 0.5 mg / kg of nerabolil can be administered intramuscularly every week or 1 - 1.5 mg / kg of retabolil once a month.

These drugs improve appetite and mood in patients. Blood production improves, and the patient becomes more resilient.

Dispensary observation. Conducted depending on the period of the disease, taking into account periods of activity and clinical remission.

The tendency of glomerulonephritis to relapse and become chronic requires long-term, staged dispensary supervision. This method allows to somewhat reduce the patient's hospitalization period and monitor the patient's condition at home. All patients with glomerulonephritis need dispensary supervision. The patient should be examined by a local doctor within a week after discharge from

the hospital, blood pressure should be measured, and special documents should be kept, taking into account the dispensary supervision.

Patients who have fully recovered (clinically and biochemically) from the hospital are examined every 3 months for the next year. If the disease does not recur during this time, it is enough to examine the patient once every 6 months. A general urine and blood test must be performed every month.

clinically and biochemically free from the disease should be examined once a month, and a complete blood count should be performed every 2 months, if residual changes in urine persist. If recovery is incomplete, the patient should be examined monthly, while continuing to take glucocorticoid hormones, and a complete blood count and urine should be performed weekly.

The level of activity of the nephritic process can be judged based on the proteinogram, hemogram, blood lipids, DFA, AST, ALT test results, and the state of kidney function based on endogenous creatinine clearance, Zimnisky test, and the amount of protein, amino nitrogen, phosphorus, carbohydrates, ammonia, and titratable acids excreted in the urine.

During the dispensary examination, the doctor determines the daily routine and the level of permissible physical exertion, depending on the patient's condition.

In cases where the patient is discharged from the hospital with residual changes in urine, he is allowed to attend kindergarten after 2-3 weeks, exempted from labor and physical education classes, and school with an additional day off. If the disease does not reactivate within a year in this manner, such a patient is allowed to participate in physical education training groups with monitoring of urine analysis.

Patients under observation are advised to eat a balanced diet at home, avoid eating pickled and salty foods, and completely avoid allergenic foods (chocolate, oranges, coffee, etc.).

The measures taken in the dispensary depend on the form of the disease and the level of activity at the time of the hospital response. In nephrotic and mixed forms of glomerulonephritis, treatment with glucocorticoids, which was administered in the hospital, is continued. If the patient is taking hormones every day during this period, they are gradually transferred to taking them in the first half of the day with a break (every other day or 3-4 days a week).

In cases of glomerulonephritis resistant to glucocorticoid hormones, treatment with cytostatics and aminoquinolines, which was started during the dispensary period, can be continued. Such treatment usually lasts 6-19 months, sometimes 1-2 years. In cases of relapse of the disease, the patient may need to be transferred to a hospital for active treatment.

suprastin) are prescribed for 10-14 days , and the amount of hormones is slightly increased. At the end of this illness, a urine test is mandatory.

During follow-up, it is important to cleanse the body of foci of purulent inflammation after complete remission of the disease in children who are being monitored. Children who have previously received hormone treatment for tonsillectomy are prescribed 15-20 mg of prednisolone in the morning on the

day of surgery and for 7-10 days thereafter, while others are prescribed desensitizing agents.

In the complex treatment of glomerulonephritis, climatic treatment plays an important role; especially during the period of residual symptoms of the disease, warm and dry climatic conditions are recommended. Patients who have had glomerulonephritis should immediately stop preventive vaccination for at least a year upon discharge from the hospital. In the future, the issue of vaccination is decided depending on the course of the disease, but at least a year must pass after the complete clinical and biochemical remission of the disease.

Dispensary supervision is continued for two years after the patient has completely recovered from the hematuric form of glomerulonephritis, and for the nephrotic form, it is recommended to observe for 5 years after the last attack of the disease, but it is more expedient to continue constant supervision and transfer the child to the supervision of a therapist when he reaches the age of 15.

CHAPTER V
URINARY TRACT INFECTION AND PYELONEPHRITIS

Pyelonephritis (according to ICD-10: N11.0 - Chronic non-obstructive, reflux-related pyelonephritis; N11.1 - Chronic obstructive pyelonephritis) - microbial inflammation of the renal interstitium and calyx - is the most common type of kidney disease. According to our observations, when analyzing the total number of kidney diseases in children under 14 years of age, pyelonephritis is the most common type, accounting for 43.4%. The incidence of pyelonephritis is to some extent related to the sex and age of the child. In particular, this indicator is 39.1% in boys and 49.2% in girls. In early childhood, these rates are close to each other (34.1 and 35.2%), while in school-age children, they are 34.7% and 58.1%. The results of a general examination of children for kidney diseases confirm the widespread prevalence of pyelonephritis in them (32.1 ±1.79 out of every 1000 children examined) and its tendency to be latent in most cases (18.9 ±1.92: 1000).

Etiology and pathogenesis. There is no specific causative agent of pyelonephritis. In most cases (60-80%) its development is caused by Escherichia coli (E CoLi). However, staphylococcus, streptococcus, proteus, and sometimes a combination of the above can cause pyelonephritis. The disease occurs more often in children with malnutrition, hypotrophy, and exudative diathesis due to acute digestive disorders. Infectious agents enter the kidneys in three ways - ascending through these routes in inflammatory diseases of the urinary tract, hematogenous and lymphogenous. The ability of microbes to settle in the kidneys and multiply and cause an inflammatory process is facilitated by defects in the renal and urinary tracts, stone retention, congenital and secondary narrowing of the urinary tract, which lead to the accumulation of urine. Allergic reactions that occurred before the onset of the disease, damage to the renal interstitial tissue under the influence of some nephrotoxic drugs and pathological metabolic products, and a decrease in the body's reactivity are also among the causes that create conditions for the development of pyelonephritis. According to the concepts of modern medicine, pyelonephritis should not occur as a primary disease in an immunologically perfect and absolutely healthy kidney, since it has been confirmed in experimental conditions that even if pathogenic microbes are introduced into the renal pelvis of animals in pure form, they are washed out with the natural flow of urine and pyelonephritis does not develop . Therefore, there must be some additional conditions for the development of pyelonephritis, and the concept of a secondary disease in its essence is currently prevailing. The phrase "urinary tract infection" is a general concept , meaning the presence of microbial inflammation in some area of the urinary system and requires a nephro-urological examination for nosological diagnosis . This is because treating a kidney infection (pyelonephritis), a bladder infection (cystitis), or a urinary tract infection (urethritis) requires a different approach.

The following tests are performed to determine microbial inflammation and its activity:

- Objective examination (provided that the external genitalia are visible)
- Arterial blood pressure measurement
- Biochemical analysis of urine (protein , oxalates , urates , calcium , phosphorus excretion per day)
- Determining immunological status
- test for dysbacteriosis
- General urine analysis (on days 1, 3, 7, 14 , then individually) or Nechiporenko test if there are minimal changes in urine
- Microbiological analysis of urine, with the condition of determining sensitivity to antibiotics
- Blood test
- Determination of SRO in blood
- Blood biochemical analysis (total protein and fractions , creatinine, urea, uric acid)
- Determination of ball filtration according to the Schwarz formula
- UTI of the kidneys and bladder
- Urine test for urogenital infections (chlamydia, mycoplasma, ureaplasma)
- Virological tests (HIV , CMV, Epstein-Barr virus)
- culture for fungi and anaerobic infections

List of additional screening procedures :

Special examination methods performed during periods of remission or clinical and laboratory remission of the pathological process (if indicated)

- the rhythm and volume of urination (taking into account the amount of fluid consumed)
- urodynamic examination methods
- furosemide and water loading
- ε secretory urography (not performed in cases of CF T and creatininemia)
- micturition cystography
- Zimnisky test
- determination of titratable acidity in urine
- determination of urine osmolarity
- Detection of microalbumin, β2-microglobulin, α1-microglobulin in urine
- determination of enzymes (LDG, GGT, ЩФ, etc.)
- dynamic renoscintigraphy
- static renoscintigraphy (after a period of clinical-laboratory remission of pyelonephritis lasting at least 6 months)

Diagnostic criteria for pyelonephritis

Complaints and anamnesis:

- fever, body temperature exceeding 38°C ;
- general malaise , weakness, loss of appetite
- There may be pain in the lower back area.
- Dysuria and edema may be observed.

Physical examination results :

- body temperature is subfebrile or normal
- positive syndrome on palpation , positive

Pasternask symptom

Laboratory test results

- E ChT 20 mm/s oat ;
- SRO 10-20 mg/l;
- Serum PCT is 2 ng/ml.

Instrumental examination results

- Kidneys UTT : congenital anomalies , cysts, stones
- Cystography – bladder - urethral reflux or antireflux condition after defecation
- Nephroscintigraphy - foci of damage in the renal parenchyma
- In tubulointerstitial nephritis – renal puncture biopsy for diagnostic purposes (with parental consent)

Indications for specialist consultations : Examinations by a urologist, a pediatric urologist . Examinations by an andrologist, an ophthalmologist, an otolaryngologist, a phthisiatrician, an immunologist, a dentist, and a neurologist, as indicated.

Classification. Several classifications have been proposed based on the causes of pyelonephritis and the diversity of its clinical presentation. The currently widely used classification of pyelonephritis is as follows:

Classification of clinical pyelonephritis in children

(Minutes of the meeting of the Expert Commission of the RF State Committee for the Development of Healthcare dated December 12, 2013 No. 23) .

The course of pyelonephritis can be recurrent:

- less frequent – <2 recurrences in 6 months or <4 recurrences in a year;
- frequently recurring – 2 recurrences in 6 months or 4 recurrences in a year;
- The recurrent course of pyelonephritis depends on :
- With reinfection (re- infection);
- Persistence of the pathogen – in cases of biofilm formation (in urolithiasis, in cases of permanent urinary catheterization , in urostomy , etc.) ;

– In infections that have not been completely cured .

Working scheme for diagnosing pyelonephritis :

– Acute or chronic;

– Non-obstructive (without urodynamic disturbances) or obstructive (with urodynamic disturbances);

– of remission or period of exacerbation (number of exacerbations must be indicated);

– Renal function : preserved or impaired .

Hospitalization instructions

– social conditions

– X-ray urological radionuclide examinations

– for surgical treatment

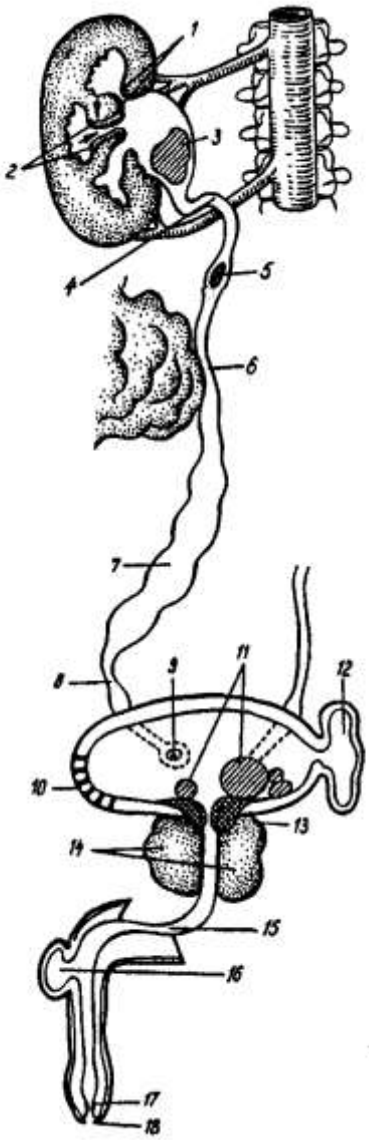
– if there are purulent complications

acute and chronic renal failure

– if there are risk factors

According to this classification , **"Primary pyelonephritis" means** that the cause of the microbial inflammatory process in the interstitial tissue of the kidney and the calyceal system has not been identified, despite the use of all modern diagnostic methods .

Secondary obstructive pyelonephritis is a microbial inflammatory disease of the kidneys that develops on the basis of impaired urodynamics due to congenital, hereditary or acquired causes of the kidneys (Fig. 13). A number of other causes are also known that lead to the accumulation of urine in the excretory tract (urostasis). If such causes are identified, pyelonephritis is considered secondary (obstructive). However, there are cases that cannot be classified as either “obstructive” or “non-obstructive”. In particular, the development of secondary pyelonephritis is characteristic of metabolic disorders (hyperoxaluria, uraturia, etc.), which are important conditions for the development of pyelonephritis, as well as primary tubulopathies. Therefore, as examination methods improve, it can be expected that the diagnosis of primary pyelonephritis will be made less and less often (Fig. 5).



7-rasm. Siydik to'xtalishi(urostaz)ning sabablari.

1. *Buyrak kosachalari boýinchalarining diskineziyasi;*
2. *Kosachalari boýinchalarining torayishi;*
3. *Jomda tosh hosil bo'lishi;*
4. *Qo'shimcha qon tomiri;*
5. *Siydik yo'lidagi tosh;*
6. *Siydik yo'lini to'sib turgan o'sma;*
7. *Siydik yo'linining axalaziyasi;*
8. *Siydik yo'linining torayishi (strikturasi);*
9. *Uretrosele;*
10. *Siydik pufagining mioneyrogen atoniyasi;*
11. *Siydik pufagining toshlari;*
12. *Siydik pufagining divertikuli;*
13. *Siydik pufagi boýin qismining sklerozi;*
14. *Prostata bezining tu-ma kasalligi;*
15. *Siydik chiqarish yo'linining*

At this point, we think it would be appropriate to dwell on the essence of pyelonephritis. For many years, there has been a question of whether pyelonephritis exists as an independent disease or only as a secondary syndrome (MS Ignatova 1989; Shulutko, Makarenko, 2002, 2006). In recent years, the majority of those studying the issue have come to view pyelonephritis as a secondary syndrome, and believe that urologists, nephrologists, and pediatricians who consider pyelonephritis to be independent treat the patient with antibiotics and uroantiseptics for many months and years, putting the patient at additional risk and providing little benefit to the patient. On the contrary, those who consider pyelonephritis not to be an independent disease, but a secondary syndrome, conduct a thorough urological, immunological, and metabolic examination of the patient, identify the underlying cause of pyelonephritis, eliminate this cause, and thereby eliminate pyelonephritis, which is a secondary syndrome. For example, obstructive syndromes are treated surgically, dysmetabolic changes are treated with diet and medication, and if

necessary, immunorehabilitation is performed. In this case, there is no need for years of antibacterial treatment.

Usually, acute pyelonephritis refers to the first clinical manifestation of a microbial inflammatory process in the interstitial tissue of the kidney and the calyceal system , followed by a cyclical course of the disease, followed by complete clinical and laboratory remission.

Chronic pyelonephritis is characterized by a prolonged and recurrent course of the microbial-inflammatory process in the kidneys, lasting more than a year.

At the same time, they can also coexist, for example, obstruction + dysmetabolic changes. The final identification of such cases is important for treatment.

Thus, to confirm that pyelonephritis is not secondary, it is necessary to confirm the absence of reflux, primary immunological deficiency, obstructive and dysmetabolic nephropathies.

Due to the involvement of interstitial tissue in the inflammatory process, the disease is chronic and prone to relapse. Depending on the degree of its activity, there are periods of activity, remission and remission (partial and complete), when all symptoms are present. Periods are distinguished when renal function is completely preserved (BFE $_0$) or impaired (BFE $_{1,2}$) and chronic insufficiency occurs (BFE $_3$).

Clinical picture. In young children, especially in cases of weakness for various reasons (diathesis, hypotrophy, anemia), pyelonephritis occurs as a complication of a number of diseases (sepsis, septicemia, etc.). In infants, this disease is accompanied by signs of general intoxication of the body - an increase in body temperature to 39 - 40 ^{0} C, vomiting, diarrhea, dehydration, neurotoxicosis. Therefore, the diagnosis of pyelonephritis in young children is somewhat complicated. The clinical picture of pyelonephritis in young and older children has a number of features.

Older children usually complain of constant pain in the lower back, lower back and during urination (dysuria). At this time, there is an increase in body temperature to 37.5 - 38 ^{0}C, headache, weakness, that is, signs of intoxication of the body, as in any microbial inflammatory disease. The patient does not have swelling on the body and face, and there is no increase in blood pressure. The patient's urine may be cloudy. When examining the urine, a slightly increased protein content (0.33 - 0.99%), the presence of a large number of white blood cells in the urine sediment (pyuria) and bacteria are detected. Erythrocyturia is not a characteristic sign of pyelonephritis. However, since pyelonephritis is in most cases a secondary disease, if it develops in children with developmental defects of the kidneys and urinary tract, urolithiasis or hyperoxal, urate, cystine, hypercalciuria, it is natural that erythrocyturia is also found. In this case, it is also necessary to differentiate it from hematuric glomerulonephritis. In acute pyelonephritis, a blood test reveals characteristic signs of a microbial inflammatory process - leukocytosis, neutrophilosis, and an increase in ESR. Pain when palpating the lumbar region - over the XII rib - is a characteristic sign of pyelonephritis (Pasternasky test).

Due to the greater damage to the interstitial tissue in pyelonephritis, the disease is prone to relapse and protracted course. It was believed that the use of antibacterial agents for 30-40 days in the treatment of pyelonephritis is not enough, the treatment should be continued for years. However, such treatment is not free from the risk of various complications from the kidneys and other internal organs. Currently, such treatment is recommended to be selected individually, that is, in cases where the disease develops on the basis of vesicoureteral reflux, in order to prevent relapse of pyelonephritis, it is continued until the child reaches 5 years of age or until the reflux disappears. In the period of relapse and exacerbation of chronic pyelonephritis (in cases where clinical and laboratory signs of the disease persist for more than a year), the same clinical picture is observed as in acute pyelonephritis. However, in this case, antibacterial agents (alternately) are administered for at least 1-1.5 months and are continued with the help of uroseptics against relapse.

In chronic pyelonephritis, the patient's condition remains almost unchanged for a long time, and sometimes during exacerbations of the disease, there may be a slight fever, mild pain in the lower back, and it may be asymptomatic. When examined during the exacerbation of the disease, leukocyturia and bacteriuria are detected in the urine. However, over time, the child begins to lag behind in growth, kidney function is impaired.

of pyelonephritis is its latent course. In this case, the disease proceeds without obvious clinical symptoms, moderate body temperature, dysuria, and even leukocyturia is weak and not constant. With careful questioning, it is revealed that patients have rapid fatigue, loss of appetite, and occasional abdominal pain, but these are not enough to diagnose pyelonephritis. Therefore, such children are detected either by chance during examination, or during the period of impaired renal function, because the disease sooner or later leads to chronic renal failure.

For the diagnosis of the disease, the patient's complaints, persistent leukocyturia and the excretion of 100,000 or more microbes per ml of urine are the main signs of the disease. Leukocytes in the urine sediment are determined to be mainly granulocytic neutrophils. It is also necessary to remember that leukocyturia may be associated with cystitis and vulvitis, and to consult a gynecologist. To identify latent forms of the disease, bacteriological and quantitative determination of blood cells in the urine sediment (Nechiporenko, Amburge, Kakovsky - Addis) methods are used. For this purpose, it is useful to induce a latent focus in the kidney by applying paraffin to the kidney area for 2-3 days or by giving the patient prednisolone, and then using the above quantitative examination methods.

In all cases of unexplained fever, abdominal pain, and seemingly random changes in urine, it is advisable to examine children for kidney disease with X-rays and ultrasound. Pyelonephritis is characterized by asymmetry, enlargement, and malformation of the urinary tract, especially the renal pelvis. These examination methods should be used in any patient with pyelonephritis, since the cause of its occurrence must be determined and, if necessary, surgically removed (congenital defects, stones).

Considering that secondary pyelonephritis can also occur in the presence of metabolic disorders (uraturia, cystinuria, etc.), appropriate examinations are required.

Treatment. Treatment goal :

- elimination of the infectious process
- elimination of causative factors
- urodynamics and kidney function restoration
- Nephroprotective treatment in progressive nephropathy
- to heal the patient and prevent complications.

Treatment tactics :

Non- drug treatment methods:

- Regimen : complete bed rest during the period of elevated body temperature, then as prescribed in the general procedure.
- Diet #7:
  - Age-specific , adapted to basic food products, proteins are not limited ;
  - restriction of extractive products, pastries , marinated products , smoked products , spicy products (garlic , onion , cilantro) and products with a high sodium content ;
  - Drink plenty of fluids (50% more than normal) , alternating with alkaline mineral waters .
- Establish a " regular " urination pattern (every 2-3 hours , depending on age);
- Daily hygiene procedures (shower, bath, toweling , toilet of the external genitalia);

Drug treatment

- Symptomatic treatments : antipyretic , detoxification, infusion agents - usually administered for 1-3 days of illness ;
- Antibacterial treatment procedures are carried out in 3 stages :

Stage 1 -1 Antibiotics for 0-14 days ;

Empirical (initial) choice of antibiotics :

- " Protected " penicillins : amoxicillin/clavulanate, amoxicillin/sulbactam;

Third generation cephalosporins : cefotaxime, ceftazidime, ceftriaxone, cefixime, ceftibuten .

In severe cases :

- Aminoglycosides : netromycin , amikacin, gentamicin;
- Carbapenems : imipenem, meropenem ;
- IV generation cephalosporins (cefepime).

Indications for parenteral therapy :

- Patient age <3;
- Severity of the patient's condition : high activity of the infectious-inflammatory process or suspicion of sepsis, severe intoxication or dehydration;

- Dyspepsia (vomiting) and malabsorption in the gastrointestinal tract ;
- Inability to take medications ;
- of resistance of microorganisms to oral αντιβιοτιχσ .

oral drug delivery regimens :

- Clinically positive changes and no fever in the last 24 hours ;
- Absence of vomiting .

antibiotic treatment in patients with pyelonephritis depends on the severity of the disease .

Duration of antibiotic treatment course in patients :

- Severe course (fever ≥39°, dehydration, repeated vomiting): antibiotics are administered intravenously until the temperature returns to normal (average 2-3 days) , then switched to oral administration (stepwise treatment) for 10-14 days ;
- Mild cases (moderate fever, no severe dehydration, adequate fluid intake): Take oral antibiotics for at least 10 days.

If the treatment is effective, the following will be observed :

- Positive clinical changes in the course of the disease are observed 24-48 hours after the start of treatment ;
- 24-48 hours, eradication of microflora is observed ;
- A decrease or disappearance of leukocyturia is observed 2-3 days after the start of treatment ;
- of an antibacterial drug after 48-72 hours is carried out after the results of microbiological examination and the determination of the sensitivity of microorganisms to antibiotics.

Table 12.

Dosages of antibacterial drugs for the treatment of pyelonephritis in children

(LSStrachunsky, Yu.B. Belousov, SN Kozlov, 2007)

Name of the drug	Distribution mode	
	Quantity	Delivery method and mode
Amoxicillin/clavulanate*	40-60 mg/kg/24 hours (as Amoxicillin)	Oral and intravenous 2-3 times a day
Amoxicillin/sulbactam	40-60 mg/kg/24 hours (Regarding Amoxicillin)	Oral , intramuscular and intravenous 2-3 times a day
Third generation cephalosporins		
Cefotaxime	Children up to 3 months – 50 mg/kg/8 h 3 months : 50-100 mg/kg/24 h	Oral and intravenous 2-3 times a day

Ceftriaxone	Children up to 3 months – 50 mg/kg/8 h 3 months : 50-100 mg/kg/24 h	IM and IV 1-2 times a day
Ceftazidime	Children up to 3 months: 30 – 50 mg/kg/8 h Children older than 3 months3 0-100 mg/kg/24 h	IM and IV 2-3 times a day
Cefoperazone/sulbactam	40-80 mg/kg/24 hours (as Cefoperazone)	IM and IV 2-3 times a day
Cefixime	Children >6 months old — 8 mg/kg/24 h	Oral 1-2 times a day
Ceftibuten	For children >12 months: when weighing <45 kg - 9 mg/kg/24 hours; when weighing >45 kg - 200-400 mg/24 hours	Oral 1-2 times a day
Fourth generation cephalosporins		
Cefipime	>2 months: 50 mg/kg/24 h	3 times a day
Aminoglycosides		
Gentamicin	Children up to 3 months - 2.5 mg/kg/8 s; children older than 3 months - 3-5 mg/kg/24 s	IM and IV 1-2 times a day
Netilmycin	Children up to 3 months - 2.5 mg/kg/8 hours; children older than 3 months - 4-7.5 mg/kg/24 hours	IM and IV 1-2 times a day
Amikacin	Children up to 3 months - 10 mg/kg/8 h; children older than 3 months - 15-20 mg/kg/24 h	IM and IV 1-2 times a day
Carbapenems		
Imipenem	Children up to 3 months - 25 mg/kg/8 h; children older than 3 months : if weight <40 kg - 15-25 mg/kg/6 h . if weight >40 kg – 0.5-1.0 g/6-8 h, not more than 2.0 g/24 h.	IV 3-4 times a day
Meropenem	Children older than 3 months : 10-20mg/kg/8	IV 3 times a day

	hours (max 40 mg/kg/8 hours) In an amount not exceeding 6g/24h.	

" PROTECTED " PENICILLINS

the 2012 European guidelines (M.Grabeet. al. Guidelines on urological infections. Uroweb, 2012), the dose of amoxicillin/clavulanate is 3 months. In children under 12 years of age:

- parenteral - 60-100 mg/kg/day 3 times ;
- oral - 37.5-75 mg/kg/milk in 2-3 times .

2 - stage – uroseptic treatment (14-28 days).

1. 5-nitrofuran derivatives:
   - Furagin – 7.5-8 mg/kg (no more than 400 mg/24 hours) 3-4 times ;
   - Furamag - 5 mg/kg/24 s (in an amount not exceeding 400 mg/ 24 s) 2-3 times .
2. Fluoroquinolones:
   - Negram, nevigramon (for children older than 3 months) – 55 mg/kg/24 h 3-4 times ;
   - Palin (for children over 12 months) – 15 mg/kg/24 hours 2 times .

3 - stage- preventive treatment against relapses .

Long-term antimicrobial prophylaxis for urinary tract infections (UTIs) in children is administered :

- ≥3 relapses of SYI during the year ;
- Bladder and urethral reflux , SY anomalies , severe neurogenic dysfunction of the bladder ; young children who have experienced an episode of pyelonephritis ;
- All girls who have experienced urinary tract stones , dysuric conditions, and episodes of SYI .
- Nitrofuran derivatives (furagin, furamag) 1/3-¼ of the daily dose (1-2 mg/ kg) in the evening for 1-12 months . The duration of treatment depends on the duration of clinical and laboratory remission :
- 6 months – if the interval between relapses is from 3 weeks to 3 months ;
- 12 months - if the interval between relapses is less than 3 weeks ;
- Bladder and urethral reflux can occur over a long period of time .

Other types of treatment:

- Phytotherapy .
- Probiotics

no surgical treatments or specific preventive methods .

Recommended :

- Strengthening the body ;
- Sanitation of chronic foci of infection .

Dispensary observation: Dispensary observation for children who have experienced an episode of acute pyelonephritis Dispensary observation for

children who have experienced an episode of acute pyelonephritis is carried out for 5 years , and in the case of a recurrent course of the disease - until transfer to an adult clinic (up to 19 years of age) (Table 13).

