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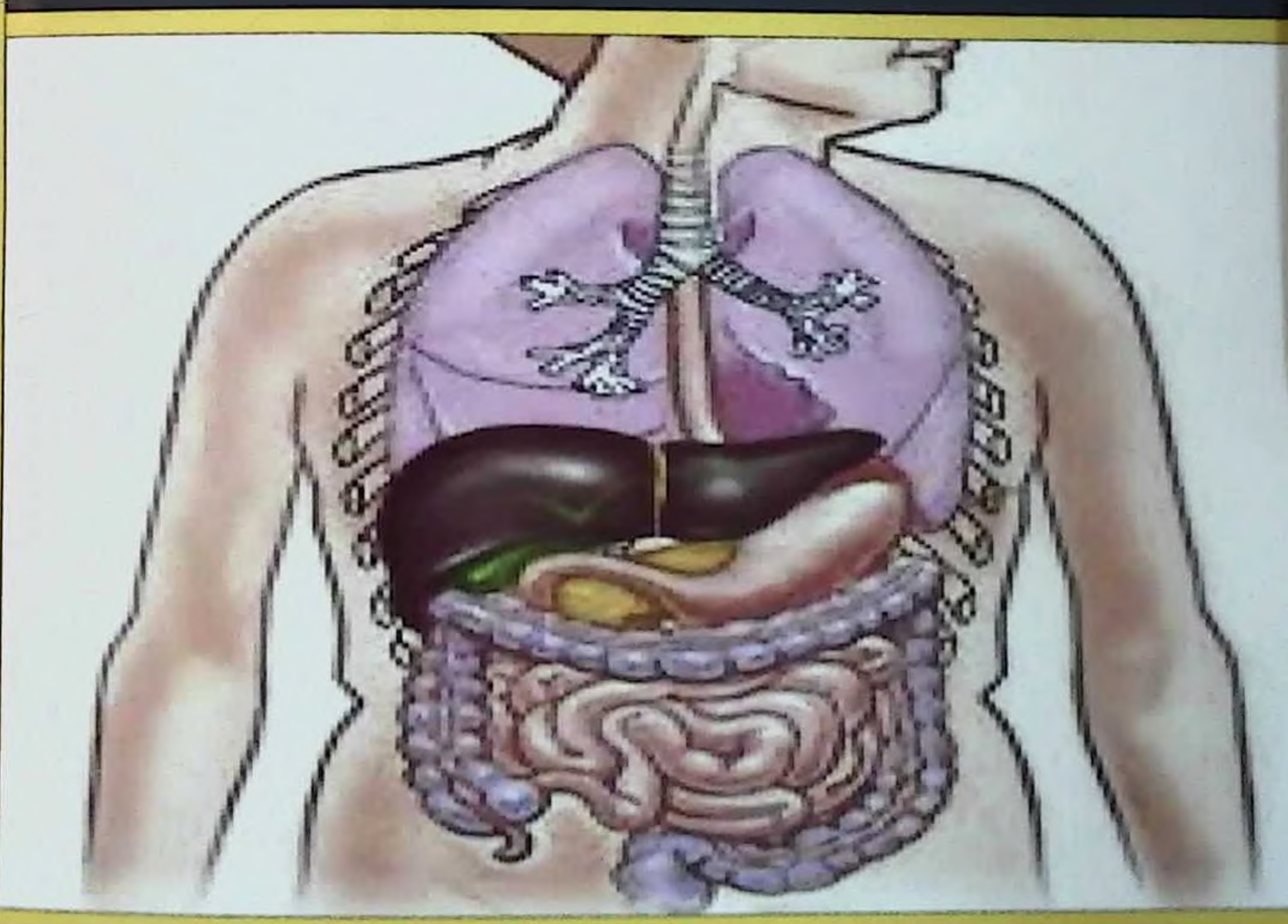
FUNCTIONAL STATE OF THE LIVER IN CHILDREN WITH CHRONIC GASTRODUODENAL PATHOLOGY



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MINISTRY OF HEALTH CARE OF THE REPUBLIC OF UZBEKISTAN

SAMARKAND STATE MEDICAL UNIVERSITY

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The monograph presents data on the functional state of the liver in children suffering from chronic gastroduodenal pathology: chronic gastritis, chronic gastroduodenitis and duodenal ulcer. The clinical and biochemical assessment of nitrogen and carbohydrate metabolism, in particular detoxification and gluconeogenic liver function in children with diseases of the gastrointestinal tract is given in detail. Based on the results of the work, a systematized complex therapeutic tactics has been developed, adapted in the conditions of practical healthcare and implemented in practice.

The monograph is intended for general practitioners, pediatricians, gastroenterologists and hepatologists, doctors of related specialties, masters and students of medical institutes.

Ilmiy kengash kotibi t.f.n. **U.U. Ochilov**

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LIST OF ABBREVIATIONS

UPDT — upper parts of the digestive tract

DGR — duodenogastric reflux

GERD — gastroesophageal reflux disease

CGDP – gastroduodenal pathology

GIT — gastrointestinal tract

PPI - proton pump inhibitor

AFFS — acid-forming function of the stomach

MPC — maximum permissible concentration

GM — gastric mucosa

USE - ultrasound examination

FEGDS— fibroesophagogastroduodenoscopy

FFA - free fatty acids

CAG - chronic active hepatitis

CPG - chronic persistent hepatitis

CG – chronic gastritis

CGD – chronic gastroduodenitis

CGDP - chronic gastroduodenal pathology

EGDS — esophagogastroduodenoscopy

DU — duodenal ulcer

PUS — peptic ulcer of the stomach

SGTT - standard glucose tolerance test

INTRODUCTION

This monograph contains a modern understanding of the functional state of the liver in children suffering from chronic pathology of the gastroduodenal zone. The digestive system is a complex digestive process, the well-coordinated work of which largely depends on the condition of the child and his health. Since the digestive process is a single, integral one, there are close relationships between the activities of individual organs. A disorder of the functions of one of the departments of the digestive tract can lead to a violation of the functions of other organs. Age-related changes in the structure of the digestive system and its functions are inextricably linked with the peculiarities of the vital activity of the body at each stage of its development, with energy and plastic needs, with the peculiarities of nutrition.

This monograph discusses the issues of comorbidity in diseases of the gastrointestinal tract, i.e., combined damage to the organs of the gastroduodenal zone and the hepatobiliary system in children of a functional and organic nature, and also emphasizes the need for timely diagnosis and adequate therapy of combined disorders.

Working on this monograph, we tried to reflect as fully and systematically as possible all the current information about the clinical and biochemical features of chronic gastritis, gastroduodenitis and duodenal ulcer in children. Particular attention was paid to a detailed assessment of detoxification and gluconeogenic liver function in children with chronic gastroduodenal pathology, description of the dynamics of clinical, laboratory-instrumental and special biochemical studies, the interpretation of which is the key not only to correct clinical diagnosis, but also to adequate individual selection of optimal treatment methods for children with chronic gastroduodenal pathology.

CHAPTER 1. CHRONIC GASTRODUODENAL PATHOLOGY IN CHILDREN AND CLINICAL CHARACTERISTICS OF SOME ASPECTS OF METABOLISM

Diseases of the digestive organs are quite widespread in the children's population and occupy a leading place in the structure of somatic pathology of childhood, being in the first place in the structure of chronic pathology in children 0-18 years old. Over the past 20 years, the prevalence of digestive diseases in children has increased by 30% and, according to various authors, ranges from 79.3 to 100 per 1000 children [10, 22, 24, 25, 72, 73].

Among the diseases of the digestive system, the leading place is occupied by gastroduodenal pathology, as well as functional disorders and inflammatory diseases of the biliary tract. In modern conditions, chronic diseases of the gastroduodenal region and the biliary system have an early onset (they begin already at preschool age) and subsequently take a continuously recurrent course, significantly reducing the quality of life of children and adolescents. According to Russian researchers, only 7.9% of children aged 1.5-7 years are registered at a dispensary in a children's polyclinic with digestive diseases, which is 5.9 times lower than the indicators according to active diagnostics [17, 24, 25, 27, 28, 162].

Despite the clearly expressed anatomical and physiological division of the digestive tract into departments, the digestive process is a single, integral one, there are close relationships between the activities of individual organs, since the digestive system is a complex digestive process, the well-coordinated work of which largely depends on the condition of the child and his health [24, 25, 37, 72, 99, 123].

A disorder of the functions of one of the departments of the digestive tract can lead to a violation of the functions of other organs. Age-related changes in the structure of the digestive system and its functions are inextricably linked with the peculiarities of the vital activity of the body at each stage of its development, with energy and plastic needs, with the peculiarities of nutrition [25, 54, 72, 74, 80, 115, 135, 145, 148].

The main function of the digestive system is the digestion and absorption of nutrients, but the digestive system performs other important functions, among which are:

- motor and associated transient tow truck;
- secretory and excretory, which regulate the constancy of the internal environment of the body and the homeostasis of the enteral environment;
- endogenous digestion and utilization of endogenous substances due to hydrolysis and subsequent absorption of endogenous substrates and metabolites secreted into the intestinal lumen;
- metabolic, carrying out the transformation and biosynthesis of substances not only from exogenous substrates that are transported through it during absorption, but also endogenous, entering the mucous membrane from blood serum, as well as into the intestinal lumen as a result of secretion;
- protective, in which epithelial and mucosal barriers play an important role, as well as the immune system of the mucous membrane and other protective factors are actively involved;
- regulatory, which is carried out with the help of substrate, nervous and endocrine regulation, ensuring the interconnection of various parts of the digestive process, assimilation and metabolism of food substrates [25, 72, 140].

All these functions of the gastrointestinal tract are closely related to each other and are aimed at achieving the main goal - optimizing the processes of digestion and absorption of nutrients that are necessary to ensure the energy needs of the body's vital activity, physical activity and growth of the child [25, 72, 81, 86].

The digestive system is a complex and at the same time well-organized and stable system that has ample opportunities for rapid adaptation to various changes in the environment. The child's digestive system is less adaptive because it is in the process of development. In addition, children can observe an individual rate of development of certain functions of the digestive system, which may lag behind or outstrip the average values characteristic of a particular age [25, 73, 150].

The normal functioning of the digestive system depends on providing it with nutrients, energy substrates and oxygen. Despite the fact that a huge flow of both exogenous and endogenous nutrients passes through the gastrointestinal tract, the supply of all its departments with energy and plastic substrates is carried out from the blood. Therefore, any violation of systemic or peripheral blood circulation can not only cause trophic disorders in the digestive organs, but also disrupt or completely block the absorption of nutrients [24, 156].

For the functioning of such a complex and well-functioning mechanism as the digestive system, perfect regulation mechanisms are needed, which are presented and repeatedly duplicated at the level of nervous, hormonal and substrate regulation and are able to work offline. In the gastrointestinal tract, there is a fairly perfect system of hormonal regulation, which is tuned and adapted to a certain type and diet [24, 73, 190, 198].

An important role belongs to the substrate regulation of digestion, so any changes in the chemical composition of the diet, its volume, osmolarity, pH, etc. they cause changes in the system of commands sent to the digestive receptor apparatus. Disorders of nervous regulation primarily affect the motility of the digestive organs. Thus, disturbances in the functioning of the regulatory system underlie the development of many diseases of the digestive system [200].

Traditionally, all pathological conditions that occur in any system of the human body are divided into organic and functional. Organic pathology is associated with damage to the structure of an organ, the severity of which can range from a gross anomaly to a subtle enzymopathy. In functional disorders, these injuries are not found, and the resulting changes lie outside the affected organ and are associated with altered regulation of the impaired function [24, 200].

In general, diseases of the digestive system arise as a result of the interaction of genetic factors and the environment. Ecology also plays a certain role in this regard. Unfavorable ecology aggravates hereditary predisposition, increases the influence of negative family factors, which

leads to more frequent occurrence and more severe course of digestive diseases.

The deterioration of the environmental situation, an increase in the frequency of allergic diseases, the regime of increased neuropsychiatric stress experienced in the children's collective, a sedentary lifestyle, an unbalanced diet and a violation of the regime are currently considered as the main factors that lead to an increase in the frequency of diseases of the digestive tract. Over the past 10 years, the role of the neuropsychic factor in the formation of pathology of the digestive organs has increased significantly. The psychosomatic genesis of gastroenterological diseases can be traced to one degree or another in 40-50% of patients [24, 204].

Children have age periods when the probability of problems from the digestive system is much higher. Thus, an increase in the incidence of digestive organs in children is noted at the ages of 5-6 and 9-12 years, i.e. during periods of the most intense morphofunctional changes in the child's body, when growth imbalances and organ dysfunction occur due to uneven growth and maturation of individual organs and systems [74, 249].

It has been established that up to 30% of the diseases detected in these age periods are nothing more than functional disorders, of which more than half pass without a trace without any treatment. At the same time, part of the functional disorders, provided that the child is constantly exposed to adverse environmental factors, progresses and turns into a chronic process. Accordingly, the proportion of functional disorders of the digestive system decreases with age in children, and at the same time the frequency of organic diseases increases, in the structure of which gastroduodenitis is in the first place, intestinal diseases are in the second, diseases of the hepatobiliary system are in the third [25, 209, 225, 240]. Thus, the attitude to functional diseases should be serious enough, and the treatment should be adequate [204, 205, 235, 248].

Of the organic diseases of the digestive organs in children, chronic gastroduodenitis (inflammatory-dystrophic lesion of the mucous

membrane of the stomach and duodenum) is most common, the causes of which are diverse. At the present stage, data on the role of Helicobacter pylori as one of the main causes of the development and progression of gastroduodenal pathology are of great scientific and practical interest. Among children with chronic diseases of the stomach and duodenum, Helicobacter pylori infection is up to 81% in chronic gastritis and 90-100% in erosive and ulcerative lesions of the gastroduodenal zone. The role of Helicobacter pylori is explained by the fact that under its influence, mucosal atrophy develops and / or intestinal metaplasia or dysplasia develops, which increases the risk of malignant processes. Recently, Helicobacter pylori has been detected in newborns in 5.4% of cases, and by the age of 13-15, the infection rate reaches 58-72%.

In addition, immunological disorders (20%) and toxic damage by various substances (5%) are important in the development of gastroduodenitis. Predisposing to the disease of gastroduodenitis are factors such as a burdened hereditary history, the quality and quantity of food, dry eating, the use of seasonings, spices, irregular diet (1-2 times a day), the presence of food allergies.

Acquired non-infectious (inflammatory) diseases of the digestive system in children differ in polymorphism and a tendency to generalize the pathological process and have some features (unlike adults) that make it difficult to timely diagnosis and, accordingly, adequate treatment:

- Often erased beginning;
- Most immediately acquire a chronic, recurrent or latent course;
- Exacerbations are expressed dimly (subclinically);
- They are characterized by clinical diversity: in addition to pain, dyspeptic, intoxication syndromes, there may be a delay in development;
- It is not uncommon to involve associated organs in the pathological process;
- A combination of inflammatory and functional changes in the gastrointestinal tract (occurs in 2/3 of children).

For the diagnosis of organic and functional diseases of the digestive system, the analysis of complaints, hereditary history, knowledge and consideration of anatomical and physiological features of the gastrointestinal tract of the child, as well as laboratory and instrumental examinations are of paramount importance. The main difficulty in the diagnosis of functional disorders is the need to exclude all possible organic pathology. Only after that it is possible to speak with confidence about the functional nature of the disease [24, 74, 263].

The use of instrumental examination methods in pediatric gastroenterology is a separate topic, due to the insecurity of many methods for the patient and limited information content.

Diagnosis of a number of chronic diseases of the gastrointestinal tract became possible after the introduction of endoscopy and endoscopic biopsy into medical practice. All organs of the digestive tract are available for endoscopy in children: esophagus, stomach, initial and terminal sections of the small and all sections of the colon, biliary tract, liver. Morphological examination of the mucous membrane taken during endoscopic examination (biopsy) allows to diagnose diseases such as gastritis, duodenitis, celiac disease, ulcerative colitis, Crohn's disease, etc., to determine the severity of chronic inflammation and its activity, to monitor treatment [24].

Paediatric gastroenterology also uses a wide range of modern radiological diagnostic research methods, mainly X-ray, ultrasound and computed tomography. The X-ray method and computed tomography are the main and decisive in the diagnosis of congenital anomalies, malformations of the digestive tube and acquired organic lesions of the digestive organs, as well as in the detection of complications of peptic ulcer disease.

One of the leading methods in pediatric gastroenterology today is ultrasound. Accessibility, harmlessness and absence of contraindications make ultrasound a screening, rather informative method that clarifies structural changes in abdominal organs in children [74, 274].

Of the laboratory methods of investigation, various biochemical methods for studying liver and pancreatic function are most often used [10, 283].

The tendency to increase the frequency of diseases, their prolonged and recurrent course, involvement of other organs and systems in the pathological process, multiple metabolic disorders, lagging children in physical development indicate the need to search for new aspects of pathogenesis and improve treatment methods for this category of patients [3, 119, 127, 168].

The high prevalence of digestive diseases in childhood, their tendency to increase (by 37% over the last decade), their long and recurrent course indicates the need to search for new aspects of pathogenesis and improve treatment methods for this category of patients [69]. In children with chronic diseases of the gastroduodenal zone, functional and morphological changes from the internal organs, multiple metabolic disorders are often detected, which is pronounced in patients with a long course of the disease.

Currently, the problem of chronic diseases of the digestive system has attracted the attention of not only therapists, but also pediatricians, due to the almost universal increase in the frequency of the disease among children and its tendency to chronic recurrent course [3, 21, 30, 34, 124, 130, 160].

The prevalence of gastroenterological diseases is 79.3-109.2 per 1000 children, and the indicators are approximately the same in different regions [3, 18, 118, 129, 202], of these, the share of CGDPs accounts for 57-76% [34, 160].

A.V.Mazurin and A.M.Zaprudnov [128] note that the most common form of CGDP is CGD. According to their observations, it occurs in 50-60% of children who complained of abdominal pain, who underwent gastroduodenoscopy with a targeted biopsy of the gastric mucosa and duodenum, followed by its morphological examination.

According to V.B.Shifrin [220], DU was diagnosed in 1.8 per 1000 examined children of primary school age, but its frequency increased in

older school-age children - 6.2. Among all hospitalized children with DU, averaged 6.3% [128].

M.R.Rustamov [168] found that in children in a sharply continental climate, CGDP occupies a high proportion and amounts to 59.4 per 1000 children, including CG -27.5, CGD -23.0, DU - 6.3 and functional stomach disorders - 2.6 per 1000 children.

Numerous studies claim that chronic pathology of the gastroduodenal zone is formed from the age of 5-6, but as a result of incorrect diagnosis and treatment, the pathological process leads to difficult-to-reverse changes in the gastroduodenal system in children and manifests itself in adults [3, 20, 26, 43, 44, 149, 174].

Considering the features of the age-sex prevalence of digestive diseases in children, many researchers come to the conclusion that girls are more likely to get sick than boys, which is associated with a hereditary predisposition on the maternal side [49, 165]. A.A.Baranov, O.V.Grinina [20] note that the frequency of detection of the disease in girls is higher than in boys, respectively - 96.3 and 61.8 per 1000 children. According to A.Mryglodowic [259], the incidence of diseases falls at the age of 11-14 years and more often falls on the female sex.

Due to the current use of the latest research methods, combined lesions of the digestive organs are detected more often (from 37.6 to 56.4%) than isolated ones [121]. A.V.Mazurin et al., [131] note that more than half of children with diseases of the digestive organs have combined lesions of the stomach and duodenum. The authors also note that hyperacid condition is more often detected in children with CGDP compared to normal and hypoacid, in 98% of children with peptic ulcer, the pathological process was localized in the duodenal bulb.

CG, CGD and DU are a classic example of a polyethological disease, and the etiology and pathogenesis are so interrelated that it is not always possible to separate them. In addition to the suffering of the whole organism, there are factors that play a significant role in the development of CGDP [132].

The role of feeding and nutrition disorders starting from infancy in the genesis of gastroenterological diseases is indicated by numerous authors [40, 23, 61, 108, 125, 257, 269].

When analyzing anamnestic data in children with diseases of the gastrointestinal tract, N.V.Dmitrieva et al., [62], irregular nutrition and dry eating were found in 50% of cases.

I.G.Abbasov et al. report on the role of irrational distribution of food during the day, in particular, the intake of a large amount of food during dinner, the use of food inappropriate to age, dry eating, overeating, the use of spicy dishes in the development of the disease [1]. J.Wolff, L.Lauter [287] attach great importance to the state of the chewing apparatus in the digestion of food and the development of the disease.

Another equally important factor in the development of gastroenterological diseases is a change in neurohumoral regulation. As N.N.Gridneva rightly points out [57], their significance in the origin of DU in children becomes even more obvious if we take into account the insufficient differentiation of the central and autonomic nervous systems, as well as the associated lability and vulnerability of the emotional sphere of the child.

Exacerbations of diseases of the gastroduodenal zone, often their occurrence, occur mainly due to psycho-emotional trauma, overstrain from mental and physical work. A special role is assigned to stress, the nervous pathway of which runs schematically along the axis: the cerebral cortex - the higher centers of the visceral nervous system (hypothalamus, pituitary gland) - the autonomic nervous system (N.vagus) – the stomach [58]. An increase in the tone of the vagus nerve activates the activity of the glandular apparatus in the interstitial period with an increase in juice production in the complex reflex nervous phase, at the same time gastrin secretion increases, motility and stomach tone increase.

Gastroduodenal diseases are also among the psychosomatic, in the occurrence of which 3 main structures of the brain play an important role: the neocortex, hypothalamus and limbic system. Two endocrine

systems are also connected with these structures: the pituitary gland-the adrenal cortex and the hypothalamus-the cerebral part of the adrenal cortex. The trigger mechanism for the development of DU and CGD may be the true depletion of these regulatory systems, or diseases are a response to stress, emotions, and others. They are accompanied by a number of physiological reactions, stimulation of the brain, limbic system, hypothalamus with subsequent changes in the autonomic and endocrine systems, violation of all types of metabolism, vaso- and visceromotor, secretory and circulatory reactions [128].

Currently, there are several other factors that are important in the development of CGDP: Campylobacter pylori [12, 118, 122, 172, 178, 185], immunological disorders [116, 208]; endocrine factors - STH, TTH, ACTH, sex hormones, insulin [16, 38, 133, 179]; gastrointestinal hormones - gastrin, secretin, glucagon [95, 126, 250]; biogenic amines - serotonin, histamine [239]; intestinal microflora [8, 68, 203]; microcirculatory disorders [31];

It is necessary to dwell on one more important issue. A large mosaic of gastroenterological diseases in childhood [71], frequent addition of an immunological component [208, 215], the presence of various disorders of the nervous system [193, 211, 217], a high degree of allergization and the incidence of chronic focal infection [66, 78, 158, 182], significant frequency of deviations in the emotional sphere [5The involvement of many organs and systems in the pathological process makes it very difficult to diagnose any of the gastroenterological diseases [97, 111, 184].

Despite the many proven methodological approaches to this issue, the problem is still not solved. To a large extent, this is due to the lack of clear criteria for the progression of gastroenterological diseases in the clinic, while, as a rule, the functional state of internal organs is not taken into account [213]. Insufficient attention to this problem entails a certain schematicity in the treatment of CGDP in children. Thus, A.M.Zaprudnov, emphasizing the polysyndromic nature of gastroenterological diseases, believes that the therapy and prevention of gastrointestinal diseases in children should affect not only the mechanisms of the chronic pathological process, but also the metabolic

disorders and functional disorders of internal organs that occur in this case. Only such a comprehensive approach to the treatment of gastroenterological diseases is able to restore the disturbed homeostasis of patients [71].

Diseases of the gastroduodenal zone are by no means isolated, since the functional state of other organs and systems is simultaneously disrupted [127, 199].

Quite often, children (1/2 - 2/3) with CGD and DU have intestinal disorders in the form of constipation, alternating with unstable chairs. According to A.V. Mazurin and A.M.Zaprudny [127], neuromuscular dyskinesia of the large intestine of vagal origin is assumed to be the basis of these intestinal symptoms. It is impossible to exclude the role of a sparing diet, poor in fiber, and therefore insufficiently irritating to the intestines.

According to Yu.I.Fishzon-Ryss [199], gastritis with preserved or increased secretion is characterized by a tendency to spastic constipation, and for gastritis with secretory insufficiency, the most characteristic is considered to be a tendency to diarrhea [52, 137, 273]. A.A.Levin [112] with the help of biopsy revealed eunitis in 28 of 43 patients HG.

In patients with diseases of the gastrointestinal tract, there is a violation of the resorption of nutrients, which is confirmed by a decrease in the total absorption function of the intestine. In the last decade, the attention of researchers has been attracted by a problem caused by a violation of the transport of nutrients in the intestine [186]. The discovery of membrane digestion played an important role in this [195, 196].

Noteworthy is the work of V.L.Paikov et al., [159], who studied in detail the ultrastructure of the duodenal mucosa in children with CGD. The authors revealed ultrastructural changes in the enterocytes of the duodenal mucosa in CGD in the period of exacerbation, which gave the researchers reason to assume the possibility of a violation of their transport function.

Anatomical proximity of the stomach, duodenum, liver, bile ducts, pancreas cause rapid involvement in the pathological process of these organs in diseases of the upper digestive tract. So, unlike adults, 50% of children with CGDP mainly have functional dyskinetic disorders in combination with the syndrome of "subhepatic" cholestasis, which is manifested by a moderate increase in the liver (by 1-2 cm) and an increase in excretory enzymes in the blood [79, 93, 180].

Turning to the discussion of the effect of chronic diseases of the stomach and duodenum on the hepatobiliary system, it should be emphasized that such a relationship has attracted the attention of researchers for a relatively long time [117, 146, 221]. However, despite this, the involvement of the liver in the pathological process in chronic GDP is still insufficiently studied and the information is quite contradictory. Thus, I.M.Lipetsk [117] showed that during experimental gastritis in animals, known morphological changes in the liver are detected - "gastrogenic hepatitis".

The involvement of the liver in the pathological process is associated by a number of clinicians with the entry into the portal vein of harmful substances from pathologically altered gastric mucosa. Similarly, toxic products absorbed from the affected intestine, and in particular, of microbial origin, can affect.

A.L.Myasnikov [146] regarded the resulting lesions as serous hepatitis. However, as shown by puncture hepatobiopsy B.I.Shulutko et al., [221], liver changes detected in some patients with atrophic gastritis were predominantly degenerative in nature.

The liver lesions in question are most likely not purely gastric, but gastroenterogenic in nature. In this regard, literary references to the development of serious changes in liver tissue and an increased frequency of liver cirrhosis in patients with a resected stomach attract attention [223]. The liver, in such cases, suffers due to the general disorganization of digestive processes, deficiency of plastic substances and vitamins, as well as the influence of other factors mentioned above.

According to A.V.Mazurin [125], in children with CGDP, mainly functional disorders of the hepatobiliary system are detected due to

damage to the duodenal mucosa. which produces interstitial hormones that regulate the activity of the digestive tract.

According to B.K.Reinhard, A.M.Spivak [166], 37.1% of patients have impaired protein-forming and antitoxic liver function in case of DU and CGD with a disease duration of up to 5 years. In patients with frequent exacerbations of DU and CGD, with the addition of complications, disorders in pigment, enzyme and lipid metabolism are also observed.

So, in patients with chronic GDP, deep morphological and functional changes of the liver can be observed, which are a consequence of the multifactorial nature of the pathological process. At the same time, the data presented above are not without contradictions, often in these studies the study of liver function was not the main one.

There is practically no information in the literature about changes in children's specific liver functions, such as urea-forming, gluconeogenic, bile-forming, etc., the effect of drugs on these liver functions in children with CG, CGD and DU has not been studied.

Some issues of early diagnosis and differential diagnosis of chronic gastritis (CG), chronic gastroduodenitis (CGD) and duodenal ulcer (DU) are still insufficiently developed, there are no clear criteria for monitoring the effectiveness of the treatment, the reserve capabilities of the body and the functional state of internal organs, especially the liver, with this pathology are practically little studied. A number of these problems can be solved by deeply studying the specific functions of the liver, which include gluconeogenesis - that is, the ability of hepatocytes to synthesize glucose from non-carbohydrate compounds - amino acids, glycerin, lactate, pyruvate and others.

At the same time, gluconeogenesis, as a fundamental process supporting normoglycemia, replenishing the deficit of mobilized glycogen, regulating the pool of intermediate compounds of carbohydrate, nitrogen and lipid metabolism, increasing in a number of physiological conditions (pregnancy, intense muscle work, emotional stress) and occurring mainly in the liver, with diseases of the gastroduodenal zone, practically does not investigated.

Meanwhile, the elucidation of changes in gluconeogenesis can reveal new prospects in the study of the pathogenesis of gastroduodenal pathology (CGDP) in children and help in the development of objective differential diagnostic criteria for these diseases, in assessing the effectiveness of treatment and in finding pathogenetically justified therapeutic measures. Based on this, the study of carbohydrate, nitrogen metabolism and gluconeogenic liver function in chronic GDP in children substantiates the urgency of the problem.

Undoubtedly, in the pathogenesis of chronic diseases of the gastrointestinal tract in children, the main place belongs to the violation of metabolic processes. Based on this, modern data on changes in certain types of metabolism (lipid, carbohydrate, nitrogen, and enzymes) in patients suffering from CGDP will be presented below.

Recently, the issues of lipid metabolism in chronic diseases of the gastrointestinal tract have been studied in more detail [2, 7, 9, 39, 41, 48, 76, 89, 90, 91, 110, 113, 138, 151, 167, 170, 171, 173, 187, 212, 230, 232, 236, 252, 260, 272, 275, 277, 279, 282, 284, 286].

Having studied the role of lipids in the human body, a number of authors have established their participation in the construction of biological membranes and immunological processes [32, 102].

It should be noted that the imbalance in the lipolysis-lipogenesis system towards the latter is essential in the genesis of dyslipidemia, along with the increased transition of carbohydrates to fats [183]. The authors, in order to identify characteristic changes in the carbohydrate-fat metabolism system, examining lipidograms, revealed a tendency to decrease phospholipids, free cholesterol, triglycerides and to increase free fatty acids (FFA) and cholesterol esters, which indicates the inclusion of glucose in energy metabolism and a decrease in lipid consumption.

Similar data were obtained by G.I.Zaitseva et al., [67], when studying lipid metabolism in patients suffering from chronic eating disorders, in particular in children with hypostatic type dystrophy and paratrophy, a decrease in the level of total lipids, phospholipids and an increase in the concentration of FFA in blood serum was revealed.

Investigating the state of lipid metabolism in CG, CGD and DU, M.I.Borisenko [32] notes a violation of lipid metabolism in the early stages of the disease. In the period of exacerbation, the author established hypercholesterolemia, a decrease in the level of phospholipids, FFA, triglycerides (with an increase in free) and cholesterol esters. All these shifts in lipid metabolism are associated with impaired liver function, neuroendocrine regulation, changes in hydrolysis and absorption of fats. It should be noted that individual works are devoted to the correction of impaired lipid metabolism in CGDP [42, 89, 63, 157, 164, 168].

In chronic diseases of the gastrointestinal tract, carbohydrate metabolism also suffers, as evidenced by numerous works of researchers.

A.G.Oparin [155], in patients with DU, he found hypoglycemia, a decrease in the level of glycoproteins in the gastric mucosa and gastric juice, most pronounced with reduced acid-forming function of the stomach.

A.R.Zlatkina et al. [77], investigating the features of carbohydrate metabolism in patients with DU, come to the conclusion that disorders of carbohydrate metabolism are characterized by hyperinsulinemia with normal fasting glycemia, - the most pronounced in the acute stage.

L.N.Rybina et al. [169], conducting a standard glucose tolerance test (SGTT), 45 children with CGD revealed a tendency to fasting hypoglycemia, more pronounced in patients in the phase of incomplete clinical remission. Fasting glycemic indices in 39 children were below 80 mg%, of which 16 were below 70 mg%. The maximum glycemia after glucose loading in 31 patients was in the range of 120-170 mg%, in 1 - 175 mg%, in 13 - below 120 mg%. Maximum glycemia was more often observed at 30 minutes (32 patients), less often at 60 minutes (13 patients). The study showed that only 25.5% of patients with CGD have a normoglycemic type of the SGTT curve, 64.5% of patients have impaired glucose tolerance, which indicates, according to the authors, the expediency of periodic monitoring of SGTT in patients with CGD.

There are a sufficient number of works in the literature by researchers who have studied the level of glycemia, hydrolysis and absorption of carbohydrates, regulation of carbohydrate metabolism, the influence of emotional stress on carbohydrate metabolism in normal and in the pathology of the gastrointestinal tract [5, 6, 11, 13, 75, 77, 79, 83, 100, 101, 105, 106, 107, 154, 179, 181, 219, 226, 244].

In a comprehensive plan, studying carbohydrate metabolism and its hormonal regulation in the dynamics of glucose-tolerant and insulin-tolerant tests, M.A.Sklyarova [179] revealed a decrease in glucose and insulin tolerance in patients with DU and CG, which is associated with dishormonal shifts depending on the duration and severity of the course of the disease.

S.S.Galaeva [47] found in 72% of children with DU and 73% of children with CGD a decrease in the maximum increase in glycemia after oral lactose loading, as evidenced by low values of the hyperglycemic Baudouin coefficient, absorption and utilization coefficients. Similarly, the maximum increase in glycemia decreased after loading with monosaccharides (glucose ± galactose) in 56% of children with DU and in 63% of children with CGD. The excretion of D-xylose in urine was reduced in 85% of children with DU in the first 2 hours, in 78% - in 5 hours, and the lowest rates of D-xylose excretion were observed in multiple duodenal ulcers. Similar deviations of D-xylose excretion were observed in children with CGD, especially erosive.

V.V.Loboreva [120], in children with pathology of the gastroduodenal system revealed a low increase in sugars after exercise, which indicates a slowdown in hydrolysis and absorption of sugars. She noted a sharp decrease in the excretion of D-xylose in the urine.

A comprehensive examination of children with CGDP by B.K.Pchelin [163] showed a violation of the hydrolysis and absorption of sugars with functional disorders of the gastroduodenal system in 5.1%, with CGD in 21.3% and with DU in 41% of patients. Studies by E.G.Livshits [116] have shown more pronounced disorders of intestinal absorption function in children with DU, compared with CGD.

V.N.Toparskaya [192] revealed disorders of carbohydrate metabolism in diseases of the stomach, which was expressed in changes in the nature of glycemic curves after sugar loading. With hyperacid gastritis, there is a low curve with hypoglycemic lowering of sugar, with anacid gastritis. on the contrary, the sugar curve has a higher rise. On the other hand, hyperglycemia (of alimentary origin) inhibits gastric secretion, and hypoglycemia after insulin increases it. The mechanism of these interactions between sugar levels and gastric secretion, according to the author, is carried out through the parasympathetic nervous system. since they do not manifest themselves when the vagus is cut. A wide variety of sugar curves is observed in DU: low, flat, high, irritative or diabetoid curves. The author explains this by a disorder of the regulation of carbohydrate metabolism in connection with a violation in the central nervous system underlying the pathogenesis of peptic ulcer disease.

According to the observations of H.Meyers [256], with the rapid release of the stomach from glucose (for example, after gastric resection or with duodenal administration of sugar), the author received a high type of glycemic curve with a shortening of the duration of hyperglycemia and vice versa, with delayed gastric emptying (for example, with pylorostenosis), he observed a slowdown in hyperglycemia, that is, a protracted nature curve.

There are data in the literature regarding changes in lactic and pyruvic acids in CGDP in children. Thus, according to A.B.Asatryan et al. [13], the content of lactic acid in the blood of children with DU is increased by 30-40% of the level of healthy ones. Investigating the indicators of energy metabolism L.G.Komarova et al. [94], an increase in the concentration of lactic acid in the blood serum was found in children with CGD and with DU, the content of pyruvic acid did not deviate from the level of healthy. Aerobic glycolysis weakens in the body of patients and toxic metabolic products, including lactic acid, accumulate.

In the literature there are reports of a violation of protein-nitrogen metabolism in the pathology of the gastroduodenal system in children [4, 45, 60, 87, 126, 136, 166, 184, 197, 206, 207, 280]. Many of these

authors emphasize that diseases of the gastrointestinal tract in children are accompanied by minor hypoproteinemia, hypoalbuminemia, hypergammaglobulinemia and, consequently, dysproteinemia.

M.S.Abdullakhodzhaev et al. [4], hypoproteinemia was detected in 128 out of 156 children suffering from DU, and hypoalbuminemia was observed in 100% of patients. N.A.Klants [87] in patients with DU and CG, he revealed hypoalbuminemia, and in 83% of cases hypogammaglobulinemia.

A.V.Khodykin et al. [207], examining the indicators of nitrogen metabolism, found an increased content of amine nitrogen in blood serum in patients with DU and CGD, which averaged, respectively, 17.4± 0.65 mg% and 16.3± 0.43 mg% (normal - 11.3± 0.37 mg%). An increase in the level of tryptophan, a decrease in the content of lysine and leucine in the blood serum was revealed. The authors attribute the revealed shifts in nitrogen metabolism to disorders of the functions of the stomach and duodenum, the protein-forming function of the liver and increased catabolism.

In the acute stage, a significant decrease in the amount of histidine, proline, alanine, glycine, valine, tyrosine, threonine, a slight decrease in the content of methionine, phenylalanine, leucine, isoleucine and arginine is noted in the serum of I.I.Degtyarev and E.V. Solodov [60]. The amount of serine and glutamic acid remains within the normal range, and lysine, and especially aspartic acid, increases sharply.

Interesting data are presented in the works of A.V.Mazurin et al., [126, 132], G.K.Gulyaev [59], A.M.Zaprudnov [70], who studied the proteins of the "acute phase of inflammation" in CGD and DU. Thus, the level of prealbumin, haptoglobin, ceruloplasmin, β2-glycoprotein I, III, CRP increased, and the concentration of α1-glycoprotein, on the contrary, decreased. The increase in prealbumin, β2-glycoprotein I, III and CRP, a generally recognized marker of inflammation, was in a certain dependence on the severity of morphological changes in the gastric mucosa and duodenum. The authors suggest that the decrease in α1-acid glycoprotein containing a large amount of sialic acid (12.1%) is due to high acidification in CGD. The increase in haptoglobin, which is

a natural component of cathepsin B, is due to its release during inflammatory and destructive processes. The role of ceruloplasmin is determined not only by its participation in the synthesis of transferrin, but also in the catalysis of biologically active substances - histamine, serotonin, adrenaline, norepinephrine. Based on the data obtained, the authors consider these tests to be highly sensitive, indicating the severity of inflammatory and dystrophic mucosal processes in the examined patients.

All of the above contributes to some extent to a profound violation of the homeostasis of patients with CGDP. However, the true state of affairs is not fully known. In this regard, the study of gluconeogenesis, more precisely, the gluconeogenic function of the liver, which reflects the integrative relationship in the body of proteins, fats and carbohydrates, may be more informative. Unfortunately, this specific liver function has not been studied in CGDP, which gives us a reason to briefly present the features of glucose synthesis from non-carbohydrate compounds, in conditions of norm and pathology of organs and systems adjacent to the stomach.

As is known, normally, the formation of glucose from non-carbohydrate compounds plays an important role in maintaining the homeostasis of carbohydrate metabolism in the body: lactate, pyruvate, glycerin, glucogenic amino acids, etc. Up to 200 grams of glucose can be synthesized in a day in the body of a starving person in this way [234, 251]. However, gluconeogenesis as a process is necessary not only to maintain normal blood glucose levels in a starving body, but also to maintain normoglycemia during sleep, pregnancy, heavy physical work, insufficient intake of carbohydrates from the gastrointestinal tract, depletion of glycogen reserves in the liver and muscles with normal and especially excessive production of lactate and glycerin.

In other words, it is one of the fundamental processes aimed at providing the body with carbohydrates in conditions of norm and pathology. The synthesis of glucose from non-carbohydrate compounds occurs mainly in the liver and the cortical layer of the kidneys, partly in the mucous membrane of the gastrointestinal tract. The liver, by changing the rate of gluconeogenesis, in accordance with fluctuations in

the composition of the internal environment of the body, is able to effectively maintain normoglycemia. At the same time, the kidneys, in the conditions of liver shutdown, are also able to maintain blood glucose concentration within normal limits for a long time [114, 241, 243, 251].

Gluconeogenesis, as a process, is provided by four key enzymes: Glucose-6-phosphatase (G-6-Phase, 3.1.3.9.), Fructose-1,6-diphosphatase (F-1,6-DPase, 3.1.3.11.), Phosphoenolpyruvate Carboxy-kinase (FEPASE, 4.1.1.32.) and Pyruvate Carboxylase (PKase, 6.4.1.1.). The relationship between these and some other enzymes, intermediate compounds of the breakdown of proteins, carbohydrates and the endocrine system, ensures the synchronicity of all links of gluconeogenesis in the body.

Pyruvate plays a central role in the biosynthesis of glucose from non-carbohydrate compounds, which in the mitochondria of animal and human cells is carboxylated into oxaloacetate [270], under the influence of ATP-dependent PCase [249] in the presence of Mg, HCO and biotin. The latter, in the process of decarboxylation and phosphorylation under the influence of PHEPase, turns into phosphoenolpyruvate (PHEP), a compound with a high value of free standard energy. Such a workaround for pyruvate synthesis is considered to be the main one [285, 246, 247]. FEP through a series of reactions turns into fructose-1,6-diphosphate, which under the influence of F-1,6-dphase turns into fructose-6-phosphate. The latter thermodynamically irreversible reaction is catalyzed by glucose-6-phosphatase, as a result of which glucose and inorganic phosphorus are formed from glucose-6-phosphate [104. 114, 134, 142, 152, 194].

The course of gluconeogenesis reactions, changes in the activity of key enzymes, the role of activators and inhibitors in this process, shifts in *de novo* glucose synthesis under normal and pathological conditions are presented below in the works of domestic and foreign researchers [53. 84, 85, 216. 234, 236, 242, 284]. The main precursors of newly formed glucose are glucogenic amino acids. In the perfused liver of rats, a mixture of amino acids increases glucose synthesis in proportion to their concentration [33, 241, 242, 255].

The most important amino acid in this regard is alanine. K.Snell [279], J.Zaleski et al., [290], it was found that in rat liver sections the rate of gluconeogenesis is a linear function of alanine concentration. With an increase in the concentration of C-1-alanine from 0.3 to 1.0 mmol / l, this corresponds to the transition of rats from starvation to a state of normal nutrition, the inclusion of a radioactive label in glucose is accelerated 2.3 times. Alanine is effectively transformed into glucose and in the kidneys [224, 231, 254, 267].

This amino acid in physiological concentrations inhibits the activity of pyruvate kinase in the liver and kidneys, contributing to the switching of glycolysis metabolites to the pathway of gluconeogenesis [285]. In addition to alanine, other amino acids are easily converted into glucose. Thus, glycine, serine, asparagine and proline are able to significantly increase glucose production in the liver of rats in vitro. In isolated hepatocytes, phenylalanine, arginine, ornithine, tyrosine, and especially lysine, effectively stimulate the synthesis of glucose from lactate. Tryptophan, cysteine and histidine decrease, valine, methionine and glutamine do not change the rate of gluconeogenesis in rat hepatocytes [35, 237, 238, 245, 258, 262, 278, 286].

At the same time, excess glucose inhibits the processes of glycogenolysis and gluconeogenesis, while the processes of glycolysis and glycogenesis are enhanced [51, 253, 261, 264, 266, 281].

With a decrease in the concentration of glucose in the blood, the processes of glycogenolysis and gluconeogenesis are activated and the processes of glycolysis and glycogenesis are weakened [246, 271]. These metabolic shifts are observed even in conditions of short-term glucose deficiency [233, 268]. Among the activators of gluconeogenesis can be noted vasopressin, angiotensin and oxytocin [286], nicotinic acid [257], morphine [288, 289], acetylsalicylic acid [228], etc. On the contrary, de novo glucose synthesis rate inhibitors include ethanol [144], halothane [227], phenformin [238], tryptophan [265], digitonin [266], insulin, nonsteroidal anti-inflammatory drugs, cardiac glycosides, narcotic drugs, etc.

In recent years, the process of gluconeogenesis has been studied in various diseases in close connection with metabolic disorders. Thus, V.S.Shapot, V.A.Blinov [216], studying some aspects of the pathogenesis of tumor growth, for the first time established the relationship between the rate of biosynthesis of newly formed glucose and the level of glycemia in various tumors.

B.S.Mirzaev [144], N.R.Minullina [143] associate an increase in blood glucose in the first hours of observation in patients with acute myocardial infarction with an increase in the synthesis of this metabolite from non-carbohydrate compounds.

N.V.Blinova [29] observed shifts in gluconeogenic function in patients with chronic hepatitis and cirrhosis of the liver, which changed in strict accordance with the clinical picture. In patients with chronic active hepatitis, fasting blood glucose was always significantly higher than in chronic persistent hepatitis - 3.81±0.09 and 3.45±0.08 mmol/l, respectively ($P<0.001$). This turned out to be a consequence of a more intense neoplasm of glucose from glycerin in CAH than in CPH.

In addition, there are studies confirming the strengthening of gluconeogenic liver function in various conditions: in infants with pneumonia occurring against the background of hypotrophy and with "pure" hypotrophy [36], with stress [92], in patients with rheumatoid arthritis and deforming osteoarthritis [188], in patients with acute cholecystitis [14], with experimental hepatitis and liver cirrhosis in rats [201]. At the same time, in patients with infiltrative pulmonary tuberculosis [189] and in burned [85, 218] - gluconeogenesis is suppressed.

The chronic and often recurrent course of CGDP in children dictates the need to search for improving the therapy of patients with this pathology. In this, as A.A.Baranov notes [19], the use of biochemical research methods, which open up prospects for the development of specific therapy and prevention, is of great help.

As stated above, various metabolic disorders are observed with CGDP, in particular, disorders of protein, carbohydrate, and lipid metabolism, which indicate that methods of treating diseases of the

gastroduodenal system should be aimed at correcting impaired metabolic processes pathogenetically justified. Thanks to this turn of modern ideas about the treatment of patients with CGDP. it is possible to achieve a certain therapeutic effect.

Based on these ideas. sedatives, antispasmodics, antacids. vitamins, mineral waters, physiotherapy procedures, reparants, adsorbents, antibiotics. stimulants, etc. have recently been used for the medical treatment of patients with CGDP.

Currently, there are several groups of anti-ulcer agents with a specific mechanism of action: antacids and adsorbents; antisecretory drugs; cytoprotectors; antibacterial agents [210].

The most widely used in pediatric practice are antacid drugs that have the ability to neutralize hydrochloric acid (almagel, vikalin, gastropharm); antisecretory drugs: central and peripheral M-cholinoblockers (atropine, platyphylline, metacin, belladonna preparations, gangleron, quaterone, etc.), histamine H-receptor blockers (cimetidine, 2 ranitidine, famotidine), selective M-holinoblocker - gastrocepin; cytoprotectors: locally acting antipepsin agents - sucralfate (venter), antepsin; true cytoprotectors are synthetic analogues of prostaglandins [157]; substances with antipepsin and antibacterial activity (de-nol); opioid peptides of peripheral type of action (dalargin); antibacterial agents, etc. [46].

Colloidal bismuth subcitrate (de-nol) is currently considered the most effective anti-ulcer agent. Its high anti-ulcer efficacy is primarily associated with antibacterial action against Helicobacter pylori. There are numerous studies on the pathogenetic role of this microorganism in CG and DU [55, 56, 88, 98, 222, 276].

Other agents that have a pronounced antibacterial effect against Helicobacter pylori are metronidazole (trichopol), vikalin, furagin, a number of antibiotics (oxacillin, penicillin, erythromycin).

There are data in the literature on the effective use of mineral waters [64], antioxidants [89, 139], protein hydrolysates [60, 214], vegetable fats [42, 157, 191], biologics [161], infant formula, various dishes,

vitamins, neuroleptics, enterosorbents and others in the treatment of CGDP [141].

In particular, H.T.Khamraev [203], using enterosorbents (activated charcoal and pectin) in the complex therapy of children with CGD and DU, established their positive effect on redox processes and intestinal microbiocenosis. There are many works on the use of essentiale in liver pathology, enterocolitis, salmonellosis [50, 96, 138, 147, 177]. The work of E.A.Zhukova et al., [65] is devoted to the use of essentiale in the complex therapy of children with DU. The authors observed a positive clinical effect in 75% of patients, the process of lipid peroxidation improved, relapses decreased by 1.7 times compared to the control group.

In the literature available to us, we have not found any works devoted to the use of glycerin in the treatment of diseases of the gastroduodenal zone. There are isolated works on its use in violation of cerebral circulation, in liver diseases, its effect on hematopoiesis has been studied [15, 103, 149].

As is known, one of the main links in the complex treatment of CGDP, according to many authors, is diet therapy, which provides for mechanical, thermal, chemical treatment of the gastrointestinal mucosa. Thus, according to M.A.Samsonov [175], an anti-ulcer diet can stop the clinical symptoms of PBC in 86% of cases, not only that, the author states about the positive dynamics of protein and lipid metabolism in patients with this pathology.

The analysis of literary sources indicates that there are absolutely no works devoted to the complex correction of the functional state of the liver and impaired metabolic processes in children with CGDP. A review of the literature also reveals a lack of information on the correction of lipid, carbohydrate and nitrogen metabolism and gluconeogenesis in patients suffering from diseases of the gastroduodenal system - essentiale and medical glycerin. Therefore, the study of these issues and the development of effective therapies determines the relevance of the problem of modern gastroenterology of childhood. The stated motives served as a starting point for conducting a

clinical assessment of the functional state of the liver according to the data of gluconeogenic liver function, carbohydrate and nitrogen metabolism in children with CGDP.

In conclusion, it should be noted that gluconeogenesis is one of the important specific functions of the liver, with the defeat of the gastrointestinal tract has not been practically studied. At the same time, shifts in carbohydrate and nitrogen metabolism in children with CGDP have not been sufficiently studied. Meanwhile, a more in-depth study of the metabolism in CG, CGD and DU in children would allow the introduction of additional diagnostic, prognostic criteria into the clinic, which would contribute to the development of pathogenetically based therapy, and conduct more reliable monitoring of the effectiveness of the treatment.

Therefore, the study of these issues and the development of effective therapies determines the relevance of the problem of modern gastroenterology of childhood.

The stated motives served as a starting point for conducting a clinical assessment of the functional state of the liver according to the data of gluconeogenic liver function, carbohydrate and nitrogen metabolism in children with CGDP.

CHAPTER II. GENERAL CHARACTERISTICS OF THE EXAMINED PATIENTS AND METHODS OF THEIR RESEARCH

II.1. General characteristics of the examined patients.

To fulfill the tasks, 116 children with chronic GDP aged from 7 to 14 years who were on inpatient treatment in the children's department of the clinic No. 2 of SamMI were examined in dynamics. The control group consisted of 22 practically healthy children aged 11-13 years, from among the students of secondary school No. 1 in Samarkand. Of the 116 examined patients, there were 77 girls (66.4%), 39 boys (33.6%), 29 children aged 7-11 years, 87 12-14 years. CG was diagnosed in 34 (29.3%) patients, CGD - in 63 (54.3%) and DU - in 19 (16.4%). The age-sex composition of sick children is presented in Table 1, from which it can be seen that the number of girls is almost twice (1.97 times) predominant over boys. In the age aspect, out of the total number of patients, the majority (70.0%) were aged 12 to 14 years. There is an increase in morbidity with an increase in the age of children, which is noticeable in relation to CGD.

Table 1.

Distribution of examined patients by nosology, gender and age.

Groups surveyed	Total		Gender				Age			
			boys		Girls		7-11 years		12-14 years	
	N	%	n	%	n	%	n	%	n	%
CG	34	29,3	12	35,3	22	64.7	11	32,3	23	67,7
CGD	63	54,3	21	33.3	42	66,7	19	30,1	44	69,9
DU	19	16,4	6	31,6	13	68,4	6	31,6	13	68,4
Total with CGDP	116	100	39	33,6	77	66,4	36	31,0	80	69,0

Data on the seasons of the year and the prescription of the disease are presented in Table No.2. 40 children were examined in winter, 26 in spring, 17 in summer and 33 in autumn.

Table 2.

Distribution of the examined patients according to the season of the year and the prescription of the disease.

Distribution of patients by:		Examined patients							
		CG (n= 34)		CGD (n= 63)		DU (n= 19)		Total with CGDP (n=116)	
		N	%	n	%	n	%	N	%
Season of the year	Winter	12	35,3	20	31,7	8	42,1	40	34,5
	Spring	7	20,6	14	22,2	5	26,3	26	22,4
	Summer	6	17,6	11	17,4	-	-	17	14,6
	Autumn	9	26,5	18	28,7	6	31,6	33	28.5
Duration of the disease	Up to 1 year	19	55,9	30	47,6	5	26,3	54	46,6
	1-3 years	13	38,2	28	44,4	11	57,9	52	44,8
	More than 3 years	2	5,9	5	8,0	3	15,8	10	8,6

The prescription of the disease in children with CGDP in most cases (89%) ranged from one month to three years, of which in more than half of cases (54.0%) did not exceed 1 year, and in 8.6% of children, more than three years. All children were admitted to the hospital during the period of exacerbation of the disease. and 16 (13.8%) of them for examination and clarification of the diagnosis.

Along with the generally accepted clinical and laboratory research methods, a detailed examination of sick children was carried out, according to a specially developed map by the staff of the Department of Children's Diseases No. 1 SamMI, which allows taking into account

climatic, seasonal features, genetic factors, the results of instrumental and laboratory research methods.

Out of 116 patients, 69 (59.5%) children were previously on outpatient and inpatient treatment for diseases of the gastroduodenal system, 5 (4.3%) children were admitted to hospitals of another profile (surgical, helminthological, infectious) for examination. For the first time, 47 (40.5%) children applied for diseases of the gastroduodenal zone. In the referral form, the diagnoses most often appeared: acute and CG, CGD, DU and for examination. In 34 (29.3%) sick children, there was a discrepancy between the directional and clinical diagnoses.

Dental caries (22.2%), chronic tonsillitis (24.8%), adenopharyngosinusitis (6.0%), acute inflammatory respiratory diseases (5.4%), anemia (5.2%), polyhypovitaminosis (5.2%), pyelonephritis (4.6%), gallbladder dyskinesia (1.7%) were noted as concomitant diagnoses.

In 104 children, we conducted a genealogical survey for 3-4 generations along the ascending and lateral lines from the proband (Table 3). The burden of the family history of CGDP was 65.3%. The most common indication of hereditary burden was in children with CG (68.7%), compared with patients with DU (66.6%) and CGD (62.9%), and the I degree of kinship prevails over the II degree, almost 2.37 times.

The total number of sick relatives, including Proband, was 151 for 56 families. Of these 151 patients, female persons turned out to be 1.5 times more than male persons, and in case of DU, this ratio was 2.5: 1.

In addition to the above, secretory and acid-forming functions of the stomach were studied in 106 (91.3%) sick children. 108 (93.1%) were subjected to X-ray examination, endoscopy was performed in 85 (73.2%) sick children. Correction of violations of the functional state of the liver by hepatotropic agents was carried out in 80 (68.9%) patients. Catamnestic observation in the near future after discharge from the hospital was carried out on 38 children, of whom 6 (5.1%) were hospitalized again due to a re-exacerbation of the disease.

Table 3.

Genealogical analysis of pedigrees of sick children.

№	Analysis of relatives of children with CGDP	N	%
I.	Studied pedigrees	104	100
II.	Hereditary burden of gastroduodenal pathology	68	65.3
III.	The number of sick relatives	95	100
	- including, on the mother's side	16	16,8
	on the father's side	25	26,3
	Mother	20	21,1
	Father	15	15,8
	Brother	8	8,4
	Sister	11	11,6
IV.	Males	38	40,0
	Female	57	60,0
	M : W	1 : 1,5	
V.	I degree (father, mother, brothers, sisters)	26	29.9
	II degree (grandmother, grandfather, uncles, aunts)	11	12.6
	III degree (great-grandfather, great-grandmother, cousins, brothers)	2	2,3
	I+II degree	15	17,2
	I+III degrec	1	1,1
	II+III degree	1	1,1
VI.	I generation	12	13,8
	II generation	26	29,9

	III generation	6	6,9
	I + II generation	2	2,3
	II + III generation	5	5,7
	I + II + III generation	5	5,7

II.2. Research methods

The examined children were subjected to a thorough general clinical and laboratory-instrumental examination. The general clinical examination included the collection of anamnesis, genealogical analysis of pedigrees, anthropometry with an assessment of the physical development of children, objective data. Laboratory and instrumental methods included general and biochemical analyses of blood, urine, feces, examination of gastric and duodenal contents, X-ray and endoscopic examinations, etc.

The study of gastric contents was carried out in the morning, on an empty stomach, by fractional method. The volume of individual portions (in ml.), the concentration of free hydrochloric acid and total acidity (in titration units) in each portion were determined and, based on the data obtained, the absolute indicators of the acid-forming function of the stomach - the "flow rate" of hydrochloric acid for the lean, basal and stimulated phases of secretion were calculated, according to the nomogram of S.B.Korostovtsev.