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Plan for dispensary monitoring of children with pyelonephritis

Medical treatments	Number of observations
1. Medical examinations	Once every 3 months
2. Clinical blood analysis	6 once a month
3. General urine analysis (or Nechiporenko test)	In case of intercurrent diseases, once a month before vaccination
4. Bacterial analysis of urine	6 once a month
5. UTT of abdominal organs and kidneys	Once every 12 months
6. Zimnisky test	6 once a month
7. Biochemical blood analysis	12 once a month
8. Determination of ball filtration rate	12 once a month

CHAPTER VI.
HEREDITARY KIDNEY DISEASES (TUBULOPATHIES)

Over the past 30-40 years, the widespread application of genetic and clinical membranological research methods in nephrology practice has led to a sharp expansion and deepening of the scope of medical scientific understanding of the causes and processes of kidney and urinary tract diseases. Observations in this regard show that in recent years the weight of hereditary and congenital diseases in the nosological structure of kidney diseases in children has been increasing: 14-20% of kidney diseases are associated with hereditary factors, and in more than 50% of cases, a hereditary predisposition has been identified. Hereditary kidney diseases can manifest themselves in infancy, but in most cases they are detected in infancy or later. In cases where the disease is detected late, it is often possible to see that kidney function is already impaired. It was initially proposed to divide hereditary kidney diseases into 2 groups: “nephritic syndromes”, which are clinically similar to advanced kidney diseases (glomerulonephritis, pyelonephritis), and “chronic tubulopathies”, which are associated with hereditary deficiencies in organic and electrolyte transport in the renal tubules (Table 14).

Table 14 .

Kidney and urinary tract diseases

Uploaded	Hereditary
1. Glomerulonephritis 2. Pyelonephritis, tubulo-interstitial nephritis . 3. Defects of the kidneys and urinary tract that occur during embryogenesis and fetogenesis. 4. Kidney damage in systemic diseases.	1.Glomerulopathies . 2.Tubulopathies . 3.Hereditary defects of the kidneys and urinary tract. 4.Kidney damage in hereditary metabolic disorders.

The distinction between acquired, congenital and hereditary kidney diseases in this classification is of great importance both theoretically and practically, since it is clear that a number of drugs used in acquired kidney diseases (antibiotics, corticosteroids and cystostatics) are not only useless in other cases, but can even be harmful. MS Ignatova and Yu.E. Veltishev proposed a more complete classification of hereditary kidney diseases, distinguishing three large groups (Tables 15, 16).

Table 15

Main groups and forms of congenital and hereditary kidney diseases

(M.S.Ignatova , 2011).

Disease groups.	Shapes
Kidney structural defects	1. Renal parenchymal insufficiency (agenesis, hypoplasia). 2. Excess kidney tissue (extra kidneys). 3. Defects in the position, shape and location of the kidneys. 4. Defects in the integrity of kidney tissue. 5. Cystic kidney diseases.
Nephritic-like hereditary diseases	1. Hereditary nephritis (Alport syndrome) with preserved hearing and its decline. 2. Congenital nephrotic syndrome. 3. Familial nephrotic syndrome.
Tubulopathies	1. Primary tubulopathies (renal glucosuria, renal diabetes insipidus, phosphate diabetes, congenital vitamin D-resistant rickets, pseudohypoaldosteronism, De-Toni-Debre-Fanconi disease, renal tubular acidosis, glycinuria, cystinuria, xanthinuria) 2. Secondary tubulopathies (primary hyperoxaluria, primary hyperoxaluria, cystinosis, etc.).

Table 16.

Classification of hereditary and congenital nephropathies

(M.S.Ignatova , 2011).

Type of nephropathy	Nosological form
Anatomical anomalies of the urinary system	a) Anatomical anomalies of the kidney: - numerically (agenesis, aplasia, additional kidney); - by location (dystopia, nephroptosis, rotation); - shape anomaly (horseshoe-shaped, S- and L-shaped kidneys); b) Anomalies of the urinary tract, bladder and urethra; c) Anomalies of the structure and location of the renal blood vessels (including the lymphatic system); g) Anomalies of the nervous system supply of the urinary system (neurogenic bladder).

Histological dysembryogenesis of the kidneys	a) With cysts: polycystic disease, Fanconi nephronophthisis, Senior's disease, "Finnish form" of congenital nephrotic syndrome, other types of cystic (cavity-forming) disease; b) Cystless: oligonephronia, segmental hypoplasia (Ask-Upmark disease), hypoplastic dysplastic nephropathy (snigeneris), associated with developmental anomalies of the urinary system or dysmetabolic changes: associated with glomerulonephritis or interstitial nephritis.
Hereditary nephritis	a) With hearing loss; b) Hearing loss is not reduced.
Tubulopathies	a) Primary: damage mainly to the proximal part of the renal tubules (renal glucosuria - renal diabetes, phosphate - diabetes, de Toni - Debre - Fanconi disease, cystinuria, aminoglycosuria, II - various renal tubular acidosis, etc.); Types that develop mainly in the distal part of the body (diabetes insipidus, diabetes mellitus, type I renal tubular acidosis) . b) Secondary: in hereditary metabolic disorders (galactosemia, cystinosis, gout, Fabry angiokeratoma); c) Crystalluria (oxalate and urate nephropathies) observed in familial cell membrane disorders.
Nephropathies among monogenic and chromosomal syndromes	
Embryonic kidney tumor	Wilms' tumor

SAA heredity and their significant weight in the nosological composition of SAA diseases require the widespread use of medical-genetic examination methods in nephrological practice.

Tubulopathies

In medical practice, a group of diseases associated with the secretion and reabsorption of one or more substances in the renal tubules and with a variety of clinical manifestations are collectively referred to as tubulopathies. Depending on the nature of their origin, primary and secondary tubulopathies are distinguished.

Primary (hereditary) tubulopathies :

1) changes in the composition of proteins that ensure the reabsorption of substances;

2) enzymopathies, that is, deficiencies in the activity of enzymes that ensure the active transport of substances through membranes;

3) loss of sensitivity of the cells of the mucous membrane of the urinary tract to the effects of the corresponding hormones;

4) occurs as a result of defects in the general structure of tubular cells.

Secondary tubulopathies: develop as a result of metabolic disorders that are not directly related to the nephron and tubules. A thorough analysis of the medical history and family tree is crucial in differentiating primary and secondary tubulopathies. Definitive diagnosis of tubulopathies requires specific clinical and biochemical examinations. However, a number of clinical signs and their complexes (syndromes) are known that allow not only to predict the presence of tubulopathies in a child in general, but even their specific features (Table 16).

Tubulopathies with skeletal anomalies . Primary (hereditary) tubulopathies causing rickets include de Toni-Debre-Fanconi syndrome, phosphate diabetes, and renal tubular acidosis. Ricket-like changes in the skeleton are also observed secondary to hereditary disorders of calcium-phosphorus metabolism (vitamin D-dependent rickets, celiac disease, pseudohypoparathyroidism, chronic renal failure) (Table 17).

Table 17.

Classification of tubulopathies based on the main clinical symptoms

(according to Yu.E. Veltishev)

Main character set	Primary tubulopathies	Appearance (phenotypically similar cases)
Skeletal anomalies (renal osteopathies)	Phosphate-related diabetes, de Toni-Debre-Fanconi disease, renal tubular acidosis.	Vitamin D-dependent rickets, hypophosphatasia, celiac disease, pseudohypoparathyroidism
Polyuria	Renal glucosuria, renal diabetes insipidus, renal salt diabetes (pseudohypoaldosteronism)	Fanconi's disease (nephronophthisis), pyelonephritis, cystinosis, tyrosinemia, chronic renal failure
Nephrolithiasis	Cystinuria, glycinuria, and aminoglycinuria	Oxalosis, secondary hyperoxaluria, xanthinuria, Lesch-Nyhan syndrome

De Toni – Debre – Fanconi disease . De – Toni – Debre – Fanconi disease (glucose – amino – phosphate diabetes) is a primary (hereditary) renal enzymopathy, transmitted from generation to generation in an autosomal recessive manner. This disease is the most severe proximal tubulopathy. Due to the primary impairment of the reabsorption of phosphorus, glucose and amino acids in the renal tubules, they are excreted in large quantities in the urine (phosphaturia, glucosuria, hyperaminoaciduria). Since these substances are not reabsorbed in the renal tubules, the osmotic pressure of the urine increases, and as a result, a large amount of urine is excreted (polyuria), the body becomes dehydrated (dehydration), and thirst appears (polydipsia). These reasons lead to an increase in body temperature. Large amounts of phosphorus are lost in the urine, leading to hypophosphatemia, and hypokalemia develops. The above-mentioned set of chemical changes in urine can sometimes develop secondary to

cystinosis, chronic renal failure of various causes. In this case, it is called the De-Toni-Debre-Fanconi complex (syndrome), as opposed to an independent hereditary disease.

Clinical picture . Symptoms of De – Toni – Debre – Fanconi disease, especially biochemical changes, can be felt in the first months of a child's life, but the clinical picture begins to fully manifest itself after the age of one year. The child begins to lag behind his peers physically and mentally. Ricket-like changes appear in the bones of the skeleton, especially the small bones , which are prone to hardening, pain appears in the bones. Sick children are prone to infectious diseases, they easily develop otitis media, pyelonephritis. In young children, a large amount of urine is excreted, and older children complain of thirst. As a result of frequent urination, hypokalemia develops - leading to hypotension, hyporeflexia. ECG - shows characteristic signs of hypokalemia. Metabolic disorders, in turn, lead to acidosis, and the patient has signs of general intoxication - pallor, weakness. Urinalysis reveals hyperaminoaciduria, hyperphosphaturia, and glucosuria. Since glucose and amino acids are a favorable nutrient medium for the growth of microbes in the kidneys and urinary tract, favorable conditions are created. Therefore, they often develop recurrent, chronic pyelonephritis. Despite the large excretion of glucose and more than 10 amino acids in the urine, their content in the blood remains practically unchanged. These factors are important for comparative diagnosis. X-ray examination shows osteoporosis in all skeletons, bones are very soft for their age. As a constant biochemical criterion, it should be noted that the clearance of inorganic phosphorus in the blood is high, tubular reabsorption of phosphorus is up to 45% and lower. The level of phosphorus in the blood is two times lower than in healthy children, and the level of calcium in the urine is almost unchanged.

Treatment . Specific treatment measures are not known. There is no special diet, but products rich in sulfur-containing amino acids (cysteine, cystine), carbohydrates should be limited, and fluids and proteins should be given in sufficient quantities. It is advisable to recommend potassium-containing products to the patient's diet. It is necessary to monitor the acid-base balance, and in case of acidosis, 4% sodium bicarbonate solution should be administered intravenously or orally in a dose of 45-60 ml with the addition of 2 g of citric acid, 3 g of sodium citrate, 3.3 g of potassium citrate per 100 ml. Vitamin D in the amount of 20,000-30,000 IU per day under the control of the amount of calcium excreted in the urine gives a significant clinical result. Gradually (over 4-6 weeks) the amount of vitamin D should be brought to 75,000-100,000 IU. Treatment is carried out with constant monitoring of the level of calcium and phosphorus in the blood and urine, as well as the state of osteogenesis. The use of vitamin D requires the use of calcium preparations. For this purpose, it is advisable to use one of the complex calcium preparations (Table 18).

Table 18.

Calcium-sparing combination drugs

(Novikov PV, 2003)

Pharmacological drugs	Composition
Calcevita (Cal - C - vita)	1 chewable tablet contains 300 IU of vitamin D and 250 mg of calcium.
Kalsinova	1 tablet contains 100 IU of vitamin D, 100 mg of calcium, and 77 mg of phosphorus.
Ca – D – 3 Nikomed (Calcium – D – 3 Nykomed)	1 tablet contains 200 IU of cholecalciferol and 500 mg of ionized calcium.
Calcium - sandoz forte (Calcium - sandoz - forte)	1 tablet contains 500 mg of ionized calcium.
Calcium glycerophosphate	1 tablet contains 500 mg of calcium glycerophosphate.
Osteochea	1 tablet contains 400 mg of calcium, 150 mg of magnesium and 100 IU of cholecalciferol.
Vitrum	1 tablet contains 400 IU of vitamin D, 162 mg of calcium, and 125 mg of phosphorus.
Pikovit	1 dragee contains A, C, B1 , B2 , B6 , B12 , panthenol, folic acid , nicotinamide, 100 IU vitamin D3 , 12.5 mg calcium and 10 mg phosphorus. Children under 7 years of age are prescribed 5 dragees per day, and after 7 years of age, 7 dragees per day.

When prescribing calcium supplements, it is necessary to take into account the amount of calcium in the blood and urine, as well as the daily calcium requirement of children (Table 19).

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The need for phosphorus and calcium during the day and night

(Novikov PV, 2003)

Age	Calcium	Phosphorus
0 – 4 months	450 mg	30 mg/kg
4 – 12 months	500 mg	50 mg/kg
1 – 6 years old	1000 mg	1500 mg
7 – 10 years old	1200 mg	2000 mg
11 – 14 years old	1500 mg	2500 mg

In addition, 0.1 - 0.3 mg/kg of methylandrostenolone per day is prescribed, which, being an anabolic hormone, improves the absorption of amino acids and relatively reduces aminoaciduria. At the same time, the administration of phosphates (phytin 0.5 - 1.0) allows you to reduce the daily dose of vitamin D required for treatment to 25,000 - 30,000 IU. As in all cases of hypokalemia , caution should be exercised when administering glucose solution intravenously,

as this can lead to a sharp dilation (collapse) of blood vessels and a decrease in blood pressure.

Familial hypophosphatemia, Fanconi-Girare renal phosphate diabetes (phosphate renal diabetes, familial phosphate diabetes, hereditary phosphate diabetes). The essence of this disease is a violation of the reabsorption of phosphates in the renal tubules and is accompanied by the development of rickets-like symptoms that are resistant to the effects of vitamin D.

Etiology and pathogenesis . The causes of the disease are not fully understood. However, the role of a sharp decrease in the reabsorption of phosphates in the renal tubules in the development of the disease (pathogenesis) is undoubtedly important. The cause of the violation of phosphate reabsorption is associated with a deficiency in the amount (or their activity) of the enzymes that provide this process. At the same time, a secondary decrease in calcium absorption in the intestines is also observed. In this disease, the level of vitamin D in the blood of patients does not differ from that of healthy children. However, in these patients, the amount of vitamin D usually used in practical medicine does not significantly affect the absorption of phosphates either in the renal tubules or in the intestines. This is also evidenced by the fact that the level of vitamin D in the patient's blood does not differ significantly from that of healthy children . The importance of hereditary factors in the development of the disease is also confirmed by the presence of similar sick individuals in the families of sick children. One of the parents will definitely have hypophosphatemia and rickets. Gender is not a factor in hypophosphatemia, but rickets are more common in males.

Clinical signs of the disease are not noticeable in infancy and childhood. However, when the child reaches 1 year of age and begins to stand and walk, a rickets-like curvature of the legs resembling O and X is observed. These changes occur despite the usual intake of vitamin D to prevent rickets. The child has difficulty walking, and in severe cases may become unable to walk. There may also be “late” forms of this disease, in which the child’s legs become painful when walking, and their curvature begins to be felt when the child reaches 6-8 years of age. In such patients, the eruption of teeth is delayed beyond the usual period, their enamel loses its strength and becomes prone to caries. The physical growth of children also lags behind . Sometimes this disease is observed in combination with conditions such as albinism, congenital nystagmus, xanthomatosis, craniosynostosis.

Phosphate diabetes should be distinguished from simple rickets, which is observed in vitamin D deficiency, from other tubulopathies and osteopathies that develop as a result of chronic renal failure. The causes of rickets-like diseases are diverse, and they can be manifested in hereditary (primary tubulopathies) and acquired kidney diseases (chronic renal failure), metabolic disorders (cystinosis, tyrosinosis, Wilson's disease, glycogenoses), diseases of the gastrointestinal system (malabsorption, congenital hepatitis and cirrhosis of the liver, congenital defects of the biliary tract) and endocrinopathies (primary hyperparathyroidism).

Rickets-like changes are also observed in disorders of nutrient absorption in the intestines (malabsorption). In the metabolism of calcium and vitamin D, the intestines perform two functions: 1) absorption of vitamin D in the intestines, 2) synthesis of calcium-binding protein in enterocytes. Malabsorption syndrome is based on changes in the structure and function of enterocytes, both of which are impaired and rickets-like changes in the bones develop. Rickets-like changes can be observed in all diseases accompanied by malabsorption, and are especially pronounced in cystic fibrosis and celiac disease.

Primary hyperparathyroidism leads to calcium leaching from the bones, hypercalcemia and hypercalciuria. As a result, osteoporosis develops, bones become easily flexible and brittle. As a result, rickets-like changes develop. Affected children suffer from bone pain. Prolonged hypercalciuria can lead to the formation of kidney stones, calcium deposits in the kidney tissue - nephrocalcinosis.

The reason for the development of rickets-like changes in severe hepatopathies (chronic hepatitis, cirrhosis of the liver) is that vitamin D is a fat-soluble vitamin, and its absorption from the small intestine requires the participation of bile acids. Failure to enter the intestine or insufficient absorption of bile can cause vitamin D deficiency in infancy.

Phosphate - a sharp decrease in the level of phosphate in the blood is a characteristic sign for the diagnosis of diabetes. In addition, an increase in the enzyme alkaline phosphatase is also observed in the blood. Although the level of calcium in the blood is normal, the absorption of calcium from the intestines is impaired (Table 20).

Table 20.

Inorganic phosphate levels in the blood of healthy children

(Novikov PV, 2003)

Age	Serum level	
	M mol /l	Mg%
Babies	1.6 – 2.7	4.8 – 8.0
1 – 3	1.3– 2.1	3.8 – 6.5
4 – 7	1.2 – 1.8	3.7 – 5.6
8 – 11	1.1 – 1.7	3.6 – 5.5
12 – 15	0.9 – 1.6	2.9 – 5.4

Phosphate is the most important biochemical marker for diabetes, which is characterized by the excretion of large amounts of phosphate in the urine and a decrease in the amount of phosphorus in the blood, which is explained by impaired phosphate reabsorption in the renal tubules.

Treatment. The safe doses of vitamin D are ineffective in treating such patients. Currently, the only treatment for phosphate diabetes is the use of large doses of vitamin D and its analogues. The effectiveness of treatment is monitored by radiological monitoring of bone maturation (Table 21).

21-jadval .

Pharmacological preparations of vitamin D

(P.V. Novikov, 2003)

Pharmaceutical preparations	Release	Dosage
Vitamin D_2 (ergocalciferol)	0.0625% oil solution 0.125% oil solution 0.5% oil solution 0.5% alcohol solution	25,000 IU in 1 ml 50,000 IU per ml 200,000 IU in 1 ml 200,000 IU in 1 ml
Vitamin D_3 BON (cholecalciferol)	0.05% oil solution to drink or m/o	200,000 IU in 1 ml
Vigantol (cholecalciferol)	0.05% oil solution	200,000 IU in 1 ml
Rocaltrol (calcitrol)	Capsules Solution	0.25 mcg in 1 capsule 0.5 mcg in 1 capsule 1 mcg in 1 ml
Van-alpha (alpha-calcidol)	Tablet	0.25 or 1 mcg in 1 capsule
Alpha D_3 – Teva	Capsules	0.25, 0.5 mcg in 1 capsule or 1 mcg
AT10 (dihydrotaxysterol)	0.1% oil solution	40,000 IU per ml (1 ml contains 1 mg of the drug)
Taxistin (dihydrotaxisterol)	0.1% oil solution	40,000 IU in 1 ml (1 ml = 24 drops contains 1 mg of the drug)

The initial dose of vitamin D is 10,000 - 15,000 IU per day. The maximum dose of vitamin D used in this disease is from 50,000 IU to 300,000 IU per day. It is important to note that vitamin D and its preparations are currently classified as hormones (secosteroids). Therefore, their use requires caution, as with other hormones (e.g. corticosteroids). Some preparations belonging to the vitamin D group do not have the activity of vitamin D (dihydrotaxisterol, taxin), but they have the property of enhancing the absorption of calcium from the intestines.

Dihydrotaxisterol is usually prescribed in a dose of 10-20 drops, and taxin in a dose of 500 mcg to 1.5 mg once a day after meals, and it is necessary to monitor the level of calcium in the blood and urine. The daily dose of vitamin D, which is the main treatment for this disease, is increased every 2 weeks, and this is done by monitoring the level of calcium and inorganic phosphates in the blood and urine, the level of alkaline phosphatase in the blood, and the restoration of the bone structure in X-ray examinations (Table 22).

Table 22.

Vitamin D used in the treatment of tubulopathies daily amount (B/kg) (M.S.Ignatova , 2011).

Age (in years)	Vitamin D levels (TB/kg/milk) and nosological forms			
	Phosphate – diabetes	Vitamin D-dependent rickets	De - Tony - Debre - Fanconi	Renal tubular acidosis
0 – 3	7000 – 6000	4000 – 3000	4000 – 2000	3000 – 2000
4 – 7	6500 – 4000	3500 – 2000	7000 – 2000	4500 – 2000

The use of large amounts of vitamin D, while not indifferent to the body, poses a risk of vitamin D poisoning (hypervitaminosis D). During treatment with vitamin D, this category of patients sometimes develop signs of poisoning (the patient's appetite decreases, thirst, diarrhea, at the same time increased urine output, calcium excretion in the urine of more than 3 mg / kg per day, hypercalcemia), which necessitates the cessation of treatment. After the signs of poisoning disappear, it is necessary to continue treatment with a smaller amount of vitamin D than before . At this time, it is also advisable to prescribe neutral inorganic phosphorus to patients as the sodium salt of phosphoric acid. Usually, inorganic phosphates are prescribed at a dose of 70-100 mg / kg (relative to phosphorus) per day. For this purpose, Osteogenon can be prescribed (1 tablet contains 178 mg of Sa, 82 mg of P) - 1-3 tablets per day. Centrum 1-2 tablets per day (1 tablet contains 162 mg Sa, 125 mg R, 100 mg Mg). Vitrum (1 tablet contains 162 mg Sa, 125 mg R, 100 mg Mg). As a result of such treatments, rickets-like symptoms disappear, and the growth of the sick child also improves. When such patients reach adulthood, the symptoms of the disease subside without treatment, but the risk of recurrence of phosphorus metabolism disorders during puberty and pregnancy remains. In individuals who have had phosphate diabetes in childhood, hypophosphatemia persists even in adulthood, while the level of alkaline phosphatase may be normal. If we compare the symptoms of this disease in several generations, we can determine that skeletal changes are more severe in men than in women.

The category of polyuric tubulopathies includes primary enzymopathies: renal glucosuria (glucodiabetes), renal diabetes insipidus, renal salt diabetes (pseudohypoaldosteronism), and Fanconi nephronophthisis. Secondary tubulopathies accompanied by polyuria are also observed in neurohypophyseal diabetes insipidus.

Renal glucosuria (renal glucodiabetes). The disease is caused by a deficiency of enzymes that ensure the reabsorption of glucose in the renal tubules. The disease is transmitted from generation to generation in an autosomal dominant manner. In this case, the loss of glucose through the urine reaches 10-20 grams (sometimes up to 100 grams) per day.

The clinical picture is not rich in external signs, sometimes it occurs without external signs. Clinical signs such as excessive appetite, weakness, growth retardation, thirst are observed in severe cases of the disease. Thirst, frequent urination, the presence of acetone and sugar in the urine raise suspicion of diabetes mellitus. There are the following main indicators for comparing these diseases: 1) in patients with renal glucosuria, along with the excretion of large amounts of glucose, the blood sugar level remains normal; 2) regardless of the amount of carbohydrates consumed, the excretion of sugar in the urine is maintained; 3) regardless of the amount of carbohydrates consumed, the blood sugar level does not change; 4) the blood sugar level does not change even when glucose is loaded. Special methods of treating the disease have not been developed. If mild hypoglycemia develops as a result of urinary glucose loss, 3.0 - 5.0 g. of additional glucose per day is administered, and hypokalemia developed due to polyuria is moderated (taking Panangin tablets 2 - 3 times a day - 1 tablet contains 140 mg of potassium). Energotropic drugs are recommended in moderate doses: coenzyme Q - 50 - 90 mg per day, cytochrome C intramuscularly or intravenously 4.0, nicotinamide 100 - 300 mg, vitamin C - up to 1.0 g, L - carnitine 50 mg / kg.

Renal diabetes insipidus. The disease is linked to the X chromosome and is transmitted from generation to generation in a sex-linked recessive manner. Boys are mainly affected. The basis of the disease is the loss of sensitivity of the renal tubules to antidiuretic hormone (ADH), which reduces water excretion in the kidneys. At the same time, the level of ADH in the blood of such patients does not decrease, the osmolar pressure in the blood is within the norm, only the relative density of urine is sharply reduced. In addition to the primary forms of renal diabetes insipidus, secondary forms are observed in renal and extrarenal diseases (chronic pyelonephritis, nephrosclerosis, renal amyloidosis, hypokalemia, hyperparathyroidism, hypervitaminosis D and others). Disturbances in homeostasis in diabetic nephropathy lead to changes in serum osmotic pressure and hyperelectrolyteemia (increased serum sodium to 180 mEq/L, and chloride to 160 mEq/L).

Clinical picture. The disease begins with symptoms such as polyuria, constipation, vomiting, and as a result, the child lags behind in development. In severe cases of dehydration, body temperature increases due to dehydration of the central nervous system cells, and in some cases, seizures may occur. The daily urine output in an infant can be 2 liters or more. Due to dehydration, the sodium content in the blood increases to 150–200 mmol/l, and chlorides to 150–170 mmol/l. In severe cases, potassium and residual nitrogen may also increase. Sugar and protein are not detected in the urine, and its specific gravity is 1001–1004.

to compare neurohypophyseal and renal diabetes insipidus . In neurohypophyseal diabetes, when the hormone is administered, a decrease in diuresis and an increase in urine osmolality are observed, while in renal diabetes insipidus, no changes are observed. ADG is injected intramuscularly in an amount of 5–8 units, depending on the age of the patient. Pituitrin can also be

used, 5 units per milliliter of which are injected intramuscularly, 0.1–0.15 ml up to 1 year, 0.2–0.4 ml at 2–5 years, and 0.4–0.6 ml at 6–12 years. In renal diabetes insipidus, the paradoxical (opposite) effect of sulfonamide diuretics (hypothiazide) is characteristic: when 25–100 mg of hypothiazide are prescribed per day, urine output decreases by 50–70% and the specific gravity of urine increases.

Treatment: 25-50% glucose and Ringer-Locke solutions are administered intravenously or oral rehydration methods are used. Unlike diabetes insipidus caused by brain damage, the use of pituitrin does not give a positive result, on the contrary, hypothiazide (25-100 mg per day), which is considered a diuretic, leads to a decrease in urine output. Indomethacin (metindole) also has an antidiuretic effect when administered at a dose of 2-3 mg / kg per day. Such treatment is carried out as courses of 7-10 days. The use of pharmacological agents in renal diabetes insipidus is carried out under the control of the level of electrolytes in the blood and urine. Due to the risk of excessive potassium loss due to polyuria, hypothiazide is sometimes used in combination with spironolactone (veroshpiron).

Renal salt diabetes (pseudohypoaldosteronism). As a result of a decrease in the sensitivity of the renal tubules to aldosterone, the level of sodium in the blood serum is less than 130 mEq/L. In infants, symptoms such as polyuria, vomiting, and loss of appetite appear on the first day of life. The child lags behind in growth, mental and physical development, the process of ossification in bone tissue slows down, and the level of sodium in the blood decreases. Treatment mainly uses table salt (up to 5 grams per day). Drugs that affect the vascular system (cordiamine, mezaton) are also used as needed.

Fanconi nephronphthisis. Nephronphthisis is a familial disease characterized by impaired urine-concentrating function of the kidneys, polyuria, polydipsia, and gradually leads to chronic renal failure. The disease is transmitted from generation to generation in an autosomal recessive manner. Its initial symptom is a decrease in the urine-concentrating function of the kidneys. The specific gravity of urine decreases to 1010 and below, and all clinical and biochemical signs of renal failure gradually develop. For comparative diagnosis, chronic renal failure caused by chronic glomerulonephritis, pyelonephritis, and other kidney diseases should be considered.