Duodenal probing was carried out by the classical method. The X-ray examination of the stomach and duodenum was carried out according to the generally accepted method, after preliminary preparation of patients. The endoscopic examination was performed with an Olympus GFB-4 gastroduodenofibroscope, after preliminary premedication. Immediately before the introduction of the endoscope, the pharyngeal ring was anesthetized with a 1% solution of dicaine.

There are many methods for studying the synthesis of glucose from non-carbohydrate compounds. However, most of them are acceptable only in the experiment (tissue sections, organ perfusion, the use of

labeled compounds). To study gluconeogenesis in clinical conditions, we used the technique of N.V.Blinova.

We used glycerin as a glucose precursor. The method for determining the rate of glucose synthesis from non-carbohydrate compounds was as follows: in practically healthy children, as well as in patients with chronic CGDP, on an empty stomach, 3.0 ml of blood was taken from the ulnar vein, and then orally they received a single load - glycerin, at the rate of 0.5 mg / kg of weight (specific weight - 1.26; the dose was derived at the Department of Biochemistry of SamMI), which was dissolved in 200.0 ml of warm water. After 0.5, 1 and 2 hours in the capillary blood of the examined healthy and sick children, the glucose content was determined, and 3 hours after the load, 3.0 ml of blood was taken from the vein again. In the blood taken twice (on an empty stomach and 3 hours after exercise), the content of not only glucose, but also ammonia, urea and glutamine was determined. Examination of patients according to this method was carried out in dynamics: upon admission and discharge from the hospital.

The proposed technique made it possible to observe the dynamics of the conversion of a non-carbohydrate compound into glucose and, at the same time, the effect of glycerin, as a precursor of gluconeogenesis, on some indicators of nitrogen and carbohydrate metabolism in healthy children and patients with chronic CGDP.

The blood glucose content in children was determined by a highly specific enzymatic method in the glucose oxidase-peroxidase-orthotoluidine system. The principle of this method is that glucose is oxidized by air oxygen in the presence of the enzyme glucose oxidase (glucose: oxyreductase, 1.1.3.4.) with the formation of a colored compound during the reaction, the intensity of which is proportional to the concentration of glucose.

The rate of gluconeogenesis was calculated by the average hourly increase in glucose levels. The tolerance of peripheral tissues to glucose of gluconeogenic genesis was determined by calculating the difference between the initial and final data of blood glucose levels before and after glycerin loading.

The concentration of urea in the blood serum was determined by the diacetyl monoxime method according to S.B.Barker, a set of reagents from the company "La chema" [229]. The principle of the method is that urea forms a yellow staining with diacetyl monoxide. The color is stabilized by potassium persulfate and its intensity is proportional to the content of urea in the blood serum.

To determine ammonia and glutamine, we used a micrometer developed by A.I.Silakova et al. [176]. The method is based on the principle that free ammonia (or ammonia formed from glutamine) interacts with sulfuric acid, and the color of the ammonium sulfate solution is then manifested by Winkler reagent.

The optical density of the colored solutions was measured on FEK-56 with appropriate light filters and the thickness of the cuvette layer. The data obtained resulted in a true indicator using pre-constructed calibration curves. In the diagnosis of chronic gastroduodenal pathology in children, the classification of A.V.Mazurin (1985) was used.

All the digital data of our observations were processed statistically, according to a specially compiled computer program BK 0010-01 "Electronics" using the Student's reliability criterion [82, 153]. In addition, the correlation coefficient and confidence intervals of the average value were calculated [109]. The differences were considered significant at $P<0.05$.

II.3. Clinical characteristics of sick children.

As is known, the characteristics of patients with CGDP are diverse. However, the most characteristic complaints of children with chronic diseases of the stomach and duodenum, upon admission to the hospital, were abdominal pain. The vast majority of patients with CG (29 children, 85.3%) noted pain in the epigastric region, less often pain was in the epigastric and right subcostal regions (2 children), in the epigastric and paraumbilical zone in 1 and in the remaining 2 children - in other combinations.

The characteristics of the pain syndrome, depending on the nosological form of the disease, are presented in table 4, from which it can be seen that the pain was more often of a combined nature. The

combined nature of pain was found in CGD 2 times more often than in CG and more than 2.5 times than in DU. If 12 out of 19 patients with DU and 31 out of 63 children with CGD had complaints of the combined nature of pain, then in children suffering from CG, dull, aching prevailed.

Table 4

Pain syndrome in the observed patients.

№	Clinical symptom of	CG (n=34)		CGD (n=63)		DU (n=19)		Total with CGDP (n=116)	
		n	%	n	%	n	%	n	%
	I. Localization of pain:								
1	Epigastric region	29	85,3	-	-	-	-	29	25,0
2	Pyloroduodenal area	-	-	9	14,3	4	21,0	13	11,2
3	Epigastrium and right hypochondrium	2	5,9	7	11,1	2	10,5	11	9,5
4	Epigastrium and umbilical region	1	2,9	39	61,9	12	63,3	52	44,8
5	Other combinations	2	5,9	8	12,7	1	5,2	11	9,5
	II. The nature of pain								
6	Stabbing	-	-	12	19,0	2	10,5	14	12,1
7	Cramping	-	-	7	11,1	-	-	7	6,0
8	Dull, aching	18	52,9	3	4,7	-	-	21	18,1
9	Spilled	1	3,0	6	9,5	3	15,7	10	8.6

10	Combined character	15	44,1	31	49,2	12	63,3	58	50,0
11	Compressive	-	-	4	6,3	2	10,5	6	5,2
	III. Duration of pain								
12	Short-term (up to 30 min)	32	94,1	35	55,5	5	26,3	72	62,0
13	Long (30 min – 2 hours)	2	5,9	24	38,2	8	42,2	34	29,4
14	Permanent (more than 2 hours)	-	-	4	6,3	6	31,5	10	8.6
15	Related to food intake	20	53,8	42	66,7	10	57,7	72	62,1
16	Unrelated -//- - //-	6	17,6	9	14,3	7	36,8	22	19,0
17	For no reason	8	23,6	12	19,0	2	10,5	22	19,0
	IV. Time of pain occurrence								
18	Before meals	10	29,4	6	9,5	4	21,0	20	17,2
19	During or after a meal	8	23,6	34	53,9	6	31,5	48	41,3
20	- including, after rough food	7	20,5	20	31,7	4	21,0	31	26,7
21	Before and after meals	16	47,0	8	12,7	9	47,3	33	28,4
22	Night	-	-	6	9,5	14	73,7	20	17,2
23	Hungry	19	55,8	44	69,8	19	100	82	70,6
24	Moyningan rhythm of pain	6	17,6	17	27,0	10	52,7	33	28,4

Of the 116 patients, 72 (62.0%) had short-term pain syndrome, 34 (29.4%) had longer-lasting (from 30 minutes to 2 hours) and 10 (8.6%) had constant pain, and their pain intensity was different. Thus, pain syndrome of intense, non-intense and moderate intensity occurred, respectively. in 26 (22.4%), 39 (33,6%) and 51 (44.0%) patients, i.e., moderate intensity pain was 2 times more common than intense pain and 1.35 times more common than non-intense.

In children with CG, the pains were mostly short-term (94.1%) and non-intense (70.6%), whereas with CGD more often (57.2%) they had an average intensity, although in half of the cases their duration was short-term. In contrast, in the case of DU, the pain syndrome in every second child was intense and prolonged.

An analysis of anamnestic data presented in the same table showed that the majority of 72 (62.0%) sick children associate the appearance of pain with eating. In particular, 41.3% of children had pain syndrome during or after meals, including 31 (26.7%) pain increased after eating coarse, fatty (after pilaf) food, which was 1.5 times more common in CGD. Of 116 children with CGDP, the appearance and intensification of abdominal pain before meals occurred 2.4 times less often than after meals (17.2%, versus 41.3%). The rest of the patients had pain both before meals and after meals.

14 out of 19 children with DU complained of night pains. Hunger pains were noted in the anamnesis in 82 (70.6%) patients, including all children suffering from DU and in more than half of patients with CG (55.8%) and CGD (69.8%). Moyningan rhythm of pain (hunger-pain-eating-relief-hunger-pain..) It was detected in 33 (28.4%) patients, while being detected in half of cases among children with DU, its frequency in this disease exceeded that in CGD and almost 3 times (2.99 times) than in CG.

In most of the patients we observed, pain syndrome was combined with dyspeptic disorders, the frequency of which, depending on the nosology, is shown in Table 5. The table clearly shows that 86 (74.1%) patients had a decrease in appetite, and nausea, heartburn, belching occurred with almost the same frequency in every second child.

Vomiting was observed somewhat less frequently (43.1%). 75 children (64.6%) had irregular chairs, 56 of them (48.2%) complained of constipation, 11 (9.4%) patients had constipation disorder and 8 (6.8%) had unstable chairs. Irregular chairs, including a tendency to constipation, were found with the greatest frequency in DU than in CG and CGD. 29 patients complained of bad breath and 48 children complained of bitterness in the mouth, of which 29 (60.4%) were with CGD.

Table 5.

The frequency of dyspeptic disorders and symptoms of general intoxication, depending on the nosological form of the disease.

	Clinical symptoms:	CG (n=34)		CGD (n=63)		DU (n=19)		Total (n=116)	
		n	%	n	%	n	%	n	%
I. Dyspeptic disorders:									
1.	Decreased appetite	21	61,7	51	81,0	14	73.7	86	74,1
2.	Nausea	18	53.0	39	61,9	12	63,1	69	59,4
3.	Vomiting	16	47.6	23	36,5	11	58,0	50	43,1
4.	Heartburn	12	35.3	46	73,0	8	42,1	66	56,9
5.	Belching	16	47,6	34	54,0	12	63,1	62	53,4
6.	Irregular chair	15	44,1	43	68,2	17	89,1	75	64,4
7.	including, - constipation	13	38,3	29	46,0	14	73,7	56	48.2
8.	- unstable chair	1	2.9	6	9,5	1	5,2	8	6,8
9.	- change of constipation by diarrhea	1	2,9	8	12,7	2	10,2	11	9,4

II. Симптомы общей интоксикации:									
10	Headache and dizziness	11	32,3	36	57,1	9	47,3	56	48,2
11.	General weakness and fatigue	19	56,0	38	60,3	14	73,7	71	61,2
12	Irritabilit	3	8,7	12	19,4	7	36,8	22	18,9
13.	Rapid excitability	2	5,8	3	4,7	2	10,2	7	6,0
14.	Sweating	1	2,9	4	6,3	4	22,2	9	7,7
15	Restless sleep	2	5,8	4	6,3	3	15,8	9	7,7

An analysis of anamnestic data showed that in 54 (46.5%) children, the pain syndrome intensified after physical, mental stress, after excitement, in the remaining 62 (53.5%) children, with the combined effect of these factors.

In patients with CGDP, symptoms of general intoxication often accompanied the disease. Thus, general weakness, fatigue, malaise were found in 71 (61.2%) children, headache, dizziness - in 56 (48.2%), irritability - in 22 (18.9%), restless sleep and sweating in 18 (15.5%), rapid excitability in 7 (6.0%) children.

Among the examined patients, the overwhelming majority – 79 (68.1%) had not previously observed the correct diet. Of these, 52 (44.8%) children ate irregularly, ate more food late in the evening, which is due to national traditions, and this fact was revealed in every second patient with DU and CGD and almost every third - CG.

In addition, it was found out from the anamnesis that 27 (23.2%) patients had regular dry eating and fast, hasty eating. Abuse of rather hot food was noted in 7 children suffering from CG and in 6 - cold food.

An analysis of anamnestic data showed that 1/3 of the children's daily diet was dominated by dishes such as pilaf, roast, etc., which,

being greasy, coarse, often contributed to the appearance of heaviness and discomfort: 35 (30.1%) - baked and sweets, every seventh child - flour products. 40.8% of patients liked and quite often consumed salty, and almost every fifth child liked spicy and bitter. Almost 1/3 (32.5%) of patients with exacerbation of chronic CGDP had a seasonal character.

The general condition of the majority of patients (89.3%) was assessed as moderate, and the rest - as severe. An objective study of 116 children with CGDP, in every second patient (51.7%) revealed a reduced diet, in 44.9% - satisfactory and in 4 (3.4%) children it was elevated.

On examination, attention was drawn to the pallor (46.5%) and dryness (31.0%) of the skin, sometimes blue under the eyes.

In a significant majority (93.1%) of patients, the tongue was overlaid with a white or yellowish coating, in 7 (6.0%) patients atrophy of the papillae of the tongue was noted.

Palpation of the abdomen revealed an increase in the liver from 1.0 to 2.5 cm in 16 (13.7%) patients. Rumbling was noted in 8 children with CGD and DU, while palpation of the abdomen was noted, in 5 - moderate tension of the abdominal muscles.

The study of the neuropsychic sphere revealed emotional lability in most patients. 7 (6.0%) children were withdrawn, difficult to make contact.

No abnormalities were detected on the part of the respiratory organs.

In the study of the cardiovascular system, the vast majority of patients (76.7%) revealed muffled heart tones and often (7.7%) listened to systolic murmur at the apex of the heart.

All the examined patients underwent conventional laboratory tests of blood, urine, feces, both upon admission to the hospital and in the dynamics of the disease.

Analysis of hemograms showed that 1/4 of the patients had an acceleration of ESR. 13 children had moderate eosinophilia and 7 had a significant decrease in the number of erythrocytes and hemoglobin. On

the part of the genitourinary system, the phenomenon of pyelocetasia, moderate leukocyte and cylindruria was found in 3 patients.

During a coprological study, neutral fat was determined in the chair of 18 patients, fatty acids - in 8 children, muscle fibers - in 29, digestible - in 28 children and indigestible - in 60 children, vegetable fiber, from small amounts to significant, in most patients small detritus was detected, in 14 children - iodophilic flora, 27 patients have starch. Grigersen's reaction was positive in 18 cases.

In general, almost all of the listed clinical indicators were found in each nosological form of the disease, but with varying frequency, did not have strictly specific features for each pathology, and therefore could not be used as a diagnostic criterion. In this connection, for the purpose of differential diagnosis, additional laboratory and instrumental research methods were carried out: fractional examination of gastric juice, duodenal probing, gastrointestinal X-ray, gastroduodenofibroscopy and others.

The study of gastric juice by fractional method showed that out of 106 patients with chronic GDP, 73 (68.8%) patients have a hyperacid state, 23 (21.7%) have a normocid state and 10 (9.5%) patients have a hypoacid state of gastric juice.

The level of hydrochloric acid production in patients with CGDP in the basal and stimulated phases of secretion is 3.3 and 9.3 times higher, respectively, than in the lean portion.

Radiological data in 23 cases gave a discrepancy with the clinical diagnosis, which is associated with the identity of radiological signs of diseases of the gastroduodenal system.

The endoscopic picture in CG and CGD was characterized by superficial, hypertrophic and erosive changes in the mucous membranes of the gastroduodenal zone. Edema, hyperemia, and a defect of the mucous membranes ranging in size from 0.3 to 0.8 cm were found in DU. 1/3 of the examined patients had an increased tone of the upper gastrointestinal tract in the form of convoluted, poorly straightening folds and pyloric spasm.

Although the above methods are the most reliable and highly informative in the diagnosis of CGDP, we have tried to identify additional differential diagnostic criteria for CG, CGD and DU in the early stages of development by studying specific liver functions using special biochemical research methods, which we will discuss in more detail in the next chapter.

CHAPTER III. THE STATE OF GLUCONEOGENIC LIVER FUNCTION, SOME INDICATORS OF CARBOHYDRATE AND NITROGEN METABOLISM IN HEALTHY CHILDREN AND PATIENTS WITH GASTRODUODENAL PATHOLOGY

III.1. Some indicators of gluconeogenic liver function, carbohydrate and nitrogen metabolism in healthy children.

Currently, in the field of studying protein-nitrogen, carbohydrate and lipid metabolism, gluconeogenic liver function both in healthy and in a number of pathological conditions, there is quite extensive data. However, there is no information about the indicators of gluconeogenic liver function in children with CGDP in it.

In this regard, when starting to study some indicators of carbohydrate, nitrogen metabolism and gluconeogenesis in chronic GDP in children, it is necessary to have data on the content of complexes involved in the processes of gluconeogenesis, carbohydrate and nitrogen metabolism in normal.

We considered it expedient for a better comparison to obtain our own data on the content of glucose, ammonia, urea and glutamine in the blood of healthy children, before and after loading with glycerin, conducted under strictly identical conditions as in children of the same age suffering from CGDP.

The criteria for selecting healthy children were the absence of a history of intestinal infection, other diseases, as well as various dysfunctions and abdominal pain for 3-6 months before the examination and data that the child did not take medications during these periods. The conclusion about the child "Practically healthy" was made on the basis of anamnesis and objective research data. The children did not complain, had a steady appetite, and ate regularly.

Their physical development was assessed as average, above average and high, and their neuropsychiatric development corresponded to their age. All this met the requirements for the selection of the control group. Thus, the control group consisted of 22 practically healthy children - students of secondary school No. 1.Samarkand, aged 11-13 years, there

were 10 boys (45.5%). 12 girls (54.5%). To study the gluconeogenic function of the liver in healthy children on an empty stomach, the level of glucose in the blood was determined. Then, all children orally, as a load, took glycerin, at the rate of 0.5 mg / kg of weight dissolved in 200.0 ml of warm water.

As our studies have shown, their fasting blood glucose was 2.76±0.11 mmol/l (Fig.1). After 30 minutes after loading with glycerin, this level increased by 1.27 times (3.51±0.11 mmol/l; P< 0.001), after 1 hour the glucose level increased by 1.45 times and reached a maximum (4.02±0.10 mmol/l; P<0.001), then began to gradually decrease and by the third hour of observation was 2.94± 0.10 mmol/l.

These data indicate that in healthy children glycerin is effectively converted into glucose, and the increase in this newly formed glucose (the difference calculated between the glucose content in the blood after loading with glycerin on average per hour and the fasting glucose level) is on average 0.70 mmol/l/h. The tolerance of peripheral tissues to glucose of gluconeogenic genesis (the difference between the initial and final blood glucose levels) was 0.18 mmol/l.

After we found out the rate at which glycerin was converted into glucose in healthy children in the liver, we studied their changes in nitrogen metabolism, which are closely interrelated with the process of gluconeogenesis.

Shifts of some nitrogenous compounds in the blood of practically healthy children on an empty stomach and 3 hours after glycerin loading are also presented in Table 6, from which it can be seen that on an empty stomach the level of ammonia in healthy children was equal to 126.14±11.1 mmol/l, urea - 4.03± 0.13 mmol/l, and glutamine - 738.14±17.4 mmol/l. After 3 hours after loading with glycerin, these indicators decreased: - ammonia by 1.5 times (83.11±10.3 mmol/L; urea by 1.4 times (2.84±0.14 mmol/L; P<0.001) and glutamine by 1.03 times, which was 714.19±19.5 mmol/L (P>0.5).

We found that the introduction of glycerin leads to a statistically significant decrease in the blood levels of ammonia and urea in healthy children, as well as a slight decrease in the concentration of glutamine in

the blood. This may be a consequence of the fact that under the influence of glycerin, on the one hand, the processes of amination are intensified, and on the other, under conditions of an additional amount of glucose formed de novo, its nitrogen-sparing effect is more clearly manifested.

Based on the tasks set, after determining the normative indicators in practically healthy children, we conducted a similar examination of patients with CGDP.

III.2. The effect of glycerin load on the indicators of gluconeogenic liver function, carbohydrate and nitrogen metabolism in children with gastroduodenal pathology.

We examined 116 children with CGDP, of which 34 patients had CG, 63 had CGD and 19 had DU.

In patients with CG, when they were admitted to the hospital, the concentration of glucose in the fasting blood was higher than in the control group and was on average 2.91 ± 0.12 mmol/l (P>0.5). 30 minutes after loading with glycerin, as a factor exhibiting gluconeogenic liver function, the blood glucose level in patients increased by 1.13 times (P> 0.2) and reached a maximum by the second hour (3.95± 0.12 mmol/ L; P<0.01). By the end of the study (3 hours), the glucose level was 3.44±0.11 mmol/l (P<0.01) (Fig.1).

Figure 1. Dynamics of blood glucose in healthy children and patients with chronic gastritis, before and after glycerin loading. On the ordinate axis: on the left - the concentration of glucose in the blood in

mmol /L. The arow indicates the glycerin load. Vertical columns: hourly increase in blood glucose levels in healthy children and patients in mmol/l/h.

In this group of patients, the increase in newly formed glucose by 1, 2 and 3 hours of follow-up was 0.89, 1.04 and 0.53 mmol/l, respectively, averaging 0.82 mmol/l/h. The tolerance of peripheral tissues to glucose of gluconeogenic genesis was 0.53±0.05 mmol/l. These data suggest that the gluconeogenic function of the liver in patients with CG is enhanced compared to practically healthy children.

From the data given in Table 7, it can be seen that nitrogen metabolism is also disrupted in patients with CG: the concentration of ammonia ($P<0.05$) and urea ($P<0.001$) in their blood increases statistically significantly on an empty stomach, and the level of glutamine is 750.45 ± 1.10 mmol/l ($P>0.5$).

Table 7.

The effect of glycerin load on the content of nitrogenous compounds in healthy children (I) and in patients with CG (II).

Fasting	On an empty stomach		After 3 hours	
	I	II	I	II
Ammonia (in mmol/l)	126,14±11,1	153,0±7,89	83,11±10,5	93,9±5,15
P			<0,01	
P_1		<0,05		
P_2				<0,001
Urea (in mmol/l)	4,03±0,13	4,68±0,09	2,84± 0,14	4,11±0,07
P			<0,001	
P_1		<0,001		
P_2				<0,001
Glutamine (in mkmol/l)	738,14±17,4	750,45±11,0	714,19±19,5	738.8±15,0
P			>0,5	
P_1		>0,5		
P_2				>0,5

The concentration of nitrogenous fractions (ammonia, urea) underwent very significant changes in patients with CG after loading

with glycerin. Thus, the content of ammonia in their blood decreased by 59.1 mmol/l, urea - by 0.57 mmol/l, and glutamine by 11.65 mmol/l. A comparative analysis with the data of the control group showed that in patients with CG under the influence of glycerin, the concentration of ammonia in the blood decreases significantly more than normal, respectively, by 1.62 and 1.5 times. In other words, the administration of glycerin to CG patients caused a more significant decrease in the content of ammonia in the blood than in healthy children. These results indicate that glycerin should be used as a means to reduce the level of highly toxic ammonia in the blood of patients with CG.

As an example of the above, we give an extract from the medical history of a patient who was in the clinic under our supervision.

Patient Dilnoza N., case history No. 2880/1493, 9 years old, was admitted to the clinic with complaints of pain in the epigastric region, nausea, bitterness in the mouth, decreased appetite, lethargy. The child has been ill for 3 months. She has not been treated for this disease before. A girl from the first pregnancy and childbirth. She grew and developed in satisfactory material and living conditions. As a child, she suffered from viral hepatitis B, often had sore throats. 5 months ago I was treated in a hospital for a worm infestation. Heredity is not burdened.

Objectively: general condition of moderate severity. The skin is pale in color, clean. A girl of the right physique, low nutrition. Peripheral lymph nodes are not enlarged. Bone and joint system without deformities. Above the lungs there is a percussive – clear pulmonary sound, auscultative – vesicular breathing. The boundaries of the heart are within the age norm. Auscultation: heart tones are muted, there are no noises. The pulse is rhythmic, satisfactory filling and tension, 84 beats per minute.

The tongue is moist, overlaid with a whitish coating, the throat is pink. The abdomen is of the usual shape, soft, with deep methodological palpation according to the Obraztsov-Strazhesco method, soreness in the epigastric region is noted. The liver and spleen are not palpable. The

chair is regular, decorated, prone to constipation. Urinary system without features. Sleep is calm. The emotional state of the child is labile.

*Laboratory indicators: General blood test – Hb – 110.0 g/l, red blood cells – 3.6*1012 / l, color index – 0.9, white blood cells – 4.0*109 / l, SOE – 8 mm/h. Analysis of urine and feces without pathology. Biochemical blood tests: total bilirubin – 8.2 mmol/l, AsAT – 0.17 mmol/l, ALAT – 0.28 mmol/l.*

Analysis of gastric contents: the flow rate of free hydrochloric acid in the lean portion is 8.8 mg, in the basal portion – 40 mg, in the stimulated portion – 86 mg. Microscopy of the sediment – the nuclei of leukocytes – a small amount in mucus, the epithelium is flat – 002-1-002.

Fluoroscopy of the stomach and duodenum: the esophagus is freely passable. On an empty stomach is a secret. The folds of the mucosa are rough, covered with a secret. Stomach tone is reduced, peristalsis is alive. The gatekeeper is free to pass. The bulb of the duodenum with clear contours is completely emptied. Palpation is determined by soreness in the epigastric region.

Gastroduodenofibroscopy: the esophagus is freely passable, the gastric mucosa is smooth, pale pink in color, folds of medium size, fine-spotted areas of hyperemia are noted throughout. The gatekeeper is freely passable, the mucous membrane of the duodenal bulb is pale pink in color, the bulbous part of the duodenum is free.

Based on complaints, anamnesis, clinical, laboratory and instrumental data, the diagnosis was established: Chronic gastritis with increased secretory function of the stomach, the stage of exacerbation.

Special research methods: the patient's gluconeogenic liver function was determined. It was found that the fasting blood glucose level was 3.33 mmol/l, 30 minutes after the glycerin load it was 3.84 mmol/l, and after 1, 2 and 3 hours – 4.30, 4.49 and 3.87 mmol/l, respectively. On average, the increase in newly formed glucose in this patient was equal to 0.89 mmol/l/hour.

From this example, it follows that under the influence of glycerin, a patient with chronic gastritis formed more glucose than in practically healthy children, in whom its increase averaged 0.70±0.14 mmol/l/h, i.e. gluconeogenic liver function in this patient was increased. The tolerance of peripheral tissues was 0.54 mmol/l, which also exceeded the norm.

The content of ammonia in the blood 3 hours after glycerin loading in this patient decreased by 76.36 mmol/l and amounted to 99.8 mmol/l. The level of urea on an empty stomach was 4.80 mmol/l, after exercise, this indicator decreased by 0.42 and amounted to 4.38 mmol /l. The concentration of glutamine in the blood after exercise did not undergo significant changes.

We observed significant changes in the gluconeogenic function of the liver in patients with CGD.

It was found that the blood glucose level in 63 patients with fasting CGD was equal to 3.10±0.11 mmol/l. Compared with practically healthy children, these data were statistically significantly higher (2.76±0.12 mmol/l; $P<0.05$). 30 minutes after the glycerin load, the glucose concentration in the blood began to increase and gradually increased by the 2nd hour of the study to 4.24 ± 0.09 mmol/l ($P<0.001$). By the third hour of the study, their blood glucose remained higher than the baseline ($P<0.001$), but there was already a downward trend (Fig.2). The increase in glucose formed from glycerin in patients with CGD averaged 0.92 mmol/l/hour.

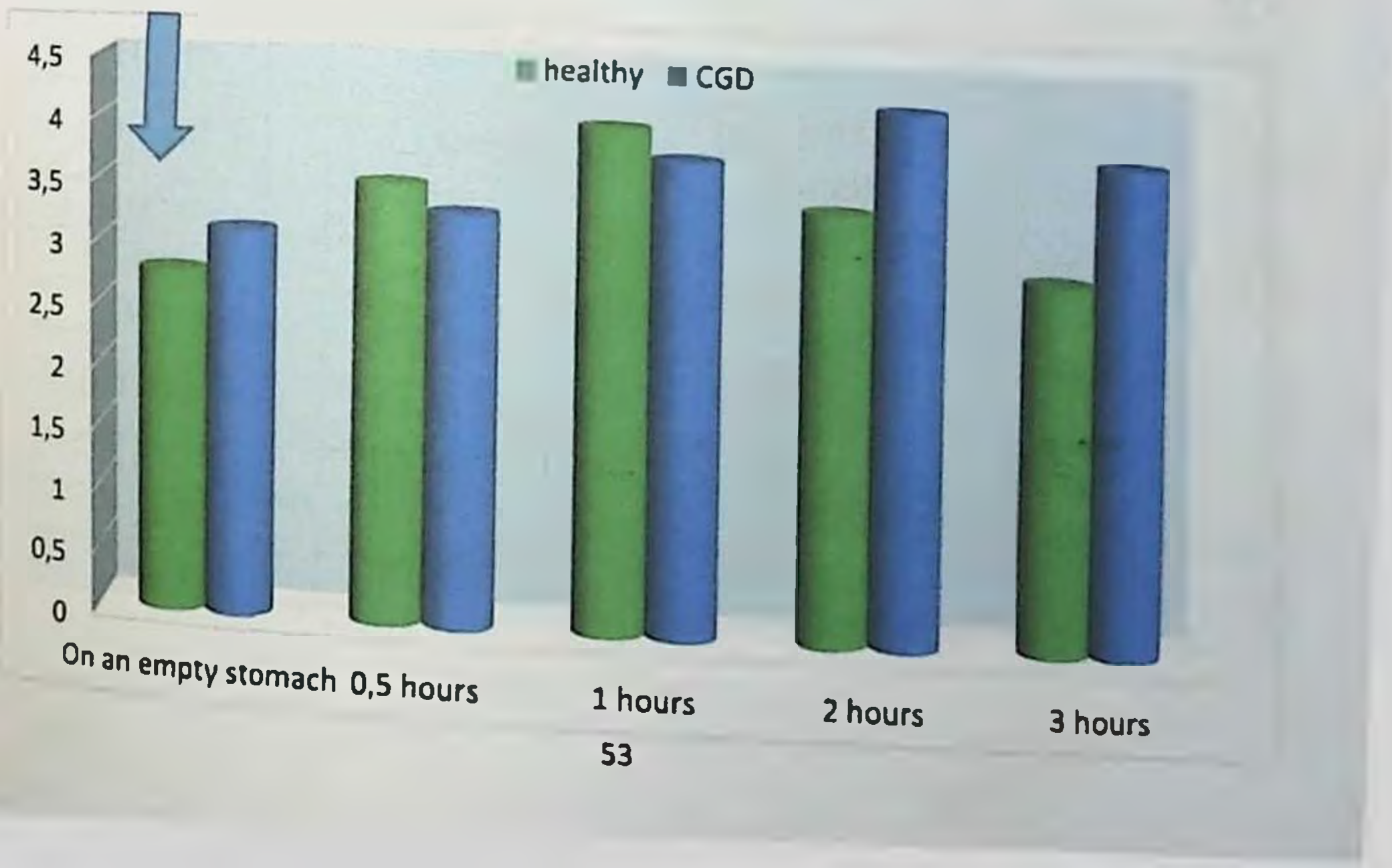

Figure 2. Dynamics of blood glucose in healthy children and patients with chronic gastroduodenitis after loading with glycerin. The remaining designations are as in Figure 1.

The tolerance of peripheral tissues to gluconeogenic glucose in patients with CGD was 0.76±0.06 mmol/L and was statistically significantly higher than in practically healthy children (0.18±0.018; P<0.001).

Summarizing the results presented above, it can be noted that the liver of patients with CGD synthesizes glucose from glycerol 1.3 times more than the liver of practically healthy children.

As for nitrogenous compounds in the blood (Table 8), in patients with fasting CGD, the ammonia content was significantly higher than in the control group (126.14±11.1 mmol/l; P<0.001).

Table 8.

The effect of glycerin on the content of nitrogenous compounds in the blood of healthy children and patients with CGD.

Indicators	Examined children	Healthy	Patients with CGD
Ammonia (in mmol/l)	On an empty stomach	126,14±11,1	208,3±7,3
	P		<0,001
	After 3 hours	83,11±10,5	145,1±8,4
	P		<0,001
Urea (in mmol/l)	On an empty stomach	4,03±0,13	4,78±0,08
	P		<0,001
	After 3 hours	2,84±0,14	4,42±0,09
	P		<0,001
Glutamine (in mkmol/l)	On an empty stomach	738,14±17	788,6±6,0
	P		<0,01
	After 3 hours	714,19±19,5	754,5±5,7
	P		<0,05

Note: P - the reliability of the differences between the indicators in the group of patients with CGD and in healthy children.

The level of urea in patients with fasting CGD was 4.78± 0.08 mmol/l. This is 0.75 mmol/l higher than the concentration of this metabolite typical for practically healthy children (P<0.001). The high content of ammonia, apparently, potentiates the synthesis of glutamine in patients with CGD, the content of this usually little-changing compound in patients was statistically significantly higher (P<0.01) than normal.

Glycerin load caused a decrease in the blood content of ammonia and urea in patients with CGD, respectively, by 63.2 mmol/l and 0.36 mmol/l. Moreover, the decrease in ammonia was more significant (30.3%) than urea (7.5%). These data may indicate a more intensive excretion of ammonia in the urine, an increase in the process of reducing amination, slowing down the catabolism of tissue proteins. The content of glutamine in the blood of patients with CGD under the influence of glycerin decreased slightly and amounted to 34.1 mmol/l (4.3%).

So, we found that patients with CGD have increased gluconeogenic liver function, compared with practically healthy children. In addition, it was established for the first time that glycerin has pronounced hypoammonium and hypoureoemic effects in patients with CGD. Consequently, the use of this load, on the one hand, makes it possible to adequately judge the gluconeogenic function of the liver in patients with CG and CGD, and on the other - to use glycerin as a means of reducing the content of ammonia and urea in the blood in the pathology of the gastrointestinal tract in children.

In confirmation of what we have stated above, we give a brief extract from the medical history.

Patient Dilafruz R., case history No. 2189/935, 14 years old, was admitted to the clinic with complaints of pain in the epigastric region immediately after eating, nausea, vomiting, belching air, constipation, decreased appetite, weakness.

From the anamnesis, the child has been ill for 1 year, she has not been treated for this disease before. At the age of 2, she suffered from

viral hepatitis A. As a child, she was often ill with colds. It was found out that the father suffers from chronic gastritis.

Objectively: general condition of moderate severity. The skin is pale in color, clean. A girl of moderate nutrition. Peripheral lymph nodes are not enlarged. Bone and joint system without deformities. Breathing through the nose is free. There is a percussive – clear pulmonary sound above the lungs, vesicular breathing is heard auscultatively. The heart tones are muted, there are no noises. The pulse is rhythmic, satisfactory filling and tension, 78 beats per minute.

The tongue is moist, overlaid with a whitish coating. The throat is pink, the pharyngeal tonsils are not enlarged. The abdomen is of the correct shape, soft, with palpation by the Obraztsov-Strazhesco method, soreness is noted in the epigastric and umbilical regions. The liver and spleen are not palpable. Chair once every 2-3 days. Urinary system without features. The girl is calm, sleep is not disturbed.

General tests of blood, urine and feces without features. Biochemical analyses without pathology.

Analysis of gastric contents: the flow rate of free hydrochloric acid in the lean portion is 4.5 mg, in the basal portion – 25.2 mg, in the stimulated portion – 87.6 mg. There is mucus in all portions. Microscopy of the sediment: leukocytes in mucus in large numbers, the epithelium is flat – single in the field of view.

Duodenal probing: in portion "A" epithelium in large quantities, leukocytes 10-12 in the field of vision.

Roentgenoscopy of the stomach and duodenum: we pass the esophagus freely, the contents of the secret in the stomach on an empty stomach, the folds of the mucous membrane are rough, with a tight contrast filling, the stomach is shaped like a hook, with smooth, clear contours, peristalsis is good, the pylorus canal is freely passed. The bulb of the duodenum is triangular in shape, the contours are even. Palpation is determined by soreness in the epigastric and pyloroduodenal areas.

Gastroduodenofibroscopy: the esophagus is freely passable, the gastric mucosa is smooth, pale pink in color, fine-spotted areas of

hyperemia are noted throughout. The gatekeeper is freely passable, the mucosa of the duodenal bulb is hyperemic, slightly edematous, the bulbous part of the duodenum is free.

Clinical diagnosis: Chronic gastroduodenitis with increased secretory function of the stomach, acute stage.

The gluconeogenic function of the liver was studied in the patient. It was found that the fasting blood glucose content is 3.59 mmol / l, 0.5, 1, 2 and 3 hours after the administration of glycerin, respectively, 3.91, 4.24, 4.64 and 3.91 mmol / l. The increase in newly formed glucose was 0.67 mmol/l/hour, the tolerance of peripheral tissues was 0.32 mmol/l.

The content of ammonia, urea and glutamine in the fasting blood was 176.1 mmol/l, 4.32 mmol/l and 801.88 mmol/l, respectively. After loading with glycerin, the level of ammonia decreased by 102.7 mmol/l, urea – by 0.99 mmol/l, glutamine concentrations decreased slightly, by only 35.30 mmol/l.

Thus, we observe in this example a slight increase in glucose, a significant decrease in the concentration of ammonia (by 58.3%) and urea (by 22.9%) under the influence of glycerin in the blood, and a slight decrease in the level of glutamine (by 4.4%).

Studying the gluconeogenic function of the liver in patients with DU, we revealed its more significant changes compared with practically healthy children with CG and CGD.

It was found that in patients with DU, upon admission to the hospital, the concentration of glucose in the blood on an empty stomach was higher than in the group of healthy children and was on average 3.00± 0.09 mmol/l (P>0.1). 30 minutes after glycerin loading, the blood glucose level in patients with DU began to increase and was equal to 3.40 ± 0.09 mmol/l (P<0.01). Subsequently, it was kept above the level determined on an empty stomach (Fig.3). The increase in newly formed glucose by 1, 2 and 3 hours of observation was 0.90, 1.26 and 1.20 mmol/l, respectively, averaging 1.12 mmol/l/h, which is statistically significantly higher than the rate of gluconeogenesis in the control group (0.70±0.14 mmol/l/h; P<0.01).

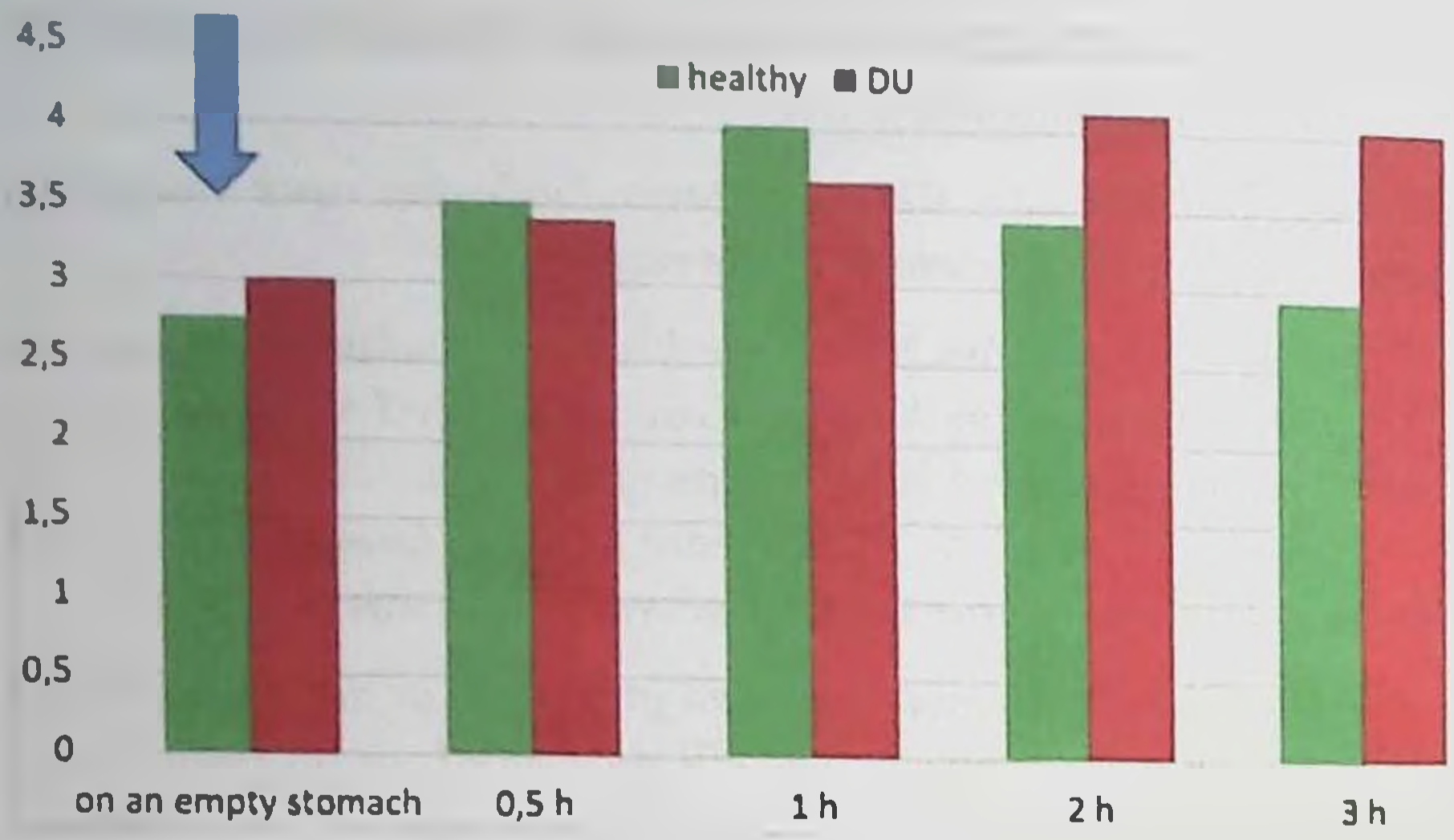

Figure 3. Dynamics of blood glucose in healthy children and in patients with duodenal ulcer disease after glycerin loading. The remaining designations are as in Figure 1.

In this group of patients, newly formed glucose penetrated the peripheral tissues more intensively than in practically healthy children by 6.6 times and was equal to 1.20± 0.09 mmol/l (P<0.001).

So, according to the results presented above, it can be noted that the liver of patients with DU synthesizes glucose from glycerol 1.6 times more than the liver practically healthy children, 1.36 times more than the liver of patients with CG and 1.22 times more than CGD.

After that, we studied the changes in nitrogen metabolism in patients with DU. As can be seen from Table 9, the content of ammonia in fasting blood in patients with DU was 228.5±8.5 mmol/ l, and was statistically significantly higher than in practically healthy children (P<0.001).

Table 9.

The effect of glycerin load on the content of nitrogenous compounds in the blood in healthy children and in patients with duodenal ulcer.

Indicators	Examined children	Healthy (n=22)	Patients with DU (n=19)
Ammonia (in mmol/l)	On an empty stomach	126,14±11,1	228,51 ± 8,5
	P		<0,001
	After 3 hours	83,11±10,5	163,8 ± 5,5
	P		<0,001
Urea (in mmol/l)	On an empty stomach	4,03±0,13	5,04 ± 0,069
	P		<0,001
	After 3 hours	2,84±0,14	4,66±0,10
	P		<0.001
Glutamine (in mkmol/l)	On an empty stomach	738,14±17	827,79±6,8
	P		<0,001
	After 3 hours	714,19±19,5	772.32±4.9
	P		<0,001

Note: P is the reliability of the differences between the indicators in the group of healthy and sick children.

The content of urea in the blood on an empty stomach was 5.04± 0.069 mmol/l, which is 1.01 mmol/l higher than its concentration in practically healthy children (P< 0.001). As for glutamine, its content in the blood of patients with fasting DU was also increased compared to practically healthy children (P<0.001).

As in our previous observations, glycerin load in patients with DU caused a decrease in the blood content of ammonia, urea and glutamine, respectively, by 64.71 mmol/l, 55.47 mmol/l and 0.38 mmol/l. Moreover, the most significant reduction was in ammonia (28.3%) than in glutamine (6.7%) and urea (7.5%).

These data, as in patients with CG and CGD, indicate intensive excretion of ammonia in the urine and an increase in the process of restorative amination, a slowdown in the catabolism of tissue proteins in patients with CGDP.

The revealed hyperammonio- and hyperureoemia in patients with DU, in our opinion, indicates a pronounced violation of nitrogenous homeostasis, increased formation of nitrogenous slags in the body and increased urea formation in this category of patients.

As an example to the above, we will give an extract from the medical history:

Patient with Dilorom S., 14 years old, medical history No. 1200/654. She was admitted to the clinic with complaints of nocturnal, hungry stabbing pains in the epigastric region, nausea, vomiting, heartburn, decreased appetite, weakness. The girl has been ill for 1 year. 7 months ago, she was treated at the SamMI clinic No. 1 for duodenal ulcer. The girl from V-pregnancy and childbirth, in childhood often had sore throats, colds.

Objectively: general condition of moderate severity. The skin and visible mucous membranes are pale, subcutaneous fat is moderately developed. Breathing through the nose is free. Percussion over the lungs - a clear pulmonary sound, auscultative vesicular breathing is heard. The heart tones are muted, there are no noises, the pulse is rhythmic, medium filling and tension, 82 beats per minute.

The tongue is moist, overlaid with a white coating. Palatine tonsils are hypertrophied. There are carious teeth in the oral cavity. The abdomen is soft, with palpation there is pain in the epigastric region. The liver and spleen are not palpable. The chair is regular, there is a tendency to constipation. Urinary system without features.

General tests of blood, urine and feces without features.

Examination of gastric contents: the flow rate of free hydrochloric acid in the lean portion is 10.2 mg, in the basal portion – 53 mg, in the stimulated portion – 83.2 mg. In all portions, mucus and leukocytes are in large quantities, the epithelium is flat – single in the field of vision.

Fluoroscopy of the stomach and duodenum: the esophagus is freely passable. The folds of the mucosa are coarse, thick, filled with a secret, with a tight contrast filling, the stomach is shaped like a hook with smooth and clear contours. Peristalsis is alive. The gatekeeper is passing through. There is a persistent barium spot (niche) on the surface of the duodenal bulb. On palpation, soreness is noted.

Gastroduodenofibroscopy: the esophagus is freely passable. The gastric mucosa is pale pink in color, folds of medium size. The gatekeeper is passing through. There is an ulcer in the bulb of the duodenum, rounded in shape, up to 0.4 cm in diameter, the bottom of the ulcer is covered with a grayish coating, the edges are edematous, hyperemic. The bulbous part of the duodenum is free.

The conclusion of the ENT doctor is chronic decompensated tonsillitis. Dentist's conclusion – dental caries. Ultrasound – liver, gallbladder, pancreas without features.

Clinical diagnosis: the main one is duodenal ulcer with increased secretory function of the stomach, the stage of exacerbation; concomitant - chronic decompensated tonsillitis, dental caries.

The gluconeogenic function of the liver was studied in the patient. The fasting blood glucose content is 2.93 mmol / l, 0.5, 1, 2 and 3 hours after the application of glycerin, respectively, 3.06, 3.59, 4.35 and 4.24 mmol / l. The rate of gluconeogenesis or the increase in newly formed glucose in this patient was equal to 1.13 mmol/l/hour, the tolerance of peripheral tissues to newly formed glucose was 1.31 mmol/l.

The content of ammonia in the blood on an empty stomach was equal to 252.5 mmol / l, and 3 hours after loading with glycerin decreased to 176.1 mmol / l. The level of urea in the blood before the load was 5.24 mmol / l, and 3 hours after the application of glycerin was equal to 4.99 mmol / l. The concentration of glutamine in the blood on an empty stomach was 839.54 mmol/l, after loading with glycerin it also decreased to 789.84 mmol/l.

In this patient, there is an increase in the rate of gluconeogenesis, i.e., a more intensive formation of glucose from glycerin and a high need for peripheral tissues in it. Hyperammonemia was also noted, due to the

destruction of the mucosa, and increased urea formation - as a mechanism for neutralizing ammonia.

So, we found that in patients with DU there is an increase in the gluconeogenic function of the liver, which is reflected by a significant transformation of glycerin into glucose, an increase in the tolerance of peripheral tissues to glucose of gluconeogenic genesis. As for changes in nitrogen metabolism, hyperammonemia is characteristic of patients with DU, apparently as a consequence of increased protein breakdown and subsequent deamination of amino acids. This toxic metabolite is successfully neutralized in practically healthy individuals in the form of urea and glutamine, whereas in patients with CG, CGD and DU - mainly in the form of urea. We believe that the glutamic acid-glutamine system in these patients does not work as effectively in ammonia neutralization as in healthy children.

III.3. Characteristics of gluconeogenic liver function in patients with gastroduodenal pathology, depending on the duration of the disease.

Studying the functional state of the liver according to some indicators of carbohydrate and nitrogen metabolism, according to the gluconeogenic liver function in children with CGDP, and having found certain metabolic changes, we were interested in their dependence on the clinical manifestations of the disease: the duration of the disease, the activity of the pathological process and the state of acidity of gastric juice.

Depending on the duration of the disease, as indicated in the second chapter (Table 2), all patients with CGDP were divided into 3 groups. The first group included patients with a disease duration of up to 1 year, the second - those who have been ill for 1-3 years, and the third - over 3 years. Since the number of patients with a disease prescription of more than 3 years was small, which created difficulties for statistical processing of the data obtained, we found it necessary to compare patients with a disease prescription of up to 1 year (group I) and 1 year or more (group II).

We found that in patients with CG in the second group, the fasting blood glucose content was on average 3.16±0.09 mmol/L, which is significantly higher than in patients of the first group (P<0.02).

Our further observations showed that all the time after glycerin loading. the increase in newly formed glucose, as and the content of previous glucose in the blood remained higher in patients of the second group (Fig.4).

From the data presented in Figure 4, it follows that in patients with CG with a disease duration of up to 1 year, the increase in newly formed glucose, after loading with glycerin, averaged 0.76 mmol/l/h, and in patients of the second group, the rate of gluconeogenesis was 0.91 mmol/l/h (P<0.05). Tolerance of peripheral tissues in patients of group I it was 0.42, and in the second group it was slightly higher (0.55 mmol/l).

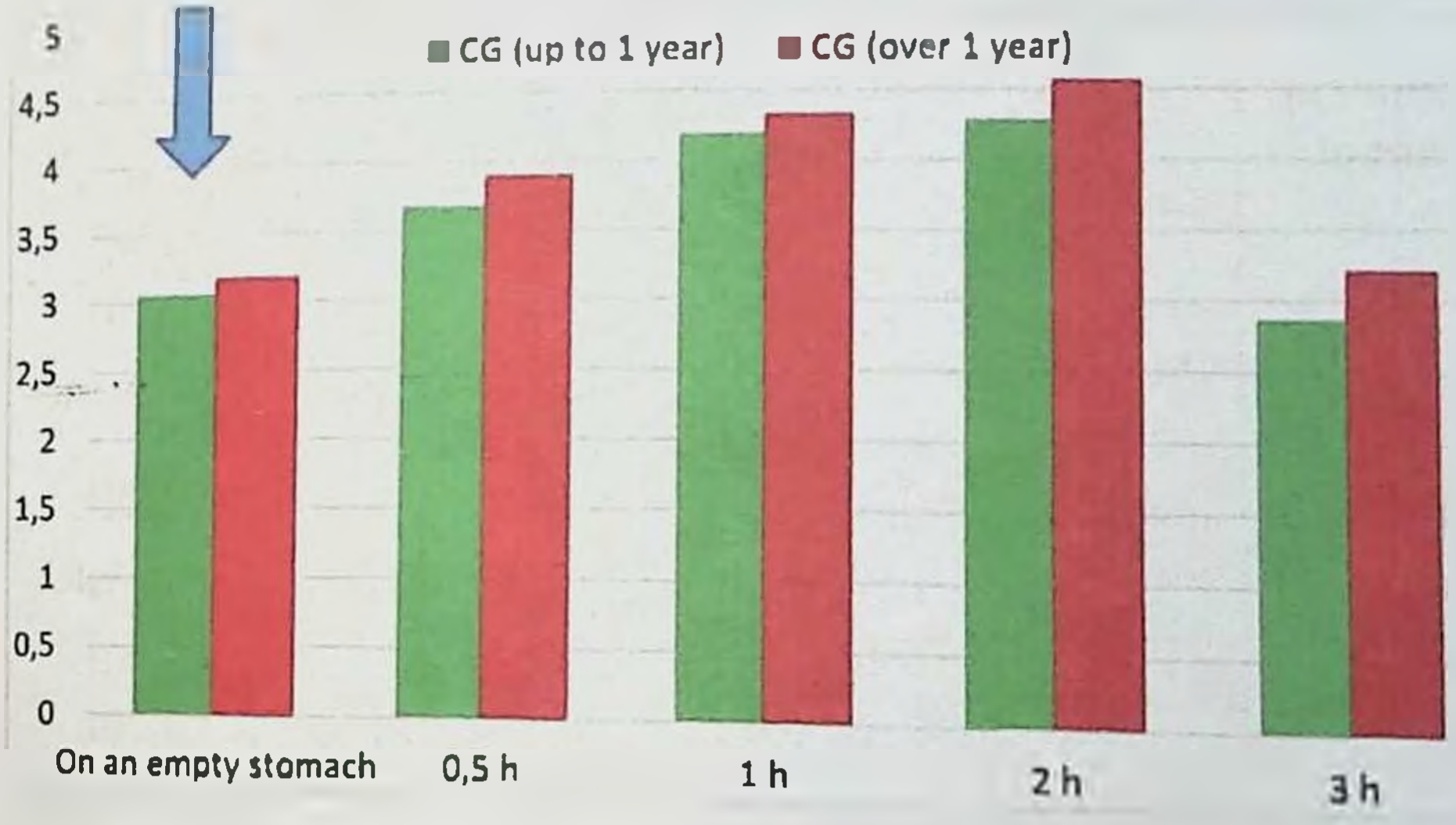

Figure 4. Dynamics of blood glucose in children with chronic gastritis with a disease duration of up to 1 year and over 1 year. The remaining designations are as in Fig.1.

The results obtained indicate that with an increase in the duration of the disease, the intensity of gluconeogenic liver function in patients with CG increases.

Let us now proceed in a similar sequence to discuss the shifts in nitrogen metabolism in patients with CG, depending on the prescription

of the disease. The level of ammonia in the blood of patients with CG, with a disease duration of more than 1 year (group II) on an empty stomach, was significantly higher than its level in patients of group I, respectively, 172.28±8.32 and 134.42±6.88 mmol/l (P<0.001) (Table 10).

Table 10.

The content of nitrogenous compounds in the blood of patients with chronic gastritis, depending on the duration of the disease.

Indicators	Examined children	Healthy (n=22)	Patients with CG (n=34)	
			to 1 year (n=19)	over 1 year (n=15)
Ammonia (in mmol/l)	On an empty stomach	126,14±11,1	134,42±6,88	172,28±8,32
	P		>0,5	<0,001
	After 3 hours	83,11±10,5	104,06±6,26	134,42±5,37
	P_1		>0,1	<0,001
	P_2	<0,05	<0,001	<0,001
Urea (in mmol/l)	On an empty stomach	4,03±0,13	4,46 ± 0,09	4,82 ± 0,08
	P		<0,01	<0,001
	After 3 hours	2,84±0,14	4,20 ± 0,08	3,91 ± 0,07
	P_1		<0,001	<0,001
	P_2	<0,001	<0,05	<0,001
Glutamine (in mkmol/l)	On an empty stomach	738,14±17	742,36±12,4	760,48±13,2
	P		>0,5	>0,5
	After 3 hours	714,19±19,5	746,5±11,1	726,62±11,6
	P_1		>0,2	>0,5
	P_2	>0,5	>0,5	<0,05

Note: where P is the reliability of differences in relation to the corresponding group of healthy children on an empty stomach; where P1 is in relation to the level of healthy children after exercise; P2 is in relation to the corresponding group before and after glycerin load.

We noted that with an increase in the duration of the disease, the body of CG patients is increasingly exposed to the toxic effects of ammonia, the level of which in the blood of children of the second group was 1.28 times higher than in the first. After glycerin loading, the content of ammonia in the blood of patients with a prescription of the disease up to 1 year decreased by 36.7%, and in patients of group II, by only 22.5%. This indicates the resistance of the body of long-ill children to the hypoammonemic action of glycerin.

As for urea, in patients of group I and II before loading with glycerin. its content in the blood, depending on the duration of the disease, fluctuated within narrow limits, and averaged 4.46± 0.09 and 4.82±0.08 mmol/l, respectively. Under the influence of glycerin, the level of urea in the examined groups of children decreased, and in patients of the second group, this decrease was more intense, compared with patients of the first group, by 3.5 times (0.91 and 0.26 mmol/l).

The level of glutamine in the blood, both before and after loading with glycerin, did not change significantly in both groups. It follows from this that the neutralization of ammonia in the body of HCG patients occurs mainly due to urea formation, and with an increase in the prescription of the disease, this process increases.

Let's back up these average statistical data with concrete examples. To do this, we will briefly give a few observations, focusing only on the passport part of the medical history, the prescription of the disease and the special studies we conducted:

Patient M., 14 years old, medical history No. 2086/902. Diagnosis: main – Chronic gastritis with increased secretory function of the stomach, the stage of exacerbation; concomitant – Chronic decompensated tonsillitis. Tooth decay. He has been ill for 6 months.