There are a number of characteristic symptoms of chronic renal failure caused by hereditary kidney diseases:

- Symptoms of the disease appear as early as 2–3 years of age, as polyuria and thirst;
- At this time, the child lags behind in development;
- The child loses weight and develops anemia.

The diagnosis of the disease is made based on a careful study of the patient's family history, as well as clinical and urine specific gravity.

Treatment. Symptomatic treatment methods are used: Treatment aimed at restoring water-electrolyte balance and normalizing metabolism is recommended.

Tubulopathies with concomitant damage to the kidneys and bone system

Primary renal tubular acidosis (Lightwood syndrome). A hereditary disease of the renal tubules, characterized by metabolic acidosis, severe osteopathy, nephrocalcinosis, and nephrolithiasis. The basis of the above changes is the insufficiency of the distal renal tubules: the process of exchanging sodium ions for hydrogen ions in the cells is lost. As a result, a deficiency of bicarbonates in the blood and hyperchloremic acidosis develop. Also, in renal tubular acidosis, polyuria and a decrease in the specific gravity of urine occur. A decrease in the amount of hydrogen ions leads to the cessation of the formation and diffusion of ammonia in the distal tubules, the titratable acidity of urine decreases. Due to the loss of bicarbonates with urine, chloride ions accumulate in the blood and hyperchloremia increases. The disease is transmitted from generation to generation in most cases in an autosomal recessive manner.

The main clinical signs of renal tubular acidosis are: 1) polyuria, polydipsia, hypo-isosthenuria; 2) metabolic acidosis with a deficiency of bicarbonates in the blood; 3) changes in the blood ionogram (hypokalemia, hypocalcemia, hyponatremia, hyperchloremia, etc.); 4) lag in physical development. Nephrocalcinosis and nephrolithiasis occur in 30% of patients with tubuloacidosis. Two forms of the disease are distinguished. **The first is "infantile", "transient" (Lightwood syndrome)** - associated with a lag in the development of acetogenetic activity of the kidneys (Welty et al. Yu.E. 1989). The disease begins to manifest itself from the first months of a child's life, often with the introduction of complementary foods: the child experiences vomiting, thirst, exsicosis, increased body temperature, weakness, polyuria, hypokalemia, and sometimes convulsions. The outcome is good when the disease is uncomplicated and adequately treated.

The second form is Butler-Albright syndrome. In the primary form of this syndrome, there is a genetic link to impaired acidogenesis in the distal renal tubules, the secondary form is found in Fanconi syndrome, Sjögren syndrome, galactosemia, Willson-Konovalov disease, chronic glomerulonephritis, myeloma, etc. The disease occurs only in girls.

of the disease are : osteoporosis, X-shaped curvature of the bones, pain in the legs. The disease occurs in children of early age, they lag behind in physical development. In most cases, it is complicated by nephrocalcinosis, nephrolithiasis and pyelonephritis.

The diagnosis is made on the basis of clinical and laboratory data. To detect latent acidosis, an ammonium chloride test is performed (in the norm, the pH in the urine decreases to 5 and below), in tubular acidosis, the pH of the urine does not fall below 5.4.

Treatment. Correction of metabolic acidosis is carried out with sodium bicarbonate. In the first 12 hours, 1/3 of the bicarbonate deficit should be eliminated, and the remaining deficit should be eliminated over the next 36 hours.

To correct hypocalcemia, 10 ml of 10% calcium gluconate solution is administered intravenously every 4 hours until the Ca content in the blood is normalized , and vitamin D is prescribed. Urolithiasis, which occurs in tubular acidosis, is treated in a general manner. Magnesium salts, phosphate salts are recommended to improve the solubility of calcium salts. The prognosis is poor if the disease is accompanied by pyelonephritis and urolithiasis. In fact, glucosuria, aminoaciduria, and phosphaturia indicate damage to the proximal part of the nephron and are the result of accumulation of cystine crystals in the tubular epithelium, chronic inflammation, or a hereditary defect in the enzyme system that ensures the reabsorption of the above substances.

Clinic . After the child reaches the age of two, symptoms such as weakness, loss of appetite, vomiting, subfebrile temperature, constipation, thirst, polyuria, excessive loss of body water - dehydration occur, and the child becomes susceptible to various infectious diseases. Due to hypokalemia, signs of muscle hypotonia and hyporeflexia are observed, and arterial pressure tends to decrease. Later, signs characteristic of rickets are observed: osteoporosis, curvature of long tubular bones. The disease is transmitted from generation to generation in an autosomal dominant manner.

Treatment. If the disease is detected in a timely manner, a large dose of vitamin D and a complex treatment that restores metabolism can give a positive result. If acidosis is observed, a citrate mixture (Albright-Sholya mixture) should be prescribed.

Cystinuria is a common type of **nephrolithiasis-associated tubulopathies , resulting from a hereditary defect in amino acid transport in the kidneys and intestines. The disease is characterized by excessive excretion of cystine in the urine, urolithiasis, hematuria, and pyuria.**

of cystinuria is based on clinical findings (kidney and bladder stones, cystine crystals in the urine) and biochemical tests.

Treatment. Dietary therapy - restriction of foods containing uric acid, drinking plenty of fluids, and increasing urine alkalinity. To prevent cystine crystallization, D-penicillinamine (0.5 to 6 grams) is prescribed daily for 5 to 25 months.

Oxalosis and hyperoxaluria are hereditary metabolic disorders characterized by the accumulation of oxalic acid salts in various parenchymal organs. Sometimes this process is observed in the kidneys and manifests itself as primary hyperoxaluria - urolithiasis, nephrocalcinosis. The development of oxalosis is likely to be associated with increased synthesis of oxalic acid in the tissues. The cause of hyperoxaluria is a violation of the reabsorption of oxalates

in the renal tubules. The disease is transmitted from generation to generation in an autosomal recessive manner.

Clinic. The disease is characterized by the predominance of symptoms of nephrocalcinosis or nephritis. In nephrolithiasis, attacks of kidney pain, hematuria, and urinary tract infections are observed. The stone is located in both kidneys and is prone to recurrence. The excretion of oxalic acid in the urine is up to 10 times higher than normal. In diffuse oxalosis, growth retardation, osteoporosis, and arthralgia are observed, and family history reveals relatives with "rheumatism", urolithiasis, or pyelonephritis.

Treatment. Diet - products rich in oxalic acid are limited: these include vegetables, chocolate, cocoa, tea, etc. To prevent the formation of oxalate - calcium crystals, magnesium oxide - 150 - 200 mg per day, vitamin B_6 (40 - 60 mg in milk) are prescribed for 15 - 20 days. Products containing calcium are limited. These issues are specifically covered in the section on oxalate nephropathy.

CHAPTER VII
DYSMETABOLICAL NEPHROPATHIES

Dysmetabolic nephropathies are understood in a narrower sense as primary (hereditary) disorders of metabolism, and in a broader sense as nephropathies caused by various metabolic disorders (diabetes, diarrhea, etc.), as well as disorders of environmental influences (heavy metals, etc.). Our observations have ±shown that dysmetabolic nephropathies are observed in 21.5 out of 1,000 children examined in the climatic conditions of Uzbekistan. Of these, 40.9% of the children were found to have hyperoxaluria, 30.8% to have uraturia, and 14.9% to have cystinuria by screening. In addition, hypercalciuria, disorders of tryptophan metabolism, and other less common conditions are also known to cause the development of dysmetabolic nephropathies. Thus, nephropathies caused by metabolic disorders are relatively common, and this is actually the basis of interstitial nephritis, pyelonephritis, and urolithiasis, which are pathogenetically close to them (VA Tabolin et al., 1981). Dysmetabolic nephropathies are most often manifested in early childhood (up to 5 years old), without extrarenal symptoms, in particular, changes in urine (microhematuria, weak proteinuria), which creates certain difficulties for diagnosis. Due to the absence of noticeable clinical symptoms, they are often detected accidentally (during pneumonia, acute respiratory viral infection, or when examining urine during a child's placement in kindergarten or school) as an "incidental" finding (MM Akhmedova, 2001). Observations have shown that dysmetabolic nephropathies have a gradual course, and as the disease progresses, their clinical picture also becomes richer in symptoms. In particular, initially, only biochemical changes (hyperoxaluria, uraturia, cystinuria, etc.) are observed in the child, and as a result of various stresses (flatulence, hypoxia, unbalanced nutrition, etc.), changes in urine (hematuria, proteinuria) develop in the later period. The cause of changes in urine during this period can only be determined on the basis of an analysis of the patient's family tree and biochemical tests. On this basis, interstitial nephritis and pyelonephritis may later be added. This requires enriching the treatment with appropriate measures and medications. In terms of the development of the disease, the child may also develop urolithiasis and autoimmune processes at an early age (IM Balkarov, 1991). Since such a process gradually leads to chronic renal dysfunction in the patient, it is possible to determine the type of metabolic disorder in a timely manner and, on this basis, to conduct appropriate dietary and drug treatment. Therefore, a multi-stage clinical, genetic, and biochemical program is recommended for the diagnosis of dysmetabolic kidney disease. This program of examination is presented in the section "Renal function examination" of this book.

Existing studies on the role of heredity in kidney diseases mainly concern hereditary nephritis, and it is estimated that hereditary nephropathies account for 24.4–26.5% of all kidney diseases, of which 42% are hereditary nephritis (VVFokeeva hm1976). It has been established that hereditary nephritis is a monogenic disease transmitted from generation to generation in a dominant manner, partially linked to the sex chromosome.

In all other cases, the study of multigenic (multifactorial) kidney diseases that develop due to hereditary predisposition continues. In particular, we, having studied the family tree of 80 healthy children growing up in families complicated by kidney diseases, 39 children with primary glomerulonephritis, 94 children with oxalate nephropathy, 60 children with urate nephropathy, and having performed a genetic and statistical analysis of the data obtained using a number of special methods, came to the following conclusions: in cases of dysmetabolic nephropathies, in families with a high level of hereditary predisposition to kidney diseases (54.8 - 74%), their inheritance pattern is not consistent with monogenic models, but mainly consistent with the polygenic (multifactorial) model (Table 23).

Table 23.

Compatibility of data on the inheritance of dysmetabolic nephropathies with the criteria for multifactorial diseases

(M.S.Ignatova , 2011).

Criteria for multifactorial hereditary diseases.	Information received.
Typically, multifactorial diseases are relatively common in the population (0.1% or more), regardless of geographic, ethnic, and social factors.	The overall prevalence of nephropathy in the pediatric population we examined was 7.4% (3.57% in the artificially selected control group).
The prevalence of the disease among blood relatives depends on their degree of kinship to the patient under study.	The incidence of kidney disease is 4 times higher among parents and 3 times higher among siblings compared to the control group. It is 3.5-4 times higher among first-degree blood relatives than among more distant (II-III) blood relatives.
It has a variety of clinical presentations .	It has clinical manifestations ranging from biochemical changes without external signs to interstitial nephritis, dysmetabolic glomerulonephritis, stone disease, and other conditions.
The more such patients in the family, the greater the risk of the child getting sick.	If the parents in the family are "healthy x healthy", the incidence of kidney disease in the family tree is 10.1%, if "healthy x sick" - 16.2%, if "healthy x sick" and one or more second-degree relatives are sick - 20.3%
The laws of disease inheritance do not conform to the simple Mendelian laws.	Various hereditary - indicators obtained as a result of statistical analysis (8.6 - 14%): dominant (50%)

	and recessive (25%) are sharply different from hereditary diseases
The influence of environmental factors is clearly felt in the manifestation of the disease due to hereditary predisposition.	The share of hereditary predisposition in the development of the disease is on average 61.8% for first-degree blood relatives ±, and the share of environmental factors is 38.2% - 43%.

Based on the presented data, it can be noted that environmental factors play an important role in the development of dysmetabolic nephropathy. In this case, in order to develop the prevention of diseases on a scientific basis, general pediatrics, in particular pediatric nephrology, sets the task of thoroughly studying environmental factors (climatic conditions, ecological and social situation, etc.). As can be seen from the presented data, modern diagnosis of kidney diseases is a rather complex task, requiring special knowledge and skills. Recognition of the staged course of dysmetabolic nephropathy, in turn, also indicates that each stage requires its own specific treatment. In particular, in the initial biochemical stage, neither clinical signs nor changes in urine are observed in the child, but early correction of metabolic changes is crucial to prevent the development of subsequent stages.

Treatment of dysmetabolic nephropathies must take into account the stage of the disease, the state of kidney function, and the type of metabolic disorder that caused the disease.

The basis of the treatment of nephropathies that have developed due to metabolic disorders should be a special diet. For all types of metabolic disorders, substances that activate metabolism at the cellular level (cocarboxylase, adenosine triphosphate) and vitamin B6 (pyridoxine) are generally recommended . There is no reason or need to use antibiotics in the treatment of dysmetabolic nephropathies (however, the development of their complications is not far-fetched). The general rule for prescribing a diet is that certain products are limited in consumption, depending on the type of metabolic disorder, but the growing child's body should be adequately supplied with protein, electrolytes, drugs and fluids. When prescribing a diet, it is advisable to take into account the daily rhythm of kidney function, that is, animal proteins should be prescribed in the first half of the day, and vice versa, fruit and vegetable products in the second half of the day. However, it is also a medical fact that each type of metabolism requires its own specific diet and medication (Table 24).

Table 24.

Stages of development of dysmetabolic nephropathies and general principles of treatment

(DIIshkabulov, SKAbdurakhmanova SK, 1997) **.**

Stages of hereditary and DZMN	Treatment methods
Biochemical stage leading up to clinical manifestation	Correction of latent metabolic disorders: diet, balneological treatment methods, drug treatment
With changes in urine (nephritic phase)	Treatment of metabolic disorders, taking into account the above treatment methods and the state of kidney function
Period of (partial) impairment of some kidney functions	A light potato diet (2-3 weeks). If there is mellituria, switch to consuming carbohydrates, and if there is proteinuria, switch to consuming animal proteins in the morning. Measures that reduce kidney function and crystalluria (pyridoxine, ATP) are prescribed.
Hepatorenal syndrome	Drainage without a probe (according to the Demmyanov method) with mineral water, xylitol or a 25% solution of magnesium sulfate. Treatment and diet to stimulate kidney function
Pyelonephritis	Antibacterial treatment (antibiotics and ntrofurans)
Interstitial nephritis.	Delagil, aspirin
The addition of an autoimmune process, glomerulonephritis	Heparin, delagil, aspirin, antihistamines, glucocorticoids, immunosuppressant drugs
Chronic renal failure	Conservative management, hemodialysis, and kidney transplantation

Urate nephropathies

Urate nephropathies occur as a result of impaired uric acid metabolism in the body. It is known that uric acid in the body is mainly formed as a product of the metabolism of purine bases, which are components of nucleic acids. This process consists of three stages, including the formation of uric acid from substances other than purines (de novo), formation during the breakdown of purines, and synthesis in the body with the participation of purines. Children are characterized by a high clearance of uric acid, which in children causes an increased incidence of hyperuraturia in cases where uric acid is formed in large quantities in the body

(CameronJ.S . , etal., 1993). Usually, inosinic acid acts as a direct starting product for the formation of uric acid. This process can also occur through the indirect formation of uric acid through nucleic acids. However, uric acid can also be formed in the body through a “short exchange pathway” without the participation of nucleic acids, in the presence of the amino acid glycine, which is usually considered a source for the synthesis of oxalates (VA Tabolin, VP Lebedev, 1975). Therefore, uraturia and hyperuricemia can also be observed in patients with oxalate nephropathy. During the day and night, about 750 mg of uric acid (about 10 mg per kg of body weight) is formed in the human body, and the same amount is excreted in urine, saliva, gastrointestinal fluids, and bile . 85-90% of the daily amount is excreted by the kidneys. The amount of uric acid in the blood serum of healthy children is 0.12-0.27 mmol/l. Its level in the urine is a variable indicator and depends on the quality of nutrition, the state of filtration in the renal tubules and the processes of secretion and reabsorption in the renal tubules. Normally, an average of 10% of uric acid filtered in the renal tubules is excreted in the urine, and 90% is reabsorbed in the renal tubules. The excretion of uric acid in the urine of a child is 0.27 mmol/kg per kg of body weight up to 7 years, and 0.19 mmol/kg from 8 to 14 years. In various pathological conditions, the balance between the formation and excretion of uric acid in the body is disturbed, and its level in the blood increases, leading to hyperuricemia (GU) and hyperuricosuria (GUZ). The increased excretion of uric acid salts (urates) in the urine is called uraturia.

Three types of primary "familial" gout are known - metabolic, renal, and mixed (increased urinary excretion with decreased uric acid production). It has been established that the development of primary gout through the metabolic pathway is due to a complete or partial deficiency of the enzymes hypoxanthine-guanine-phosphoribose-transferase (GGPRT), glucose-6-phosphatase, and phosphoribosyltransferase (FRST), which lead to increased endogenous purine production (CameronJ.S. et.al., 1993; CeronAet.al., 1987; MerdzD.Pet.al., 1989). One of the most severe clinical forms of this group is Lesch, Nyhan (1964) disease, which is inherited in an X-linked recessive manner.

This disease manifests itself in early childhood as a complex of mental retardation, autoaggressive behavior of the patient, choreoathetotic tremor, gouty arthritis, urolithiasis. Secondary hyperuricemia is more common and can occur in a number of diseases with increased cell division (leukemia, treatment of tumors with cytostatics), with a decrease in the excretory function of the kidneys, in various metabolic disorders (lactic acidosis, ketosis, dehydration, etc.), and under the influence of a number of drugs (thiazide diuretics, catabolic hormones and cytostatics, etc.). These things have been sufficiently covered in the special literature in numerous generalizing articles on this topic (K.M. Sadovsky, 1965; V.A. Abolin, V.P. Lebedev, 1975; D. Ziyaev, 1988; T. Soliev, 1990; J. Eshkobulov, 1991; Ch. Raderecht, 1975; J.B. Wyngaarden, et.al., 1978).

external clinical symptoms has a prevalence of 20-25% among the population. Gout is an important factor increasing the risk of developing diseases such as ischemic heart disease, hypertension, atherosclerosis, and bronchial

asthma. It is known that Gout is the basis for the development of gout, and therefore the importance of Gout in medicine has been constantly studied in connection with gout. At the same time, it is known that the kidneys, as the main organ for excreting uric acid from the body, are damaged in 50-100% of cases in Gout (NAMukhin, 1985; OVSinyachenko, 2006; IMBalkarov, 1999).

The amount of urate in the urine exceeding 1 mg / ml per day always poses a risk of kidney and urinary tract damage, and crystallization of salts. As a result of epidemiological studies of children under ten years of age for GU, it was determined that the prevalence of hyperuricemia among children is 1-5%. Among the adult population, asymptomatic GU accounts for 20% or more in some regions of the world (DP Mertz, 1980). GU has its own characteristics in certain periods of childhood: renal infarction in infancy, neuro-articular diathesis in preschool age, secondary pyelonephritis, urate stone formation, and later urate (dysmetabolic) interstitial nephritis and pyelonephritis, recurrent urate stone formation disease (Table 25).

Table 25.

Clinical manifestations of uricopathy in relation to age (M.S.Ignatova , 2011).

Age periods	Clinical manifestations
Infancy	External signs of connective tissue dysembryogenesis include renal infarction, and in certain situations (dehydration, hypoxia, drug exposure) renal tubular blockade and acute renal failure.
Breast age	Uric acid diathesis, Lesch-Nyhan syndrome, interstitial nephritis, secondary pyelonephritis.
Early and preschool age	Nervous - arthritic diathesis, "arthritis", acetonemic vomiting, interstitial nephritis, secondary pyelonephritis, urate urolithiasis.
School and adolescence	Vegetative vascular dystonia, psychoasthenia, acetonemic vomiting, arthropathy, gastroduodenitis, interstitial nephritis, urate nephrolithiasis and urachaliasis, secondary pyelonephritis, hyperuricemic glomerulonephritis, tubular disorders of renal function .
Older people	Chronic interstitial nephritis, nephrogenic anemia, hyperuricemic glomerulonephritis, urate urolithiasis, nephrolithiasis, secondary pyelonephritis, gout, nephrosclerosis, chronic renal failure, neuroses, gastro-duodenal diseases, salt deposits in the joints and spine, obesity, ischemic heart disease, predisposition to diabetes.

By hyperuricemia and uraturia have recently been covered in the special literature under the term "urate nephropathies" (LAShangutova, 1984; LN Astakhova, 1980; SV Malsev et al., 2006). Urate nephropathies account for 9.9–26% of kidney diseases observed in childhood (AKSybysheva, 1976; J.

Eshkobulov, 1980). Urate nephropathies are based on damage to the intercellular tissues of the kidneys due to the accumulation of urates (B.Ya. Reznik et al., 1991). Therefore, morphological changes in the kidneys are observed much earlier than the clinical symptoms of the disease. Sometimes, however, they can occur for a long time without external clinical symptoms (including in the urine), and this is the main difficulty in the clinical diagnosis of the disease. The disease, unlike glomerulonephritis and pyelonephritis, often manifests itself as temporary changes in the urine. Sometimes when examining the urine for the purpose of placing a child in kindergarten, school, sometimes during acute respiratory diseases, when examining the urine before preventive vaccination, proteinuria, microhematuria are found. At the same time, urates, uric acid crystals are found in large quantities in the urine sediment. The protein in the urine of such a patient is not very high (0.078-0.004 ±g / 24 h), the degree of hematuria may vary, but the erythrocytes remain unchanged, which also allows us to rule out glomerulonephritis. Sometimes pain is observed in the lumbar region, reaching the level of a true kidney attack. With urate nephropathy, patients have oliguria, diuresis decreases to 200-300 ml per day, a dark gray precipitate can be observed in the urine (especially in the summer heat). It is clear that there are few specific clinical signs and complaints that allow us to diagnose urate nephropathy. However, in such cases, a thorough study of the patient's family tree and special biochemical tests are useful. Since purine metabolism disorders usually have a familial nature, diseases belonging to the so-called "uricopathies" group (gout, obesity, urate urolithiasis, gallstone disease, tumors, spondylosis) are more common among the blood relatives of such a patient (proband). Analysis of the patient's family tree, if carried out in conjunction with biochemical analysis, makes it easier to distinguish hereditary nephropathies from glomerulonephritis. Signs of neuro-articular diathesis in children: the child's excessive stubbornness and activity, accompanied by general symptoms such as pain in the joints for no apparent reason (arthropathy), acetonemic vomiting, dystonia of the vegetative nervous system (Yu.I. Rovda, 1986). These general clinical signs are associated with the peculiarities of uric acid metabolism in the body and, to some extent, allow us to justify the commonality in the origin of diseases that are grouped under the term "uricopathy".

Of the disease diagnosis determination for above cited methods not applicable quite a bit to difficulties face bride.

Such comparative diagnosis that's why for also important, one how much in diseases application necessary (in glomerulonephritis – hormones, non-steroid to inflammation against drugs, pyelonephritis far term antibacterial therapy) treatments prevent to take possible. Currently, there are options for limiting the formation of uric acid, increasing its solubility, and treating it with a diet. Therefore, for treatment without causing discomfort to the patient, the diagnosis must be perfect, since such a patient requires long-term treatment and constant monitoring. Timely detection of glomerulonephritis that has developed on the basis of GU is of great importance for treatment. Some experts recommend distinguishing the GU type of glomerulonephritis (NAMukhin 1985, 1986). The

cause of chronic interstitial and tubular pathological processes observed in hyperuricemia and urateria is that the solubility of urates accumulated in the interstitial fluid in the acidic environment of the renal tubules decreases and, crystallizing, injures the mucous membranes, precipitates and forms stones (Ni AN, et al., 2004; MV Lebedeva et al., 1997).

There are also conditions for damage to the interstitial tissue, since in the relatively alkaline environment of the renal medulla and due to the high concentration of sodium, sodium monourate salt is formed, forming crystals. This phenomenon can be clearly observed when examining patients with urate nephropathy with ultrasound. In this case, any cause that causes dehydration in the child's body (exicosis, prolonged heat, hypoxia) leads to a sharp decrease in urine output, a sharp increase in urate and oxalate crystals in its composition, that is, the risk of stone formation. It follows that it is not accidental that in such families there are urolithiasis, salt accumulation in the spine, arthropathies, radiculitis, chronic nephritis-like conditions. The most frequently and early affected organs in individuals with hyperuricemia and urateria are the kidneys, and in 69.2% of gout patients, kidney changes are detected in the first five years of the disease, especially when the level of uric acid in the blood exceeds 0.08 g/l, it exceeds 83.3%. Damage to the interstitial tissue and renal tubules, in turn, creates favorable conditions for the development of a microbial inflammatory process - pyelonephritis.

In the absence of appropriate treatment measures, all of the above cases lead to sclerosis of the renal tissue and chronic impairment of all its functions. From the above, it is clear that the development of urate nephropathy is gradual, gradually increasing in clinical symptoms (Table 54). Due to these processes, the altered renal tissue, as a foreign body, triggers an immunological process in the body, and the resulting complex of "antigen + antibodies" affects the basal membrane of the glomeruli, leading to glomerulopathy, i.e. secondary glomerulonephritis. Such glomerulonephritis, unlike primary (streptococcal) glomerulonephritis, is accompanied by impaired renal concentrating function (hypostenuria) at the very beginning of the disease. In primary glomerulonephritis, this condition is observed only in the period of advanced chronic renal failure. Another characteristic feature of uricopathy is that, if carefully examined, even before the formation of glomerulonephritis, signs of neuro-articular diathesis can be detected in a child (hyperactivity, irritability, shooting headaches - migraine, acetonemic vomiting), intestinal colic, joint pain in the evening (arthritis), vegetative dystonia of blood vessels, etc. The combined course of glomerulonephritis and pyelonephritis is observed in 4-15% of cases, and the opposite is true for the addition of pyelonephritis to existing glomerulonephritis, i.e., the development of glomerulonephritis in a patient with pyelonephritis is 3-4 times more common than the development of glomerulonephritis (ANShpigel, 1991). However, glomerulonephritis resulting from metabolic disorders can often develop on the basis of microbial or non-microbial interstitial nephritis or secondary chronic pyelonephritis (Table 26).

Table 26.

Stages of development of urate nephropathy

(M.S.Ignatova , 2011).

Development stages	Main and additional factors causing nephropathy	Main clinical signs
Primary or secondary disorders of uric acid metabolism	Latent (biochemical) signs of metabolic disorders, tubulopathy	Pathological changes in urine
Abacterial and bacterial interstitial nephritis	Accumulation of urates in the interstitial tissue of the kidney, blockage of the renal tubules with urates	Tubulo – Clinical signs of interstitial nephritis
Hepato-renal syndrome	Biliary drainage dysfunction, reactive hepatitis	Liver enlargement, dyslipidemia, urinary syndrome
Pyelonephritis	Addition of microbial inflammation to abacterial interstitial lesions	Bacteruria, proteinuria, leukocyturia
Nephrolithiasis	Microbial inflammatory diseases of the kidneys, dehydration, effects of prolonged heat on the body	Intoxication, abdominal and lower back pain, dysuria, urinary changes
OBE	Urinary tract obstruction (dehydration, acute intercurrent illness, drug effects)	Oliguria, edema, hypertension, hyperazotemia
Involvement of an autoimmune process	Changes in kidney tissue due to microbial or non-microbial inflammation, leading to an immunological process	Glomerulonephritis with tubulo-interstitial changes
Prospect	Development of generalized sclerosis due to involvement of glomeruli in tubulo-interstitial inflammation	Chronic renal failure

Treatment of urate nephropathy. In case of high excretion of urates in the urine (uraturia), a diet is also effective. In this case, it is forbidden to eat products containing a large amount of purine bases: brain, liver, pate, kidneys, meat stew,

nuts, cocoa, chocolate, legumes, peas (see table 55). Mainly dairy and green foods are recommended. Eggs can be eaten. Fresh fruits, cereals, rice, eggs and potatoes do not contain any purine substances at all. Fresh fruits, juices and vegetables create an alkaline environment in the urine, under such conditions urates dissolve better. On hot summer days, urine is excreted too little, a thick precipitate forms in it. Therefore, in the summer it is necessary to drink more fluids (increase the amount by 1.5 or 2 times). Considering that urates dissolve well in an alkaline environment, it is recommended to drink low-mineralized, hydrocarbonate-magnesium mineral water. The best varieties of grapes grown in Uzbekistan should be widely used in the treatment of uraturia. Because grape juice creates a more alkaline environment, 1 liter of grape juice replaces 6 grams of soda. In addition, grape juice is somewhat diuretic, rich in pectin substances, which is why it promotes bowel movements. Grape juice is 70-80% water, and contains a little vitamin C. In order for the patient to urinate more, he should eat watermelon as much as possible, because watermelon is diuretic. It is advisable to boil meat, because 50% of the purine in boiled meat goes into the soup. It is better for the patient to eat meat in the first half of the day. 1-2 lemons eaten per day will help dissolve urates (Table 27).

Table 27.

Recommended dietary foods for high urate excretion

(DIIshkabulov, SKAbdurakhmanova SK, 1997) **.**

Products name	**Food products and types of food**	
	Recommended	**Forbidden**
Meat and poultry	Boiled lean beef, chicken, boiled meat dumplings, manti.	Meat stew, brains, liver, canned goods, pork.
Fish	Boiled, lean fish. Served 1-2 times a week, in the morning.	Fatty, fried, salted fish and canned fish.
Dairy products	All types of products. Even when added to food.	_____
Egg	In any form.	_____
Oils	Butter and vegetable oils are also used in their pure form for food.	_____
Vegetables and fruits	Freshly picked: carrots, pumpkin, cucumber, apple, peach, pear, quince, cherry, melon, watermelon.	Salt is added sparingly. Nuts, beans, peas, mushrooms, green peas.

Cereal, pasta	It can be prepared in any way.	If you have a tendency to gain weight, it is better to eat less.
Soup	Dairy-free, meatless cabbage soup with potatoes and cereals, various vegetables, topped with sour cream.	Meat, fish, chicken soup, scallion soup.
Where?	Meatless, vegetable broth soup, with cream or tomato.	Meat stew, garam masala, mustard, paprika. Pickles, apples. Chocolate.
Fresh fruits, dessert	All fresh fruits can be eaten in moderation. Compote, jelly, marmalade.	_____
Drinks	Liquid tea, fruit juices, watermelon, grape juice. Alkaline mineral waters.	Cocoa, coffee, decoction of nettle, bitter tea are not allowed if there is a lot of oxalate excretion in the urine.
Bread and flour products	Wheat and rye bread.	It is better to eat less of the types prepared with added oil, eggs, and sugar.

Therefore, patients with urate nephropathy need constant monitoring, and treatment should be organized taking into account the cause of hyperuricemia: in case of hyperuricemia directly related to purine metabolism in the body, uricosuric agents (allopurinol) that limit the synthesis of uric acid are prescribed, in case of hyperuricemia caused by poor renal excretion, uricosuric agents (anturan, etamide, magurlit, bimaren, soluran), and in case of mixed causes, the combined use of the above agents is required. Allopurinol (milurit) - 4 - oxypyrazolone 3,4 - pyridimine, chemically similar to hypoxanthine, sharply limits the activity of the xanthine oxidase enzyme and ultimately reduces the formation of uric acid. The daily dose is prescribed at the rate of 5 - 10 mg / kg (up to 6 years old - 0.15 per day, after 6 years old - 0.2) for 3 - 4 weeks. To maintain the achieved result after the level of uric acid in the blood has been normalized, during the change of seasons, in case of any intercurrent diseases (diarrhea, acute respiratory viral infections), it is necessary to re-prescribe 50-75 mg of allapurinol for 1-2 weeks for maintenance purposes (VP Lebedev et al., 1978). At the same time, the appointment of vitamin E at the rate of 3 mg per kg of the child's weight and vitamin A at the rate of 1000 units per year prevents the increase in uric acid through excessive oxidation of fats and ensures the stability of cell membrane function.

Uricosuric agents (anturan, etamide) limit the reabsorption of uric acid from the renal tubules into the blood, increase its excretion in the urine, and as a result, the concentration of uric acid in the blood decreases, reducing the risk of its deposition in tissues, connective tissues, and joints. However, uric acid excreted in large quantities with urine crystallizes in the renal tubules and interstitial tissue, damaging them, and increasing the risk of stone formation. Given this situation, the patient is recommended to consume fluids in an amount that allows urine output to be at least 1.0–1.5 ml/min (or 2 liters per 1.73 m^2 of body surface area). Given the increased solubility of urate salts in an alkaline environment, watermelon, fruit juices, especially grape and its juice, are recommended. For this purpose, low-mineralized hydrocarbonate-magnesium waters are useful. Among the local cultural waters, the waters of Semashko, Zangi-ota, and Chinabod (Tashkent), Khavatog (Syr Darya), Galla-orol, Gagarin (Jizzakh), Tortkul (Karakalpakstan), Chimyon (Fergana), and Kushogoch (Samarkand) may meet these requirements, but they have not yet been studied in this sense (Ismailov ZT, et al., 1992). In the treatment of uraturia and urate nephropathy, medicinal plants with uricolytic and uricosuric properties (mountain yam, yam, mountain basil, cornflower) and preparations prepared from them should be widely used (urolesan, avisan, systenal, phytolysin). At the same time, if secondary pyelonephritis has developed, antibacterial treatment under bacteriological control is also carried out until the urine is purified. The occurrence of interstitial (abacterial) nephritis requires the use of nonsteroidal anti-inflammatory drugs (aspirin, ortofen), aminoquinoline drugs, since if the sclerotic process is not prevented, chronic renal failure will eventually occur. In particular, the autoimmune process in hyperuricemic children, the occurrence of secondary glomerulonephritis, creates a complex situation for treatment, since there are a number of measures that are usually widely used to treat glomerulonephritis, without taking into account hyperuricemia (for example, thiazide diuretics, glucocorticoids), which can lead to a further increase in the level of uric acid in the blood and, as a result, a number of dangerous complications (encephalopathy, acute renal failure due to the filling of the renal tubules with urates).

Oxalate nephropathies

There are a number of substances known to cause urolithiasis when excreted in large quantities with urine (cystine, tryptophan metabolites, calcium, etc.), which also have a negative effect on kidney tissues, causing abacterial inflammation of the connective tissue, pyelonephritis, and pathological changes in urine (hematuria, proteinuria). Often, this condition is caused by an increase in the amount of oxalates.

40% of oxalates in the body are formed from glycine, 35-44% from ascorbic acid. Purines, serine, tryptophan, tyrosine and tryptophan can also be additional sources for the formation of oxalates in the body. Therefore, there is a close relationship between the violation of the metabolism of these substances and the process of oxalate formation in the body. Hyperoxaluria can also develop due to endogenous and exogenous deficiency of vitamin $B_{6\ (pyridoxine),}$ which participates

as a coenzyme in the metabolism of these substances , and due to deficiency of pantothenic acid. Hyperoxaluria can occur alimentarily, when foods are rich in oxalates - oxalic acid, ascorbic acid. Considering that 88-98% of the oxalates absorbed from the intestines and excreted in the urine by the kidneys, it is advisable for such patients to follow a special hypooxalogenic diet. Most researchers believe that the main cause of hyperoxaluria is its biosynthesis in large quantities in the body. Recent studies have shown that phosphoethanolamine, which is formed as a result of the imperfection of cell membranes, including those of kidney tissues, and the increased oxidation of fats in their composition under various influences, is a major source of oxalate formation in the body. (Yu.E. Velti et al., 1991; EA Yureva, 1979; JE Eshkobulov, 1980).

Currently, there is also a monogenic primary hereditary hyperoxaluria, which is transmitted from generation to generation, and this disease is less common. There are two types of primary hyperoxaluria, the first of which is caused by a deficiency of the enzyme carboglycase, which is involved in the metabolism of glyoxylic acid, resulting in the excretion of oxalic, glycolic and glyoxylic acids in the urine.

In patients with hyperoxaluria, a deficiency of magnesium in biological fluids also plays a role in the formation of kidney and urinary tract stones, since oxalic acid forms water-soluble compounds with magnesium. Increased urinary calcium excretion (hypercalciuria) is observed in 44-62% of patients with kidney stones (Hallson PC et al., 1977; Nenhas T. et al., 1992).

In such patients, a violation of the reabsorption of a number of amino acids (serine, glycine, tryptophan) in the proximal renal tubules is observed. To a certain extent, the deficiency of vitamin B_6, which acts as a coenzyme in the violation of the metabolism of these amino acids, also plays a role. Hyperoxaluria underlies the diseases of oxalosis, oxalate nephritis, oxalate stone formation, and their clinical symptoms are also diverse depending on the stage of their development (Figure 14). Before the development of symptoms of oxalate nephropathy, the appearance of oxalate-calcium salt grains and erythrocytes in the urine sediment is observed . The large amount of excreted oxalate-calcium grains injure the urinary tract and cause the excretion of erythrocytes with urine.

Therefore, abacterial inflammation of the connective tissues - interstitial nephritis and microbial inflammation (pyelonephritis) also occur, since it is easier for microbes to settle in the affected tissues. All this creates conditions for the formation of stones in the kidneys and urinary tract.

For oxalate nephropathy, pathological inclusions in the urine are a characteristic feature in the early stages, which can be detected only when examining the urine sediment. This condition is often detected by chance, and is characterized by up to 0.066% (0.28 ± 0.04%) protein in the urine, hematuria (from 2-3 grains per field of view of the microscope to the level of macrohematuria, which is clearly visible to the eye) (VA Bondarenko et al., 2005). Sometimes patients with hyperoxaluria note abdominal pain, vomiting, and the release of small stones with feces. Sometimes changes in the joints,

headaches can be observed. As mentioned above, children with hyperoxaluria often have a high probability of developing secondary pyelonephritis. In such cases, sick children develop signs of general intoxication of the body (increased body temperature, malaise, pallor) and characteristic signs of pyelonephritis (lumbar pain, bacteriuria). One of the characteristic signs of secondary pyelonephritis observed in children with hyperoxaluria is that in addition to bacteriuria, the urine of such patients may contain leukocyturia, and even more pronounced hematuria, which makes the changes in the urinary sediment similar to glomerulonephritis. The presence of edema, increased blood pressure, and biochemical markers in the blood (hypoproteinemia, the number of immune cells against streptococci) characteristic of glomerulonephritis allow us to rule out glomerulonephritis.

In addition, GN mainly appears after the age of 5, while oxalate nephropathy appears in 65% of cases before the age of 5, and in 12% of patients before the age of 1. A study of the genealogy of patients with oxalate nephropathy shows that in 20-50% of cases this disease has a familial nature, that is, there are individuals in the family with one or more types of kidney diseases. The fact is that nephropathies that develop due to dysmetabolic, including hyperoxaluria, develop in several stages:

Biochemical imbalance and hyperoxaluria → hematocrit syndrome → pyelonephritis and interstitial nephritis → urolithiasis → eventually chronic renal failure may develop. Treatment with antibiotics, if metabolic measures are not taken, will not have a positive effect on renal processes and changes in urine, but on the contrary, can lead to various complications (allergies, nephrotoxic effects, dysbacteriosis).

In this case, we are talking about the primary (familial) type of oxalic acid metabolism, and the analysis of the family tree gives grounds to say that the factor causing hyperoxaluria is transmitted from generation to generation in a hereditary way, because although the family tree shows widespread, at first glance, diseases affecting various organs (hepato-biliary, gastroduodenal system, vegetative dystonia, diseases of the urinary system), they may all be based on a single factor - hyperoxalemia and hyperoxaluria. The almost identical results of the morphobioptic examination also confirm our opinion.

In this case, the development of profound morphological changes in both children, which led to chronic renal failure, was considered to be "chronic pyelonephritis" based on changes in urine. However, the main sign of pyelonephritis - bacteriuria - was never observed in pathological quantities. It should be noted that the misdiagnosis and the unjustified, prolonged use of the selected treatment regimens (mainly antibiotics) were the cause. Here, we should highlight several cases:

1. The mistake begins with not thoroughly studying the patient's family tree. Family tree analysis should be the basis for studying the patient's life and disease history.

2. Based on the analysis of the family tree and the complex of existing diseases, it is not difficult to confirm that in this case the familial nephropathy is

of a dysmetabolic nature. In addition, it is known that this complex of diseases is typical for hyperoxaluria, and this is shown by biochemical tests.

3. Due to the general practitioners' lack of knowledge of dysmetabolic and other hereditary nephropathies, they have been using antibacterial treatment for a long time (more than ten years), which is unreasonable and, in a certain sense, dangerous for patients. As a result, profound morphological changes have developed in the children's kidneys.

4. At present, every physician involved in treatment and prevention should be deeply aware of the fact that, while it is not possible to eliminate the genetic changes that cause hyperoxaluria, it is possible to prevent a number of diseases associated with this factor and ensure the effectiveness of treatment by reducing the synthesis of oxalates in the body , using measures that increase the solubility of oxalates, and prescribing a hypooxalemic diet.

Treatment of oxalate nephropathy. Diet is also a key factor in this type of kidney disease. Oxalates, which are the end product of metabolism in the body, are excreted mainly in the urine. Therefore, it is not recommended to give these patients foods containing oxalic acid.

Four groups of foods are distinguished based on the amount of oxalate they contain:

1. Products containing 1 to 10 g/kg of salicylic acid include cocoa, chocolate, beets, celery, spinach, scallions, parsley, and rhubarb.

2. Products containing 0.3 to 1.0 g/kg of salicylic acid: carrots, beets, onions, green beans, tomatoes, tea.

3. Products containing 0.05 to 0.3 g/kg of oxalic acid: freshly picked cabbage, apricots, bananas, currants, Brussels sprouts.

4. Eggplant, mushrooms, cauliflower, cucumbers, peas, and pumpkin are very low in oxalic acid. By following these restrictions, you can reduce the excretion of oxalates in the urine by 40% or more.

In some metabolic disorders, the excretion of oxalates in the urine can be achieved by changing the way glycine is formed. For this, it is necessary to introduce sodium benzoate into the body (it is abundant in cranberry juice). Glycine metabolism can also be accelerated by taking vitamins B_1 and B_6 in the morning and afternoon. In the treatment of oxaluria, it is of great importance to prevent excessive saturation of urine with calcium salts of oxalic acid. In this case, it is important to determine the amount of urine excreted during the night and day. Drinking plenty of fluids (especially in summer, during hot days), drinking mineral water (mostly in the evening) prevents microcrystallization. In addition, taking 100-300 mg of magnesium oxide per day also reduces crystallization. If urolithiasis and pyelonephritis are accompanied by oxaluria, patients are recommended to eat foods made from potatoes and cabbage, such foods do not strain the kidneys. A variety of dishes are prepared from potatoes, cabbage, and vegetables with a low oxalic acid content (except for bitter tea, broad-leaf vegetables). Vegetable oil, butter, and cream are also recommended in small quantities. Not too sweet wet fruits, fruit juices, and various liquids are appropriate, which increase the amount of urine excreted during the day and

night. According to the physiological rhythm of the body, urine thickens in the evening and at night, creating conditions for the crystallization of various salts. The table below lists the names of dishes recommended for hyperoxaluria (Table 28).

Table 28.

Recommended products for high urinary oxalate excretion

(DIIshkabulov, SKAbdurakhmanova SK, 1997) **.**

Products name	Food products and types of food	
	Recommended	Forbidden
Vegetables and cucumbers	Potatoes, eggplant, cabbage, turnips, beets, cucumbers, melons, pumpkins, vinaigrettes, watermelons.	Broccoli, spinach, rhubarb, spinach, black currants. Tomatoes can be given in small quantities.
Soups	Chicken, lean fish (with cereals, vegetables, cabbage)	Soup with spinach and chard
Sweets, wet – fruits	Sugar, honey, nectar, apple, grape, cherry, sour cherry, quince, peach, pear, compote, jelly fruit juices	Chocolate, figs
Meat and poultry meats	Lean meat, mostly boiled, lean varieties of sausage, sausages, beef. It is recommended to give it in the first half of the day.	____
Fish	Lean fish, boiled (sardine, bream, cod, carp can be cooked in any way)	Oily fish, salted, smoked, canned fish, boiled soup.
Egg	A warmed, slightly steamed egg, fried in oil. Can be used as a meal.	Raw or hard-boiled eggs.
Dairy products	Milk can be consumed as a food, and kefir and cream can be consumed on their own or in food.	Cottage cheese and cheese can be eaten in small amounts without being used as food .
Bread , butter, vegetable oil, fats	A common type of bread that is rolled up. It can be eaten on its own or used in food. Vegetable oil and fats are used in food.	____

Cereals, pasta, legumes	Various cereals, vermicelli, pasta, and soups made from buckwheat.	Legumes are rarely given.
Drinks	Liquid lemon tea, grape, apple, watermelon juices, alkaline mineral waters.	Hot tea, coffee, cocoa, milk tea.

If the above diet is followed for 2-3 weeks, the excretion of oxalates in the urine decreases by 45.5%, and at the same time, the urinalysis also improves. The key to effective treatment of oxalate nephropathy is, first of all, taking measures to normalize metabolism, and then organizing treatment taking into account the stage of development of the disease, that is, its form (the period of only biochemical changes, the period complicated by aseptic inflammation in the renal tubules and intercellular tissues, the formation of stones, etc.), the state of kidney function. For example, when metabolic disorders are detected early in a patient and pathological changes in the kidneys have not yet occurred, diet plays a key role in treatment. In hot climates, it is especially important to monitor the child's sufficient fluid intake so that urine excretion does not decrease sharply and the concentration of oxalates and other salts does not increase. In this regard, mineral waters with a slightly alkaline environment are useful to increase the solubility of oxalates. Especially, the world-famous Uzbek grapes should be widely used, because 1 liter of grape juice corresponds to 6 grams of soda (ZI Umidova, 1975).

Therefore, it changes the body's environment to a more basic one, is a diuretic, and has the property of increasing intestinal motility due to its rich content of pectin. It is also useful because it contains a small amount of vitamin C, which is a source of oxalate synthesis in the body . For this purpose, watermelon and its juice should be widely recommended.

In order to prevent crystallization in patients with hyperoxaluria, it is important to reduce the excretion of calcium in the urine, along with the above measures. For this purpose, it is recommended to limit the consumption of calcium-rich foods (milk, spinach, carrots , beans), and to reduce the absorption of calcium from the intestine, the patient is prescribed phytin (inosinhexaphosphoric acid). The use of enterosorbents in the treatment of dysmetabolic nephropathies has been shown to give positive results (EA Gordieva et al., 2005).

The essence of the step-by-step treatment of dysmetabolic nephropathies is that, for example, if a child has damage to the tubules and interstitial tissue, the above measures are not enough - in this situation, nonsteroidal anti-inflammatory drugs, aminoquinoline drugs, and if pyelonephritis is observed, short-term antibacterial therapy should be used, taking into account bacteriuria and the sensitivity of microbes. Any other complications (autoimmune process, stone formation, impaired renal function) require the introduction of appropriate additions to the treatment.

To improve the solubility of oxalate salts, it is recommended to administer 100-200 mg of magnesium oxide per day, as well as mediators that activate cellular metabolism (50-100 mg of cocarboxylase, 20-40 mg of pyridoxal phosphate, or 0.2-0.5 mg of pyridoxal phosphate per μmol of urinary oxalate) for 2-4 weeks.

Sodium benzoate reduces oxalate synthesis by altering glycine metabolism in the body. Piperazine, which forms a soluble salt with oxalates present in the body, prevents the crystallization of oxalates, reducing their harmful effects.

Associated with metabolic disorders other nephropathies

In a number of other cases, dysmetabolic nephropathies may develop, which in all cases do not have specific symptoms, but are mainly manifested by changes in the urine (proteinuria, hematuria). These changes in the urine are a sign of the presence of some kidney disease, but cannot be the basis for a nosological diagnosis, since they are observed in various kidney diseases. In all cases, the main cause of nephropathy can be determined on the basis of family history analysis and special biochemical tests. On this basis, it is possible to determine the initial, biochemical stage of metabolic disorders (i.e., before clinical symptoms appear). Later, all the stages mentioned above - oxalate, urate nephropathies (interstitial nephritis, pyelonephritis, etc.) also occur in this case. As the changes in the kidneys deepen, clinical signs of renal failure appear, initially partial, and then general.

Cystinuria is a common enzymopathy, which is second only to hemoglobinopathies in terms of prevalence (VV Dlin et al., 2005). Biochemical cystinuria is detected in 1 in 340 school-age children and in 1 in 140 preschool children (VP Lebedev, 1971). It has been determined that 4% of the population are carriers of the cystinuria gene (V. Krzhijek, 1972). Biochemically detectable cystinuria occurs in 12.9–40% of nephro-urological patients (NK Vasilenko, 1976). In healthy children, cystine clearance is 2.0–0.34 ml/min. 1.73 m^2, and daily urinary excretion is 22–52.7 mg. In cystinuria, its excretion is more than 100-120 mg, and its clearance is sharply increased. The amino acid cystine, which contains sulfur and sulfur, causes degenerative changes in the interstitial tissue of the kidney, tubules, causing interstitial nephritis, pyelonephritis, and the formation of stones in the renal tract. The younger the child, the stronger the harmful effect of cystine - pathological changes observed in the kidneys (proteinuria, hematuria) in cystinuria are manifested in 50% of cases by the age of 3. In interstitial nephritis and pyelonephritis caused by cystine, a large amount of cystine, lysine, and arginine are constantly excreted in the urine, regardless of the stage of the disease (active period, remission period). In this sense, a number of clinical signs observed during the initial period of the disease are also useful. In particular, instead of the symptoms occurring 2-3 weeks after an infectious disease, which is typical for glomerulonephritis, in this case, changes in urine are detected on the first day of the intermediate illness, the sensitivity to streptococci remains unchanged, and signs such as edema, hypertension, and oliguria are not observed, which should actually be considered important criteria

for comparative diagnosis. In the family tree of families with cystinuria, urolithiasis, interstitial nephritis, pyelonephritis, gallstones and inflammation of the bile ducts, and diseases of the gastro-duodenal system are often found. Observing hexagonal, characteristic cystine crystals in the urine of patients under a microscope does not cause any difficulties. The diagnosis of intermittent cystinuria is made by determining the amount of cystine in the blood and urine using a chromatographic method.

intake of amino acids containing sulfur and sulfur . At the same time, such a diet involves the use of alkaline treatment to dissolve cystine and drinking plenty of fluids to reduce the concentration of cystine in the urine and eliminate it from the body more quickly.

Methionine in food forms cystine in the body. Therefore, it is advisable to eat foods that are low in methionine. Salt should be used in moderation. While maintaining the ratio of essential nutrients, it is not recommended to eat fish, cheese, mushrooms, eggs, beans, peas. Because these products contain a lot of methionine. Dietary dishes made from potatoes are useful for patients who excrete cystine in their urine.

Below is a sample of food (in grams) recommended by EA Yureva for one day.

200 grams of white bread, 20 grams of butter are given per day. The above diet meals contain 2500 - 2700 calories of energy, 60 grams of protein, 0.7 - 0.3 grams of methionine. Animal proteins are consumed in the first half of the day. Taking into account the chemical properties of cystine and its poor solubility in an acidic environment, Albright's mixture, bicarbonates, alkaline mineral waters are widely used. These should be given especially in the evening, before going to bed, because during sleep the patient does not go out to urinate much, and at this time his acidic environment creates the most favorable conditions for the crystallization of cystine.

In addition, it is recommended to increase the amount of fluid consumed during the day and night to 2 liters or more, especially in the evening. Then the concentration of cystine in the urine will decrease by 1.5-2.5 times. During the period of dietary treatment, it is advisable to prescribe pyridoxine (B_6) and ascorbic acid.

A number of products formed **in the violation of tryptophan metabolism can also have a strong nephrotoxic effect (tryptophan and its derivatives - kynurenine and xanthurenic, anthranilic acids).** The three most important pathways of tryptophan metabolism in the body are known:

1. the pathway of nicotinic acid formation;
2. the serotonin pathway, which leads to the formation of serotonin, 5-oxindole acid, and melatonin;
3. In the presence of tryptamine and indole 3-acetic acid-forming pathways, 85% of tryptophan metabolism in the body occurs through the formation of kynurenine (Barashnev Yu.I., 1987).

B_6 as a coenzyme of kynureninase at several stages (in the conversion of tryptophan to anthranilic acid, oxykynurenine to oxyanthranilic acid, etc.) , and

its deficiency can lead to the accumulation of all the listed derivatives in the body and their various (including nephrotoxic) effects. Allergic diseases are common in families with hereditary disorders of tryptophan metabolism (since non-immunological allergic conditions occur due to an increase in serotonin). Under the influence of tryptophan derivatives of the indole and tryptamine categories, anemia and tumor diseases increase, and the harmful effects of anthranilic and xanthurenic acids on the nervous system cause a tendency to convulsive states. Vascular diseases and nephropathies are especially common. In the treatment of nephropathies caused by disorders of tryptophan metabolism, vitamin $_{B6}$ is used in doses ranging from 5-10 mg to several 100 mg, along with an appropriate diet - it is usually recommended to take it in the morning 30 minutes before breakfast, since pyridoxine hydrochloride is absorbed through the intestines in the form of phosphates and is thus active. For this purpose, the use of the phosphated form of vitamin B6 - pyridoxal phosphate - is more suitable. Treatment begins with the appointment of one mg of vitamin $_{B6}$ for each mg of xanthurenic acid excreted in the urine , and under clinical and biochemical control, it can sometimes be increased by 2 times, since not all patients have the same sensitivity to $_{B6}$.

A study of the state of tryptophan metabolism in the body shows the following: urinary excretion of kynurenine is 68.5 mmol/s, kynurenic acid – 46.9 mmol/s, xanthurenic acid – 57.9 mmol/s, while urinary excretion of N_1 - methylnicotinamide is reduced to 15.4 mmol/s.

CHAPTER VIII
ACUTE RENAL FAILURE

Acute renal failure is a reversible, but very short-term (several hours or days) violation of the homeostatic function of the kidneys. In acute renal failure, its excretory function is impaired, which is accompanied by the accumulation of toxic products in the blood that are usually excreted in the urine - the balance of water, electrolytes, acid-base imbalance is disturbed, hyperazotemia is observed. The causes of acute renal failure are diverse and are divided into three large groups that are important in terms of providing medical care to the patient: prerenal (before the kidney), renal (in the kidney itself) and postrenal (outside the kidney) harmful factors . Although prerenal factors are diverse (dehydration, bacteremia, kidney injury due to a stroke), they are mainly based on insufficient blood supply to the kidney (ischemia). Cases of renal failure directly related to kidney damage (renal form) include damage by heavy metal salts, manganese , certain drugs - mercury, sulfonamides, antibiotics, and blockage of the renal tubules with urate salts. Sometimes it is also observed in acute glomerulonephritis, collagenous secondary glomerulonephritis. Postrenal (postrenal) factors include causes that prevent the outflow of urine - urinary stones, tumors of the pelvis and bladder, and narrowing of the urinary tract due to inflammation.

Clinical picture. Acute renal failure occurs in 4 stages: onset, oliguria, recovery of urine output, and resolution.