Patient Z., 13 years old, medical history No. 1958/1148. Diagnosis: main – Chronic gastritis with unchanged secretory function

of the stomach, the stage of exacerbation; concomitant – Chronic decompensated tonsillitis. He has been ill for 4 years.

The increase in glucose in these patients after administration of glycerin was, respectively, 0.53 and 1.01 mmol / l / hour. In a patient with a 6–month prescription of the disease, the decrease in the concentration of ammonia in the blood after loading with glycerin was 25.2%, urea – 8.2%, and in a patient with a 4-year prescription of the disease, respectively - 43.3% and 18.6%. The content of glutamine in patients with different duration of the disease did not undergo significant changes.

So, in children suffering from CG, we found a direct correlation between the duration of the disease and profound metabolic disorders, in the form of an increase in the level of ammonia (r= 0.79) and urea (r= 0.84) in the blood. With the increase in the prescription of the disease, there is an increase in the glucose-forming function of the liver in patients with CG, and also, there is a more intense breakdown of tissue proteins, which leads to hyperammonemia and an increase in the ornithine cycle of urea formation. The glutamic acid-glutamine system, as expected, does not undergo significant changes.

After the dependence of the gluconeogenic function of the liver and some indicators of nitrogen metabolism in patients with CG on the duration of the disease has been determined, we proceed to consider this issue in patients with CGD. As noted earlier in Chapter II (Table 2), all patients with CGD, depending on the prescription of the disease, were divided into three groups: the first group – with a prescription of the disease up to 1 year - 30 children; the second - with a prescription of 1-3 years - 28 children; the third group - sick over 3 years - 5 children. But, as mentioned earlier in the case of CG, for a more reliable assessment of the rate of gluconeogenesis and changes in nitrogen metabolism, we found it necessary to divide all patients with CGD into two groups: the first - with a disease duration of up to 1 year and the second - over 1 year (Fig.5).

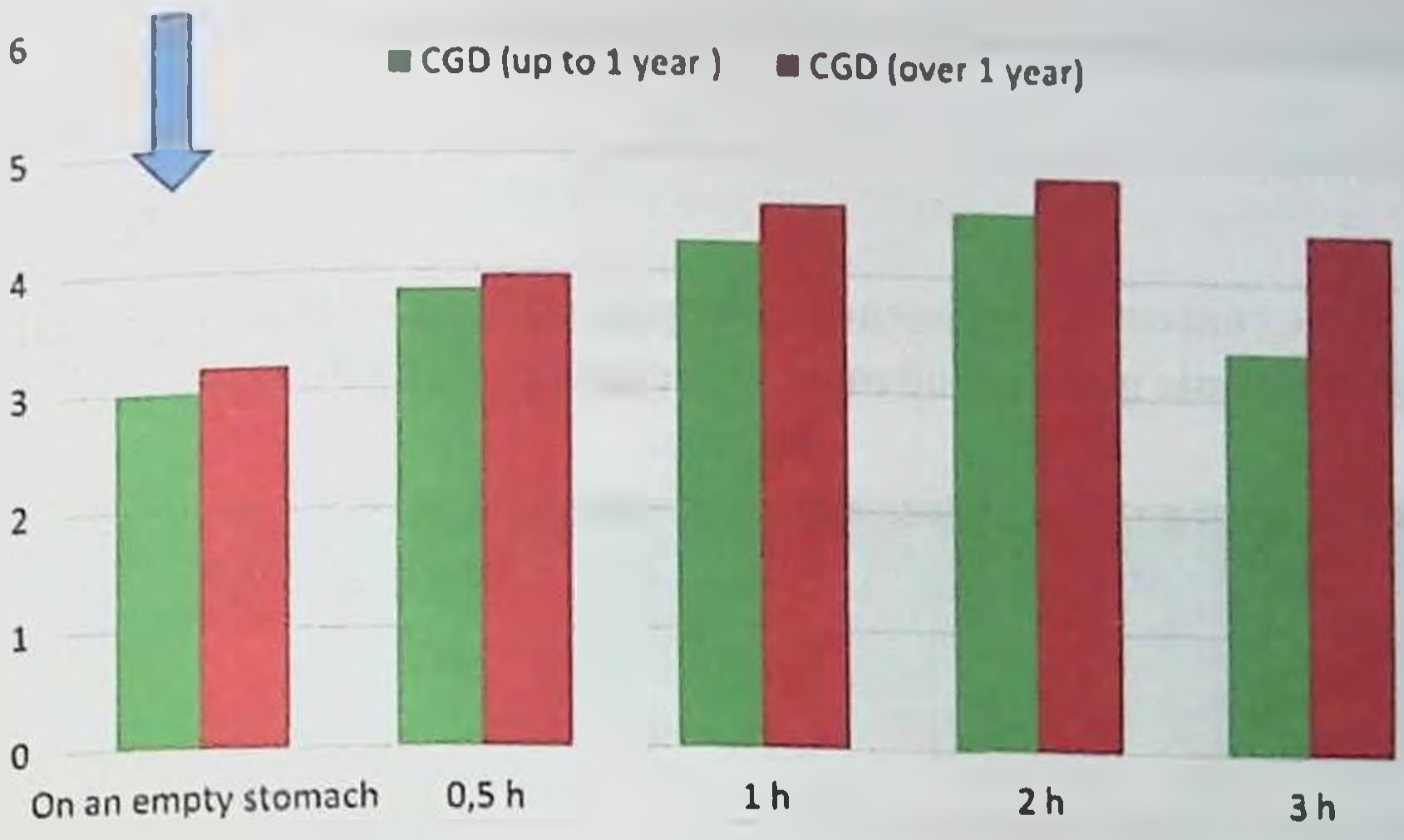

Figure 5. Dynamics of blood glucose in children with chronic gastroduodenitis with a disease duration of up to 1 year and over 1 year. Columns - an increase in newly formed glucose with a disease duration of up to 1 year, unshaded - over 1 year. The remaining designations are as in Fig.1.

In the first group, the fasting blood glucose content was 2.98±0.11 mmol/l, and in the second, with a disease duration of more than 1 year, this indicator was 3.19±0.11 mmol/l, which is 0.21 mmol/l higher. The increase in newly formed glucose after glycerin loading in patients with CGD of the first and second groups was, respectively, 0.80 and 1.04 mmol/l/hour. The tolerance of peripheral tissues to gluconeogenic glucose was 0.62 and 0.91 mmol/l, respectively.

These data indicate that the intensity of gluconeogenic liver function, in patients with CGD, increases with increasing duration of the disease. The results obtained show that the need of peripheral tissues for newly formed glucose is higher in patients with a long course of the disease.

As for the shifts in nitrogen metabolism in patients with CGD, in the second group of subjects, the level of ammonia in the fasting blood

was 226.45±7.62 mmol/l, which is statistically significantly higher than in patients of group I (187.63±6.91 mmol/L; P<0.001) (Table 11).

Table 11.

The content of nitrogenous compounds in the blood of patients with chronic gastroduodenitis, depending on the duration of the disease.

Indicators	Examined children	Healthy (n=22)	Patients with CGD (n=63)	
			to 1 year (n=30)	over 1 year (n=33)
Ammonia (in mmol/l)	On an empty stomach	126,14±11,1	187.63±6,91	226,45±7,62
	P		<0,001	<0,001
	After 3 hours	83,11±10,5	159,4±8,72	131,16±8,1
	P_1		<0,001	<0,001
	P_2	<0,05	<0,01	<0,001
Urea (in mmol/l)	On an empty stomach	4,03±0,13	4,61±0,07	4,92±0,09
	P		<0,001	<0,001
	After 3 hours	2,84±0,14	4,49±0,08	4,32±0,09
	P_1		<0,001	<0,001
	P_2	<0,001	>0,2	<0,001
Glutamine (in mkmol/l)	On an empty stomach	738,14±17	777,6±5,2	798,5±6,9
	P		>0,05	<0,001
	After 3 hours	714,19±19,5	766,2±4,9	745,3±6,6
	P_1		>0,01	>0,2
	P_2	>0,5	>0,1	<0,001

Note: where P and P1 are the reliability of differences in relation to the corresponding group of healthy children on an empty stomach and

after glycerin load; P2 is the reliability in relation to the corresponding group before and after glycerin load.

After glycerin loading, the ammonia content in the blood of patients of both groups significantly decreased, and more intensively in children with a disease duration of more than 1 year. Consequently, in patients with CGD with an increase in the prescription of the disease, the ability of glycerin to have a hypoammonemic effect increases (r=0.63).

Loading with glycerin effectively reduced the concentration of urea in the blood of patients with CGD of both groups. So, in patients with a disease duration of up to 1 year, the level of urea in the blood decreased by 0.12 mmol/ l after exercise, and in patients suffering from CGD over 1 year. after the introduction of glycerin, the level of urea decreased by 0.60 mmol / l (r=0.73). This reflects a significant increase in the urinary function of the liver in patients with CGD with an increase in the duration of the disease.

As can be seen from Table 11, the concentration of glutamine in the blood, after glycerin loading, did not undergo significant changes in patients in both groups.

Below, using specific examples, we present the results of our special studies, which studied the effect of glycerin on the gluconeogenic function of the liver and on some indicators of nitrogen metabolism in patients with CGD, with different disease duration:

Patient Ch., 9 years old, medical history No. 2754/1173. Diagnosis: Chronic gastroduodenitis with increased secretory function of the stomach, acute stage. The prescription of the disease is 4 months. The fasting blood glucose level was 2.61 mmol/l, 0.5; 1; 2 and 3 hours after loading with glycerin, respectively – 2.93; 3.59; 3.26 and 2.93 mmol/l. The rate of synthesis of newly formed glucose was 0.65 mmol/l/h, the tolerance of peripheral tissues to gluconeogenic glucose was 0.32 mmol/l. The level of ammonia, urea and glutamine in the fasting blood corresponded to 176.1 mmol /l, 4.38 mmol / l and 743.86 mmol / l, 3 hours after taking glycerin, respectively – 151.5 mmol / l, 3.94 mmol / l and 732.36 mmol / l.

Patient H., 13 years old, medical history No. 3005/1555. Diagnosis: Chronic gastroduodenitis with unchanged secretory function of the stomach, acute stage. The prescription of the disease is 6 years. The fasting blood glucose level is 3.26 mmol / l, after oral administration of glycerin in 0.5; 1; 2 and 3 hours, respectively – 3.59; 4.03; 4.64 and 4.24 mmol / l. The rate of growth of newly formed glucose on average per hour was 1.04 mmol/l. The tolerance of peripheral tissues to glucose of gluconeogenic genesis was 0.98 mmol/l. The concentration of ammonia, urea and glutamine in the fasting blood corresponded to 226.6 mmol/l, 5.24 mmol/l and 801.88 mmol/l, and 3 hours after loading with glycerin, this level corresponded to 111.0 mmol/l, 4.16 mmol/l and 766.58 mmol/L.

From these examples, it follows that with an increase in the duration of the disease, the ability of the liver of patients with CGD to synthesize glucose from glycerin increases. It should also be noted that with an increase in the duration of the disease, newly formed glucose is poorly absorbed by peripheral tissues. At the same time, the hypoammonemic and hypoureoemic effect of glycerin increases.

Then we studied the state of gluconeogenic liver function and some indicators of nitrogen metabolism in patients with DU depending on the duration of the disease. To do this, we divided all patients with DU, as in the case of CG and CGD, into two groups: with a disease duration of up to 1 year and over 1 year.

The level of glucose in the blood, in patients suffering from DU before 1 year on an empty stomach, was equal to 2.85 ± 0.09 mmol/l. Statistically significantly, compared with practically healthy children, it increased in patients of the second group, averaging 3.26±0.10 mmol/l ($P<0.001$). In all patients, the load of glycerin contributed to an increase in the concentration of glucose in the blood.

The increase in newly formed glucose in patients of the first group was equal to 0.93, and in patients of the second group - 1.17 mmol/l /h. These data indicate that the gluconeogenic function of the liver increases with an increase in the prescription of the disease (Table 12).

Table 12.

The effect of glycerin load on the blood glucose content of patients with DU (mmol/l), depending on the duration of the disease.

Duration of the disease	On an empty stomach	Time in hours after exercise	Glucose content after glycerin load
Up to 1 year (n = 9)	2,85 ± 0,09	0,5	3,27 ± 0,12
		1	3,64 ± 0,14
		2	3,90 ± 0,13
		3	3,81 ± 0,11
Over 1 year (n = 10)	3,26 ± 0,10	0,5	3,62 ± 0,08
		1	4,25 ± 0,07
		2	4,58 ± 0,07
		3	4,48 ± 0,05

Studying the changes in nitrogenous compounds depending on the prescription of the disease, we found that in the blood of patients with DU of the second group on an empty stomach, the level of ammonia was equal to 242.63±7.52 mmol/l, and in patients of the first group, it was significantly lower and amounted to 192.84±11.4 mmol/l (P<0.001). (Table 13).

Table 13.

The effect of glycerin load on the content of nitrogenous compounds of patients with DU with different disease duration.

Indicators	Examined children	Healthy (n=22)	Patients with DU	
			to 1 year	over 1 year
Ammonia (in mmol/l)	On an empty stomach	126,14±11,1	192,84±11,4	242,63±7,5
	P		<0,001	<0,001
	After 3 hours	83,11±10,5	176,18±7,6	152,46±5,2
	P_1		<0,001	<0,001
	P_2	<0,05	>0,2	<0,001
Urea (in mmol/l)	On an empty stomach	4,03±0,13	4,98±0,08	5,08±0,06

	P		<0,001	<0.001
	After 3 hours	2,84±0,14	4,72±0,11	4,54±0,08
	P_1		<0,001	<0,001
	P_2	<0,001	>0,05	<0,001
Glutamine (in mkmol/l)	On an empty stomach	738,14±17	813,42±8,4	841,2±6,1
	P		<0,001	<0,001
	After 3 hours	714,19±19,5	802,12±6,2	760,8±4,1
	P_1		>0,001	<0,02
	P_2	>0,5	>0,5	<0,001

Note: 1- on an empty stomach; 2 – 3 hours after glycerin load; P and P1 – reliability of differences in relation to the corresponding group of healthy children on an empty stomach and after glycerin load; P2 – reliability in relation to the corresponding group before and after glycerin load.

As can be seen from Table 13, in these groups of patients with DU, the content of urea in the blood on an empty stomach was approximately the same, and significantly exceeding its level in practically healthy children by 0.95 and 1.05 mmol/l ($P<0.001$), and the concentration of glutamine was significantly higher in patients with a disease prescription of more than 1 year ($P<0.05$).

Glycerin loading led to a statistically significant decrease in the content of ammonia and urea ($P<0.001$ and $P<0.01$) in the blood of patients suffering from DU for more than 1 year. This cannot be said about the patients of the first group, where the reduced tolerance of the body to the hypoammonemic and hypoureoemic action of glycerin was clearly manifested. After glycerin loading, the content of glutamine in the blood of patients of the second group decreased by 80.4 mmol/l, which is 7.1 times higher than those of patients of the first group.

To confirm the above, we briefly present two clinical observations, where the prescription of the disease was up to 1 year and over 1 year:

Patient A., 14 years old, medical history No. 1004/380. Diagnosis: Duodenal ulcer with increased secretory function of the stomach, acute

stage. The prescription of the disease is 6 months. The fasting blood glucose level was 2.93 mmol/l, 0.5; 1; 2 and 3 hours after loading with glycerin, respectively – 3.26: 3.59; 4.03 and 3.91 mmol/l. The rate of glucose synthesis was 0.91 mmol/l/h. The tolerance of peripheral tissues to newly formed glucose was 0.98 mmol/l. The level of ammonia, urea and glutamine in the blood before the load corresponded to 202.6 mmol/l, 4.80 mmol/l and 815.36 mmol/l, and 3 hours after the glycerin load, respectively – 176.1 mmol/l, 4.61 mmol/l and 789.84 mmol/l.

Patient T., 10 years old, medical history No. 2049/898. Diagnosis: duodenal ulcer with increased secretory function of the stomach in the acute stage. The prescription of the disease is 3 years. The fasting blood glucose level is 3.16 mmol / l, after oral administration of glycerin in 0.5; 1; 2 and 3 hours, respectively – 3.65; 4.18; 4.49 and 4.35 mmol / l. The growth rate of newly formed glucose averaged 1.18 mmol/l per hour. The tolerance of peripheral tissues to gluconeogenic glucose was equal to 1.19 mmol/l. The content of ammonia, urea and glutamine in the blood before the load was 252.5 mmol/l, 5.24 mmol/l and 848.20 mmol/l, respectively, and 3 hours after the glycerin load, respectively - 176.1 mmol/l, 4.38 mmol/l and 766.58 mmol/l.

From these examples, it follows that in patients with DU, with an increase in the duration of the disease, the ability of the liver to synthesize glucose from glycerol increases. At the same time, with an increase in the duration of the disease, the hypoammonemic and hypoureoemic effect of glycerin increases.

Concluding this chapter, it can be noted that in practically healthy children, we have detected a low content of glucose in the blood on an empty stomach and a slight increase in its concentration in patients with chronic GDP, which, in our opinion, indicates an increased need for energy material in the body of patients.

For the first time, the gluconeogenic function of the liver in children with chronic GDP has been studied in various variants. An azo-free compound, glycerin, was used as a precursor of the newly formed glucose.

We have noted that in children with chronic GDP, the gluconeogenic function of the liver increases, which is probably one of the compensatory and adaptive acts to provide the body with an additional amount of glucose. The consequence of increased gluconeogenesis in these patients is a significant transformation of glycerin into glucose, increased tolerance of peripheral tissues to glucose. Moreover, a more pronounced increase was recorded in children with CGD and DU.

Studying some indicators of nitrogen metabolism, we found that hyperammonemia is characteristic of patients with chronic GDP, apparently as a consequence of increased protein breakdown and subsequent deamination of amino acids. This toxic metabolite is successfully neutralized in practically healthy children in the form of urea and glutamine, whereas in patients with CGDP only in the form of urea, as evidenced by hyperureoemia in patients on an empty stomach.

We believe that in patients with CGDP the synthesis of urea in the liver varies widely. The biosynthesis of this final product of nitrogen metabolism, with this pathology in children, is not disturbed and the reserves of the liver in this respect are significant. This fact is of great practical importance, as can be judged by using glycerin loading.

We have discovered the ability of glycerin to have a hypoammonium and hypoureoemic effect, which is more pronounced in patients with chronic GDP than in practically healthy children.

When clarifying the question of how the gluconeogenic function of the liver changes depending on the duration of the disease, it was found that it is least disturbed in children with a disease duration of up to 1 year, and more in patients with a disease duration of more than 1 year for all nosological forms of CGDP.

At the same time, it has been shown that newly formed glucose is equally poorly absorbed by peripheral tissues in patients with chronic GDP with a disease duration of up to 1 year and over 1 year than in healthy children.

For all nosological forms, as the duration of the disease increased, the hypoammony and hypoureoemic effect of glycerin increased.

So, with CGDP in children, there are significant violations in carbohydrate and nitrogen metabolism. The increase in glucose, ammonia, urea and glutamine levels in the blood of patients with CGD and DU was especially clear.

Here it should be emphasized once again that shifts in these final products of nitrogen metabolism, depending on the nosological form and duration of the disease, in chronic GDP in children, could be established only with the use of an exogenous stimulus - glycerin load.

CHAPTER IV. CORRECTION OF DISORDERS OF GLUCONEOGENIC LIVER FUNCTION, CARBOHYDRATE AND NITROGEN METABOLISM UNDER THE INFLUENCE OF TREATMENT OF PATIENTS WITH GASTRODUODENAL PATHOLOGY.

This chapter will present the data obtained by us in 34 patients with CG, 63 patients with CGD and 19 patients with DU, in whom the gluconeogenic function of the liver and the state of nitrogen metabolism were studied after treatment. The data obtained from these patients upon admission to the clinic are presented in the previous chapter.

Despite significant achievements in the study of the pathogenesis of gastroduodenal diseases in children (CG, CGD, DU), the results of treatment of this pathology require further research.

Our research has shown that one of the pathogenetic mechanisms of the formation of these diseases are multiple disorders of carbohydrate, nitrogen metabolism and the functional state of the liver. Increased gluconeogenesis and a high level of nitrogenous parameters were caused by a violation in the body of patients, between the pathological focus and the liver.

In this regard, the problem of developing effective methods of treating CGDP in children is urgent. In the literature available to us, we have not found any works concerning the correction of disorders of gluconeogenic liver function and nitrogen metabolism in CGDP in children and adults.

As corrective agents, we used hepatoprotectors: essentiale and medical glycerin for the entire period of treatment. Essentiale - as a complex drug that actively affects tissue metabolism and improves the functional state of the liver, and glycerin - as a hypoammonic and hypoureoemic agent.

To assess the effectiveness of the above-mentioned drugs on the course of clinical and biochemical parameters in sick children with CGDP, we compared the results of studies against the background of various treatment methods.

The patients were divided into four groups:

Group I consisted of 36 patients: with CG - 10, CGD - 20, DU - 6 patients who were treated with conventional methods of treatment, including diet, sedative therapy, antispasmodics, drugs with reparative properties. antacids, vitamins, physiotherapeutic procedures; group II consisted of 27 patients: CG - 9, CGD - 14, DU - 4 patients who received essentiale along with conventional treatment (2 capsules 2 times a day after meals); Group III consisted of 25 patients: CG- 7, CGD - 14, DU - 4 patients who received medical glycerin in a complex of medicinal products (at a dose of 0.5 mg / kg of weight dissolved in 200.0 ml of boiled water every 2 days); group IV included 28 patients: with CG - 8, CGD - 15, DU - 5 patients who received complex treatment, including conventional methods of treatment, essentiale and glycerin, for the entire period of treatment.

In all the examined patients, except for the generally accepted clinical and laboratory-instrumental methods of research, the gluconeogenic function of the liver and the state of nitrogen metabolism were studied at admission and after the therapy methods.

The criteria for judging the therapeutic effect of corrective agents were clinical manifestations (the timing of the disappearance of subjective and objective manifestations of the disease) and indicators of glucose, ammonia, urea and glutamine in the blood.

In general, it was found that as a result of the therapeutic measures carried out, the fasting blood glucose in patients with CG decreased by 0.08 mmol/l, averaging 2.83± 0.08 mmol / l, which is almost equal to those of healthy children (2.76± 0.12 mmol/l). Moreover, in patients of group II and IV, it was almost the same, being equal to 2.80±0.06 and 2.82±0.07 mmol/l, respectively, and lower than in patients of group III and I, whose glucose level was equal to 2.84±0.11 and 2.86±0.08 mmol/l, respectively. The concentration of glucose in the blood of group I patients after the conventional method of treatment was the highest compared to other methods of treatment and exceeded its level in healthy children by 0.10 mmol/l ($P>0.5$).

Before discharge, patients with CGD had a decrease in fasting blood glucose by 0.12 mmol/l, which averaged 2.98± 0.09 mmol/l, which is 4.0% lower than they had before treatment and only 7.4% (P>0.1) higher than the data of practically healthy children. Moreover, their fasting blood glucose levels varied with various combinations of drug treatment. Thus, in patients with traditional treatment (group I), the fasting blood glucose concentration was 3.04± 0.12 mmol/l, and in patients of group IV with complex treatment, it was 2.90± 0.08 mmol/l.

In patients with DU, the fasting glucose content before discharge in all groups averaged 2.98±0.07 mmol/l, which is significantly higher than its level in practically healthy children (P<0.05) and was almost the same as fasting glucose in patients before treatment.

As for the clinical picture, we obtained the greatest effect in patients with CG, CGD and DU in the IV treatment group. These patients indicated not only a significant improvement in their general condition, but also a decrease in abdominal pain, the disappearance of nausea, vomiting and an improvement in appetite. They also had improved laboratory blood counts, endoscopic and X-ray picture, while group I patients continued to complain of abdominal pain, headaches and gastrointestinal dysfunction.

We obtained additional information about shifts in carbohydrate metabolism, in particular about changes in the gluconeogenic function of the liver, in patients with chronic GDP using glycerin loading.

In patients with CG after treatment, the increase in newly formed glucose under the influence of glycerin load by 0.5 and 1 hours was 1.44 and 1.24 times lower than in practically healthy children and, at the same time, 1.33 and 1.13 times higher than those in patients before treatment. By the 2nd and 3rd hours of the study, the increase in newly formed glucose in patients before discharge exceeded by 1.27 and 2.05 times these indicators in healthy children and decreased by 1.13 and 1.53 times, compared with patients with CG, when they were admitted to the hospital (Fig.6).

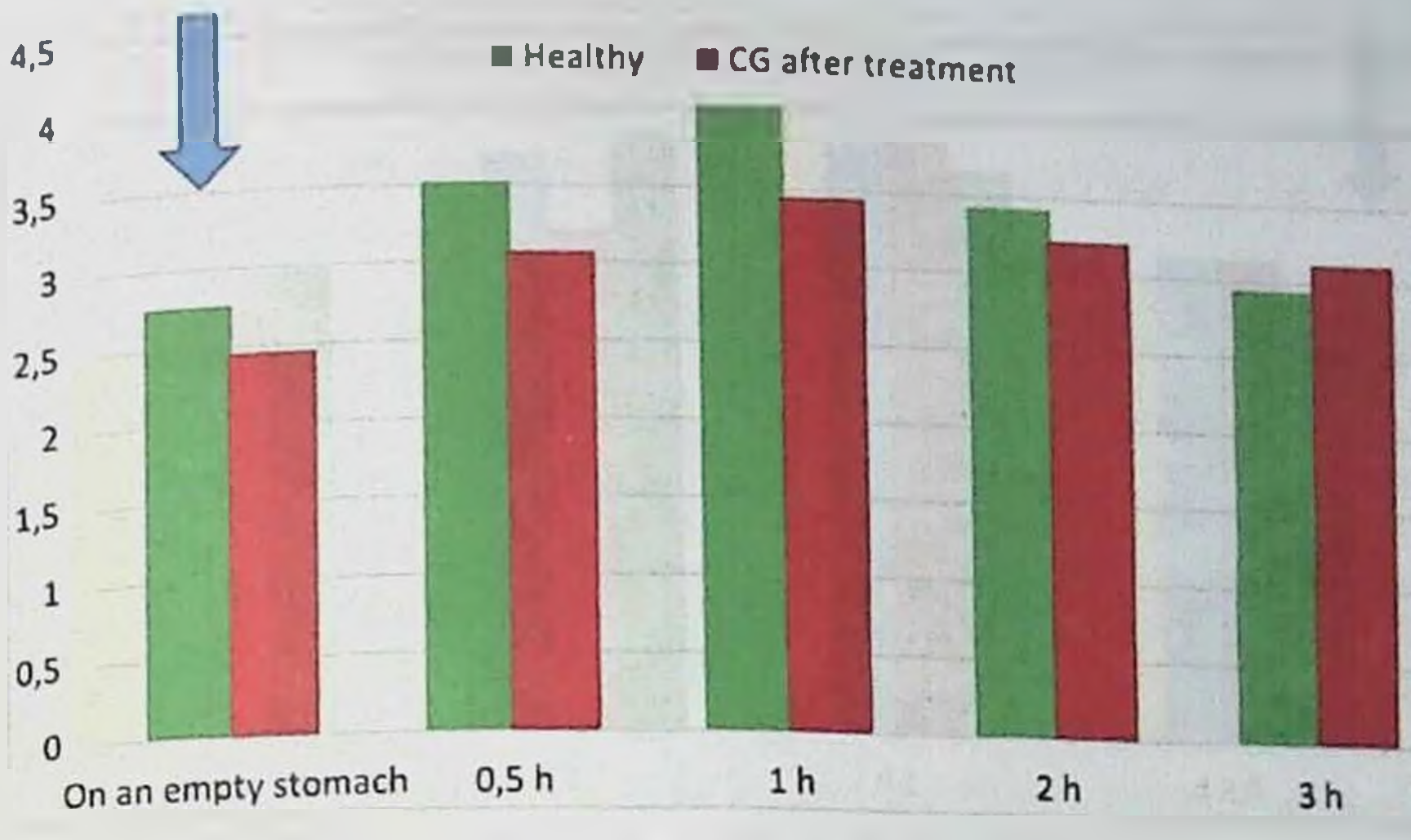

Figure 6. The effect of glycerin load on blood glucose in healthy children and patients with chronic gastritis after treatment. Columns - hourly increase in glucose in practically healthy children and in patients with chronic gastritis, before their discharge from the hospital.

Further, we found that the drug treatment of CG patients reduced the rate of glycerin transformation into glucose in them. Indeed, before discharge in these patients, the rate of glucose biosynthesis decreased by 0.08 mmol/l/h, equaling an average of 0.74±0.06 mmol/l/h. At the same time, the hourly increase in newly formed glucose by 0.5, 1, 2 and 3 hours of observation was 0.25, 1.015, 0.84 and 0.37 mmol/l, respectively, and was almost the same as in healthy children. In addition, in patients with CG after treatment, the resistance of peripheral tissues to glucose of gluconeogenic genesis decreased by 0.16 mmol/l.

So, after the treatment, the gluconeogenic function of the liver was normalized in patients with CG and the threshold of sensitivity of peripheral tissues to newly formed glucose decreased.

We discussed generalized data for all groups of patients with CG. However, the results obtained in these patients, taking into account different treatment regimens, differed (Fig. 7).

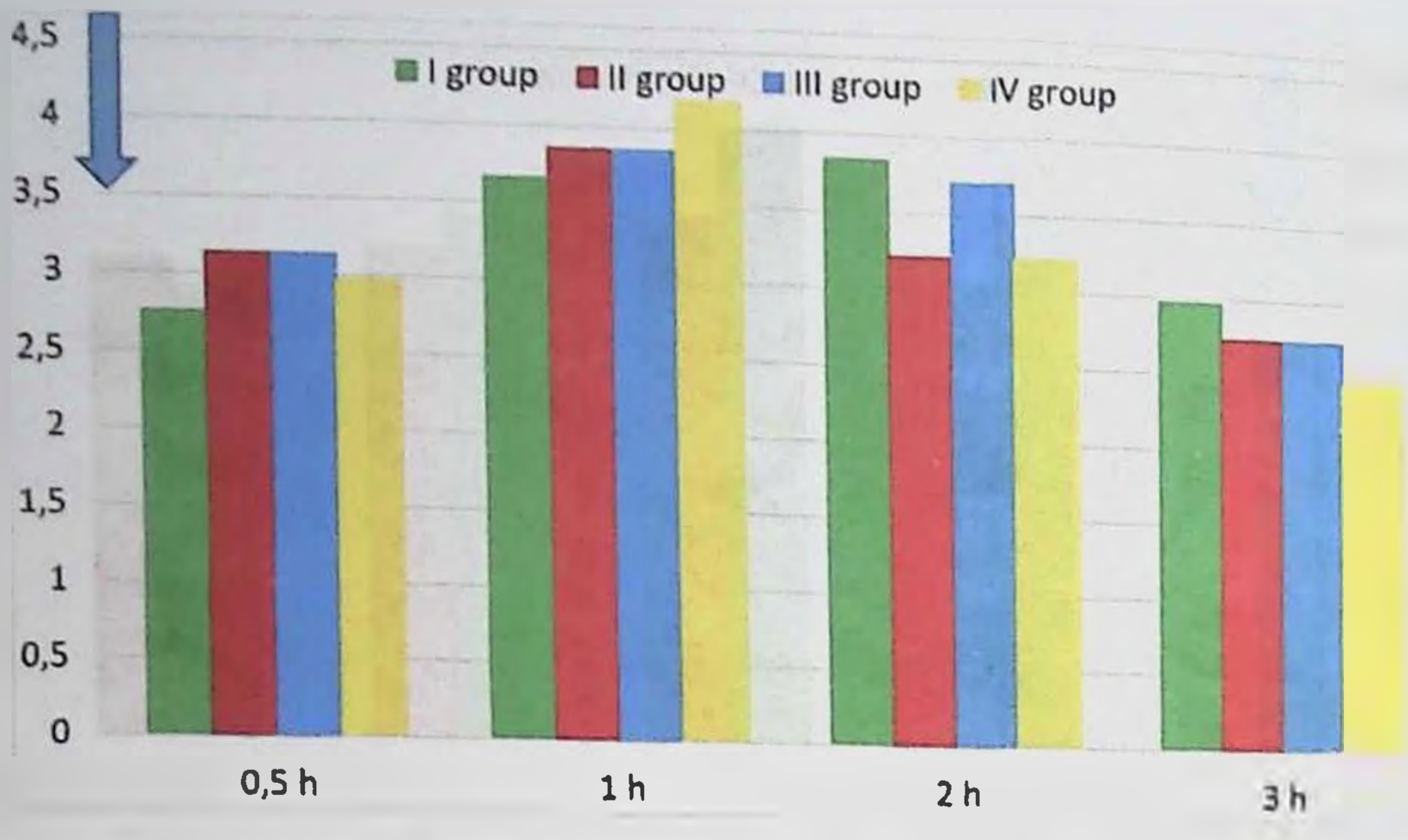

Figure 7. The effect of glycerin load on changes in blood glucose levels in patients with chronic gastritis, depending on treatment methods. Columns - hourly increase of newly formed glucose in patients with chronic gastritis of groups I, II, III and IV before discharge. The remaining designations are as in Fig.1.

Figure 7 shows that after glycerin loading, the blood glucose content was the lowest by 0.5 and 1 hour of the study in patients with the traditional method of treatment. On the contrary, in the following hours of the study, the glucose level in this group of patients was the highest, compared with other groups of patients, before their discharge from the hospital.

In our opinion, this fact indicates an improvement in the absorption of glycerin in the stomach during the initial hours of the study in patients of the II, III, IV treatment groups before their discharge from the hospital, compared with patients who received traditional treatment. The glycemic curve after glycerin loading in patients of group II, III, IV was similar and almost identical to the glycemic curve of healthy children, which was especially pronounced in patients of group IV with complex treatment, additionally receiving essentiale and medical glycerin. They also had the lowest rate of conversion of glycerin into glucose - 0.69 mmol/ l / h, and was equal to the rate of gluconeogenesis in practically healthy children (0.70 mmol/l/h). As for the tolerance of peripheral

tissues to glucose of gluconeogenic genesis, it was the lowest in group IV patients and was equal to 0.28± 0.07 mmol/l, which is only 1.55 times higher than these indicators in healthy children (0.18 mmol/l; P<0.05).

In patients in groups I, II and III, the tolerance of peripheral tissues was 0.46, 0.36 and 0.38 mmol/l, respectively. Consequently, in this case of observation, complex treatment, taking into account clinical manifestations, turns out to be justified and effective.

As follows from Figure 8, before discharge, in patients with CGD 0.5, 1, 2 and 3 hours after taking glycerin, the increase in newly formed glucose was equal, respectively - 0.495, 0.945, 0.852 and 0.587 mmol / l, on average - 0.794 mmol / l / h, and the blood glucose content was equal to 3,475±0,085, 3,925± 0,072, 3,832±0,067 and 3.567±0.095 mmol/l. This is significantly lower than in patients with CGD before treatment, but at the same time higher than in practically healthy children of the control group. After treatment, the tolerance of peripheral tissues to glucose decreased in patients with CGD, averaging 0.587 mmol/l, although this is still 3.2 times more than in healthy children.

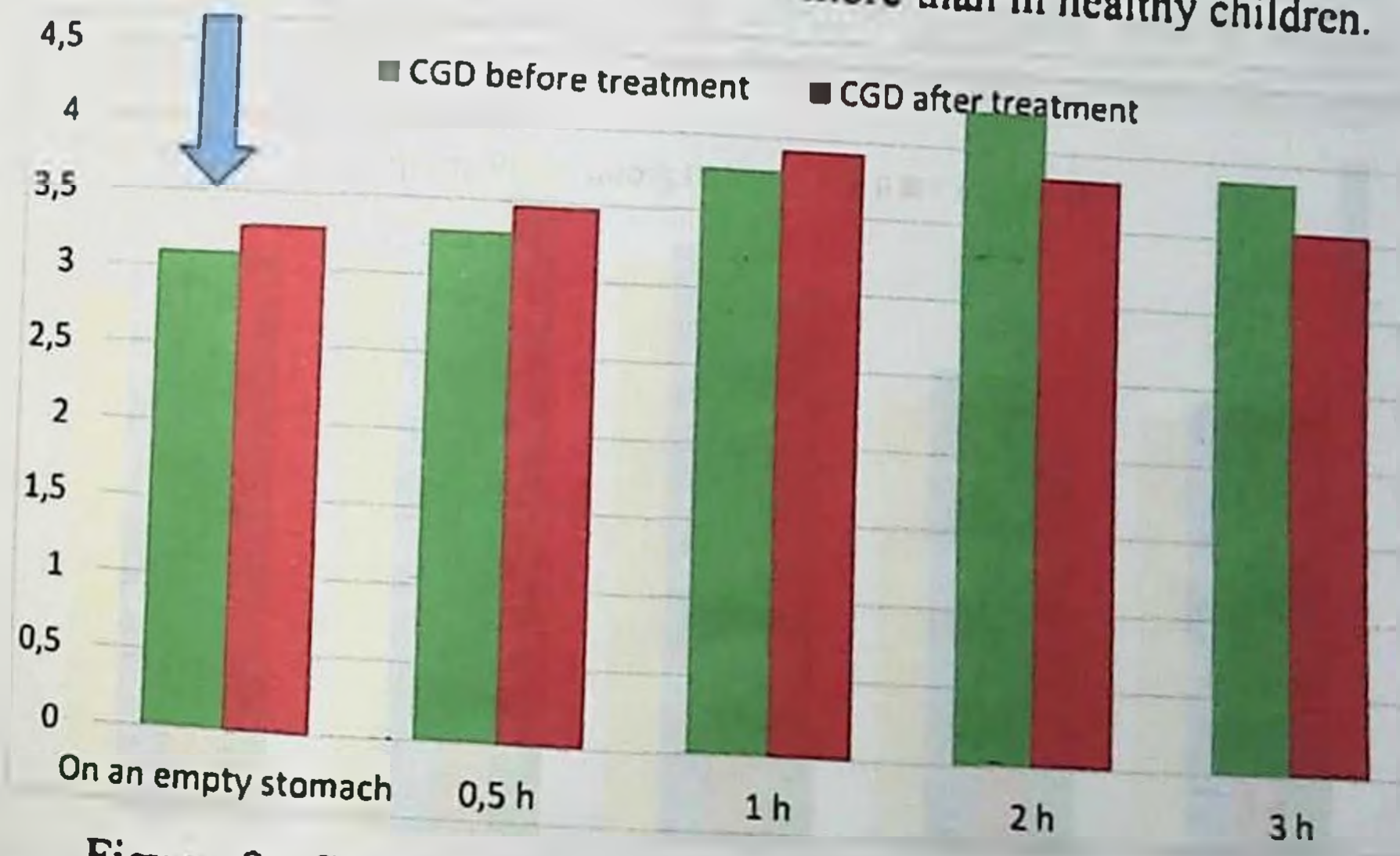

Figure 8. Comparative effect of glycerin load on gluconeogenic liver function in patients with chronic gastroduodenitis before and after treatment. Columns - an increase in newly formed glucose in patients

with chronic gastroduodenitis upon their admission to the hospital and before their discharge. The remaining designations are as in Fig.1.

Consequently, the treatment of patients with CGD significantly reduces their gluconeogenic liver function, increases the entry of glucose formed from glycerin into peripheral tissues.

We found some differences in these indicators in patients with CGD, differing in the medications used. These data are presented in Figure 9, from which it can be seen that on an empty stomach, the blood glucose level was lowest in patients of group IV of treatment - 2.90 ± 0.085 mmol/l, and the highest in patients of group I - 3.04± 0.12 mmol/L. After glycerin loading, the blood glucose level by 0.5 and 1 hour of the study was higher in patients of treatment groups II and III. By 2 and 3 o'clock, glucose values were again highest in group I patients, and lowest in group IV, respectively, 4.15±0.09, 3.65±0.07 mmol/L and 3.72±0.17, 3.41±0.07 mmol/l.

The increase in newly formed glucose in patients with group I, II, III and IV treatment, on average, was 0.74 mmol/l. The tolerance of peripheral tissues to newly formed glucose also varied by treatment groups, amounting to 0.68, 0.59, 0.57 and 0.51 mmol/l, respectively.

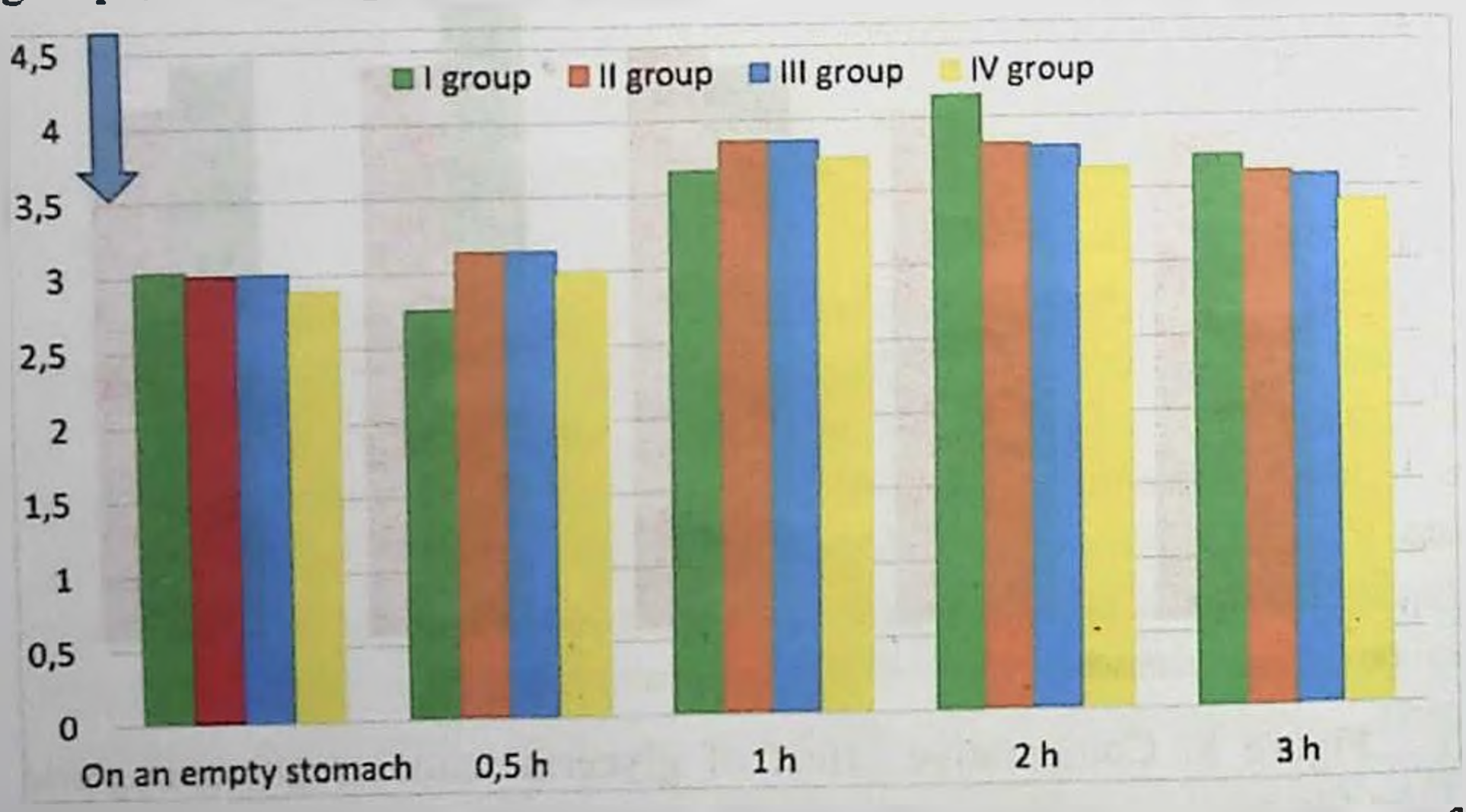

Figure 9. The effect of various types of drug treatment on the rate of transformation of glycerin into glucose in patients with chronic

gastroduodenitis. Columns - the increase in newly formed glucose in groups (group I, II, III and IV, depending on the combination of drug treatment).

Summarizing the above data, with regard to CGD, we emphasize that after a comprehensive treatment, including traditional treatment in combination with essentiale and medical glycerin, fasting blood glucose levels significantly decrease in patients, the rate of glucose neoplasm from glycerin in the liver decreases and glucose assimilation by peripheral tissues improves, compared with other corrective groups treatment, especially in relation to traditional conventional treatment.

Using glycerin loading in patients with DU after treatment, we obtained additional information about changes in the gluconeogenic function of the liver (Fig.10). These data are presented in Figure 10, from which it can be seen that in patients with DU before their discharge from the hospital 0.5, 1, 2 and 3 hours after taking glycerin, the increase in newly formed glucose was equal, respectively - 0.562, 1.017, 0.947 and 0.795 mmol / l, on average - 0.915 mmol / l /h, a blood glucose content - 3,54±0,08, 3,99±0,065, 3,927±0,061 and 3.775±0.065 mmol/l.

Figure 10. The effect of glycerin load on the gluconeogenic function of the liver in patients with duodenal ulcer disease before and after treatment. The remaining designations are as in Fig.1.

The rate of gluconeogenesis in patients with DU before discharge is significantly lower than at admission, but significantly higher than in practically healthy children (P<0.001). After treatment, peripheral tissue tolerance to gluconeogenic glucose decreased by 34.3% in patients with DU, averaging 0.79±0.058 mmol/L, although this figure is 4.38 times higher than in children of the control group.

So, in patients with DU after treatment, there was a slight tendency to decrease the level of glucose in the blood on an empty stomach than when admitted to the hospital, but still significantly exceeded the norm. This means that the need for glucose is still high in patients with DU after treatment, and a pronounced therapeutic effect occurs with a relatively high content of glucose in the blood on an empty stomach.

It follows from the above that the treatment of patients with DU significantly reduces their gluconeogenic liver function, increases the entry of glucose formed from glycerin into peripheral tissues against the background of high fasting blood glucose.

As for these indicators in patients with DU, depending on the method of drug treatment used, we found some differences here (Fig.11). From Figure 11 it can be seen that the fasting blood glucose level in patients with DU before their discharge almost did not change and was the same with different combinations of drug treatment. The increase in newly formed glucose in the groups, depending on the combination of therapeutic agents, was, respectively, 1.00, 0.88, 0.94 and 0.84 mmol/ l / h, from which it can be concluded that the rate of gluconeogenesis in patients with DU after various methods of treatment in groups decreased from 0.12 to 0.28 mmol/ l / h, which it was especially significantly expressed in patients of the IV treatment group (P<0.001).

The tolerance of peripheral tissues to newly formed glucose by groups was 1.01, 0.71, 0.74 and 0.70 mmol/L, respectively, which also clearly confirms the effectiveness of glucose uptake by peripheral tissues formed from glycerin in patients, after treatment with essentiale,

medical glycerin, and especially in their complex, compared with patients with DU, who received exclusively only traditional treatment.

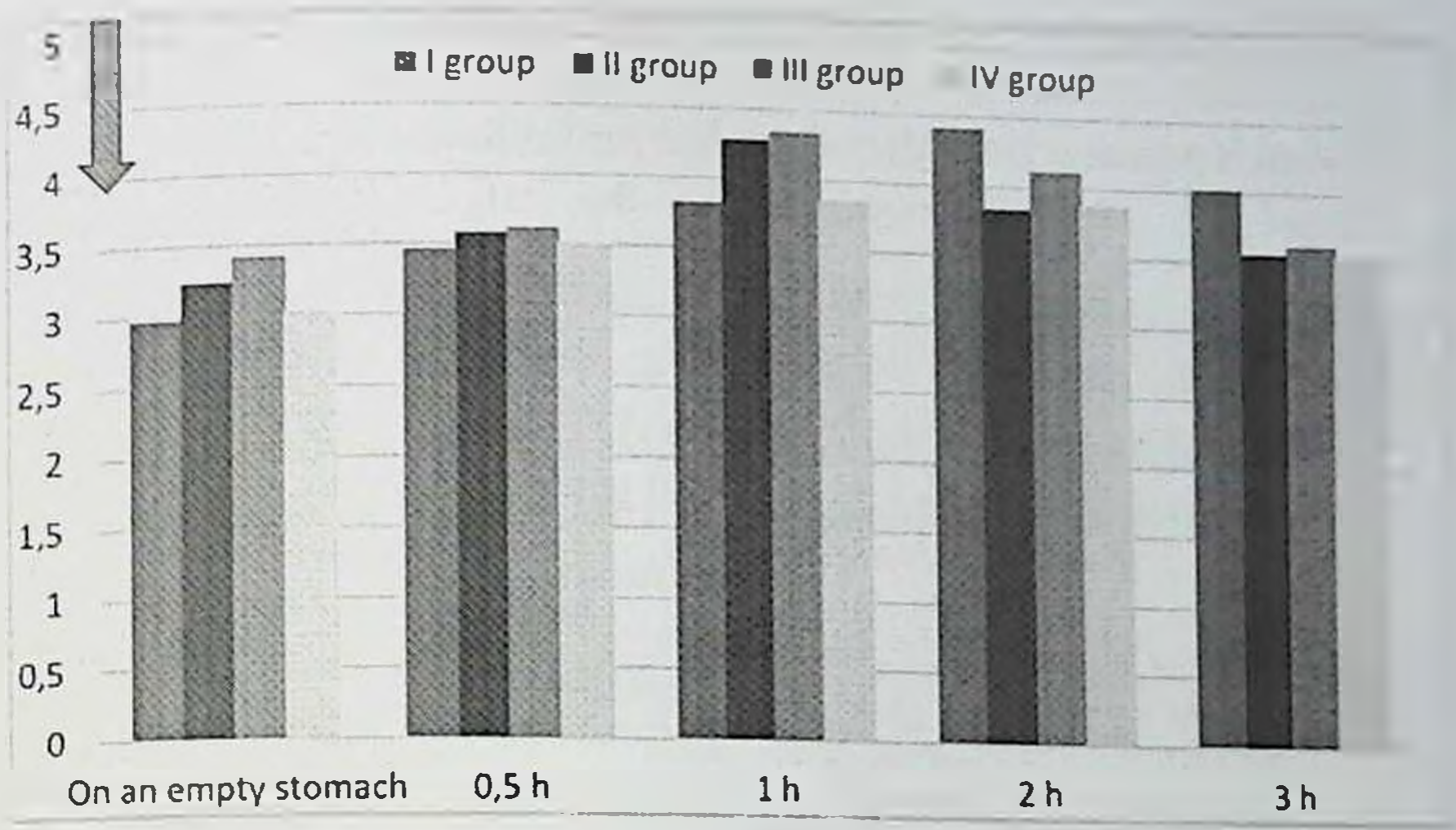

Figure 11. The effect of various types of drug treatment on the rate of transformation of glycerin into glucose in patients with duodenal ulcer. The columns are the increase in newly formed glucose by groups (groups I, II, III and IV, depending on the combination of drug treatment). The remaining designations are as in Fig.1.

Summarizing the above data, we emphasize that after the treatment, the rate of glucose neoplasm from glycerol in the liver decreases in patients with DU and the absorption of glucose by peripheral tissues improves, and all this was most pronounced in group IV patients.

Taking into account the close relationship between carbohydrate and nitrogen metabolism, we will now discuss the data we have obtained with respect to some indicators of nitrogen metabolism in patients with CG, CGD and DU.

We found that in patients with CG before discharge, the ammonia content in the blood decreased by 21.7 mmol/l and amounted to 131.29 ±7.1 mmol/l, which was slightly higher than in healthy children, and in patients of group IV it corresponded to the norm (125.95±6.43 mmol/l). According to our data, this decrease in the concentration of ammonia in

the blood occurs mainly due to increased urea formation. Indeed, after treatment, the urea level in patients decreased by 0.58 mmol/l and amounted to 4.1±0.075 mmol/l. In this respect, the glutamic acid-glutamine system turns out to be less effective. In fact, in patients with CG after a course of treatment, the content of glutamine in the blood on an empty stomach decreased by 8.8 mmol /l and averaged 741.65 ± 8.1 mmol / l, which corresponded to the indicators of healthy children (Table 14).

Table 14.

The effect of glycerin load on some indicators of nitrogen metabolism in the blood in patients with chronic gastritis after treatment.

Indicators	Examined children	Healthy (n=22)	Patients with DU (n=34)
Ammonia (in mmol/l)	1	126,14±11,1	131,29 ± 7,1
	P		>0,5
	2	83,11±10,5	85,45 ± 5,9
	P_1		>0,5
	P_2	<0,05	<0,001
Urea (in mmol/l)	1	4,03±0,13	4,10 ± 0,075
	P		>0,5
	2	2,84±0,14	3,56 ± 0,065
	P_1		<0,001
	P_2	<0,001	<0,001
Glutamine (in mkmol/l)	1	738,14±17	741,55 ± 8,1
	P		>0,5
	2	714,19±19,5	721,5 ± 5,65
	P_i		>0,5
	P_2	>0,5	>0,05

Note: 1 – on an empty stomach; 2 – 3 hours after glycerin load: P and P1 – the reliability of differences in relation to the corresponding group of healthy children; P2 - in relation to the corresponding group before glycerin load.

3 hours after glycerin loading in patients with CG, the concentration of ammonia in the blood decreased by 8.45 mmol/l, averaging 85.45 ± 5.9 mmol/ l and no longer differing from similar data in healthy children. The content of glutamine decreased by 17.2 mmol/l, averaging 721.5±6.65 mmol/l, and urea - by 0.55 mmol/l and amounted to 3.56± 0.065 mmol/l, which is 1.25 times higher than this indicator in healthy children.

Consequently, as a result of treatment in patients with CG, the concentrations of ammonia, glutamine and urea in the blood were leveled to normal. Moreover, a special role in the neutralization of ammonia, apparently, belongs to the urea-forming function of the liver. Once again, we confirmed the previously established fact: glycerin load has a pronounced hypoammonium and hypoureoemic effect. This allows us to recommend the administration of glycerin to patients with CG, to restore the disturbed metabolism of ammonia and urea.

As for the changes in these indicators of nitrogen metabolism in patients with CG of all four groups, under the influence of different treatment regimens, they differed among themselves (Table 15). As can be seen from Table 15, patients who were on the traditional method of treatment (group I) had the highest levels of ammonia and urea in their blood before discharge from the hospital, compared with other groups. The level of glutamine in the blood before discharge is the same in all groups. 3 hours after glycerin loading, the level of ammonia in the blood significantly decreases in all groups. So, in patients of group I, the decrease occurs by 1.47 times ($P<0.001$); in patients of group II - by 1.56 times ($P<0.001$); in patients of group III - by 1.57 times ($P<0.001$); in patients of group IV, with complex treatment, it decreases by 1.64 times ($P<0.001$). So, the decrease in the level of highly toxic ammonia under the influence of glycerin load in patients of groups II, III and IV occurs more intensively than in patients who received the traditional method of treatment.

Table 15.

The effect of glycerin load on some indicators of nitrogen metabolism in the blood of patients with chronic gastritis, depending on the treatment methods.

Indicators	Time	Patients with chronic gastritis			
		I–group (n=10)	II–group (n=9)	III-group (n=7)	IV-group (n=8)
Ammonia (in mmol/l)	1	139,40±8,4	128,54±6,4	131,3±7,7	125,95±6,4
	2	94,53±6,5	82,4±5,7	83,6±6,5	81,3±4,9
	P	<0,001	<0,001	<0,001	<0,001
Urea (in mmol/l)	1	4,33±0,07	4,12±0,07	3,99±0,08	3,96±0,08
	2	3,74±0,06	3,61±0,05	3,47±0,07	3,42±0,08
	P	<0,001	<0,001	<0,001	<0,001
Glutamine (in mkmol/l)	1	742,9±7,7	741,8±10,9	741,6±8,5	740,3±5,3
	2	731,1±10,8	718,3±5,6	720,1±5,8	716,6±4,41
	P	>0,5	>0,05	>0,05	<0,001

Note: 1 and 2 - indicators on an empty stomach and 3 hours after glycerin loading; P - the reliability of differences in relation to the corresponding group of patients on an empty stomach.

The concentration of urea in the blood after loading with glycerin also significantly decreases. In healthy children, the decrease in urea level was equal to 1.19 mmol/l (by 29.5%; P<0.001). In patients with CG, when they were admitted to the clinic, the urea content in the blood after glycerin load decreased by 0.57 mmol/l (by 12.1%; P<0.001). Depending on the method of treatment, before discharge, this difference was 0.59 mmol/l (13.6%; P<0.001) in patients of group I; 0.51 mmol/l (12.3%; P<0.001) in group II; 0.52 mmol/l (13.0%; P<0.001) and in group IV patients - decreased by 0.54 mmol/l (by 14.0%; P<0.001). As for glutamine, the decrease in its concentration in the blood under the influence of glycerin in group I was unreliable (P>0.5); in groups II and III - weakly reliable (P <0.05 and P<0.05), and in group IV this decrease was more pronounced - by 23.7 mmol /l (P<0.001). From the above data, it follows that in patients with CG, all therapeutic measures have the same focus. However, essentiale, medical glycerin and their complex use in the therapeutic tactics of CG to a greater extent reduce the level of

ammonia, urea and glutamine, and in the latter variant even normalize their blood levels.