The general condition of the patient depends on the duration of the disease, the cause that caused it, and the degree of uremic intoxication. The initial period is the period during which the factors that cause renal dysfunction continue to act , and often occurs in connection with various strokes, injuries, hemorrhages and hemolysis, sepsis, as a result of circulatory collapse. The patient experiences headaches, pain in the back and abdomen, palpitations, and tremors similar to a malarial attack. The initial period is usually short. This period can last from several hours to 1-2 days. For this reason, it is often overlooked. Sometimes, when the effect of the factor that caused renal dysfunction stops , the patient's condition may temporarily improve slightly. However, then renal function deteriorates sharply, urine output decreases (oliguria), and even reaches its cessation (anuria) (Figure 17). A decrease in urine output of up to 30% compared to the child's age indicates that the process has entered the oligoanuria stage. The urine excreted in small quantities is dark brown in color, has a low specific gravity, contains protein and casts.

The patient refuses food (anorexia), vomits, and has flatulence (flatulence). Toxic nitrogen-binding substances - uric acid, urea, and creatinine

- accumulate in the blood (hyperazotemia), potassium levels increase (hyperkalemia), and the clinical picture of uremic poisoning develops.

The patient becomes thirsty, due to fluid retention in the body, the patient's weight increases - the patient's body and legs swell, sometimes fluid can accumulate in the abdominal and chest cavities (ascites, hydrothorax). At this stage, neurological symptoms and headaches appear and intensify. The functioning of the gastrointestinal system is also disrupted, since the intestinal mucosa to a certain extent takes over the excretion of nitrogenous substances - vomiting and diarrhea are observed. Increased catabolism, hyperazotemia, hyperhydration and acidosis can lead to the development of coma and the death of the patient.

In the oligoanuria stage, urine output decreases for a short time (sometimes within a few hours) and drops to 50-100 ml per day. Its specific gravity drops below 1012. The patient's heartbeat accelerates, sometimes becomes irregular (extrasystole). Signs of hyperkalemia (height of the T wave, widening of the QRS complex, location of the ST segment below the midline) can be observed on the ECG. Due to edema of the brain substance, the patient may develop convulsions (eclampsia). In this regard, the existing hypocalcemia is also of some importance. A sharp increase in the amount of nitrogen-binding substances in the blood is observed (urea 10 - 20 mmol / l, creatinine up to 0.88 mmol / l, hyponatremia up to 118 mmol / l, hyperkalemia up to 7 mmol / l, hypocalcemia below 4 mmol / l and acidosis - up to pH 7.2, SB up to 8 mmol / l). The period of oligoanuria lasts up to several days (on average 3 - 5 - 7 days), and in favorable cases it gradually (over 2 - 3 weeks) passes into the period of recovery of urine output. During this period, the color of the urine becomes clear, chemical changes in the blood normalize, the patient's blood pressure decreases and the general condition improves. However, if up to 3-5 liters of urine is excreted during this period and its replacement is not completed, the patient may develop signs of dehydration (dryness of the skin and mucous membranes, thirst), signs of hypokalemia (low blood pressure, fatigue, muscle hypotension) and hyponatremia due to the large amount of potassium lost in the urine.

During the period of recovery of urine output, changes in the composition of urine (proteinuria, cylindruria) are preserved, and their duration depends on the causes that led to this condition. In particular, if acute renal failure is caused by glomerulonephritis or pyelonephritis, changes in urine also correspond to their course. The final - **terminal - period of acute renal failure** lasts 6-12 months, and kidney function is gradually restored.

Diagnosis. Early detection of acute renal failure is extremely important, as developing hyperkalemia can quickly lead to a tragedy - cardiac arrest. Monitoring urine output is important for diagnosis and allows timely detection of acute renal failure in 90% of cases. Unfortunately, a sharp decrease in urine output cannot be considered an early sign of the disease, since it often reaches its peak after 24-48 hours. Therefore, in cases of concern, it is necessary to monitor the level of potassium, urea and creatinine in the blood daily.

In order to properly organize the treatment, it is important to timely and correctly determine the cause of renal failure in each patient. Because there are specific methods of treatment for prerenal, renal and postrenal types. At this time, the history of the disease (for example, the presence of glomerulonephritis or pyelonephritis, the use of drugs that have a toxic effect on the kidneys, etc.), a general analysis of urine are important. In particular, if there are altered erythrocytes and cylinders in the urine sediment, this is more likely a characteristic sign of damage to the renal corpuscles (glomerulonephritis), while an abundance of decomposed tissues indicates the likelihood of a toxic effect on the kidneys. In acute intestinal toxicosis in children, dehydration, hypovolemia and, as a result, oligoanuria are also relatively common as a result of vomiting and diarrhea. This is especially important for hot climates.

Treatment. Treatment of acute renal failure is a complex problem and can vary depending on the cause. The general rule is: 1) to stop the action of the agent that is disrupting the kidney function; 2) to restore the balance of the body's internal environment (homeostasis); 3) to focus on the prevention and treatment of various complications.

In the early stages of renal failure, treatment should be aimed at restoring the circulating blood volume, given that its main cause is hypovolemia and impaired blood circulation in the capillary blood vessels . The volume of fluids infused is controlled by blood pressure in the central venous catheter and should not exceed 10 cm H_2O , which poses a risk of pulmonary edema. In addition to restoring blood volume, rheopoliglyukin has the property of restoring microcirculation and reducing blood clotting. This result can also be achieved by administering 10% albumin. To prevent blood clotting disorders (coagulopathy), it is necessary to start heparin administration to the patient early in the dose of 100 U/kg and monitor blood clotting. Rheopolyglukin enhances the effect of heparin. Therefore, when they are used together, it is recommended to reduce the amount of heparin by 30-50%. At the same time, measures are taken against the influencing factors: in case of mercury poisoning - unitiol, in case of an incompatible blood transfusion - exchange transfusion, etc.

Therefore, in the early stages of renal dysfunction, treatment is aimed at preventing oligoanuria. In this sense, early administration of mannitol is also important. However, if mannitol is administered uncontrolled, it can increase blood osmolality, causing fluid retention and tissue edema, since mannitol has a diuretic effect only if the osmolality of the urine is higher than that of the blood. If the patient has developed anuria, the use of mannitol poses a risk of increasing the volume of blood circulating in the blood vessels and pulmonary edema. If renal failure develops and the disease has entered the oligoanuria stage, such patients are at risk of hyperhydration, hyperkalemia, uremia, and the addition of infectious diseases. Therefore, the specific gravity of blood and urine, the content of K, Na, P, Sa, creatinine, and uric acid should be constantly monitored.

To eliminate hyperhydration, the daily amount of fluid intake should correspond to the volume of fluid excreted (urine, feces, vomit). Water balance in the body can be monitored by constantly measuring the weight of patients.

Since hyperhydration is observed in the first 2-3 days of the oligoanuria stage, a hypoosmolar state occurs in the blood, and therefore mannitol (1.0 g / kg) is prescribed as a 15-20% solution at a rate of 60-80 drops per minute. At the same time, the administration of furosemide in an amount of 5-10 mg / kg significantly increases urine output. A decrease in the amount of sodium in the blood is also a sign of hyperhydration, which requires limiting hypotonic fluids. An important condition for the use of diuretics is a systolic arterial blood pressure of more than 60 mm Hg. If blood pressure is low, albumin, rheopoliglyukin (10–15 ml/kg) or high doses of dopamine (10 μg/kg x min) are used. In smaller doses [6–9 μg/(kg x min)] dopamine has a cardiac stimulant effect, and in even smaller doses [2–5 μg/(kg x min)] it has a diuretic effect. In smaller doses [1–3 μg/(kg x min)] dopamine increases diuresis when used with furosemide (1.5 mg/kg).

Hyperkalemia observed during oligoanuria is of significant clinical importance, which requires a sharp restriction of potassium intake with food. In addition, intravenous administration of 10-20 ml of 10% calcium gluconate solution and 100-200 ml of 10% glucose is recommended. This is especially important if the potassium content in the blood exceeds 6.5 mmol/l. At this time, it is necessary to inject 3.8 ml/kg of a 3-4% solution of sodium bicarbonate. The amount of 4% bicarbonate to eliminate acidosis is calculated as follows: VE mol/l x weight, kg/3.

If it is not possible to determine the lack of bases (VE), a 4% bicarbonate solution is administered intravenously at a rate of 3 - 8 ml / kg / 24 hours. Since hyperazotemia is also observed during the oligoanuric period of the disease, protein in the patient's diet is also sharply limited, but the food should be of sufficient energy. The diet recommended by Giordano - Giovanetti meets this requirement.

In order to prevent the patient's condition from worsening due to the addition of infectious diseases, it is necessary to follow the rules of asepsis and prescribe antibiotics. Erythromycin, penicillin, chloramphenicol can be used in these cases in the usual therapeutic doses. Antibiotics that have a toxic effect on the kidneys should not be used. In the oligoanuria stage of OBE, the use of sulfonamide drugs, nitrofurans, and antibiotics belonging to the tetracycline group is absolutely impossible. Due to the change in the electrolyte ratio in OBE, the use of cardiac glycosides is also not safe. In necessary cases, it is recommended to use digoxin once, since it is excreted mainly through the intestines.

The treatment should also be specific to the cause of renal failure. For example, in the treatment of severe diseases (leukemia, myeloma) with cytostatics, mannitol and furosemide are prescribed when the tubules are clogged with urate salts, if it occurs as a result of blood transfusion - blood exchange, if urine excretion has stopped due to urolithiasis - measures should be taken to open the urinary tract.

Hemodialysis is necessary when the current treatment is ineffective. The patient is considered to be transferred to hemodialysis in the following cases:

- Hyperkalemia more than 7 mEq/L;

- an increase in the level of urea in the blood of more than 24 mmol/l and the appearance of uremic symptoms;
- active substances below 12 mEq/L – acidosis (blood pH less than 7.2);
- hyperhydration.

Hemodialysis can be used to correct electrolyte imbalances and nitrogenous waste in a short time. In acute renal failure, 3-5 hemodialysis sessions are sufficient (MS Ignatova, Yu.E. Veltishev, 1989). The period after the blood chemistry has normalized, the patient's uremic symptoms have disappeared, and urine output has been restored is considered a recovery period. The duration of this period also depends on the underlying cause and can last 6-12 months or even longer. Patients must, of course, be under dispensary supervision.

CHAPTER IX
CHRONIC RENAL FAILURE

Chronic renal failure (CRF) is a nonspecific syndrome that occurs in hereditary, congenital and acquired diseases of the kidneys due to a progressive decrease in their homeostatic function due to sclerotic changes in the renal corpuscles and tissue. More than 50 diseases are known that can lead to CRF with varying speed and duration (MS Ignatova, Yu.E. Velti et al., 1989). Over the next 10 years, the number of patients with CRF is expected to double (MS Ignatova, 2006). According to the special literature, the incidence of the final (terminal) stage of CRF is increasing in countries around the world (Papayan VA, et al., 2004; AdrissinoY et al., 2003 M. Mitsnefesetal, 2003, 2005).

According to various sources, the prevalence of SBE among children ranges from 3-6 to 50 per million children. In St. Petersburg, 69 people, and 10.5 patients with its terminal stage (Yu.A. Yermakov et al., 2004). The reasons for the increasing incidence of SBE among the population are: 1) the primary prevention of kidney diseases leading to SBE is still not perfect; 2) the insufficient effectiveness of the "etiological" and "pathogenetic" treatment of acquired kidney diseases (GN, PN) leads to their transition to a chronic course; 3) the low effectiveness of rehabilitation (renoprotective treatment) of patients with chronic kidney diseases leads to the formation of SBE in them; 4) In recent years, the increase in congenital, hereditary, and econephropathology in children is one of the important factors in the increase in SBE (MS Ignatova, 2005; NA Tomilina, BT Bibkov, 2005; AV Smirnov et al., 2004). The problem of SBE in children is associated, in particular, with congenital and hereditary nephropathies, which account for 41.4% of them (VI Naumova, AV Papayan, 1991).

If the development of SBE in chronic GN is associated with the occurrence of nephrosclerosis, then in congenital, hereditary nephropathies, renal dysplasia, the degree of nephrosclerosis is more important, but not the degree of nephrosclerosis, but the congenital incompleteness of nephrons. In such cases, SBE can develop progressively even in the absence of active acquired kidney diseases (GN, PN) (EF Barinov, ON Sulaeva, 2002). Studying the mechanisms of SBE development in various nephropathies, developing renoprotective treatments is a new problem in pediatrics and is of great importance, since most chronic kidney diseases leading to SBE begin in childhood and lead to SBE in adolescence and adulthood (VV Arkhipov, 2006; MS Ignatova, 2006; M. Sommermeyer 2005; S. Kimetal, 2000).

Over the past 10-15 years, significant pathophysiological, experimental and clinical data have been accumulated worldwide to study the mechanisms of SBE development, which has created opportunities for the use of new strategies for renoprotective treatment in chronic kidney disease (LeveyA.S., etal., 1998). In particular, the US National Kidney Foundation (National Kidney Chronic Kidney Disease Foundation (CKD) Kidney disease – CKD) concept (SBK), and recommended its criteria (Table 29).

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Criteria for chronic kidney disease (CKD)

(NKF, AV Smirnov and others, 2005).

Criteria	Content
1	Renal disease is a condition in which the morphological or functional status of the kidneys is impaired, with or without a decrease in glomerular filtration rate (GFR), and lasts for more than 3 months. This disease is manifested by either: - with pathomorphological changes in the renal system, or: - with changes in blood or urine composition, and when performing tests that show the renal system.
2	GFR < 60 ml/min/1.73 m2 for 3 months or more, regardless of other signs of kidney disease

The concept of SBC was introduced in therapeutic nephrology in 2002, and in pediatric nephrology in 2003 (VV Arkhipov, 2006; AV Smirnov et al., 2002, 2005). The criteria for determining SBC in adults and children are the same (DD Ivanov, 2006). SBC is considered in cases where the disease has lasted longer than 3 months, regardless of the main diagnosis (B . A . Fivush et al., 1988; S . P . McDonald et al ., 2004).

The course (stage) of SBC is determined by the degree of decrease in GFR. The NKF recommended the Schwartz formula for determining the ***Glomerular Filtration Rate (GFR) in children:*** **GFR (ml/min/1.73 m ^{2}) = [0.0484 x Height (cm)] : blood creatinine (mmol/l)** . If the child is over 13 years old, a coefficient of 0.0616 is used instead of 0.0484. The indicators of GFR used to determine the stages of SBC are presented in Table 3 0.

Table 30

Normative indicators of GFT in children and adolescents

(Nogg et al., 2003).

age (gender)	**GFR (ml/min/1.73 ^{m2})**
1 – week (boys and girls)	41 ± 15
2 – 8 weeks (boys and girls)	66 ± 25
More than 8 weeks (boys and girls)	96 ± 22
2 – 12 years old (boys and girls)	133 ± 27
13–21 years old (teenage boy)	140 ± 30
13 – 21 years old (girls)	126 ± 22

According to the classification proposed by the NKF (AV Papayan et al., 2004), five stages of SBK are distinguished (Table 31).

31-jadval

Stage	Description	GFT (ml/min/1.73 m^2)	Recommended activities
	Presence of dangerous goods	≥ 90	Monitoring, measures to reduce the risk of developing kidney disease
I	Kidney disease with elevated GFR	≥ 90	Diagnosis and treatment of underlying kidney disease, slowing its progression and the development of cardiovascular complications
II	Moderate decrease in GFT	60 – 89	Assessing the rate of disease progression
III	Moderate decrease in GFT	30 – 59	Identifying and treating complications
IV	Strong decline in GFT	15 – 29	Preparing for a kidney transplant
V	Kidney failure	< 15 or transfer to dialysis	Kidney transplant (in case of complications)

SCD , it is important to assess the rate of progression, that is, the rate of renal dysfunction, in order to slow down and prevent its progression. Direct and indirect methods of assessing renal function are used to assess the rate of disease progression (AV Papayan et al., 2004).

Methods for direct assessment of renal function:

1. Evaluation of GFT according to the clearance of exogenous substances (insulin, sodium paraaminohippurate, etc.);
2. Assessment of GFR based on endogenous creatinine clearance;
3. The amount of creatinine in the blood is 1/Cr.

Methods for indirect assessment of kidney function:

1. Need for alternative treatment;
2. 50% decrease in GFT;
3. Serum creatinine level is 2 times higher than normal .

Among the methods for direct assessment of renal function, for certain reasons, the determination of endogenous creatinine clearance (S_{cr}) is widely used in nephrology practice. For this purpose, the use of the Schwartz formula has a number of advantages in pediatrics (the child's age, the severity of the condition, the absence of the need to collect urine for many hours).

The importance of the level of creatinine in the blood and the 1/Cr indicator increases as renal insufficiency worsens, since in the initial period of SBE, the level of creatinine does not change - only when the GFR decreases by 50% does the level of creatinine in the blood increase by 2 times.

an independent factor of exacerbation of SBE and development of SBE , so it is necessary to constantly monitor it. Another independent factor that can aggravate and cause the development of SBE is proteinuria (nephrotoxin). Long-term observation of patients with hypertension has shown the following (Esayan AM, 2004): 1) even traces of protein or albumin in the urine are an early indicator of kidney damage; 2) periodically increasing (persistent) proteinuria is a sign of declining kidney function; 3) the level of proteinuria corresponds to the rate of loss of kidney function; 4) effective treatment that reduces proteinuria slows down the rate of loss of kidney function; 5) proteinuria is a reliable and independent indicator of cardiovascular damage in patients with SBE; 6) There is a correlation between the level of proteinuria and the mortality of patients from heart disease (ZA Smirnov et al., 2004;NA Shishkin et al., 2005; RS Parekh et al., 2002).

The following methods have been recommended for protein determination to monitor the daily excretion of protein in the urine in order to monitor the level of proteinuria (AV Papayan et al., 2004):

1. Brandberg – Robert – Stolnikov method
2. Method for determining protein in urine with sulfacylic acid.
3. Biuret method.

In cases where it is difficult to collect daily urine in an outpatient setting due to the patient's young age or severity of the condition, daily proteinuria can be determined by calculating the ratio of the protein concentration in the morning urine (U_{pr}) to the creatinine in the same urine (U_{cr}), where daily proteinuria is defined as g / ml / 1.73 m 2. Clinical observations confirm the following: 1) the method of daily proteinuria, determined as the ratio of U pr / U cr in the morning urine, is [a] simple and realistic criterion ; 2) this indicator serves as a criterion for the progression of the disease; 3) this method reflects the degree of decrease in GFT and the degree of disease progression to an even greater extent than proteinuria determined in daily urine (AM Yesayan, 2004; PragaM ., 2002).

In addition, patients should be considered for nephrogenic dyslipoproteinemia , as this condition also causes accelerated renal dysfunction. Statins are used in therapeutic nephrology. Due to their number of complications, they are currently not used in pediatric practice. For this purpose, antioxidants (tocopherol, emoxipin), semi-saturated fatty acids (lipostabil, essentiale, orsofolin) and their combination with enterosorbents are recommended in pediatrics.

Chronic renal failure is divided into total and partial types. The total form of the disease is characterized by the presence of homeostatic changes affecting all parts of the nephron. The partial form is characterized by a violation of one or more separate mechanisms of kidney homeostatic activity. This form of the disease is characteristic of a number of hereditary and congenital nephropathies.

However, the partial form of chronic renal failure can turn into a total form by the end of the disease. In the initial period of the pathological process, partial changes are limited, stable in nature (PB $_1$), as the disease progresses, renal failure takes on a multifaceted character , which is designated PB II. Compensated (PB IIa) and decompensated (PB IIb) periods of multifaceted partial disorders are distinguished. According to most experts, PB IIb corresponds to the initial stages of chronic renal failure. End-stage renal failure is defined as the complete failure of kidney function, in which the patient is doomed to die without the assistance of an artificial kidney.

There are various classifications of SBE based on generally accepted classification criteria. Some of them are based on the nature of the kidney damage (glomerules, renal tubules), the stage of the disease, the form. Others are based on the reserve capacity of the kidney (GFT, serum creatinine concentration) (Table 32).

Table 32

Classification of chronic renal failure in children (VI Naumova , 1991)

Stage and level (according to existing classifications)	Symptoms of SBE		Stages of SBE - international phrases
	Glomerulopathy – in the	Tubulointerstitium of the kidney - in sial diseases	
I. Renal tubular insufficiency	Arterial hypertension, anemia, increased blood urea nitrogen, decreased GPT, and partial tubular dysfunction. Creatinine in the blood in quantity	Osteopathy, anemia, acidosis, renal tubular dysfunction.	Renal insufficiency ; polyuria stage
II. Total renal failure (stages):			
1. Serum creatinine concentration 0.17 – 0.44 mmol/l	Hypertension, hemorrhagic syndrome, acidosis, decreased GPT, and impaired tubular function.	Osteopathy, anemia, acidosis, impaired GF and tubular function.	Renal insufficiency ; polyuria stage
2. Serum creatinine concentration 0.44 – 0.88 mmol/l	Hypertension , hemorrhagic syndrome, acidosis, decreased GFT and impaired tubular function, complications of internal organs.	Osteopathy , anemia, acidosis, impaired renal and tubular function , complications from internal organs, hemorrhagic syndrome.	Renal failure ; polyuria stage
1. Serum creatinine concentration > 0.88 mmol/l	Symptoms of uremia, regardless of the etiology of SBE.		Uremia, terminal, oligoanuric stage of SBE

Etiology. Among the nephropathies leading to chronic renal failure, renal dysplasia, tubulopathies and hereditary nephritis occupy the main place. According to most scientists, hereditary nephritis accounts for 20% of the causes of chronic renal failure. Among the acquired kidney diseases, mixed glomerulonephritis, bacterial and abacterial urointerstitial processes are considered to be the causes leading to chronic renal failure.

The following are at risk for chronic renal failure in childhood:

1. Severe congenital and hereditary nephropathies (primary hyperoxaluria, cystinuria, bilateral renal dysplasia, polycystic kidney disease, hereditary nephritis, etc.).
2. Forms of glomerulonephritis accompanied by sclerosing and fibroplastic processes.
3. Bilateral obstructive pyelonephritis.
4. Nephropathies developing after systemic connective tissue diseases (systemic lupus erythematosus, amyloidosis, etc.).

In addition to the above, the presence of people in the family who died from chronic renal failure, hypoimmune conditions accompanied by autoaggression, impaired cell membrane stability, and the occurrence of nephropathies accompanied by premature renal failure are also risk factors for the development of chronic renal failure. According to VI Naumova (1991), SBE in children develops in 41.4% of cases due to congenital and hereditary kidney diseases, in 40.5% glomerulonephritis, in 15.3% secondary pyelonephritis, and in 2.7% due to other reasons.

In nephrology, "azotemia" is defined as an increase in the level of urea, creatinine, and other nitrogenous wastes in the blood as a result of decreased filtration in the renal tubules. An increase in the level of urea in the blood is manifested by symptoms such as headache, lethargy, weakness, and muscle hypotonia. The mechanism of toxic action of creatinine has not been determined.

In addition to biochemical tests, ECG is also important in the diagnosis of hyperkalemia. In chronic renal failure, along with hyperkalemia, zinc levels are also reduced. These changes, in turn, lead to a delay in sexual development and the development of anorexia in the child.

In chronic renal failure, calcium-phosphorus imbalance is manifested in the form of hypophosphatemia and hypocalcemia. In addition, vitamin D metabolism disorders and secondary hyperparathyroidism are observed. Acidosis, which occurs as a result of metabolic disorders, is one of the main and initial signs of chronic renal failure.

Chronic renal failure is characterized by persistent hypertension, which is observed in the total form, and in contrast, in partial renal failure, it is almost not observed. Among the main causes of arterial hypertension, the vasoconstrictive effect of the renin-angiotensin system is the main one (AM Yesayan, 2002; Karabaeva AJ, et al., 2006).

Another major symptom of chronic renal failure is a child's failure to thrive and develop (VM Krans, 2007).

Clinical signs. During the development of SBE, the kidneys adapt to these conditions during the period of decreasing number of functioning nephrons. The high adaptability of the kidneys is evidenced by the following: 1) the vital activity of infants is ensured in conditions when only 40 thousand out of 2 million nephrons are functional (2%); 2) in patients with SBE, their life is preserved even when 5-10% of functional nephrons are functional (VINaumova, AV Papayan, 1991). Clinical signs of the disease can appear only after 60-75% of kidney function is impaired. The appearance of clinical signs of chronic renal failure depends on the course of the disease that caused the disease, the influence of external adverse factors, and the age of the child. Physical and mental stress inappropriate for the child's age, eating disorders, and the addition of intercurrent diseases lead to the worsening of renal failure and the appearance of clinical symptoms (Table 63).

The patient's skin becomes flaky, dry, and sometimes itchy, which are the first signs of this disease. In the terminal stage of the disease , the skin becomes yellowish, and hemorrhages appear on it. Nails become brittle and thicken at the edges, the hair changes color, and it sheds a lot. If the patient sweats a lot in the early stages of renal failure, then by the terminal stage of the disease, the sweat production of the skin decreases sharply. Along with muscle hypotonia, the patient develops bone pain, pathological fractures, bone deformation, calcific arthritis, and proximal myopathies. Arterial hypertension is accompanied by changes in the cardiovascular system. Signs of dystrophic changes and signs characteristic of electrolyte imbalance are detected on the ECG. Patients with total renal failure are at risk of developing heart failure and uremic pericarditis (OE Ilicheva, 2007; DE Weinere et al., 2005) (Table 33).

Table 33.

Syndromes of SBE, causes of their development and clinical manifestations (MS Ignatova, Yu.E. Veltishev, 1982)

Syndromes	Reasons for development	Clinical presentation
Child's growth retardation	Renal dysembryogenesis or nephrosclerosis, hormonal disorders, protein, calorie, vitamin deficiency, azotemia, acidosis.	Hypostatura, delayed development of secondary sexual characteristics, decreased height and weight.
Azotemia (uremia)	Due to a decrease in GF, nitrogenous metabolites are retained in the blood, increasing catabolism.	Asthenia, anorexia, psychoneurological disorders, gastroenterocolitis, pericarditis.

Anemia	Erythropoietins, iron, protein deficiency, osteopathies.	Pallor, weakness, dystrophic changes in internal organs, and the appearance of a systolic murmur in the heart due to anemia.
Disturbance of hydro-ionic balance	Glomerulo-tubular communication disorders, impaired electrolyte transport in the nephron, increased catabolism .	Clinical signs are associated with the observation of hyperkalemia, hypokalemia, hypocalcemia, hyponatremia, and the development of edema syndrome .
Acid -base imbalance (metabolic acidosis)	GF – that is, ammonium – a decrease in acidogenesis, a decrease in alkaline reserve.	Nausea, vomiting, shortness of breath.
Arterial hypertension	Increased renin release, on the contrary, decreased prostaglandin production and hydro-ion imbalance.	Headache, hypertensive crises, retinopathy.
Osteodystrophy	Disorders of the formation of active metabolites of vitamin D, hyperparathyroidism.	Bone pain, radiologically detectable changes in the bones (osteoporosis).
Vascular clotting syndrome	Tendency to thrombus formation, changes in the rheological state of the blood.	Hemorrhagic changes in various tissues and organs.
Immunodeficiency state	Protein deficiency, hormonal imbalance, primary and drug-induced immunological changes.	Susceptibility to bacterial and viral diseases, septic complications, and tumor processes.

Respiratory changes are manifested by symptoms such as hyperventilation, shallow breathing, wheezing, and shortness of breath. The process can become more severe and lead to uremic pulmonary edema.

Liver and biliary tract organs may experience compensatory liver enlargement, biliary dyskinesia, and progressive renal failure, leading to liver failure.