To illustrate the above, we will give a clinical example:

Patient Z., 12 years old, medical history No. 529/211, was admitted to the clinic with complaints of pain in the epigastric region, nausea, constipation, lethargy.

The girl has been ill for 10 months, she has not been treated for this disease before. Of the transferred diseases, he notes frequent colds, sore throats. Heredity is not burdened.

Objectively: general condition of moderate severity. The skin and visible mucous membranes are pale. Subcutaneous fat is poorly developed. Peripheral lymph nodes are not enlarged, except for the submandibular group, which are the size of beans, not soldered, slightly painful on palpation. A clear pulmonary sound is percutorially determined above the lungs, vesicular breathing is auscultatively listened to. The area of the heart is without visible changes, auscultation - tones are muted, there are no noises. The pulse is rhythmic, of medium filling and tension. The tongue is moist, overlaid with a whitish coating, the pharynx is pale pink, the tonsils are enlarged, loose, there are no plaque. The abdomen is of the correct shape, soft, with palpation there is pain in the epigastric region. The liver and spleen are not palpable. Chair once every 2-3 days. Urinary system without features.

General tests of blood, urine and feces without features.

Analysis of gastric contents: the flow rate of free hydrochloric acid in the lean portion is 14.6 mg, in the basal portion – 33 mg, in the stimulated portion – 49 mg. In all portions there is a large amount of mucus, leukocytes, squamous epithelium and starch grains.

Fluoroscopy of the stomach and duodenum: the esophagus is freely passable. The folds of the gastric mucosa are coarse, hypertrophied, filled with mucus. When tightly filled with a contrast agent, the stomach is shaped like a hook, with smooth and clear contours. The gatekeeper's channel is freely passable. The duodenal bulb is triangular in shape, evacuation is not disrupted.

Gastroduodenofibroscopy: the esophagus is freely passable, there is a small amount of fluid and mucus in the stomach. The gastric mucosa is pale pink in color, in the lower third of the body and the antrum of the stomach there are areas of fine-spotted hyperemia. The duodenal mucosa is pale pink in color. The bulbous department of the duodenum is freely passable.

Clinical diagnosis: the main one is Chronic gastritis with unchanged secretory function of the stomach, the stage of exacerbation; concomitant – Chronic decompensated tonsillitis.

Special research methods: the patient has determined the gluconeogenic function of the liver and some indicators of nitrogen metabolism. The fasting blood glucose level was 2.98 mmol/l, 30 minutes after glycerin loading – 3.46 mmol/l, after 1 hour – 3.81 mmol/l, after 2 hours – 4.16 mmol/l, after 3 hours – 3.46 mmol/L. The rate of glucose formation from glycerol is on average 0.83 mmol/l/hour, the tolerance of peripheral tissues to glucose of gluconeogenic genesis is 0.48 mmol/l.

Ammonia in the blood on an empty stomach – 151.5 mmol / l, 3 hours after glycerin load – 92.0 mmol / l; urea, respectively – 4.53 and 3.84 mmol / l; glutamine, respectively – 755.36 and 743.86 mmol / l.

The child received complex treatment, which included diet therapy, antibiotic therapy (oxacillin), antispasmodics, vitamins, stimulants, ENT doctor's prescriptions, medical glycerin and essentiale.

Before discharge from the hospital, special studies were repeated. It was found that the fasting blood glucose level was equal to 2.61 mmol/l, 0.5, 1, 2 and 3 hours after glycerin loading, this indicator corresponded to 2.86, 3.59, 3.26 and 2.86 mmol/l. On average, the increase in newly formed glucose in the patient was equal to 0.62 mmol / l / hour, and the tolerance of peripheral tissues to glucose of gluconeogenic genesis, after treatment, corresponded to 0.25 mmol / l.

The level of ammonia on an empty stomach was equal to – 126.2 mmol / l, and 3 hours after glycerin load – 81.0 mmol / l, the level of urea in the blood on an empty stomach and after glycerin load in this

patient after treatment was equal, respectively – 3.84 and 3.33 mmol / l. and glutamine – 743.86 and 732.36 mmol / l, accordingly.

So, the patient under the influence of treatment, the gluconeogenic function of the liver was normalized, the absorption of glucose by peripheral tissues improved. As for the indicators of nitrogen metabolism, the levels of ammonia, urea and glutamine in the blood of this patient after treatment did not differ from those of healthy children.

This means that as a result of the treatment in patients with CG, the intensity of gluconeogenic liver function is restored to normal, the tolerance of peripheral tissues to glucose of gluconeogenic genesis is reduced and the indicators of ammonia, urea and glutamine in the blood are normalized. The performed glycerin load leads to a decrease in ammonia levels, enhances the urea-forming function of the liver and the work of the glutamic acid-glutamine cycle.

Let us now turn to the discussion of changes in some end products of nitrogen metabolism in patients with CGD as a result of therapy. We found that in these patients, after treatment, the breakdown of tissue proteins slows down somewhat. This is evidenced, in particular, by a decrease in their fasting blood levels of ammonia (Table 16). However, as can be seen from Table 16, the quantitative ratio of ammonia, urea and glutamine in patients with CGD, even at discharge, remained higher than in practically healthy children. In other words, on average, the treatment of patients with CGD was not effective enough to fully normalize the indicators of nitrogen metabolism. The picture turned out to be different after the glycerin load was carried out. Only in this case, that is, with the combination of treatment and administration of glycerin, the ammonia content in the blood of patients with CGD decreased by 49.70 mmol / l, after which it began to correspond to the norm.

Table 16.

The effect of glycerin load on the content of ammonia, urea and glutamine in the blood of patients with chronic gastroduodenitis after treatment.

Indicators	Time	Healthy Children	Patients with CGD (n=63)		P_1	P_2
			Before	After		

		(n = 22)	treatment	treatment		
Ammonia (in mmol/l)	1	126,14±11,1	208,30±7,32	143,37±5,31	>0,1	<0,001
	2	83,11±10,5	145,14±8,39	93,67±6,08	>0,5	<0,001
	P	<0,01	<0,001	<0,001		
Urea (in mmol/l)	1	4,03±0,13	4,78±0,08	4,31±0,05	<0,05	<0,001
	2	2,84±0,14	4,42±0,09	3,91±0,057	<0,001	<0,001
	P	<0,001	<0,01	<0,001		
Glutamine (in mkmol/l)	1	738,14±17	788,68±6,04	763,67±5,52	>0,2	<0,001
	2	714,19±19,5	754,50±5,7	738,2±5,48	>0,2	<0,05
	P	>0,5	>0,001	>0,001		

Note: 1 and 2 - indicators on an empty stomach and 3 hours after glycerin loading; P - the reliability of differences in the corresponding groups before and after loading; P1 - the reliability of differences in patients with CGD treatment compared with healthy children; P2 - the reliability of differences in patients with CGD before and after treatment.

At the same time, in patients with CGD after treatment, glycerin load reduced the content of urea in the blood by 0.40 mmol/l, and glutamine - by 25.47 mmol / l.

Later we found out that various methods of drug treatment significantly contributed to the improvement of the clinical picture and indicators of nitrogen metabolism in patients with CGD.

We found that in group IV of treatment of patients with CGD, the content of ammonia on an empty stomach, under the influence of complex therapy, decreased to 132.4±4.56 mmol/l, which turned out to be significantly lower than this indicator in patients with group I of treatment (162.71±7.12 mmol/l; P<0.001), and almost the same with the level of ammonia in healthy children (126.14±11.1 mmol/l). In patients with CGD, as a result of treatment with essentiale (group II) and medical glycerin (group III), there was also a significant decrease in the level of ammonia in the blood on an empty stomach, which amounted to

140.2±5.28 and 138.23±4.29 mmol/l, respectively, which was also significantly lower than in patients with the traditional method of treatment (Group I) (Table 17).

Table 17.

The effect of glycerin load on the content of ammonia, urea and glutamine in the blood of patients with CGD, depending on treatment.

Indicators	Time	Patients with chronic gastroduodenitis			
		I – group (n=20)	II – group (n=14)	III-group (n=14)	IV-group (n=15)
Ammonia (in mmol/l)	1	162,71±7,12	140,20±5,28	138.23 ±4,29	132,4 ±4,56
	2	113,21±5,29	88,95 ±5,71	85,96 ±6,81	86,7 ±6,54
	P	<0,001	<0,001	<0,001	<0,001
Urea (in mmol/l)	1	4,58 ±0,048	4,32 ±0,046	4,19 ±0,06	4,18 ±0,059
	2	4,09 ±0,064	3,95 ±0,05	3,83 ±0,062	3,77 ±0,059
	P	<0,001	<0,001	<0,001	<0,001
Glutamine (in mkmol/l)	1	772,7 ±5.24	765,1 ±6,47	762,57 ±5,21	754,4 ±5,17
	2	743,3 ±5,88	738,74±5,63	741,26 ±4,99	729,66 ±5,43
	P	<0,001	<0,001	<0,01	<0,001

Note: 1 and 2 - indicators on an empty stomach and 3 hours after glycerin loading; P - the reliability of differences in patients with chronic gastroduodenitis before and after loading.

Glycerin loading reduced the concentration of ammonia in all four groups of CGD. Therefore, 3 hours after oral administration of glycerin, ammonia in the blood of group I patients decreased by 1.43 times (P< 0.001), in the second group - by 1.57 times (P< 0.001), in the third group - by 1.60 times (P<0.001) and in the fourth - by 1.52 times (P<0.001), compared with its skinny level. At the same time, the

hypoammonioemic effect of glycerin loading was manifested in patients of groups II, III, IV of treatment of CGD, as a result of which, the level of ammonia in the blood began to correspond to the norm.

The content of urea in the blood before discharge in patients of I, II, III and IV treatment groups, on an empty stomach were also different and were, respectively - 4,58±0,048, 4,32±0,04, 4,19±0,06 and 4.18±0.06 mmol/l. Glycerin load in all treatment groups led to a significant decrease in the concentration of urea in the blood: in patients with CGD of group I of treatment, it decreased by 1.11 times (by 0.49 mmol/l) and amounted to 4.09±0.06 mmol/l (P<0.001); group II - decreased by 1.09 times (by 0.37 mmol/l) and averaged 3.95±0.05 mmol/l (P<0.001); Group III - by 0.36 mmol/l and amounted to 3.83±0.06 mmol/l (P<0.001); and in patients of group IV of treatment, the urea concentration decreased by 0.41 mmol/l, averaging 3.77±0.06 mmol/l (P<0.001). As for the content of glutamine in the blood, it was almost the same in patients with different treatment regimens and even corresponded to those of healthy children.

In confirmation of what we have stated above, we give an extract from the medical history:

Patient Z., 10 years old, medical history No. 1016/388. She was admitted to the clinic with complaints of pain in the epigastric region 2-3 hours after eating, nausea, vomiting, a feeling of bitterness in the mouth, weakness.

From the anamnesis, the girl has been ill for 1 year, is being treated for the first time. From the anamnesis it turned out that in the last 2 years she did not observe the diet. The transferred diseases are frequent sore throats, pneumonia, a tonsillectomy operation was performed a year ago.

Objectively: general condition of moderate severity. The skin and visible mucous membranes are pale in color, clean. Subcutaneous fat is poorly developed. Peripheral lymph nodes are not enlarged. Bone and joint system without deformities. A clear pulmonary sound is percutorially determined above the lungs, vesicular breathing is auscultatively listened to. The heart area is without visible changes,

auscultation - heart tones are muffled, there are no noises. The pulse is rhythmic, of medium filling and tension, 88 beats per minute.

There are carious teeth in the oral cavity. The tongue is overlaid with a white coating. The throat is pale pink, clean. The abdomen is of the correct shape, with palpation there is pain in the epigastric and pyloroduodenal areas. The liver protrudes from under the edge of the costal arch by 1.0 cm, painless on palpation, the spleen is not enlarged. The chair is prone to constipation. Urinary system without features.

General tests of blood, urine and feces without features.

Analysis of gastric contents: the flow rate of free hydrochloric acid in the lean portion is 7.0 mg, in the basal portion – 55.8 mg, in the stimulated portion – 108.0 mg.

Duodenal probing: in portion "A" mucus, leukocytes and epithelium in small amounts.

Fluoroscopy of the stomach and duodenum: we pass the esophagus freely, on an empty stomach the contents of the secret are in the stomach. The folds of the gastric mucosa are rough, thickened, with a tight filling of the stomach with a hook-shaped contrast, we shift, with clear and even contours. The gatekeeper is free to pass. The bulb of the duodenum is triangular in shape.

Gastroduodenofibroscopy: the esophagus is freely passable. There is a small amount of mucus and secretions in the stomach. The gastric mucosa is pale pink in color, with moderate-spotted hyperemia in places. The gatekeeper is freely passing, swollen. The duodenal mucosa is finely spotted in places, hyperemic, edematous. The bulbous part of the duodenum is free.

Doctor's conclusion: Curvature of the nasal septum. Dentist's conclusion: Dental caries.

Clinical diagnosis: the main one is Chronic gastroduodenitis with increased secretory function of the stomach, the stage of exacerbation, concomitant – Polyhypovitaminosis. Curvature of the nasal septum. Tooth decay.

Own research. Fasting blood glucose is 3.26 mmol/l, 0.5, 1, 2 and 3 hours after glycerin loading, respectively - 3.59, 4.24, 4.64 and 4.24 mmol/l. The rate of synthesis of newly formed glucose is 1.11 mmol/l/hour. The tolerance of peripheral tissues to gluconeogenic glucose was 0.98 mmol/l.

The level of ammonia in the blood on an empty stomach was equal to 226.6 mmol/l, 3 hours after glycerin loading 126.0 mmol/l. Urea on an empty stomach - 4.80 mmol / l, after loading - 4.16 mmol /l. The level of glutamine in the blood on an empty stomach was 789.84 mmol / l, and 3 hours after glycerin loading - 766.58 mmol / l.

The child received complex treatment, which included diet therapy, antibiotic therapy (oxacillin), antacids, antispasmodics, vitamins, stimulants, medical glycerin and essentiale.

After the treatment, special studies of carbohydrate and nitrogen metabolism indicators were repeated. It was found that the fasting blood glucose level was equal to 3.06 mmol/l, after a glycerine load after 0.5, 1, 2 and 3 hours it was equal to 3.91, 4.03, 3.91 and 3.56 mmol/l, respectively. The rate of gluconeogenesis was 0.77 mmol/l/hour, and the tolerance of peripheral tissues to glucose of gluconeogenic genesis was 0.50 mmol/l.

The concentration of ammonia in the blood on an empty stomach was equal to 133.0 mmol / l, and 3 hours after the glycerin load was equal to 86.0 mmol /l. The level of urea in the blood on an empty stomach was 4.16 mmol/ l, and after glycerin load – 3.84 mmol / l. Glutamine in the blood on an empty stomach and 3 hours after loading with glycerin was equal to 755.36 and 743.86 mmol/l, respectively.

So, after treatment, not only the clinical picture of the disease improved in the patient, but also the indicators studied by us changed: the gluconeogenic function of the liver stabilized, glucose uptake by peripheral tissues improved, the indicators of nitrogen metabolism in the blood normalized.

Considering changes in some indicators of nitrogen metabolism in patients with DU after treatment, we found that the breakdown of tissue proteins significantly slows down in them, as evidenced by a decrease in

the amount of ammonia in the fasting blood, on average by 74.87 mmol/l (P<0.001, r= 0.77) (Table 18).

Table 18.

The effect of glycerin load on the content of ammonia, urea and glutamine in the blood of patients with DU after treatment.

Indicators	Time	Healthy (n = 22)	Patients with DU (n=19)		P_1	P_2
			Before treatment	Before treatment		
Ammonia (in mmol/l)	1	126,14±11,1	228,51±8,48	153,64 ±6,97	<0,05	<0,001
	2	83,11±10,5	163,8 ±5,5	101,24 ±6,43	>0,1	<0,001
	P	<0,05	<0,001	<0,001		
Urea (in mmol/l)	1	4,03±0,13	5,04 ±0,07	4,435 ±0,074	<0,01	<0,001
	2	2,84±0,14	4,66 ±0,10	4,01 ±0.068	<0,001	<0,001
	P	<0,001	<0,01	<0,001		
Glutamine (in mkmol/l)	1	738,14±17	827,79±6,82	789,43 ±6,74	<0,01	<0,001
	2	714,19±19,5	772,32 ±4,9	747,1 ±5,22	>0,1	<0,001
	P	>0,5	>0,001	>0,001		

Note: 1 and 2 - indicators on an empty stomach and 3 hours after glycerin loading; P - the reliability of differences in relation to the indicators on an empty stomach and after loading in the corresponding group; P1 - the reliability of differences in patients with DU after treatment compared with healthy children; P2 - the reliability of differences in patients with DU before and after treatment.

The urea content after treatment decreased by an average of 0.605 mmol/l (P<0.001, r= 0.84), and glutamine - by 38.36 mmol/l (P<0.001, r= 0.91). However, as can be seen from Table 18, in quantitative terms, the content of these indicators of nitrogen metabolism in patients with DU at discharge, as well as in patients with HCG, remained higher than in practically healthy children.

3 hours after the glycerin load, the ammonia content in the blood decreased by 62.55 nmol / l, after which it began to correspond to the norm. Glycerin load decreased the content of urea by 0.647 mmol/l and glutamine, on average by 25.21 mmol/l in patients with DU. As a result of treatment and the use of glycerin load, the concentration of ammonia, urea and glutamine began to correspond to those of healthy children.

After we have found out in total the effect of the treatment on the shifts of nitrogenous parameters in the blood, we now turn to the consideration of questions about the effect of various methods of drug treatment on the clinical picture of the disease and the state of nitrogen metabolism in patients with DU (Table 19).

Table 19.

The effect of glycerin load on the change in the content of some indicators of nitrogen metabolism in the blood of patients with DU, depending on the treatment methods.

Indicators	Time	Patients with DU (n=19)			
		I – group	II – group	III- group	IV-group
Ammonia (in mmol/l)	1	176,73±9,33	151,9±9,71	145,05±3,72	140,8±5,13
	2	128,76±7,46	93,5±6,7	92,25±6,34	90,48±5,22
	P	<0,001	<0,001	<0,001	<0,001
Urea (in mmol/l)	1	4,78 ±0.091	4,42±0,074	4,29±0.074	4,25±0,059
	2	4,19 ±0,065	4,052±0,084	3,935±0,057	3,88±0,066
	P	<0,001	<0,001	<0,001	<0,001
Glutamine (in mkmol/l)	1	807,19±6,64	787,03±7,38	789,84±4,91	773,6±7,77
	2	760,92±4,83	743,86±4,69	746,73±4,69	736,9±5,89
	P	<0,001	<0,001	<0,01	<0,001

Note: 1 and 2 - indicators on an empty stomach and 3 hours after glycerin loading; P - the reliability of differences in relation to the corresponding group of patients with DU before and after loading.

As can be seen from tables 18 and 19, we noted a positive dynamics of impaired nitrogen metabolism in the treatment of patients with DU in all groups. Thus, the level of ammonia in the blood of patients with DU with the traditional method of therapy decreased by 1.29 times and amounted to 176.73±9.33 mmol /l (P<0.001), in patients of group II, in combination with essentiale with traditional therapy, decreased by 5 times and amounted to 151.9±9.71 mmol/l (P<0.001), in patients of group III of treatment, in combination with glycerin with traditional therapy decreased by 1.57 times and amounted to 145.05±3.72 mmol/l (P<0.001) and in patients of group IV, in a complex of essentiale, glycerin and traditional treatment, decreased by 1.62 times, averaging 140.8±5.13 mmol/l (P<0.001). We have noted a positive effect in this regard in patients with DU who received essentiale, medical glycerin and especially their combination in combination with the traditional method of treatment, compared with patients of group I who received treatment only by the traditional method.

Despite the effectiveness of therapeutic measures, the level of highly toxic ammonia in the blood of patients with DU was still higher than in practically healthy children. Glycerin load reduced the concentration of ammonia in the blood to the same extent in all four groups. Therefore, 3 hours after oral administration of glycerin, the ammonia content in the blood of patients with DU by treatment groups was, respectively - 128,76±7,46, 93,5±6,7, 92,25±6,34 and 90.48±5.22 mmol/l. The high hypoammonioemic effect of glycerin loading in patients with DU led to a decrease in the level of ammonia characteristic of healthy children.

The content of urea in the blood of patients with fasting DU, depending on the method of treatment used, was, respectively - 4,78±0,091, 4,42±0,074, 4,29±0,074 and 4.25±0.059 mmol/l. Although these data exceed the level of urea in the blood of healthy children, but nevertheless, there is a positive trend in relation to urea, especially pronounced in patients of II, III and IV treatment groups. The glycerin

load resulted in a significant decrease (P<0.001) in the concentration of urea to the same extent in all groups. The hypoureoemic effect of glycerin loading led to normalization of urea levels in the blood of patients with DU in all groups. It is interesting to note the fact that in healthy children, the load of glycerin reduces the level of urea by 1.42 times, and in patients with DU after treatment - by an average of 1.1 times. In our opinion, this is probably due to the fact that in patients with DU, due to increased breakdown of tissue proteins and hyperammonioemia, this highly toxic product is neutralized by increasing the ornithine cycle of urea formation. We believe that the glycerin load was an objective criterion for monitoring changes in nitrogen metabolism in patients with DU. On this basis, we recommend glycerin loading as an additional criterion for monitoring the state of metabolism in patients with chronic CGDP.

As for glutamine, its content in the blood of patients with fasting diabetes mellitus, as a result of treatment, statistically significantly dccreased and amounted to 807.19±6.64 mmol/l in patients of the first treatment group, 787.035±7.38 in the second group, 789.84±4.91 in the third and 773.64±7.77 mmoi/l in the fourth group. (P<0.001), although its level still exceeded those of practically healthy children. This indicates that in patients with DUC, in addition to urea formation, the glutamic acid-glutamine system is also affected in the neutralization of ammonia, which is reflected in an increase in its level in the blood. Only after glycerin loading, the level of glutamine in the blood of patients decreased to standard figures, and this decrease was almost the same for all treatment groups.

Thus, the results obtained by us indicate that the drug treatment of children with DU leads to an improvement in a number of parameters of carbohydrate and nitrogen metabolism.

In confirmation of the above, we give a clinical example:

Patient L., 14 years old, medical history No. 2948/1519, was admitted to the clinic with complaints of pain in the epigastric region 20-30 minutes after eating, nausea, heartburn, belching. Abdominal pains are nocturnal, of a hungry nature. Ill for 4 years, treated for the

first time. A girl from the third pregnancy and childbirth. It was found out from the anamnesis that the father suffers from gastritis, the mother died during childbirth.

Objectively: general condition of moderate severity. The skin and visible mucous membranes are pale, subcutaneous fat is moderately developed. Peripheral lymph nodes are not enlarged. Bone and joint system without deformities. Percussion over the lungs - a clear pulmonary sound, auscultative vesicular breathing is heard. The area of the heart is unchanged, the boundaries of the heart are within the age norm. The heart tones are muted, the pulse is rhythmic, satisfactory filling and tension, 90 beats per minute.

The tongue is moist, overlaid with a whitish coating. The pharynx is pale pink in color, the tonsils are enlarged. There are carious teeth in the oral cavity. The abdomen is of a regular shape, soft on palpation, painful in the epigastric and pyloroduodenal areas. The liver and spleen are not palpable. Chair once every 2-3 days. Urinary system without features.

General tests of blood, urine and feces without features.

Analysis of gastric contents: the flow rate of free hydrochloric acid in the lean portion is 13.0 mg, in the basal portion – 36.5 mg, in the stimulated portion – 91.2 mg. In all portions, mucus, leukocytes and epithelium in large quantities.

Roentgenoscopy of the gastrointestinal tract: The esophagus is passable, the gastric mucosa is smoothed. "washed out" with a secret, with a tight contrast filling, the stomach is shaped like a hook with smooth and clear contours. Peristalsis on both curvatures is sluggish. We pass the gatekeeper's channel. The bulb of the duodenum is triangular in shape, with palpation, soreness is noted.

*Gastroduodenofibroscopy: the esophagus is freely passable. The gastric mucosa is smooth, pale pink in color, small-point hyperemia is noted in places. The gatekeeper is free to pass. There is an ulcer on the wall of the duodenal bulb, measuring 0.6 * 0.8 cm, the bottom is covered with a whitish-gray coating, the edges are hyperemic, edematous. The bulbous part of the duodenum is free.*

Doctor's conclusion: Chronic decompensated tonsillitis. Dentist's conclusion – dental caries.

The gluconeogenic function of the liver and some indicators of nitrogen metabolism were studied in the patient. Fasting blood glucose is 3.26 mmol/l, 0.5, 1, 2 and 3 hours after glycerin loading, respectively - 3.59, 3.91, 4.35 and 4.24 mmol/l. The rate of glucose formation from glycerol is 0.906 mmol/l/hour, the tolerance of peripheral tissues to newly formed glucose is 0.98 mmol/l.

Ammonia in the blood on an empty stomach is equal to - 226.6 mmol / l, and 3 hours after loading with glycerin - 151.5 mmol / l. The level of urea in the blood before the load was 4.99 mmol / l, and 3 hours after the load - 4.38 mmol / l. The concentration of glutamine in the blood on an empty stomach and after loading with glycerin corresponded to 830.88 and 777.80 mmol/l.

Clinical diagnosis: the main one is duodenal ulcer with increased secretory function of the stomach, the stage of exacerbation; concomitant - Chronic decompensated tonsillitis. Tooth decay.

The child received complex treatment, which included diet therapy, oxacillin, antacids, antispasmodics, sedatives, stimulants, vitamins, as well as additionally medical glycerin and essentiale.

After the treatment, we present the results of a study of the gluconeogenic function of the liver and indicators of nitrogen metabolism. Fasting blood glucose - 2.93 mmol /l, 0.5, 1, 2 and 3 hours after glycerin load, respectively - 3.26, 3.87, 3.76 and 3.59 mmol / l. The rate of glucose formation from glycerin is 081 mmol/l/hour. The tolerance of peripheral tissues to glucose of gluconeogenic genesis is 0.66 mmol/l.

Ammonia in the blood on an empty stomach - 138.6 mmol / l, 3 hours after loading with glycerin - 96.0 mmol / l. Urea in the blood on an empty stomach and after exercise was equal, respectively - 4.16 and 3.84 mmol / l. Glutamine in the blood on an empty stomach and 3 hours after glycerin load, respectively – 777.8 and 755.36 mmol / l.

So, in this patient, as a result of complex treatment with the use of diet therapy, glycerin and essentiale, along with conventional treatment, the gluconeogenic function of the liver has significantly improved, which is expressed in a decrease in the rate of glucose neoplasm from glycerin and improved absorption of gluconeogenic glucose by peripheral tissues. Also, her fasting blood glucose concentration approached the normative indicators.

As for the indicators of nitrogen metabolism, they also decreased and began to correspond to those of healthy children.

Thus, the positive effect of glycerin and essentiale in our studies has shown the expediency of their use in the treatment of CG, CGD and DU, from the standpoint of their active influence on the state of carbohydrate and nitrogen metabolism, and gluconeogenic liver function.

The clinical evaluation of the effectiveness of treatment of patients with CG, CGD and DU showed that the terms of disappearance of subjective and objective manifestations of relapse of the disease are different depending on the method of treatment (Table 20). Thus, with CG in the IV treatment group, the pain disappeared by 5.2 ± 2.2 days, and in the first - by 8.1± 2.1, in patients with CGD, respectively - by 6.4±2.7 and 10.4±2.3 days, and in patients with DU - by 8.3±2.4 and 13.1±2.1 days of treatment.

Table 20

Dynamics of objective-subjective data recovery in children with CGDP depending on the therapy.

№	Efficiency criterion (in days)	Treatment group	CG (M ± m)	CGD (M ± m)	DU (M ± m)
1	Disappearance of pain	I	8,1±2,1	10,4±2,3	13,1±2,1
		II	5,2±2,2	6,4±2,7	8,3±2,4
		P	>0,5	>0,2	>0,2
2	Disappearance of soreness during palpation	I	15,4±1,9	19,2±1,7	25,1±2,2
		II	9,3±1,7	12,6±1,6	19,6±1,8
		P	<0,05	<0,01	>0,1

3	Disappearance of intoxication symptoms	I	19,3±1,7	20,4±1,7	25,7±1,8
		II	13,8±1,6	16,2±1,6	19,3±1,8
		P	<0,05	<0,05	<0,05
4	Normalization of dyspeptic disorders	I	15,3±2,4	19,5±2,2	21,1±2,
		II	10,1±2,2	13,2±2,1	14,9±2,4
		P	>0,1	<0,05	>0,2
5	Bed days	I	22,3±1,1	23,4±0,8	28,1±1,0
		II	19,3±0,8	21,0±0,9	25,0±0,9
		P	<0,05	<0,05	>0,1

Note: P - the reliability of differences between the data of conventional therapy (I, CG=10, CGD=20, DU =6 patients) and complex therapy (II, CG=8, CGD=15, DU=5 patients), with the use of essentiale and medical glycerin

Dyspeptic phenomena in patients of the IV group of CG treatment disappeared by 10.1 ± 2.2, and in the first group - by 15.3± 2.4 days of treatment. In patients with CGD in similar groups - by 13.2 ±2.1 and 19.5±2.2, and in patients with DU, respectively, by 14.9±2.4 and 21.1±2.6 days of treatment.