Gastrointestinal changes may occur in the form of acute dyspepsia, duodenitis, gastroenterocolitis, and may be manifested by symptoms of pseudoperitonitis. Nervous system dysfunction is accompanied by asthenia and changes in the conductivity of motor and sensory nerve fibers.

Clinical blood tests can reveal hypochromic anemia, leukocytosis, thrombocytopenia, prolonged bleeding time, and hypofibrinogenemia.

Chronic renal failure leads to increased thirst, arterial hypotension, blood coagulation disorders (thickening of the blood) during polyuria, and dehydration. During oligoanuria, general hyperhydration, hypothermia, asthenia, and hyperkalemia occur in the body. Hyperkalemia can be accompanied by paresthesia, tachycardia, arrhythmia, and sometimes cardiac arrest during systole.

Hyperkalemia is noted on the ECG as a sharp upward shift of the T wave, but sometimes hyperkalemia is suddenly replaced by hypokalemia. In this case, the patient has general hypotension, apathy, and decreased tendon reflexes, while the T wave is reduced or even negative on the ECG. Hyperkalemia, in turn, is accompanied by hypocalcemia and hypermagnesemia, which is one of the characteristic signs of chronic renal failure (Table 34).

Table 34.

SBE – description of stages and scope of main procedures

(AV Papayan, VV Arkhipov)

SBE – of stages	**Description and main treatment measures**
Stage I (Compensated)	The volume of kidney function is 80-50% of the norm, the number of functioning nephrons is 50-25%. GFT 70-50 ml/min x 1.73m 2 · Blood creatinine 0.088-0.265 mmol/l. Microhematuria. Clinical signs of SBE do not appear until the number of functioning nephrons decreases to 30%. Conservative treatment and, if necessary, surgical treatment of the underlying disease.
Stage II (Subcompensated)	Renal function is 50–25% of the norm, the number of nephrons is less than 30%. GFR 50–30 ml/min x 1.73m 2 · Creatinine 0.12–0.53 mmol/l. The tolerance to nutrients is reduced, Ca^{++} absorption is impaired. Patients are very sensitive to intercurrent diseases, dehydration , hyperkalemia, acidosis. The risk of developing OBE increases with dehydration. The patient begins to lag behind in growth. Conservative

	treatment is aimed at preserving residual renal function.
Stage III (Decompensated)	Renal function is less than 30% of the norm , the number of functioning nephrons is less than 15% of the norm. GFR 30 – 10 ml/min x 1.73m ^{2} . Creatinine 0.485 – 0.8 mmol/l. Characteristic clinical signs: osteodystrophy, anemia, hypertension. Conservative treatment is aimed at balancing metabolic changes. Patients can continue their usual lifestyle if they strictly adhere to the daily regimen, diet, and conservative treatment requirements. Preparation for dialysis treatment is necessary.
IV – stage (Uremia)	Residual renal function is less than 5% of the norm. GFR 10 ml/min x 1.73m 2 · Creatinine 0.62 – 1.1 mmol/l. Typical clinical signs: anorexia, nausea, and weight loss. If left untreated, vomiting, convulsions, coma, and gastrointestinal bleeding may develop. Heart failure and arrhythmia are often observed. Symptoms may partially disappear with strict conservative treatment. The main treatment is hemodialysis or kidney transplantation.

Currently, a new concept has been introduced into the practice of nephrology - "Chronic Kidney Disease" (Chronic Kidney In connection with the introduction of the term and classification of chronic kidney disease (CKD), the question arises of what the relationship between this classification and the current classification of SBE will be. This issue, for example, has been officially resolved in Ukraine since 2005 as follows (Table 35).

Table 35

Stages of SBK and SBE

(D.D. Ivanov, 2006)

SBK – the first stage	**SBE – the first stage**	**GFT (ml/min/1.73 m2)**	**Blood Serum creatinine (mmol/l)**	**Urine maximum density**
I	-	≥ 90	≤ 0.104	> 1,018
II	I (tubular insufficiency)	≥ 90	≤ 0.104	≤ 1.018
	I (compensation)	89 – 60	0.105 – 0.176	< 1,018

III	II (subcompensation)	59 – 30	0.177 – 0.351	<1,018
IV	III (decompensation)	29 – 15	0.352 – 0.440	
V	IV (terminal or requiring dialysis)	< 15	> 0.440	

The inclusion of the maximum relative density of urine in the above classification allows us to take into account the concentrating activity of the renal tubules. Early and accurate assessment of the stages of SBE is important for assessing the extent of conservative treatment and renoprotective therapy.

Treatment. There are two methods of treating chronic renal failure: 1) conservative treatment; 2) treatment by dialysis and kidney transplantation. This does not mean that the first method is not necessary when the second method is used, of course, in all cases both methods are used as a complement to each other. When drawing up a treatment program for a patient with chronic renal failure, two important points must be taken into account: the first is not to harm the patient. For this, it is necessary to avoid using drugs and food products that have allergic, toxic and other undesirable effects, and to prescribe a diet in accordance with the level of SBE; the second is to ensure the mental and physical hormonal development of the growing patient throughout the entire treatment. For this: 1) first of all, it is necessary to take into account the variety of causes leading to SBE, since the effectiveness of such treatment depends on the possibilities of treating the disease that caused this condition . For example, in cases of chronic pyelonephritis caused by SBE, antibacterial agents with the least nephrotoxic effect should be prescribed, taking into account renal function (Table 66).

of congenital and hereditary diseases such as renal dysplasia, oligonephronia, the possibility of etiological influence is not yet available. In this case, symptomatic treatment and diet are prescribed, taking into account the existing pathophysiological changes (anemia, dyselectrolyteemia, acidosis, etc.). On this basis, if secondary pyelonephritis develops, antibacterial treatment is carried out in the same order as above - taking into account renal function. In cases where the development of SBE is associated with glomerulonephritis, the four-component Kincoid-Smith treatment method with a modification of the initial, stage I, SBE (cytostatic drug half the usual dose, prednisolone 10-15 mg, phenylin, curantyl) may give a certain positive result (VI Naumova, 1991). In the later stages of SBE, the possibility of such treatment is often not available. In this case, treatment is aimed at moderating the pathophysiological changes present in the patient (Table 36).

Table 36.

Basics of treatment of patients in the early stages of SBE

(MS Ignatova, Yu.E. Velti sh ev , 1982)

Task	Ways to implement
Eliminate factors that exacerbate SBE .	1. Prevention of electrolyte disturbances (hyponatremia caused by diuretic therapy and a low-salt diet, hypercalcemia caused by the use of large amounts of vitamin D, etc.). 2. Control urine output. 3. Control blood pressure (hypertension). 4. Improve microcirculation. 5. Measures against the membranolytic process. Improve the function of the renal tubular epithelium . 7. Immunomodulation.
II . Prescribing medications taking into account the functional state of the kidneys.	1. Absolutely and conditionally avoid the use of drugs with nephrotoxic properties (for example: indomethacin). 2. Assign the amount of medication relative to the GFT indicator.
III. Control of syndromes present in SBE: 1. Prevention of anemia. 2. Control of arterial blood pressure. 3. Control azotemia.	1. Avoid blood loss when taking blood for analysis, and stop unnecessary tests. 2. Control of menstruation in adult girls. 1. Hypotensive treatment. 2. Control salt and fluid intake. 1. Dietary treatment. 2. Improve the function of compensatory internal organs (gastrointestinal system, skin, lungs).

4. Prevention of decompensation of acidosis.	3. Use conservative methods that reduce nitrogenous products in the blood. 1. A treatment that improves the function of the kidney tubules. 2. Reduce the intake of acidic foods.
IV . Prevention of osteopathy.	1. Limit foods rich in phosphorus, prescribe calcium and vitamin D supplements. 2. Control of acid-base balance.
V. Preparing the patient and his family for future dialysis treatment.	1. Prevention of complications that prevent dialysis. 2. Addressing psychological issues that arise in connection with the patient's transfer to hemodialysis.

Renal failure. From the early stages of the disease, the amount of electrolytes and fluids supplied to the patient's body with food should be strictly controlled. In order to stabilize the water-electrolyte balance given to patients, it is necessary to regularly monitor the amount of fluid consumed and excreted. Usually, the amount of fluid consumed should be 10-15% more than excreted. Three types of diets are recommended for patients with SBE: unrestricted protein intake - blood urea nitrogen content below 52.0 mmol/l; partially restricted (protein 1.0-1.5 g/kg/day) with a urea level of 57-71.4 mmol/l; If the urea level is more than 71.4 mmol/l, the protein content is strictly limited (up to 0.6-0.7 g/kg/day). Strict protein restriction is defined as GFR <30 ml/min/1.73 m2 , which corresponds to stage III of SBE.

Treatment of electrolyte imbalance in SBE is of great importance. Usually, in the initial stage of SBE, in stages I-II, if heart failure, edema, arterial hypertension are not observed, the amount of fluid and table salt is not limited. On the contrary, due to a decrease in the concentrating activity of the kidneys, polyuria, polydipsia are observed, hyponatremia, hypokalemia, hypocalcemia may develop. In this case, the volume of fluid consumed by the patient should be 400 ml/ml in addition to the volume of urine from the previous day. At this stage of SBE, 2-5 g of table salt per day is recommended, while controlling arterial blood pressure.

Prevention of the side effects of drugs used in nephrology practice should be aimed at preventing 2 areas: 1) their allergic and harmful effects on internal organs (digestive, nervous, hematopoietic, etc.) and 2) their direct harmful

effects on the kidneys. In stages III-IV of SBE, oliguria develops and the risk of hyperhydration increases.

In addition to dietary methods, patients are prescribed oral sorbents and enzymes, and sometimes intestinal dialysis is also used. In some cases, forced diarrhea is induced, for which the patient is given 1 liter of liquid containing 20 mEq NSO_3 - and 60 mEq / l sodium, 4 mEq potassium, 2 mEq calcium, 180 mmol mannitol for 3 hours. Usually, diarrhea begins in the patient after 45 minutes, leading to a slight decrease in nitrogen waste products in the blood. Iron preparations are of little use in the treatment of anemia in patients. Therefore, they are used only in cases of iron deficiency (hypochromic anemia). Anabolic steroids are used to improve the erythropoietic activity of the bone marrow. Treatment with blood transfusions is not only short-term, but in most cases can also exacerbate hyperkalemia. In order to restore water-electrolyte balance, the amount of fluids entering and leaving the body and the amount of electrolytes in the blood and urine are strictly controlled. In case of prolonged hyperkalemia, glycerol, laxatives, and a 10% solution of calcium gluconate, which is a potassium antagonist, are administered 5-20 ml 3 times a day. In the treatment of hypokalemia, potassium preparations are administered intravenously.

In cases of metabolic acidosis, sodium bicarbonate, alkaline mineral waters, and sodium citrate are used. Raunatin, methyldopa, clonidine, and β -blockers are prescribed for arterial hypertension. When using antihypertensive drugs, it is important to note that in cases of hypotension, glomerular filtration may decrease (Kuzmin OV, Pugachaeva MO, 2007).

In the treatment of hypertensive crises, the following drugs are prescribed sequentially: pentamine (0.2 - 0.5 ml of a 5% solution), arfonad (0.05 - 0.1% solution - 150 - 250 mg), tropafen (1 - 2 ml of a 1 - 2% solution). These drugs are administered intravenously drip with 5% glucose or saline. If the first drug does not work (within 1.5 hours), the second is used, and if the expected result is not achieved, the third is used.

For the treatment of renal osteopathy, vitamin D derivatives are recommended in combination with calcium preparations. Hypocalcemia, hyperphosphatemia, increased alkaline phosphatase activity, and radiological signs of osteoporosis are characteristic of renal osteodystrophy. The basis of drug treatment is the appointment of calcium preparations (calcium carbonate 1.0 - 1.6 g / day, calcium acetate - 0.33 - 2.0 g / day and bq) and vitamin D and its active metabolites (rocaltrol, calcitrol, oxydevit and bq). In the initial period of the disease, diet is of particular importance: products rich in calcium and limited in phosphorus are contraindicated. Phytin, aluminum hydroxide (50 - 100 mg / kg / day) are recommended to reduce phosphorus absorption in the intestine (Dlin VV, Osmanov IM, 2003).

Measures to normalize the observed changes in homeostasis should initially be aimed at eliminating hypovolemia, acidosis and membranolytic processes. When a sharp decrease in the amount of fibrinogen in the blood is observed, reopoliglyukin (10 ml / kg), curantyl (3 - 5 mg / kg) are administered

intravenously as a disaggregant. Heparin (150 - 200 B / kg), which has an anticoagulant effect, is also prescribed.

renal failure do not help, dialysis and kidney transplantation are used. Indications for switching to dialysis treatment are: an increase in the level of urea in the blood to 20 - 33 mmol / l, creatinine to 0.64 - 1.2 mmol / l, potassium to 6.5 - 7 mmol / l: a decrease in the alkaline reserve (reserve) in the blood to 15 - 12 mmol / l and a decrease in GFT to less than 5 ml / min.

In cases where chronic renal failure develops as a result of congenital or hereditary kidney diseases, the patient may be recommended to undergo a direct kidney transplant without using dialysis.

The use of antibacterial drugs in patients with chronic renal failure poses certain difficulties and is associated with a relatively high incidence of complications. The use of antibacterial drugs in such patients is required in the following cases: 1) cases where the development of the disease is directly caused by an infectious-inflammatory process (secondary pyelonephritis, calculous pyelonephritis, etc.); 2) the simultaneous occurrence of allergic and infectious causes; 3) the secondary addition of urinary tract inflammation to an existing disease; 4) the development of concomitant infectious diseases (angina, otitis media, etc.); 5) the need to treat existing concomitant diseases (tonsillitis, carious teeth, etc.), that is, they are used to treat diseases with infectious factors that are directly related to the kidneys and are not related to them. Pyelonephritis is observed in 21.9% of patients with chronic renal failure, in 10-20% of cases, glomerulonephritis coexists with pyelonephritis, and finally, in 50-70% of cases, inflammation of the urinary tract subsequently joins.

For the above reasons, patients with chronic renal failure are at increased risk of ototoxic, neurotoxic, and allergic effects of antibacterial drugs. In chronic kidney disease, renal dysfunction is exacerbated by secondary hemodynamic and metabolic changes (SA Baccolegen et al., 2000). In particular, arterial hypertension, which causes hypertension and hyperperfusion in the glomeruli, is an accelerating factor in nephrosclerosis (SV Malsev et al., 2006; MK Alchinbaeva et al., 2001).

Such factors include increased activity of the renin-angiotensin-aldosterone system. Therefore, in renal hypertension, preference is given to angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, and calcium channel blockers (P. Kincaid-Smith et al. 2002; M. Pfeffer et al., 2003) from antihypertensive drugs (Table 37).

Table 37.

Commonly used antihypertensive agents in children

(P. Kincaid – Smithetal. 2002)

Pharmacological group	**Drugs**	**Quantity**
Angiotensin converting enzyme inhibitors	Captopril (Capoten)	0.3 – 0.5 mg/kg
	Enalapril (enap, enam, renitek)	0.1 – 0.5 mg/kg

Angiotensin II receptor antagonists	Lazarus (cozaar) Irbesartan (Aprivel) Valsartan (Diovan)	Up to 50 mg/milk Up to 100 mg/milk Up to 80 mg/milk
Calcium channel blockers	Nifedipine (corinfar, cordafen, procardia) Verapamil (Kalac, Isoptin, Finoptin) Aplodipine (Norvasc)	0.25 – 2 mg/kg Up to 80 mg/milk Up to 5 mg/milk
Diuretics	Hydrochlorothiazide (hypothiazide) Furosemide (Lasix) Spironolactone (verashpirone)	1 – 4 mg/kg 0.5 – 15 mg/kg 1 – 3 mg/kg

The fluid volume for such patients is the volume of diuresis of the previous day + the volume of fluid lost through perspiration. Loss through perspiration is 1.0 ml x kg / hour in children under 5 years of age, and after 5 years of age - 0.5 ml x kg / hour (VV Dlin, IM Osmanov, 2003).

begins when the Nv level drops to 110–100 g/l . To enhance erythropoiesis, 1–3 mg of folic acid and 1–2 mg/kg/day of vitamin B6 are prescribed. Iron preparations are useful only in cases of its deficiency (hypochromic anemia). Currently, the only effective agent is recombinant erythropoietin (recormon, epomax, epletin) (AS Woetal., 2004). If the patient's Nv level is below 60 g/l, blood, packed red blood cells, and washed red blood cells should be transfused.

III-IV of SBE, if the patient lags behind in growth, recombinant growth hormone is prescribed. Hemodialysis is performed when the patient's blood urea nitrogen increases to 20-33 mmol/l, creatinine to 0.64-1.2 mmol/l, potassium to 6.5 mmol/l, and GFR drops to 5 ml/min x 1.73 m^2, since the use of only conservative methods is no longer effective.

CHAPTER X
METHODS OF RENAL REPLACEMENT TREATMENT IN CHILDREN WITH CHRONIC RENAL FAILURE. INDICATIONS AND CONTRAINDICATIONS FOR THE USE OF RENAL REPLACEMENT TREATMENT.

Hemodialysis is a procedure that allows a patient with impaired kidney function to cleanse the body of excess fluid and toxins. In acute or chronic renal failure, the kidneys are unable to remove water and protein metabolism products - urea and creatinine, maintain potassium, phosphorus, and the acid-base balance of the body. In this case, extracorporeal, that is, unnecessary metabolic products, must be removed.

When kidney function is significantly reduced, the normal process of removing excess fluid and metabolic products (urea, creatinine, etc.) from the body is disrupted. The process of regulating blood cells (red blood cells) and blood pressure is also disrupted. Therefore, patients with chronic renal failure may experience the following symptoms:

•increased fatigue,
•nausea,
•loss of appetite,
•hiccup,
•sleep disturbance,
•dryness and itching of the skin,
• decreased muscle mass,
•muscle spasms at night,
•spontaneous hematomas
•swelling of the feet and face
•anemia (decreased hemoglobin level)
•panting
•increased or decreased blood pressure

However, these signs do not allow us to accurately understand the level of kidney function. To find out, the method of determining the glomerular filtration rate (GFR) is used. This indicator reflects the volume of primary urine that the kidney glomeruli filter from the blood at a time. Various calculators and formulas for determining GFR are widely used in practice.

The following classification is used to determine the stages of chronic kidney disease (CKD):

The development of renal failure (stage 5 of CKD) means that kidney function has almost completely disappeared and the patient's condition may worsen if alternative methods of performing their functions are not used. In this case, only alternative methods of performing kidney functions can help.

The timing of the start of planned renal replacement therapy (RRT) depends on the CFT and other parameters: the patient's general condition, the presence of additional diseases, and the reduction in muscle mass (nutritive status).

If chronic kidney disease is slowly progressing, treatment options should be discussed as early as stage 4 of CKD, when kidney function continues to decline, but there is still time to choose a method and prepare for the initiation of CKD.

Table 38.

Indications and contraindications for the use of generally accepted methods of BOD in renal failure.

(Order of the Minister of Health of the Republic of Uzbekistan No. 671. 2018)

BOD methods	Instructions	Contraindications
Hemodialysis	Renal failure (acute or chronic) is a condition that manifests itself in one of the following ways and cannot be corrected by simple methods: Fluid overload (including refractory heart failure) •Hyperkalemia •Hypercalcemia •Metabolic acidosis •pericarditis • **Symptoms of uremia:** •CFR < 10 ml/min/1.73 m2 body surface area (non-diabetic chronic renal failure) • CFR < 15 ml/min/1.73 m2 body surface area (chronic renal failure in the setting of diabetes, diabetes mellitus) •Some poisonings	Difficulty communicating with the patient or hemodynamically unstable patient.
Peritoneal dialysis	The indications are the same as those for hemodialysis, except for poisoning.	**Absolute:** Loss of normal peritoneal function Scarring of the abdominal wall that restricts the circulation of dialysate. Abdominal injuries. hiatal hernias Abdominal fistulas. Abdominal wall defects (e.g., non-reducible hiatal hernias, diaphragmatic hernias, etc.) Abdominal fistulas. The patient's condition does not allow dialysis. **Relative:**

		•Intra-abdominal infection •Recurrent diverticulitis •Unable to infuse large volumes of dialysate •Inflammatory bowel diseases Ischemic colitis •Morbid obesity •Peritoneal fluid leakage •Extreme thinness
Hemoperfusion	Poisoning or toxic effects (e.g. barbiturates, many antidepressants, ethchlorvynol, meprobamate, paraquat, glutethimide, metals – lithium and barium, toxic doses of aminoglycosides or cardiovascular drugs).	Difficulty communicating with the patient or hemodynamically unstable patient.

Complications of renal replacement therapy in children with chronic renal failure.

The average life expectancy of patients on the hemodialysis program is more than 10 - 15 years. There are cases when patients have lived for more than 20 years. In any case, hemodialysis is a serious procedure, which entails the development of a number of complications. All of them are conditionally divided into early and late. The first are associated with the hemodialysis procedure itself. The second group of complications is the result of chronic renal failure. Also, the last category includes complications that occur after several years of treatment.

Early complications:

Disequilibrium syndrome is characterized by loss of orientation to the environment and inability to maintain an upright posture. Disequilibrium syndrome occurs during dialysis treatment and the onset of severe uremia. It is based on brain edema and the difference between the osmolality of cerebrospinal fluid and blood. It is initially accompanied by nausea, vomiting, agitation, and later loss of consciousness and seizures. In most cases, this syndrome disappears after drug treatment.

Low blood pressure, or hypotension. This occurs in one in three patients during the first week of the procedure. This is caused by decreased blood flow due to the rapid removal of fluid from the blood, which leads to a drop in blood pressure.

A decrease in blood pressure may be due to insufficient vasoconstriction (overheating of the dialysis solution, nutrition - stagnation of blood in the internal organs, tissue ischemia, for example, with diabetic neuropathy).

Also, a decrease in blood pressure can be associated with diastolic myocardial dysfunction of heart failure due to left ventricular hypertrophy, coronary artery disease, etc. A decrease in myocardial contractility can be due to the patient's age, hypertension, atherosclerosis, myocardial calcification, valve damage, amyloidosis, etc. Rare causes of hypotension: cardiac tamponade, myocardial infarction, occult bleeding, septicemia, arrhythmia, allergy to dialysis solution, hemolysis, air embolism.

Fever and chills. Bacterial infections can occur in dialysis patients, which are more common, develop more quickly, and resolve more slowly.

Bacterial infections can be associated with vascular access. In 50-80% of cases, the source of bacteremia is a temporary vascular access infection (during catheter use). Permanent vascular access infections can occur.

Fever can also be caused by pyrogenic reactions.

Neurological disorders: loss of balance, dizziness, nausea and vomiting. Occurs as a result of changes in blood pressure.

Syndrome of water-electrolyte imbalance: weakness, headache, nausea, seizures.

Allergic reactions to dialysates.

Development of acute hemolysis and anemia.

Late complications:

Itching is common in patients undergoing hemodialysis (HD). Accumulation of uremic toxins in the blood, contact with synthetic materials during the HD process, excessive use of medications, skin changes, susceptibility to infections, and frequent mental disorders all contribute to the development of uremic itching.

Uremic pruritus occurs in 50–90% of patients on dialysis and peritoneal dialysis. In 25–33%, pruritus appears before the start of dialysis treatment, and in the remainder, it appears during dialysis, usually 6 months after its start. Most investigators have not reported an increase in the frequency or severity of pruritus during long-term treatment with dialysis, but there is evidence of an effect of treatment duration. Itching is somewhat less common in patients on peritoneal dialysis.

Uremic itching can be periodic and constant, local and generalized. Its intensity varies from occasional discomfort during the day and night to severe. 25-50% of patients complain of generalized itching, the rest are observed mainly in the lumbar and shoulder areas, on the forearms. A certain cyclicity of itching intensity during the GD session , decreasing the next day and increasing during the two-day break between GD sessions, was found.

In some patients (25%) itching is observed only during or immediately after a GD session, and in 42% of patients itching reaches its maximum intensity at this time. Rest, heat, dry skin, sweating, reduced activity, sleep, hot or cold showers, and cold can increase the intensity of itching.

There are many reasons why itching develops. As a result, it is often impossible to determine which factor or group of factors may have triggered it. These may include:

1.Hyperphosphatemia
2. Allergic response to dialysate
3. Uremic (mixed) polyneuropathy
4. Secondary hyperparathyroidism
5. Drug allergy (heparin)
6. Cholestatic chronic hepatitis
7.Skin diseases

The basis of the treatment of skin itching is modeling an adequate dialysis program, strict adherence to a hypophosphatemic diet, and prevention of disorders of calcium-phosphorus metabolism and drug allergies.