Pain during palpation in the epigastric region disappeared with CG in the fourth and first treatment groups, respectively, by 9.3±1.7 and 15.4±1.9 days of treatment. With CGD, palpation pains in the epigastric and pyloroduodenal areas disappeared in group IV - by 12.6±1.6 and 19.2±1.7 days, and in patients with DU, respectively, by 19.6±1.8 and 25.1±2.2 days of treatment.

It should be noted that if in patients with CG of group I who received conventional treatment, the average number of bed days was 22.3, with CGD - 23.4 and with DU - 28.16 bed days, then in patients who received additional essentiale and medical glycerin (group IV), it was equal, respectively - 19.37, 21.06 and 25.0.

Concluding this chapter, it should be noted that traditionally accepted treatment has a positive effect on the disturbed metabolism in the body of sick children with CGDP, however, complete normalization of metabolic disorders is not achieved.

Thus. the data obtained by us indicate that the appointment of additional essentiale and medical glycerin, especially in their combination, along with conventional treatment, makes it possible for a faster trend of positive changes in clinical and biochemical parameters, which is due to their peculiarity to improve the functional state of the liver in providing the body with energy material, as well as to have good neutralizing properties in relation to the products of metabolism and tissue breakdown, rapid elimination of toxic products from the body, without pronounced side effects.

Summing up the results presented in this chapter, we can note that the treatment of patients with CG, CGD and DU with medical glycerin and essentiale is pathogenetically justified, which have a beneficial effect on the course of diseases, contributing to a reduction in their hospital stay, respectively, by an average of 2.93, 2.34 and 3.16 bed days.

CHAPTER V. DISCUSSION OF THE RESULTS OBTAINED

The widespread prevalence of digestive diseases among children, their chronic recurrent course, involvement in the pathological process of adjacent organs, severe complications, often leading to disability of the patient, dictates the need to develop measures aimed at preventing and treating diseases. Despite the successes achieved in the study of the etiology, pathogenesis, diagnosis and treatment of CGDP in children, some aspects of its pathogenesis and treatment are still not fully resolved and require further study.

In the body of patients with chronic GDP disorders of lipid, carbohydrate and nitrogen metabolism develop, numerous vital liver functions are upset. All this aggravates the severity of CG, CGD and DU, complicates the prognosis, dictates the need for medical correction of impaired homeostasis, additional measures in the catamnesis, and so on. Consequently, the detailing of metabolic disorders, the development of previously unknown aspects of it in the body of patients with chronic GDP opens up new prospects for pathogenetically justified correction, treatment, disease prediction, rehabilitation measures, which is relevant for modern gastroenterology. Nitrogen metabolism is still insufficiently studied, especially in complex comparative terms, in patients with CG, CGD and DU, and one of the specific functions of the liver is gluconeogenic, in these diseases it has practically not yet attracted the attention of pediatricians and therapists.

Meanwhile, due mainly to the gluconeogenic function of the liver, glycogen accumulates in hepatocytes - the most important plastic and energy material, normoglycemia is maintained, which is necessary for the normal functioning of vital organs, cell bioenergetics, biosynthesis of interchangeable amino acids, nitrogenous bases and other metabolites.

By studying the process of gluconeogenesis, which proceeds most effectively in the liver, it is possible to reveal new aspects of the pathogenesis of chronic GDP in children, additional criteria for the diagnosis and differential diagnosis of CG, CGD and DU. In addition, the state of this liver function can be used as a control over treatment, prognosis of chronic diseases of the stomach and intestines (duodenum),

as well as for the development of appropriate drug therapy, which determines the relevance of our work.

Based on the above, we have studied the state of nitrogenous, carbohydrate metabolism and gluconegenic liver function in a comprehensive comparative plan, in various variants of observation, and also evaluated the effect of treating patients with CG, CGD and DU essentiale and medical glycerin. The interrelation of changes in the functional state of the liver with the duration of the disease was also revealed and the effectiveness of various corrective treatment methods on the clinical course of the disease and on the shifts of these types of metabolism was evaluated.

This paper presents the data of 116 sick children with chronic GDP aged 7-14 years, who were examined and treated in the children's department of the clinic No. 2 SamMI, of them with CG - 34, CGD - 63 and DU - 19 patients. The diagnosis of the disease in them was established after a thorough clinical, laboratory and instrumental examination. The selection of patients was carried out strictly individually, attention was paid to the connection of the disease with the nature of nutrition, previous illnesses, living conditions, national customs, daily routine, stressful situations and other factors that would be important in the development of gastroduodenal pathology. A special card was filled out for each patient, developed by the staff of the Department of Children's Diseases No. 1 of SamMI, in which special attention was paid to heredity.

22 practically healthy children, from among the students of secondary school No. 1.Samarkand, the same age made up the control group.

Among the examined patients, there were more girls (66.4%), which was about twice the number of boys (33.6%). In the age aspect, out of the total number of patients, the majority (75.0%) were aged 12-14 years. Further, we found that 46.6% of the examined patients suffered from chronic GDP before 1 year, 44.8% - within 1-3 years and 8.6% - over 3 years.

Generally recognized laboratory and instrumental methods of research were carried out in sick children - general analysis of blood, urine, feces, gastric and duodenal probing, X-ray and endoscopic studies. Along with the generally accepted methods, special research methods were also carried out.

To assess the state of gluconeogenic liver function, we used the technique of N.V.Blinova, developed at the Department of Biochemistry of SamMI. The blood glucose content was determined by a highly specific enzymatic method in the glucose oxidase-peroxidase-orthotoluidine system. The concentration of urea in the blood serum was determined by the diacetyl monoxime method according to S.B.Bagker, a set of reagents from the company "La-chema". Ammonia and glutamine in the blood were determined by the method of A.I.Silakova and co-authors.

Our observations confirm the significant role of eating disorders (68.1%) and stressful situations (60.2%) in the development of CGDP. Genealogical analysis of pedigrees showed that 65.3% of patients suffering from CGDP have relatives with certain diseases of the gastrointestinal tract.

All children were admitted to the hospital during the period of exacerbation of the disease. CG was mainly characterized by pain in the epigastric region, nausea was noted in 55% of patients with CG, heartburn was detected in 36%, constipation - in 38% of patients. With CG, more than half of the patients (61.9%) had pain localized in the pyloroduodenal region, the Moyningan rhythm of pain was detected in 27% of patients, the disease was characterized by dyspeptic disorders. In patients suffering from DU, the clinical picture of the disease was brighter, compared with patients with CG and CGD.

Starting to discuss the results we have obtained, it should be emphasized that the literature presents ambiguous data regarding changes in blood glucose levels in patients with chronic GDP.

As follows from our data, fasting blood glucose concentration in patients with CG (2.91±0.12 mmol/L), CGD (3.10±0.11 mmol/L) and DU (3.00±0.09 mmol/L) was statistically higher than in practically

healthy children (2.76±0.11 mmol/l; P - respectively >0.5; <0.05 and >0.1). This increase in the level of glucose in the blood of patients may be the result of several reasons: excessive consumption of carbohydrates with food, severe emotional shocks, concomitant diabetes mellitus. However, these possible causes of an increase in blood glucose levels were excluded by us already during the examination of patients. It could also be assumed that an increase in blood glucose reflects a more intensive absorption of carbohydrates in the gastrointestinal tract in patients with chronic GDP, however, we have not found any works in the literature on this issue.

It is possible, in addition, that the blood glucose level in patients with chronic GDP increases due to increased glycogenolysis, although in this case, due to small glycogen reserves in the body, hyperglycemia may be only short-term. Finally, a higher blood glucose content in patients with chronic GDP may reflect a reduced tolerance of peripheral tissues to glucose or be a consequence of enhanced gluconeogenesis.

However, until now it was not known how the threshold of sensitivity of peripheral tissues of patients with CG, CGD and DU to glucose of gluconeogenic genesis changes. Gluconeogenic liver function in chronic GDP in adults and children does not attract the attention of gastroenterologists at all. In our studies, for the first time, data on changes in the sensitivity of peripheral tissues to glucose formed from a non-carbohydrate compound (glycerin) are presented in sick children with CG, CGD and DU, as well as shifts in their gluconeogenic liver function, studied under the correct conditions.

It was found that after oral administration of glycerin during the three-hour study period, 0.70 mmol/l/h of newly formed glucose was formed in practically healthy children, 0.82 in patients with CG, 0.92 in patients with CGD and 1.12 mmol/l/h of newly formed glucose with DU. This means that the gluconeogenic function of the liver in patients with CG exceeds the norm by 1.17 times, and in patients with CGD and DU, respectively, by 1.31 and 1.60 times.

Consequently, non-carbohydrate (nitrogen-free and nitrogenous) compounds are used to maintain carbohydrate homeostasis more

intensively than normal in patients with CG, CGD and DU. Apparently, therefore, in patients on an empty stomach, the glucose content in the blood is higher than in healthy children. Moreover, the increased use of these compounds to perform the gluconeogenic function and, consequently, their misuse may underlie the often observed emaciation of patients with DU and CGD. These data reveal new aspects of the pathogenesis of chronic GDP, substantiate the need for a complete protein diet and the development of additional dietary measures aimed at weakening protein catabolism in the body of patients with CGD and DU.

Then, we conducted a dynamic study of the gluconeogenic function of the liver, where glycerin was the substrate for it. We found that throughout the entire observation, the concentration of newly formed glucose in the blood of patients with CGDP was higher than in practically healthy children. So, by the third hour after loading with glycerin, on average, the blood glucose level in patients with CG by 0.50 mmol/l, in patients with CGD - by 0.58, and in patients with DU - by 0.94 mmol/l exceeded the norm. Consequently, the tolerance of peripheral tissues not only to exogenous glucose, but also to glucose synthesized from non-carbohydrate nitrogen-free compounds is reduced in patients with CG, CGD and DU. These results were obtained by us for the first time, they may be a consequence of the action of counterinsular factors, a decrease in insulin receptors in tissues, etc. An important role in the appearance of diabetogenic curves in patients with chronic GDP, in our opinion, belongs to a high concentration of ammonia.

Later, when analyzing the intensity of gluconeogenic liver function in patients with chronic GDP depending on the prescription of the disease, the following fact was established: with an increase in the limitation period of the disease, the rate of glucose synthesis from glycerol in the liver turns out to be high in patients suffering from CG over 1 year -0.91 mmol/l/h, CGD - 1.04 and DU - 1.17 mmol/ l/h, compared with patients with a prescription of less than 1 year, in the corresponding groups (r= 0.59; r= 0.78; r= 0.64). The obtained results reveal some metabolic aspects of the evolution of CG, CGD and DU, as

well as justify the change in the diet of patients, depending on the degree of chronization of the process.

It is interesting to note that with an increase in the limitation period of the disease, the tolerance of peripheral tissues to newly formed glucose decreases (r = 0.91, 0.84, 0.76, respectively). In other words, the degree of violation of carbohydrate metabolism in these patients is directly correlated with the duration of the disease.

Thus, for the first time, we have studied one of the most important functions of the liver in a complex, multidimensional manner in patients with CG, CGD and DU. To study the gluconeogenic function of the liver, an adequate technique in clinical conditions was used, a highly active nitrogen-free glucose precursor was selected. A pathogenetically substantiated mechanism of emaciation of patients with chronic GDP is proposed, which consists in excessive relocation of the flow of nitrogen-free and nitrogenous compounds from the pathway of protein synthesis to the synthesis of glucose, an energetically advantageous and easily utilized metabolite.

The data obtained by us reveal new aspects of the pathogenesis of chronic GDP in children, pathogenetically justified corrective therapy, and can be used for a clearer diagnosis and differential diagnosis of CG, CGD and DU in children.

For the first time, it was shown that patients with CG, CGD and DU have a reduced tolerance of peripheral tissues to glucose of gluconeogenic genesis, whose contribution to the total amount of carbohydrates in the body can be very significant.

As is known, in the reactions of gluconeogenesis, the carbon skeleton of non-carbohydrate compounds, thanks to key enzymes, is used for the synthesis of glucose. If the precursor of glucose is a nitrogenous compound, then functional groups, mainly amino groups, are included in the metabolism of nitrogenous compounds, the concentration of which consequently changes. In other words, since the stages of gluconeogenesis combine the pathways of carbohydrate and nitrogen metabolism, it is necessary to trace simultaneously shifts in nitrogen compounds. Based on this, we have studied in detail the state of

nitrogen metabolism in patients with CG, CGD and DU, as well as the effect on nitrogen metabolism of a load test (glycerin).

It was found that in patients with CG, CGD and DU there are significant changes in the nitrogen parameters studied by us. Thus, with CG, the content of ammonia, urea and glutamine in the blood exceeded the norm by 21.3, 16.12 and 1.66%. In patients with CGD, the concentration of ammonia, urea and glutamine in the blood was higher than normal, by 65.13, 18.61 and 6.83%, respectively, and in patients with DU, the level of the above indicators in the blood was the highest and exceeded those of healthy children, by 81.15, 25.06 and 12.14%, respectively.

Consequently, with chronic GDP, the breakdown of tissue proteins increases, which is reflected in an increase in the content of ammonia as the final product of nitrogen metabolism. Moreover, a certain increase in urea and glutamine in patients with chronic GDP is insufficient for complete neutralization of ammonia, as evidenced by the symptoms of intoxication of the central nervous system found in these patients. This means that the patients we examined had insufficient urea-forming liver function, especially in patients with CGD and DU.

We have discovered for the first time that a single oral administration of glycerin has a powerful hypoamminemic and hypoureoemic effect. Thus, the content of ammonia in the blood of patients with CG under the action of glycerin decreased by 38.6%, and urea - by 12.1%, with CGD decreased by 30.34 and 7.53%, respectively, and with DU - by 28.31 and 7.54%, respectively. After loading with glycerin in the blood of patients with CG, the level of glutamine practically did not change, and in patients with CGD and DU decreased, respectively, by 4.3 and 6.7%.

It is very significant that there is a direct proportional relationship between the high content of nitrogenous compounds in the blood and the hypo-azotemic effect of glycerin. The fact that we have established a deep decrease in the level of ammonia and urea in the blood of patients with CG, CGD and DU under the influence of glycerin may play an important clinical role for the normalization of nitrogenous homeostasis.

As for the mechanism of action of glycerin, we believe that it enhances the processes of reducing amination in the body. This, in turn, due to the weakening of the imbalance of nitrogenous compounds, apparently creates the necessary prerequisites for the synthesis of newly formed glucose.

As for the relatively low glutamine levels in patients with CG, CGD and DU, it seems that the glutamic acid-glutamine system is not effectively involved in the neutralization of ammonia, a powerful cytotoxic agent. We believe that in patients with chronic GDP hyperammonemia was a consequence of increased destruction of the gastric and duodenal mucosa, impaired amino acid transport, lack of B vitamins, imbalance in endocrine hormones, increased synthesis of glucose from the carbon skeleton of amino acids, as well as violations of the mechanisms of ammonia neutralization.

Then, we analyzed the changes in some indicators of nitrogen metabolism in patients with chronic GDP depending on the duration of the disease. Thus, in patients with CG, CGD and DU of the intestine with a disease duration of more than 1 year, on an empty stomach, the level of ammonia, urea and glutamine was significantly higher than in patients with a disease duration of up to 1 year, and we noted the highest rates of nitrogen metabolism in patients with CGD and DU.

After glycerin loading, the differences between patients, depending on the prescription period of the disease, turned out to be better pronounced. Thus, the content of ammonia in the blood under the influence of glycerin in patients with CG with a disease duration of up to 1 year decreased by 22.58%, and in patients suffering from CGD over 1 year – decreased by 21.9%. In patients with CGD and DU, with a disease duration of more than 1 year, after glycerin loading, the concentration of ammonia decreased by 42.07 and 37.16%, respectively, and in patients with a prescription period of less than 1 year, only by 15.04 and 8.64%, respectively. Consequently, we have established for the first time the fact of a weakened response of the body of patients with CG, CGD and DU suffering from less than 1 year to the hypoammonemic action of glycerin.

At the same time, glycerin loading decreased the level of urea in the blood by 0.91 mmol/l in patients with CG with a disease prescription of more than 1 year and by 0.26 mmol/l - with a prescription of less than 1 year. After glycerin loading, in patients with CGD with a disease duration of up to 1 year, the concentration of urea decreased by 0.12 mmol / l, and in patients with a disease duration of more than 1 year - by 0.60 mmol /l. Urea levels decreased by 0.26 mmol/L in patients with DU, with a prescription of less than 1 year, 3 hours after loading with glycerin, and in patients with a disease duration of more than 1 year - by 0.54 mmol/l. Hence, the hypoureoemic effect of glycerin in these patients depends on the prescription of the disease and is more stable in patients with a longer duration of the disease. We have established a direct positive correlation between an increase in the level of ammonia in the blood and an increase in the concentration of urea (r = 0.75) in patients with DU.

The content of glutamine in patients with CG of the first and second groups did not undergo significant changes after loading with glycerin, which, in our opinion, is due to the insufficiency of the glutamic acid-glutamine system in the neutralization of ammonia. However, in patients with CGD and DU with a disease duration of more than 1 year after glycerin loading, the glutamine content decreased by 53.2 and 80.4 mmol/l, respectively, which is 4.66 and 7.11 times higher than in patients with a disease duration of less than 1 year.

The data obtained once again confirm the information we presented earlier about a more significant violation of the functional state of the liver in CGD and DU, as evidenced by the fact that after loading with glycerin, complete normalization of ammonia content does not occur, and the level of urea also does not return to the data of practically healthy children.

The deep metabolic disorders we have established in CG, CGD and DU were the basis for studying the effect of treatment on the restoration of altered metabolism. To do this, we conditionally divided all patients with chronic GDP into 4 groups who received treatment according to the schemes accepted in the literature and their combination with corrective hepatotropic agents.

The first group consisted of 36 patients (CG =10, CGD=20 and DU=6 children) who were prescribed conventional complex therapy during their stay in the hospital, including diet, sedation, antispasmodics, drugs with reparative properties, antibiotics, antacids, vitamins, physiotherapy procedures.

The second group consisted of 27 patients (CG = 9, CGD= 14 and DU=4) who, along with conventional treatment, received medical glycerin, at the rate of 0.5 mg / kg of weight diluted in 200.0 ml of boiled water every 2 days.

The third group consisted of 25 children (CG= 7, CGD= 14 and DU = 4 patients) who, in addition to conventional treatment, additionally took essentiale - 2 capsules 2 times a day after meals.

The fourth group included 28 patients (CG=8, CGD= 15 and DU= 5) who received complex treatment, including conventional therapies in combination with glycerin and essentiale, for the entire period of treatment.

The clinical evaluation of the effectiveness of treatment of patients with CG, CGD and DU showed that the timing of the disappearance of subjective manifestations of relapse of the disease varies depending on the method of treatment, and the most significant differences were observed mainly in patients of the fourth group.

With CG in the fourth group of patients, the pain disappeared by 5.2± 2.2, and in the first - by 8.1± 2.1 days of treatment, in patients with CG in the fourth group of treatment - by 6.4±2.7, and in the first - by 10.4± 2.3 days, and in patients with DU, respectively, by 8.3±2.4 and 15.1 ±2.1 days of treatment.

Dyspeptic phenomena in patients of the first group of CG disappeared by 15.3±2.4, and in the fourth - by 10.1±2.2. In patients with CGD in the fourth group, dyspeptic phenomena disappeared by 13.2±2.1, and in the first group - by 19.5±2.2, and in patients with DU, respectively, by 14.9±2.4 and 21.1±2.6 days of treatment.

Pain during palpation of the epigastric and pyloroduodenal areas disappeared with CG in the first group by 15.4 ± 1.9, in the fourth - by

9.3± 1.7 day of treatment. With CGD, palpation pain disappeared in the fourth and first groups, respectively, by 12.6±1.6 and 19.2±1.7 days of treatment, in patients with DU, respectively, by 19.6±1.8 and 25.1±2.2 days of treatment.

It should be noted that if in children with CG who received conventional treatment, the average number of bed days was 22.3, with CGD - 23.4 and with DU - 28.16, then in patients who received additional medical glycerin and essentiale, it was equal, respectively - 19.37, 21.06 and 25.0 beds / days.

We have also obtained certain shifts in some indicators of carbohydrate and nitrogen metabolism, as well as gluconeogenic liver function in children who received additional corrective treatment with hepatotropic agents.

We found that in patients with CG before discharge from the hospital, the blood glucose level decreased to 2.83 mmol/ l and practically did not differ from the norm.

In patients with CGD after treatment, the glucose level decreased to 2.98, and in patients with DU - to 2.99 mmol/l. As it turned out, the decrease in blood glucose and its normalization is associated with the stabilization of gluconeogenic liver function, improved absorption of newly formed glucose by peripheral tissues and clinical improvement of the general condition of patients. For example, in patients with CG after complex treatment, the rate of glucose synthesis from glycerin was 0.69 mmol/l/h, which is 1.18 times lower than they had before treatment. In these patients, the absorption of newly formed glucose by peripheral tissues also improved by 1.9 times.

This means that in patients with CG, after the treatment, the gluconeogenic function of the liver is stabilized and the penetration of newly formed glucose into peripheral tissues improves.

As a result of complex treatment, the synthesis of glucose from glycerol in the liver decreased significantly (by 0.18 and 0.28 mmol/l/h) in patients with CGD and DU. As a result of treatment in patients with CGD and DU, the tolerance of peripheral tissues to newly formed glucose decreased significantly and began to correspond to 0.51 and

0.70 mmol/l. Consequently, as a result of complex treatment in the hospital, it was possible to reduce the synthesis of glucose from non-carbohydrate compounds, as well as the absorption of glucose by peripheral tissues in patients with CG and CGD, compared with patients with DU.

This means that the gluconeogenic function of the liver can be used as a control over the effectiveness of treatment, and based on its changes, it can be recommended to patients in the hospital an appropriate diet.

Along with this, it was found that as a result of the complex treatment, the general condition of patients improves, depressive manifestations weaken, sleep normalizes, patients become more active, their appetite improves, in some cases the size of the liver decreases.

In addition, some of the parameters of nitrogen metabolism studied by us have normalized in patients. So, before discharge, patients with CG had a decrease in the blood content of ammonia by 14.15%, urea - by 12.4%, glutamine by 1.2%. But still, in these patients on an empty stomach, compared with practically healthy children, only the level of glutamine in the blood was the same, while the content of ammonia and urea was slightly elevated. 3 hours after the glycerin load in patients with CG, the concentration of ammonia in the blood decreased by 1.53 times, urea - by 1.15 times, and these indicators began to fully correspond to the norm.

In patients with CGD and DU, after complex treatment, there is also a decrease in the concentration of ammonia in the blood (by 31.17 and 32.76%, respectively), urea (by 9.83 and 12.0%, respectively) and glutamine (3.17 and 4.63%). However, these indicators were still significantly increased compared to practically healthy children. Only after glycerin loading, the level of ammonia in patients with CGD decreased by 1.53, and in patients with DU - by 1.51 times and practically began to correspond to the norm. This was also facilitated by a decrease in the concentration of urea in patients with CGD by 1.10 times, in patients with DU - by 1.10 times and glutamine, respectively, by 1.03 and 1.05 times. Consequently, in patients with CGD and DU,

complex treatment helped to reduce the breakdown of tissue proteins, and in patients with CG - their complete stop. In addition, the urinary function of the liver improves in all examined patients, and detoxification of ammonia in the form of glutamine also improves in patients with CGD and DU. However, in quantitative terms, the content of ammonia in the blood of patients with DU remained higher than in patients with CGD, and in patients with CG, corresponded to the norm (131.3 ± 7.1 mmol/L). The concentration of this metabolite in the blood of patients with CGD and DU after treatment still exceeded the norm.

The data obtained indicate the need to develop therapeutic, preventive and rehabilitative measures in the conditions of a children's polyclinic aimed at reducing the level of ammonia in the blood - a highly toxic cytolytic agent. One of these approaches, as can be seen from our work, may consist in the introduction of glycerin. In fact, the glycerin load on the background of treatment reduced, on average, the ammonia content in the blood of patients with CG by 45.85, in patients with CGD - by 49.7, and in patients with DU - by 52.4 mmol/L. Moreover, in patients of the second and third groups, as in the first, it also began to correspond to the norm.

At the same time, after the complex treatment in patients with CG, CGD and DU, the hypoureoemic effect of glycerin is not accompanied by such a deep drop in urea as before treatment. Based on this, we believe that as a result of the treatment, the body of patients with chronic GDP began to respond adequately to exogenous stimuli that promote detoxification of ammonia. Regardless of the duration of the disease, the comprehensive treatment of patients with CGDP pathology helps to reduce the ammonia content in their blood and improve the urinary function of the liver.

In conclusion, it should be emphasized that as a result of the complex treatment in patients with CG, the gluconeogenic function of the liver is completely stabilized, the tolerance of peripheral tissues to glucose of gluconeogenic genesis decreases, the content of ammonia, urea and glutamine in their blood is normalized. It should be especially noted here that the above-mentioned changes in metabolism can often be manifested only after a glycerin load.

Complex treatment of patients with CGD and DU also leads to an improvement in a number of parameters of carbohydrate and nitrogen metabolism in them. At the same time, after the treatment, they still had a high intensity of glucose neoplasm in the liver and a relative insufficiency of the ornithine cycle of urea formation and the glutamic acid-glutamine system, more pronounced in patients with DU.

Finally, it is very important to keep in mind that although chronic GDP is a disease with a predominant lesion of the stomach and intestines, however, internal organs are very often involved in the pathological process, various types of metabolism and functions, including the liver, are disrupted. All this significantly aggravates the severity of the course of the disease, requires a special clinical and laboratory examination of patients, including with the help of various stress tests, adding additional corrective measures to their treatment regimen.

CONCLUSIONS

1. In practically healthy children aged 11-13 years, the state of gluconeogenic liver function has been studied, which can be taken as a normative indicator of carbohydrate metabolism.

2. Gastroduodenal pathology in children is accompanied by a violation of the functional state of the liver. In all examined patients, there is an increase in gluconeogenic liver function, moreover, deeper violations occur with chronic gastroduodenitis and duodenal ulcer.

3. In patients with chronic gastroduodenal pathology, a direct relationship was found between the intensity of gluconeogenic liver function, indicators of nitrogen metabolism and the duration of the disease. In patients with an increase in the duration of the disease, the rate of gluconeogenesis increases, the tolerance of peripheral tissues to newly formed glucose increases.

4. In patients with chronic gastritis, gastroduodenitis and duodenal ulcer, glycerin loading has a pronounced hypoammony and hypoureoemic effect, the degree of manifestation of which depends on the duration of the disease.

5. As a result of complex treatment with the use of essentiale and medical glycerin in patients with chronic gastroduodenal pathology, the gluconeogenic function of the liver improves, the threshold of sensitivity of peripheral tissues to glucose of gluconeogenic genesis decreases, the content of ammonia decreases and the urinary function of the liver increases.

6. The use of essentiale and medical glycerin in combination with conventional treatment has a beneficial effect on the clinical course of the disease, contributes to the reduction of the number of bed days during inpatient treatment and the achievement of an economic effect.

PRACTICAL RECOMMENDATIONS

1. For sick children with gastroduodenal pathology, to assess the functional state of the liver, it is recommended to include a study of the gluconeogenic function of the liver and indicators of nitrogen metabolism in the blood using glycerin loading, which allows to clarify the diagnosis and monitor the effectiveness of treatment.

2. The increase in gluconeogenic liver function, increased tolerance of peripheral tissues to glucose of gluconeogenic genesis, increased ammonia content, increased intensity of urea formation differ significantly in patients with chronic gastritis, gastroduodenitis and duodenal ulcer, thereby serve as additional criteria for the differential diagnosis of these diseases.

3. In order to correct impaired carbohydrate and nitrogen metabolism, as well as improve the functional state of the liver, it is advisable to include hepatoprotectors in the complex therapy of patients with gastroduodenal pathology: medical glycerin (at the rate of 0.5 mg / kg, every 2 days, dissolved in 200.0 ml of warm water) and essentiale, for the entire period of treatment.

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