SAMPLE TESTS

1. A 4-year-old child is discharged from the nephrology department. The period of follow-up observation for a child with chronic pyelonephritis is:
   2 years of stable remission
   3 years of stable remission
   6 years of stable remission
   * 5 years of stable remission
2. A 4-year-old child is discharged from the nephrology department. Specify the period of follow-up observation for a child who has had acute glomerulonephritis:
   2 years
   4 years
   3 years
   * 5 years
3. Which of the following symptoms is characteristic of pyelonephritis:
   intoxication
   leukocyturia
   pathological bacteriuria
   * all of the above
4. In primary pyelonephritis, continuous antibacterial therapy after normalization of urine analysis is carried out for:
   1.5–3 months
   3–6 months
   1 year
   * 14 days–1 month
5. What is the doctor's tactic when leukocyturia is detected in girls:
   prescribing antibacterial therapy
   cystoscopy
   excretory urography
   * examination for helminths, exclusion of vulvovaginitis
6. Which antibacterial therapy should be preferred in the treatment of the active phase of acute and chronic pyelonephritis:
   antibiotic monotherapy
   phytotherapy
   physiotherapy
   * combined therapy (antibiotic and uroseptic)
7. Secondary pyelonephritis in children most often occurs against the background of:
   glomerulonephritis
   systemic lupus erythematosus
   tonsillitis
   * urinary tract anomalies
8. Pyelonephritis is most often caused by:
   Klebsiella

streptococcus
Proteus
* Escherichia coli

9. What is the characteristic feature of lower back pain in pyelonephritis?
diffuse
bilateral
paroxysmal
* unilateral

10. Which syndrome is decisive for the diagnosis of pyelonephritis:
intoxication
pain
dysuric
* urinary

11. What level of proteinuria is characteristic of pyelonephritis?
up to 2 g/L
up to 3 g/day
up to 1 g/day
* up to 1 g/L

12. A diagnostic criterion of pyelonephritis in a general urine test is:
crystalluria
hematuria
cylindruria
* leukocyturia

13. What microbial count (number of bacteria in 1 ml of urine) is a criterion for pyelonephritis?
5,000,000 or more
50,000 or more
1,000 or more
* 1,000,000 or more

14. On kidney ultrasound in children with primary pyelonephritis, the most common finding is:
reduced kidney size
presence of salt crystals
coarseness of kidney structure
* presence of pyelectasia

15. Which study is the most informative for diagnosing urinary tract anomalies?
ultrasound
cystography
thermography
* excretory urography

16. Which of the following factors is most likely to cause hematuria in secondary pyelonephritis?
increased permeability of glomerular capillaries
rupture of glomerular capillaries

renal intravascular coagulation
* damage to urinary tract mucosa by a stone

17. Antibacterial therapy for pyelonephritis is carried out:
14–21 days
for 6 months
until partial clinical-laboratory remission
* until complete clinical-laboratory remission

18. Which of the listed antibiotics is advisable to prescribe to a child at the onset of pyelonephritis treatment?
Kefzol
benzylpenicillin
erythromycin
* Augmentin

19. What is the duration of follow-up observation for children who have had pyelonephritis?
2 years
4 years
5 years
* 3 years

20. Which of the listed physiotherapeutic methods is indicated for the treatment of pyelonephritis?
ozokerite applications on the suprapubic area
electrophoresis with calcium chloride and vitamin C on the lumbar region
electrophoresis with novocaine on the lumbar region
* electrophoresis with furadonin on the lumbar region

21. In the clinical picture of acute pyelonephritis in young children, the dominant syndrome is:
dysuric disorders
pain syndrome
dysuric and pain syndrome
* intoxication syndrome

22. In the treatment of pyelonephritis, reserve antibiotics include:
Ampiox
2nd-generation cephalosporins
3rd-generation cephalosporins
* aminoglycosides

23. The zigzag diet in children who have had acute pyelonephritis is:
alternating non-protein and protein products
alternating diet No. 5 and diet No. 15
alternating fasting and full nutrition
* alternating foods that acidify or alkalize the urine

24. Latent course of chronic pyelonephritis is characterized by:
presence of only intoxication syndrome
presence of only pain syndrome

presence of pain and intoxication syndromes
* presence of only urinary syndrome

25. In children of the first year of life with pyelonephritis, intoxication symptoms are combined with dysfunction of the:
cardiovascular system
endocrine system
nervous system
* digestive tract

26. Chronic pyelonephritis is diagnosed when signs of pyelonephritis are observed in a child for more than:
3 months
6 months
18 months
* 12 months

27. How are aminoglycosides (gentamicin) dosed in the therapy of pyelonephritis in children?
10–20 mg/kg/day
5–10 mg/kg/day
50–100 mg/kg/day
* 4–8 mg/kg/day

28. Which of the listed diseases from the group of tubulopathies does NOT belong to rickets-like disorders:
* renal glucosuria
phosphate diabetes
de Toni–Debré–Fanconi disease
hypophosphatasia

29. Which of the following statements regarding urate nephropathies is incorrect:
more common in school-aged children, especially urban
most common clinical manifestations: dysuria, abdominal syndrome, minimal urinary syndrome, arthralgia
* based on increased synthesis and renal excretion of oxalic acid salts
pronounced uraturia (1.5–2 times higher than normal)

30. Which dietary recommendation is incorrect for urate nephropathies:
increased fluid intake
* fluid restriction
restriction of meat products
exclusion of canned foods (fish and meat)

31. Which principle of diet therapy is incorrect in hyperoxaluria:
exclusion of calcium-rich foods (cheese)
* restriction of fluids
restriction of foods that form oxalates (sorrel, spinach, citrus, chocolate, black currants)
adequate fluid intake

32.Which of the following is NOT a criterion of lower urinary tract infection:
mild leukocyturia
rapid normalization of urine tests (within 3–4 days)
* impaired kidney function
active leukocytes in urine
33.Which of the following is NOT characteristic of clinical signs of nephrolithiasis in children:
periodic abdominal pain
hematuria
dysuria
* hepato-splenic syndrome
34.Which of the following refers to anomalies of kidney position and shape:
multicystic kidney
* kidney dystopia, horseshoe kidney
duplication of kidneys, accessory kidney
all incorrect
35.What belongs to kidney structure anomalies:
* dysplasia, polycystosis, multicystic kidney
kidney dystopia
horseshoe kidney
duplication of kidneys
36.Foods richest in oxalates:
* vegetables, strong tea, carrots, tomatoes, chicory, onions, sorrel
sardines in oil, liver, sprats, kidneys, nuts, mushrooms
milk, cheese, egg yolk, salmon roe
raisins, dried apricots, baked potatoes, prunes
37.Foods richest in purines:
leafy vegetables, strong tea, carrots, chicory, onions
* sardines in oil, liver, sprats, kidneys, nuts, mushrooms
milk, cheese, egg yolk, salmon roe
raisins, baked potatoes, dried apricots, prunes
38.Foods richest in calcium:
sardines in oil, liver, sprats, kidneys
* milk, cheese, egg yolk, salmon roe
raisins, baked potatoes, dried apricots
prunes, nuts, mushrooms
39.Foods richest in potassium:
leafy vegetables, strong tea, carrots, chicory, onions
milk, cheese, egg yolk, salmon roe
* raisins, baked potatoes, dried apricots, prunes
cabbage, watermelon, eggplant, bell pepper
40.Which of the following is NOT correct regarding normal kidney size in children:
kidney length corresponds to the height of four lumbar vertebral bodies
difference in length between right and left kidney does not exceed 1

cm

kidney width is about 50% of its length

* kidney length exceeds the height of four lumbar vertebral bodies

41. For which kidney pathology is ultrasound insufficiently informative:

Wilms tumor

* acute renal failure in acute tubular necrosis

hydronephrotic transformation of kidneys

pelvic dystopia of kidney

42. What is the main mechanism of action of saluretics:

increase excretion of sodium ions, decrease excretion of potassium ions, show weak diuretic effect

* increase excretion of sodium and potassium ions, show a sufficiently strong diuretic effect

increase plasma osmotic pressure, reduce water reabsorption, increase sodium excretion and water diuresis

increase excretion of sodium and potassium ions, show insufficiently strong diuretic effect

43. What is the main mechanism of action of potassium-sparing diuretics:

* increase sodium excretion, decrease potassium excretion, show weak diuretic effect

increase sodium and potassium excretion, show strong diuretic effect

increase plasma osmotic pressure, decrease water reabsorption, increase sodium excretion and water diuresis

increase sodium and potassium excretion, show insufficiently strong diuretic effect

44. What is the main mechanism of action of osmotic diuretics:

increase sodium excretion, decrease potassium excretion, weak diuretic effect

increase sodium and potassium excretion, strong diuretic effect

* increase plasma osmotic pressure, reduce water reabsorption, increase sodium excretion and water diuresis

increase sodium and potassium excretion, weak diuretic effect

45. Which degree of bacteriuria is pathological for Pseudomonas aeruginosa:

$1:10^3$ in 1 ml

$1:10^4$ in 1 ml

$1:10^5$ in 1 ml

* any amount

46. In daily urine of a healthy child, protein content usually does not exceed:

* 60–80 mg

100–120 mg

150–200 mg

1 g

47. Which casts (single in the specimen) may be found in the urine of healthy children:

granular

* hyaline
waxy
erythrocytic

48. Excretory urography allows assessment of (specify the most complete answer):
anatomical condition of the urinary tract and urodynamics
condition of the calyceal-pelvic system
functional capacity of the urinary tract
* all of the above

49. The structural and functional unit of the kidney is:
juxtaglomerular apparatus (JGA)
- Nephron.

Proximal tubule.
Distal tubule.

50. The endocrine apparatus of the kidneys is:
system of tubules.
- juxtaglomerular apparatus (JGA).

renal glomerulus.
glomerular basement membrane.

51. Choose the incorrect statement about kidney function:
Participate in maintaining homeostasis.
Participate in regulation of arterial blood pressure (endocrine function).
Perform excretory (nitrogen-excreting) function.
- Participate in protein synthesis.

52. The first stage of active detection of children with kidney and urinary tract diseases includes:
Radiological examination of kidneys and urinary tract.
- Routine urine tests twice a year, after intercurrent diseases in children from families with nephropathy in the family history, kidney ultrasound once a year.

Study of renal functional capacity.
Kidney ultrasound once a year.

53. Glomerulonephritis is called primary if it develops:
Against diffuse connective tissue diseases.
Against chronic hepatitis C.
Against systemic vasculitis, renal tissue dysplasia.
- After ARVI, tonsillitis, childhood infections, preventive vaccinations.

54. Which of the following nosological forms is NOT included in the classification of acute primary glomerulonephritis (AGN):
AGN with nephrotic syndrome.
AGN with isolated urinary syndrome.
- Interstitial nephritis.

AGN with nephrotic syndrome, hematuria and hypertension.

55. Which of the following nosological forms is NOT included in the classification of chronic glomerulonephritis (CGN):
CGN, nephrotic form.
CGN, mixed form.
- CGN, nephritic syndrome.
CGN, isolated urinary syndrome.

56. Which period is NOT characteristic of acute glomerulonephritis:
Period of initial (manifest) symptoms.
Period of recovery.
- Period of partial remission.
Transition to chronic glomerulonephritis.

57. In what time frame, with persistent clinical and laboratory remission of glomerulonephritis, can clinical recovery be considered:
2 years
3 years
4 years
- 5 years

58. In which renal pathology may jaundice occur:
In pyelonephritis.
- In hemolytic-uremic syndrome (HUS).
In dysmetabolic nephropathy.
In Alport syndrome.

59. Which investigation evaluates the concentrating function of the kidneys:
Endogenous creatinine clearance.
- Zimnitsky test.
Blood ionogram.
Nechiporenko test.

60. For which urological disease is hematuria NOT characteristic:
Urolithiasis.
Hydronephrosis.
Renal tumor.
- Ureter duplication.

61. What is the normal endogenous creatinine clearance (ml/min) in children over 1 year:
50–70
- 80–100
90–150
200–220

62. Which blood parameter is most informative for evaluating renal nitrogen-excreting function:
Residual nitrogen.
- Creatinine.
Urea.
Protein.

63. Which cause does NOT lead to non-renal elevation of blood urea:
Protein breakdown in the body.
Severe liver diseases.
Excess protein intake.
- Excess fat intake.

64. For which condition is acute urinary retention NOT characteristic:
Phimosis.
Lower urinary tract stones.
- Acute renal failure.
Urethral trauma.

65. Which condition does NOT cause anuria:
Acute renal failure.
- Lower urinary tract stones.
Acute blood loss.
Hemolytic-uremic syndrome.

66. Most common indication for micturating cystography:
- Persistent leukocyturia.
Persistent abdominal pain.
Suspected renal artery anomalies.
Persistent lumbar pain.

67. For which renal disease is arterial hypertension NOT characteristic:
Acute glomerulonephritis with nephritic syndrome.
Renal artery anomalies.
- Hereditary phosphate diabetes.
Chronic renal failure.

68. In which condition is differential diagnosis NOT required for retroperitoneal mass:
Kidney tumor.
Hydronephrosis.
- Hereditary nephritis.
Polycystic kidney disease.

69. Which statement is least accurate for acute post-streptococcal glomerulonephritis:
Symptoms appear 6–21 days after infection.
Usually acute onset.
Urinary syndrome is mainly hematuric.
- Girls are affected more often than boys.

70. Which complication is NOT typical for acute glomerulonephritis with nephritic syndrome:
Acute renal failure.
- Optic nerve atrophy.
Brain hemorrhage.
Acute renal failure.

71. For which complication of acute glomerulonephritis is convulsive syndrome characteristic:
Acute heart failure.
- Angiospastic encephalopathy.

Acute renal failure.
All incorrect.

72. Which clinical sign is NOT characteristic of nephrotic syndrome:
- Arterial hypertension.

Edema and oliguria.
Hypoproteinemia.
Massive proteinuria.

73. Which statement about chronic glomerulonephritis is incorrect:
Develops after unfavorable acute GN.
Slowly progressive course with nephrosclerosis.
- Complete recovery.

A major cause of chronic renal failure.

74. Which is NOT characteristic of nephrotic form of chronic glomerulonephritis:
- Arterial hypertension.

Massive proteinuria, hypoproteinemia.
Hypercholesterolemia.
Dysproteinemia (alpha-2 globulin fraction).

75. Which is NOT characteristic of hematuric form of chronic glomerulonephritis:
Hematuria.
- Severe edema.

Long-term preserved renal function.
Hypertension during exacerbation.

76. Which is NOT a contraindication for prednisolone in nephrotic syndrome:
Peptic ulcer disease.
Duodenal ulcer.
Chronic renal failure.
- Cholecystitis.

77. Which diuretic is NOT used for renal edema:
Lasix.
Uregit.
Veroshpiron.
- Diacarb.

78. Which drug is NOT a microcirculation enhancer:
Heparin.
Curantil.
Nicotinamide.
- Vikasol.

79. Plants with hemostatic effect used in hematuria:

- Black chokeberry, nettle leaves, blackcurrant leaves.
  Parsley leaves and root, horsetail.
  Dandelion.
  Elderflower, coltsfoot, plantain.

80. Plants with diuretic effect:

- Parsley leaves and root, horsetail, strawberry leaves/flowers.
  Elderflower, coltsfoot, plantain.
  Bearberry, kidney tea.
  St. John's wort, wormwood, yarrow.

81. What is NOT used for osteoporosis prevention during long-term prednisolone therapy:

Vitamin D preparations.
Omega-3 fatty acids.
Calcium-containing foods.

- Citrate mixture.

82. Which diseases do NOT lead to chronic renal failure:

- Interstitial nephritis.
  Chronic pyelonephritis.
  Chronic glomerulonephritis.
  Congenital and hereditary kidney diseases.

83. In steroid-dependent nephrotic form of CGN, together with prednisolone is prescribed:

NSAIDs.
Gold preparations.
Anticoagulants.

- Cytostatics.

84. Which laboratory marker is NOT characteristic of uncomplicated glomerulonephritis:

ESR increase.
Dysproteinemia.

- Hyperbilirubinemia.
  Urinary syndrome.

85. Which is NOT a side effect of prednisolone in maximal doses:

Hypertension.

- Hypoglycemia.
  Exogenous hypercorticism.
  GI ulceration.

86. Which syndrome is NOT typical for acute GN with nephritic syndrome:

Urinary.
Hypertensive.
Edematous.

- Hemorrhagic.

87. Which is NOT characteristic of interstitial nephritis:

Toxic-allergic kidney damage.
Isolated urinary syndrome with tubular dysfunction.

- Hereditary nature.
  Diet, phytotherapy, membrane protectors used.

88. Foods NOT recommended in hyperoxaluria:
  Apples, grapes, pears, apricots.
  Potatoes, cabbage, cucumbers.
  Buckwheat, oats, wheat.
- Sorrel, spinach, legumes, cheese.

89. Hypoisosthenuria is characterized by:
- Marked decrease in urine specific gravity below plasma level with reduced daily fluctuations.
  Increased density (1020–1035) with fluctuations.
  Decreased density equal to plasma (1008–1010).
  Marked decrease below plasma.

90. Hypersthenuria is characterized by:
- Increased urine specific gravity (1030–1035) without significant fluctuations.
  Decreased density.
  Marked decrease below plasma.
  Fluctuating density.

91. Isosthenuria is characterized by:
- Loss of concentrating ability with urine specific gravity equal to plasma (1008–1010).
  High density without fluctuations.
  Marked decrease below plasma.
  Narrow daily fluctuations.

92. Which study reflects glomerular filtration:
  Zimnitsky test.
  Blood proteinogram.
- Endogenous creatinine clearance.
  Blood nitrogen waste levels.

93. Hematuria is NOT characteristic of which syndrome of acute GN:
  Nephritic syndrome.
  Isolated urinary syndrome.
- Nephrotic syndrome.
  All correct.
  All incorrect.

94. Which method is NOT anatomical-functional kidney study:
  Excretory urography.
  Radioisotope scanning.
- Micturating cystography.
  MRI of kidneys.

95. Leukocyturia is NOT characteristic of:
  Cystitis.
  Urethritis.
  Pyelonephritis.

- Polycystic kidney disease.

96. Most common infectious factors causing acute GN:
- Respiratory viral infections.

Parasitic infections.
Fungal infections.
Chlamydia.

97. Most common infectious factors causing acute GN:

Pneumocystis.
- Activation of streptococcal infection foci.

Parasitic infections.
Chlamydia.

98. Main pathogenetic mechanism of acquired glomerulonephritis:

Direct bacterial/viral damage.
Intoxication process.
Genetic factors.
- Immune complex reactions.

99. In which syndrome of acute GN are edema and hypertension NOT observed:

Nephritic syndrome.
Nephrotic syndrome.
- Isolated urinary syndrome.

Nephrotic syndrome with hematuria and hypertension.

100. What is NOT restricted in diet during acute GN:

Salt.
Fluid.
Protein.
- Carbohydrates.

101. Symptomatic therapy of acute GN does NOT include:

Diuretics.
Antihypertensives.
Antihistamines.
- Cytostatics.

102. Duration of follow-up in acute GN:

1 year
4 years
3 years
- 5 years

103. Daily proteinuria characteristic of nephrotic syndrome:

0.033 g/L
Up to 1 g/day
Up to 2 g/day
- More than 2.5–3.0 g/day

104. Main routes of infection in pediatric pyelonephritis:
- Ascending and hematogenous.

Contact.

Lymphogenous.
Vertical.

105. Which foods are excluded from diet 7a:
Rice or buckwheat porridge.
Jam and sugar.
Vegetable puree, soup, eggs.
- Meat and fish.

106. Features of diet 7a:
- Salt-free, fluid restriction in edema, protein restriction to 50–55% of age norm.

Salt-free with proteins and carbohydrates introduced.
Salt-free, protein restricted to 75%.
Salt-free, protein restricted to 25%.

107. Diet sequence in severe extrarenal GN symptoms:
- Diet 7a → 7.

Diet 5 → 15.
Diet 7 → 5.
Diet 1 → 5.

108. After 6 months of remission exclude:
- Meat, fish, mushroom broths, smoked and salted foods, allergens.

Milk, cheese, eggs, boiled meat.
Vegetable soups, fruits, boiled meat.
Fruits, vegetables, spices, boiled fish.

109. Diet in acute renal failure requires:
Sufficient energy, optimal protein.
- High energy, low protein.

Low energy, low carbohydrates.
High energy, high carbohydrates.

110. Basic therapy of nephrotic syndrome in acute GN:
Curantil.
Penicillin.
Ascorutin.
- Prednisolone.

111. Most important etiological factor of GN:
Staphylococcus.
E. coli.
Hepatitis A virus.
- Streptococcus.

112. Characteristic pathogenetic mechanism of GN:
Bacterial pelvic infection.
Immediate allergic reaction.
Vesicoureteral reflux.
- Immune complex glomerular injury.

SAMPLE SITUATIONAL TASKS

1. Your conclusion regarding the general urinalysis of Lena K., 6 years old: reaction – 4.3, volume – 35 ml, specific gravity – 1.008, color – yellow, clarity – cloudy, protein – 0.99 g/L, glucose (–), acetone (–), epithelial cells: polymorphic 2–3 per hpf, leukocytes – covering the entire field of view, "active" 80%, erythrocytes – fresh 2–3–4 per hpf, casts: hyaline 2–3 per hpf; leukocytic 2–4 per hpf, mucus: (++++) , bacteria: (++):
Urinary tract infection: urethritis or cystitis;
*Pyelonephritis;
Glomerulonephritis;
Normal urinalysis.

2. Your conclusion regarding the general urinalysis of Regina V., 6 years old: reaction – 6.3, volume – 70 ml, specific gravity – 1.018, color – yellow, clarity (+), protein (–), glucose (–), acetone (–), epithelial cells: squamous 1–2 per hpf, leukocytes 6–8 per hpf, erythrocytes 0–1 per hpf, casts (–), mucus (+), bacteria (–):
Urinary tract infection: urethritis or cystitis;
Pyelonephritis;
Glomerulonephritis;
*Normal urinalysis.

3. A 6-year-old girl presented with complaints of poor appetite, lethargy, and non-localized abdominal pain. The abdomen is soft and non-tender. Urination is not difficult, up to 12 times per day.
Blood test: Hb – 124 g/L, leukocytes – 6.3×10^9/L, ESR – 8 mm/h.
Urinalysis: protein – 0.07 g/L, leukocytes – 8–12 per hpf, salts – abundant urates.
Which additional examination is necessary?
Bacteriological urine culture
Zimnitsky test
Nechiporenko test
*Urine test for daily urinary uric acid excretion.

4. A 5-year-old boy is receiving inpatient treatment for acute pyelonephritis. After therapy, his condition normalized.
Urinalysis: specific gravity – 1012, reaction – acidic, clarity – slightly cloudy, protein and glucose absent.
Microscopy: epithelial cells – occasional per hpf, leukocytes – 2–4 per hpf,

erythrocytes – 1–2 per hpf, hyaline casts – 0–1 per hpf, mucus – slight.
Which test should be performed?
Bacteriological urine culture
Zimnitsky test
*Nechiporenko test
Urine test for daily urinary uric acid excretion.

5. A mother of a child 10 days after birth consulted the pediatrician complaining of frequent urination (20–22 times/day). Using a rubber ring and a plate, it was determined that the infant voids about 30 ml per urination. These findings indicate:
Polyuria
Pollakiuria
*Physiological parameters of a newborn
Slightly increased urination due to improper drinking regimen

6. The physician received the following urine culture for patient L., 2 years old: E. coli 200,000 CFU, slightly sensitive to ampioks and gentamicin, sensitive to cefazolin and cephalexin, markedly sensitive to rifampicin.
Evaluate the result:
*True bacteriuria
Suspected urinary tract infection
Normal
Latent bacteriuria

7. Urine culture of patient M., 2 years old:
E. coli 40,000 CFU, slightly sensitive to ampioks and gentamicin, sensitive to cefazolin and cephalexin, markedly sensitive to rifampicin.
Evaluate the result:
True bacteriuria
*Suspected urinary tract infection
Normal
Latent bacteriuria

8. Urine culture of patient D., 2 years old:
E. coli 2,000 CFU, slightly sensitive to ampioks and gentamicin, sensitive to cefazolin and cephalexin, markedly sensitive to rifampicin.
Evaluate the result:
True bacteriuria
Suspected urinary tract infection

*Normal
Latent bacteriuria

9. A 14-year-old child with a 6-year history of chronic glomerulonephritis, mixed form. Edema syndrome. BP 160/110 mm Hg. Pale skin. Blood urea – 20 mmol/L, blood creatinine – 0.12 mmol/L. Endogenous creatinine clearance – 20 ml/min.
Identify the leading renoprotective therapy:
Dialysis therapy
*ACE inhibitors
Saluretics
Corticosteroids

10. A 14-year-old child who has not received pathogenetic therapy for mixed-form chronic glomerulonephritis for years has developed chronic renal failure. Blood creatinine – 0.3 mmol/L. BP – 150/90 mm Hg. With adequate conservative therapy, the most predictable outcome is:
*Further progression of chronic renal failure
Reversal of symptoms
Recovery
Remission of chronic glomerulonephritis

11. An 8-year-old girl presented with complaints of urine discoloration. Condition is satisfactory, no visible edema. BP – 105/60 mm Hg. Urine is “meat-washing” color. Preliminary diagnosis: Acute glomerulonephritis.
To clarify the diagnosis, it is necessary to determine the presence of hidden edema. Which test should be performed?
Zimnitsky test
*McClure–Aldrich test
Endogenous creatinine clearance
Monitoring of diuresis

12. A 6-year-old girl fell acutely ill: fever up to 39°C, vomiting, abdominal pain, cloudy urine. Urination in small portions. BP 100/60 mmHg.
Urinalysis: specific gravity 1006, protein 0.58 g/L, leukocytes — covering the entire field of view, erythrocytes — 20–25 per field.
Blood test: ESR — 30 mm/h.
Establish the preliminary diagnosis:

- *Acute pyelonephritis*
- Acute cystitis
- Acute glomerulonephritis

- Tubulointerstitial nephritis

13. For 2 years, a 4-year-old child has had recurrent episodes of illness accompanied by fever, lethargy, abdominal pain, and increased frequency of urination.
Urinalysis: specific gravity — 1010, protein — 0.12 g/L, leukocytes — 1/2 per field, erythrocytes — 4–5 per field, no casts, oxalate crystals present.
Preliminary diagnosis: chronic pyelonephritis.
Which examination is necessary to determine the underlying cause?

- *Voiding cystourethrography (VCUG)*
- 24-hour urinary excretion of salts
- Three-glass urine test
- Blood culture for sterility

14. A 6-year-old girl complains of poor appetite, lethargy, and non-localized abdominal pain. Abdomen soft, non-tender. Urination not difficult, up to 12 times daily.
Blood test: Hb 124 g/L, leukocytes 6.3 $\times 10^9$/L, ESR 8 mm/h.
Urinalysis: protein 0.07 g/L, leukocytes 8–12 per field, numerous urate crystals.
Which additional examination is necessary?

- Urine culture for bacterial flora
- *Urine test for 24-hour uric acid excretion*
- Zimnitsky test
- Nechiporenko test

15. Your conclusion regarding the urinalysis of a 6-year-old girl: volume 20 mL, pH 4.3, specific gravity 1.008, yellow, cloudy; protein 0.99 g/L; glucose negative; acetone negative; epithelial cells: polymorphic 2–3 per field; leukocytes — covering entire field, “active” 80%; erythrocytes fresh 2–4 per field; casts: hyaline 2–3 per field, cylindroids 3–4 per field, leukocytic casts 2–4 per field; mucus (++++) bacteria (++).
Conclusion:

- UTI: urethritis or cystitis
- *UTI: pyelonephritis*
- Glomerulonephritis
- Normal urinalysis

16. Your conclusion regarding the urinalysis of Olga G., 6 years old: volume 100 mL, pH 7.3, specific gravity 1.012, yellow, cloudy; protein 0.99 g/L; glucose negative; acetone negative; epithelial cells: squamous 2–3 per field; leukocytes 10–15 per field; erythrocytes 0–1 per field; casts: hyaline 0–1 per

field, leukocytic 0–1 per field; mucus (+), bacteria (+).
Conclusion:

- *UTI: urethritis or cystitis*
- UTI: pyelonephritis
- Glomerulonephritis
- Normal urinalysis

17. A 7-year-old child has pronounced signs of inflammation, dysuria, pollakiuria, nocturia; leukocytosis with a left shift; significant leukocyturia with active leukocytes, mild proteinuria, bacteriuria, leukocytic casts.
This clinical picture corresponds to:

- *Acute pyelonephritis*
- Acute glomerulonephritis
- Acute cystitis
- Chronic renal failure

18. Urinalysis of Vika N., 2 years 9 months: volume 70 mL, pH 7.3, specific gravity 1.015, light yellow, slightly cloudy; protein 0.033 g/L; glucose negative; acetone negative; epithelial cells 2–3 per field; leukocytes 8–12 per field; erythrocytes 0–1 per field; casts: hyaline 0–1 per 2 fields, leukocytic 0–1 per field; mucus (+); bacteria absent.
Nechiporenko test: leukocytes 6000/mL; erythrocytes 1200/mL; casts 250/mL.
Conclusion:

- *UTI: urethritis or cystitis*
- UTI: pyelonephritis
- Glomerulonephritis
- Normal urinalysis

19. A 9-year-old girl has mild systemic inflammatory signs; pollakiuria, dysuria with sharp cutting pain and premature termination of urination; moderate leukocyturia, fresh erythrocytes, mild proteinuria, bacteriuria (25–100 $\times 10^3$/mL); mild leukocytosis.
This is the clinical picture of:

- Acute pyelonephritis
- Acute glomerulonephritis
- *Acute cystitis*
- Chronic renal failure

20. A 6-year-old child undergoing a routine school examination is found to have mild proteinuria, leukocyturia, bacteriuria.
Possible diagnosis:

- *Pyelonephritis*
- Chronic renal failure
- Acute myocarditis
- Acute glomerulonephritis

21. What diagnostic studies should be performed?

- Creatinine clearance
- *Nechiporenko test, Zimnitsky test, urine culture, renal ultrasound, biochemical blood tests (total protein, urea, creatinine)*
- Nechiporenko + Zimnitsky only
- Biochemical tests + renal ultrasound

22. A 9-year-old girl has been ill for 3 years. Three hospitalizations. On examination: pale skin and mucosa, facial edema, positive left-sided Pasternatsky sign.
Biochemistry: total protein 70.3 g/L, cholesterol 5.92 mmol/L, urea 6 mmol/L.
Urinalysis: specific gravity 1.006, protein 0.33 g/L, leukocytes 1/2 field, erythrocytes 8–10 per field.
Urine culture: *E. coli* 150,000 CFU.
Zimnitsky test: specific gravity 1.002–1.006.
Diagnosis:

- *Pyelonephritis*
- Cystitis
- Acute myocarditis
- Acute glomerulonephritis

23.A 2-year-old girl developed acute illness with high fever, lethargy, abdominal pain. Frequent urination in small portions.
Urinalysis: protein 0.18 g/L; leukocytes — entire field; erythrocytes — 4–5 per field.
Presumed diagnosis: acute pyelonephritis.
Most likely etiological agent:

- Staphylococcus
- *Escherichia coli (E. coli)*
- Streptococcus
- Pseudomonas aeruginosa

24. Your conclusion regarding the urinalysis of Lida T. (6 years old):

reaction (-), volume 45 ml, specific gravity 1.028, color – dirty red, transparency – slightly cloudy, protein 2 ‰, glucose – not detected, acetone – not detected, epithelial cells: squamous 1–2 per HPF; polymorphic 4 per HPF; renal 10–12 per HPF, leukocytes 10–15 per HPF, erythrocytes 60–80 per HPF

(dysmorphic), casts: hyaline 3–5 per HPF; hemoglobin casts 5–6 per HPF, mucus: (+), bacteria: (-)
Urinary tract infection: urethritis or cystitis;
Urinary tract infection: pyelonephritis;
*Glomerulonephritis;
Normal urinalysis;

25. A 6-year-old child was found to have lethargy, low-grade fever, edema, hypertension, urinary syndrome with oliguria, hematuria with dysmorphic erythrocytes, selective proteinuria, mild leukocyturia; hypoalbuminemia, mild leukocytosis, eosinophilia, ESR 30–40 mm/h. This is the clinical picture of:

acute pyelonephritis;
*acute glomerulonephritis;
acute cystitis;
chronic renal failure.

26. Your conclusion regarding the urinalysis of Viktor T. (age 3 days):

reaction – 7.3, volume – 12 ml, specific gravity – 1.007, color – red, transparency – slightly cloudy, protein – 0.52 ‰, glucose (+), acetone (-), epithelial cells: squamous 1–2 per HPF, leukocytes 4–5 per HPF, erythrocytes 0–2 per HPF, salts: uric acid crystals (+++)
Urinary tract infection: pyelonephritis;
Glomerulonephritis;
Normal analysis;
*Uric acid infarction.

27. Your conclusion regarding the urinalysis of 6-year-old Lena K.:

reaction – 4.3, volume – 35 ml, specific gravity – 1.008, color – yellow, transparency – cloudy, protein – 0.99 g/L, glucose (–), acetone (–), epithelial cells: polymorphic 2–3 per HPF, leukocytes – cover the entire field of view, “active” – 80%, erythrocytes – fresh, 2–3–4 per HPF, casts: hyaline 2–3 per HPF; leukocytic 2–4 per HPF, mucus: (++++) , bacteria: (++).
Urinary tract infection: urethritis or cystitis;
*Pyelonephritis;
Glomerulonephritis;
Normal analysis;

28. Your conclusion regarding the urinalysis of Regina V., 6 years old:

reaction – 6.3, volume – 70.0 ml, specific gravity – 1.018, color – yellow, transparency (+), protein (–), glucose (–), acetone (–), epithelial cells: squamous 1–2 per HPF, leukocytes – 6–8 per HPF, erythrocytes 0–1 per HPF, casts (–), mucus (+), bacteria (–).

Urinary tract infection: urethritis or cystitis;
Pyelonephritis;
Glomerulonephritis;
*Normal analysis;

29. A 6-year-old girl complains of poor appetite, lethargy, non-localized abdominal pain. The abdomen is soft, non-tender. Urination is not difficult, up to 12 times per day. Blood test: Hb – 124 g/L, leukocytes – 6.3×10^9/L, ESR – 8 mm/h. Urinalysis: protein – 0.07 g/L, leukocytes – 8–12 per HPF, salts – urates in large quantity. Which additional test is needed?

Bacteriological urine culture
Zimnitsky urine test
Nechiporenko urine test
*Daily urinary uric acid excretion test.

30. A 5-year-old boy is hospitalized for acute pyelonephritis. After treatment, the child's condition normalized. Urinalysis: relative density – 1012, reaction – acidic, transparency – slightly cloudy, protein and glucose – absent. Microscopy of sediment: epithelial cells – single per HPF, leukocytes – 2–4 per HPF, erythrocytes – 1–2 per HPF, hyaline casts – 0–1 per HPF, a small amount of mucus. Which test should be performed?

Bacteriological urine culture
Zimnitsky urine test
*Nechiporenko urine test
Urinary uric acid daily excretion

31. The mother of a 10-day-old infant consulted the district pediatrician with complaints of frequent urination in the child (20–22 times per day). Using a rubber ring and a cup, it was determined that the child urinates approximately 30 ml each time. The obtained data indicate:

Polyuria.
Pollakiuria.
*Physiological parameters of a newborn.
Slightly increased urination due to disturbed drinking regimen.
32. The physician received the following urine test result of patient L., 2 years old: E. coli flora 200,000, poorly sensitive to ampioks, gentamicin, sensitive to cefazolin, cephalexin, highly sensitive to rifampicin. Evaluate the result:
*True bacteriuria.
Suspicion of urinary tract infection.
Normal indicator.
Latent bacteriuria.

33. The physician received the following urine test result of patient M., 2 years old: E. coli flora 40,000, poorly sensitive to ampioks, gentamicin, sensitive to cefazolin, cephalexin, highly sensitive to rifampicin. Evaluate the result:
True bacteriuria.
*Suspicion of urinary tract infection.
Normal indicator.
Latent bacteriuria.
34. The physician received the following urine test result of patient D., 2 years old: E. coli flora 2,000, poorly sensitive to ampioks, gentamicin, sensitive to cefazolin, cephalexin, highly sensitive to rifampicin. Evaluate the result:
True bacteriuria.
Suspicion of urinary tract infection.
*Normal indicator.
Latent bacteriuria.
35. A 14-year-old child has been monitored for chronic glomerulonephritis, mixed form, for 6 years. Edema syndrome. Blood pressure 160/110 mmHg. Skin pale. Blood urea – 20 mmol/L, blood creatinine – 0.12 mmol/L. Endogenous creatinine clearance – 20 ml/min. Determine the leading method of renoprotective therapy for this patient:
Dialysis therapy
*ACE inhibitors
Saluretics
Corticosteroids
36. A 14-year-old child who has not received pathogenetic therapy for several years for mixed-form chronic glomerulonephritis developed chronic renal failure. Blood creatinine – 0.3 mmol/L, blood pressure – 150/90 mmHg. Against adequate conservative therapy, the most predictable disease outcome will be:
*Further progression of chronic renal failure
Reverse development of chronic renal failure symptoms
Recovery
Remission of chronic glomerulonephritis
37. A girl, 8 years old, admitted with complaints of a change in urine color. General condition satisfactory, no visible edema. Blood pressure – 105/60 mmHg. Urine the color of “meat slops.” Preliminary diagnosis: Acute glomerulonephritis. To clarify the diagnosis, it is necessary to determine the presence of hidden edema. For this purpose, the following test should be performed:
Zimnitsky test
*McClure test
Endogenous creatinine clearance
Diuresis monitoring

TESTS CONNECTED WITH PHOTOS

1. During kidney ultrasound (image 84), in children with primary pyelonephritis, the following is most commonly observed:

Рис. Fig. 84

A. presence of salt crystals;
B. increased renal tissue density;
C. uneven cortical thinning;
D. *presence of pyelectasis.*

2. For which renal pathology is the diagnostic value of this examination the highest (image 84):

Рис. Fig. 84

*A. *Kidney tumor, renal calculi*
B. Hydronephrotic changes of the kidneys
C. Glomerulonephritis
D. Renal pelvic dysplasia

3. For pyelonephritis, the following positive sign is characteristic, which is shown in image 88:

A. Voskresensky's sign
B. Babinski's sign

C. Ortner's sign
**D. Pasternatsky's sign*

4. The sign shown in image 88 is positive in:

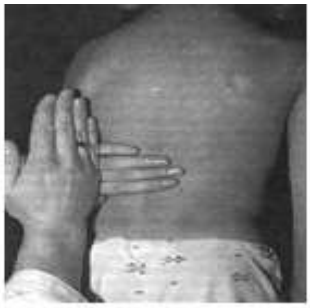

Рис. Fig. 88

A. Chronic glomerulonephritis
**B. Pyelonephritis*
C. Cystitis
D. Renal failure

5. The structure of the organ shown in image 89 is determined using:

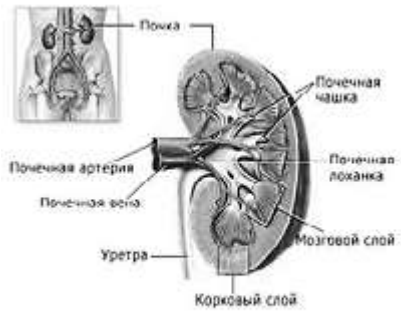

Рис. Fig. 89

A. Cystoscopy
B. Nechiporenko test
C. Endogenous creatinine clearance
**D. Ultrasound (US)*

6. Which structures of the organ shown in image 89 are most often affected in pyelonephritis?

**A. Renal tubules;*
B. Mucous membrane of the ureters;
C. Kidney capsule;
D. Glomerular capillaries.

7. Antibacterial therapy for acute bacterial infection of the organ shown in image 89 is carried out:

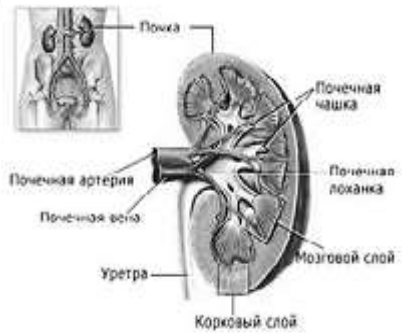

Рис. Fig. 89

A. 14–21 days;
*B. *until complete clinical and laboratory remission;*
C. for 6 months;
D. until partial clinical and laboratory remission.

8. In a 5-year-old boy, on examination (image 90), eyelid swelling predominantly appearing after sleep, pale skin, and a body temperature of +37.5°C were found. The child is lethargic and complains of abdominal pain. In the general urine analysis, leukocyturia is present. The illness has lasted for 3 days. What is the most likely diagnosis in the child:

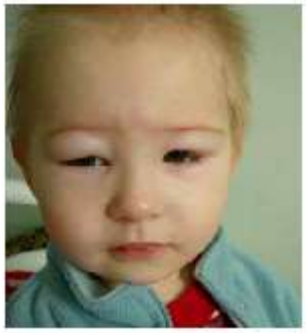

Рис. Fig. 90

A. Acute glomerulonephritis
*B. *Acute pyelonephritis*
C. ARVI: adenovirus infection
D. Acute conjunctivitis

9. In chronic pyelonephritis, ultrasound (image 84) typically shows:

**A. Dilatation of the calyceal-pelvic system and irregular outer contour*
B. Increased density/striated pattern of the renal parenchyma
C. Presence of stones in the pelvicalyceal system
D. Presence of cysts and calcifications in the renal parenchyma

10. What type of examination of a child with chronic pyelonephritis is shown in the image

Рис. Fig. 84

A. Excretory urography
B. Magnetic resonance imaging (MRI)
C. *Ultrasound examination
D. Radioisotope scanning
E. Computed tomography (CT)

11. A 10-year-old girl has been ill for 7 months. She is periodically bothered by lower back pain, subfebrile temperature, and eyelid swelling. In the general urine analysis, leukocyturia and mucus are present. During examination of the urinary system (image 84), an irregular outer contour and dilatation of the pelvicalyceal system (PCS) were found. These symptoms are characteristic of:

Рис. Fig. 84

A. Urinary tract infections
B. Acute pyelonephritis
C. *Chronic pyelonephritis
D. Chronic glomerulonephritis

12. “Photo 85 shows …”

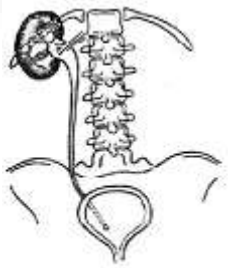

Рис. Fig. 85

A. *Aplasia of the left kidney
B. Hypoplasia of the left kidney
C. Duplication of the right kidney
D. Ectopia of the left kidney

13. The pathology shown in Photo 85 belongs to::

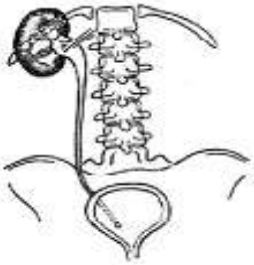

Рис. Fig. 85

14. The pathology shown in Photo 86 belongs to:

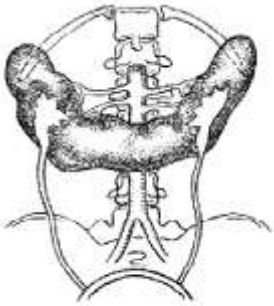

Рис. Fig. 86

A.Polycystic kidney, multicystic kidney
B. Renal dystopia, horseshoe kidney
C. *Anomalies of kidney position and shape
D. Accessory kidney

15. The pathology shown in Photo 86 may cause the following complication:

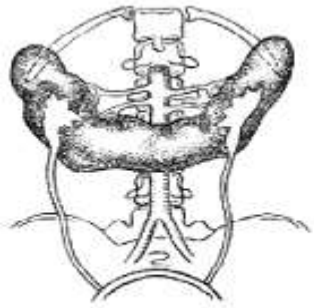

Рис. Fig. 86

A. Polycystic kidney disease
B. Glomerulonephritis
C. Primary pyelonephritis
D. *Hydronephrosis

16. Photo 86 shows:

A. Hypoplasia of the left kidney
B. Duplication of the right kidney
C. Ectopia of the left kidney
D. *Horseshoe kidney

17. The pathology shown in Photo 86 may cause the following complication:

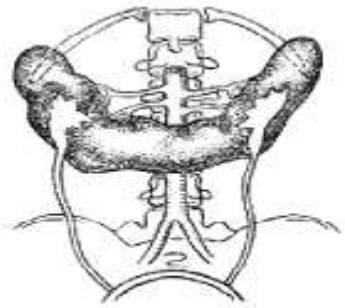

Рис. Fig. 86

A. Polycystic kidney disease
B. Glomerulonephritis
C. Primary pyelonephritis
D. *Secondary pyelonephritis

18. Which investigation is most informative for diagnosing the pathology shown in photo 86?

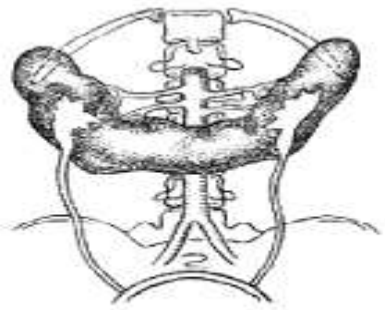

Рис. Fig. 86

A. Ultrasound
B. *Excretory urography
C. Cystoscopy
D. Cystography

19. The pathology shown in Photo 86 can be detected by additional methods of Investigation

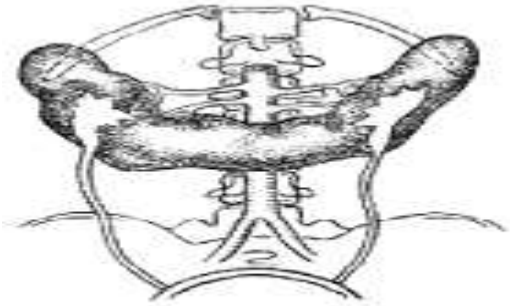

Рис. Fig. 86

A. Excretory urography
B. Ultrasound
C. Computed tomography
D. *Cystoscopy

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GLOSSARIY

1. **Albuminuria** - is the abnormal urinary excretion of albumin protein, which is a key early marker of kidney damage. Normally, the renal filters do not allow this protein to pass through, so its appearance (especially more than 30 mg/day) indicates a pathology. This is an important indicator of diabetic nephropathy, hypertension, and the risk сердечно-сосудистыхof heart disease.

2. **Anuria** - isa critical pathological condition characterized by complete cessation of urine production or its extremely small volume (less than 50-100 ml per day). At the same time, urine stops entering the bladder. This is a dangerous symptom that indicates severe kidney problems or urinary tract obstruction and requires immediate medical attention.

3. **Bacteriuria** - is thepresence of bacteria in the urine, which normally should be sterile. This is a laboratory sign that often indicates urinary tract infections (cystitis, pyelonephritis), but sometimes it is asymptomatic (asymptomatic bacteriuria). It is usually diagnosed when more than 105 colony-forming units (CFU/ml) of bacteria are detected.

4. **Hyperstenuria** - isan increase in the relative density (specific gravity) of urine above normal (usually >1.030–1.032 g / ml), indicating a high concentration of dissolved substances. The condition often indicatesdehydration, diabetes, heart failure, or kidney problems, requiring treatment for the underlying condition.

5. **Hyperuricemia** - is a pathological condition characterized by an increased content of uric acid (urates) in the blood serum (usually >360-420mmol/l). This is a laboratory sign of a metabolic disorder, which is often asymptomatic, but with a long course leads to the development of gout, urolithiasis and kidney damage.

6. **Hyperuricosuria** - is a pathological condition characterized by increased uric acid (urate) excretion in the urine(more than 800 mg / day in men and more than 750 mg / day in women). This condition often leads to the formation of kidney stones (urate nephrolithiasis) and occurs due to metabolic disorders, a diet rich in purines, or genetic factors.

7. **Hyperoxaluria** - is a pathological condition characterized by an excessive content of oxalates (oxalic acid salts) in the urine (>40 mg /day). This metabolic disorder often leads to the formation of kidney stones (urolithiasis), nephrocalcinosis, and the risk of kidney failure. There are primary (genetic) and secondary (acquired) forms.

8. **Hypoisostenuria** - is a pathological condition characterized by the release of urine with a consistently low specific gravity (usually less than 1010-1012 g / ml) in all portions during the day. This is a sign of a serious violation of the concentration function of the kidneys, often indicating chronic renal failure, nephrosclerosis or diabetes insipidus.

9. **Glucosuria** - is a pathological condition in which sugar (glucose) is detected in the urine in concentrations exceeding the norm (more than 25 mg /dl or >0.8 mmol/l). Normally, the kidneys completely reabsorb glucose back into

the blood. Glucosuria most often indicates diabetes mellitus (high blood sugar), but it can also be caused by kidney disease.

10. **Homeostasis** - isthe ability of a living organism to maintain the constancy of its internal environment (temperature, chemical composition, pressure) through self-regulation mechanisms, despite changes in the external environment. It is a dynamic balance necessary for the survival and efficient functioning of all organs and systems.

11.**Dysmetabolic nephropathies -** arekidney damage caused by metabolic disorders (most often salt). In simple terms, this is a condition where excess salts accumulate in the kidneys, turning into crystals, which leads to inflammation, cloudy urine, pain and the risk of developing stones.

12. Urinary tract infection (UTI) - thisis an inflammation in the organs responsible for the excretion of urine, caused by bacteria (most often Escherichia coli) that got inside. In simple terms, this is when germs enter the urethra or bladder, causing burning, pain, and frequent urge to go to the toilet.

13.**Isostenuria -** is apathological condition characterized by the inability of the kidneys to concentrate and dilute urine, in which its relative density remains constantly low and the same (usually 1.008-1.012) during the day. This means that the urine has the same density as the primary filtrate, without changing depending on fluid intake.

14. **Endocrine function of the kidneys** - is the ability of the organ to synthesize and release into the blood biologically active substances and hormones that regulate blood pressure, hematopoiesis and metabolism. Unlike the excretory (urine production) function, this function works as an endocrine gland.

15. **Concentration function of the kidneys** - thisis the ability of the renal tubules to return water to the blood (reabsorption) and accumulate dense substances, creating concentrated urine (density >1020 g / ml) in case of water deficiency. This is a mechanism for preserving fluid and maintaining a constant blood composition.

16. **Acid-base balance (KSHR)** - is themaintenance of a constant level of acidity (pH) of the internal environment of the body (primarily blood) in a narrow range, necessary for the normal operation of enzymes and metabolism. The normal pH value of human arterial blood is 7.35–7.45. This balance is regulated by the respiratory and excretory systems.

17**. Miction cystography** - cystourethrography) is an X-ray examination of the bladder and urethra performed during the act of urination. The procedure allows you to assess the anatomy of the lower urinary tract and identify pathologies, such as vesicoureteral reflux, by introducing a contrast agent through a catheter.

18. **Urinary syndrome** - is a complex of laboratory changes in urine tests (protein, red blood cells, white blood cells, cylinders), indicating damage to the kidneys or urinary tract. This is not an independent disease, but a sign of diseases (pyelonephritis, glomerulonephritis, ICD), manifested by deviations from the norm.

19. **Nephrology** – is a branch of medicine that studies the kidneys: their structure, function, diseases, and treatment methods.

20. **A nephrologist -** is a " kidney therapist " who treats them with pills and diet, preventing the need for surgery. He deals with kidney failure, nephritis, the effects of diabetes and hypertension on the kidneys, and leads patients after transplantation.

21. **The nephron** - is a tiny "filter" inside the kidney that is responsible for purifying blood and forming urine. Each kidney contains about 1 million of these microscopic structures. They filter out harmful substances, excess water and salts, leaving useful components in the body.

22. **Nephritic syndrome –** is a clinical and laboratory symptomcomplex that occurs in acute inflammation of the renal glomeruli (glomerulonephritis). It is characterized by a "triad": pronounced hematuria (blood in the urine, often the color of "meat slop"), arterial hypertension (increased blood pressure) and edema. Often accompanied by a decrease in the amount of urine (oliguria) and moderate proteinuria.

23. **Nephrotic syndrome** - is a complex of symptoms that occurs due to damage to the renal glomeruli and filtration disorders, leading to a massive loss of protein in the urine. Key signs: highproteinuria (more than 3 g / day), a critical decrease in protein (albumin) in the blood, severe edema (includingascites) and elevated lipid levels.

24. **Hereditary nephritis (often referred to as Alport syndrome)** - is a genetically determined non-immune kidney disease caused by a mutation of genes responsible for the synthesis of type IV collagen. It leads to damage to the renal glomeruli, persistent hematuria (blood in the urine), progressive renal failure, and sometimes — to hearing and vision disorders.

25. Acute glomerulonephritis – is a sharp inflammation of the kidney (glomerular) filaments caused by a malfunction of the immune system. Due to inflammation, the kidneys stop normally purifying the blood, retain fluid and remove protein/red blood cells. It often occurs 1-3 weeks after the infection (sore throat, flu).

26. **Acute renal failure (ARF) or acute kidney injury (AKI) isa sudden, often reversible decrease in kidney function, causing self** - poisoning of the body (azotemia), a violation of the water-salt balance. The condition requires immediate hospitalization, often to the intensive care unit, for treatment of the underlying condition. The main signs: a sharp decrease or absence of urination, swelling, nausea, vomiting.

27. **Edematous syndrome** - is apathological condition characterized by excessive accumulation of fluid in the intercellular space of tissues or serous cavities of the body. It is manifested by an increase in the volume of organs/limbs, swelling, decreased skin elasticity (testovatost) and heaviness. Most often indicates diseases of the heart, kidneys, liver, blood vessels or endocrine disorders.

28. **Oliguria** - is a pathological condition characterized by a sharp decrease in the amount of urine excreted (diuresis) to less than 400-500 ml per day in

adults, which indicates kidney problems, dehydration, or serious systemic diseases. This is not an independent disease, but a symptom that requires a search for the cause.

29. **Oligoanuria (a combinationof oliguria and anuria)** - is a critical pathological condition characterized by a sharp decrease (less than 400-500 ml/day in adults) or almost complete cessation (less than 100 ml/day) of urinary excretion by the kidneys. This is a dangerous symptom that indicates acute kidney failure, dehydration, or severe circulatory disorders.

30. **Pyelonephritis** - is an infectious inflammation of the kidneys caused by bacteria (most often Escherichia coli) that enter the organ from the bladder or from the bloodstream. In simple words, this is when they "caught a cold" or brought an infection to the kidneys, which causes them to swell, hurt, and the body temperature rises sharply.

31. **Proteinuria** - is a pathological condition characterized by an increased protein content (usually albumin) in the urine, exceeding normal values (more than 150 mg / day). This is not an independent disease, but a symptom indicating a malfunction of the kidneys or other body systems. Often accompanied by foamy urine.

32.**Renal hypertension syndrome (nephrogenic arterial hypertension –** is a persistent increase in blood pressure (BP) caused by diseases of the kidneys or their vessels. This is secondary hypertension caused by pathology of the parenchyma (inflammation), blood vessels (stenosis) or abnormalities of kidney development. It is characterized by high blood pressure, difficult to treat, and symptoms of kidney damage.

33. **Polyuria** – is a pathological increase in the daily volume of urine exceeding 2.5-3 liters in adults (with a norm of 1-1. 5 liters), which is accompanied by frequent urination. This condition often indicates a malfunction of the kidneys, hormonal disorders (for example, diabetes) or excessive fluid intake.

34. **Reabsorption –** is the process of reabsorption of water, electrolytes, and nutrients (glucose, amino acids) from primary urine back into the bloodstream through the renal tubules. This is a vital mechanism that allows you to return about 99% of the filtered fluid to the body, turning the primary urine into the final one.

35. **Retrograde pyelography** – is an invasive X-ray method of examining the upper urinary tract (kidneys, pelvis, ureters) using a contrast agent inserted "from the bottom up" (retrograde) through a ureteral catheter under the control of a cystoscope. The method allows you to visualize the anatomy in detail and identify stones, tumors, or strictures.

36. **Glomerular filtration rate (GFR)** - is the main indicator of kidney function, reflecting the volume of blood (in ml) cleared by the kidneys of metabolic products (creatinine) in one minute. GFR allows you to assess how effectively the kidneys filter blood, and is a key tool for diagnosing kidney diseases (the norm is ~ 100-125 ml / min/1.73m2).

37.**Tubulopathies** - are a group of kidney diseases in which the work of their microscopic tubules (tubules) is disrupted. Instead of purifying the blood and returning useful substances back, the kidneys begin to lose them in the urine (salts, calcium, glucose) or do not remove excess. This leads to a lack of trace elements, metabolic disorders and dehydration.

38. **Chronic renal failure (CRF)** - is an irreversible progressive decline in kidney function caused by the death of nephrons due to diseases (diabetes mellitus, hypertension,glomerulonephritis), leading to intoxication of the body. Symptoms include swelling, itchy skin, nausea, ammonia smell, and fatigue.

39. **Chronic glomerulonephritis** - is a long-term autoimmune inflammation of the renal glomeruli (filters), in which the immune system mistakenly attacks its own kidneys. This leads to gradual scarring of the tissue, reduced blood purification, and the development of kidney failure. Main signs: protein/blood in the urine, persistent high blood pressure and swelling.

40. **Cylindruria** - is a laboratory syndrome characterized by the detection of cylinders in the urine sedimentsilindrov-protein casts of renal tubules consisting of protein, epithelial cells, red blood cells or white blood cells. This is a specific sign of kidney disease, indicating a violation of their function.

41. **Erythrocyturia (or microhematuria)** - is a pathological condition characterized by the presence of an increased number of red blood cells (red blood cells) in the urine, determined only by microscopic examination. This is an important diagnostic sign that indicates kidney disease, infections (cystitis), urolithiasis, or urinary tract injuries.

43. **Excretory urography (intravenous)** - is an X-ray method of examining the urinary tract (kidneys, ureters, bladder) using an iodine-containing contrast agent administered intravenously. The method allows you to evaluate the anatomical structure and functional ability of the kidneys, as it is based on their ability to display contrast.

FOR NOTES

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PEDIATRIC NEPHROLOGY

STUDY GUIDE

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