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# PATHOGENETIC INTERACTION OF THYROID DISEASES AND GASTROINTESTINAL PATHOLOGY IN CHILDREN

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**Abstract:**

The article summarizes current data on dysfunctional disorders of the gastrointestinal tract, liver and biliary tract in children with thyroid diseases. The course of diseases of the digestive system depends on the functional characteristics of the thyroid gland.

**Keywords:** Gastrointestinal Tract, Liver, Thyroid Gland, Children.

The problem of thyroid pathology in young children remains one of the little-studied. Its relevance is due to the fact that thyroid diseases currently take the first place among all endocrine pathology in children [1]. This is of concern not only to endocrinologists, but also to pediatricians and neurologists, which is explained by the significance of the influence of thyroid hormones on the development and growth of a child, especially on the processes of neurogenesis that determine the most important cerebral functions.

Currently, it is impossible to imagine the diagnosis of thyroid pathology without ultrasound examination (ultrasound), which allows to assess its location, structure, volume. The question of the final interpretation of thyroid volume standards, especially in young children, is intensively studied, but not solved [2, 4, 5].

After the birth of a child, the importance of thyroid hormones (TG) in the development of his brain and the formation of cognitive functions does not decrease; on the contrary, thyroid hormones play a very important role in the process of differentiation of neurons, growth of axons and dendrites, in the formation of synapses, gliogenesis, maturation of the hippocampus and cerebellum (throughout the first year of life), as well as stimulate myelinogenesis and myelination of neurocyte processes (during three years of postnatal stage of development). As a result of hypothyroxinemia, dysontogenesis of higher mental functions is observed in the developing brain. TG deficiency often occurs at the end of pregnancy and in the first week of postnatal life, which dramatically worsens the intellectual and motor functions of a person, the brain stops developing, undergoes degenerative changes. Transient hypothyroidism of newborns is reversible and thyroid function is subsequently restored, but intellectual development disorders remain for life [1, 3-5]. Unfortunately, information about the consequences of congenital transient hypothyroidism (VTG) is rather fragmentary and ambiguous [2, 4-7]. Many authors have found an increase in the content of atherogenic lipoproteins in the blood and a decrease in the content of anti-atherogenic lipoproteins. Thyroxine and triiodothyronine deficiency leads to a change in cholesterol metabolism in the liver: the synthesis of low and very low density lipoproteins (LDL and VLDL) increases. Thyroid hormones ensure the effective use of lipid substrates by hepatocytes, increase the synthesis and mobilization of triglycerides (TG) of adipose tissue, increase the concentration of non-esterified fatty acids in the blood and increase the activity of hepatic lipase [4,7].

Different variants of thyroid dysfunction, due to the presence of gastrointestinal effects in thyroid hormones, often accompany disorders of motility and secretion of the digestive organs, which gives reason to talk about the important role of thyroid in the development of gastrointestinal diseases [13]. Thyroid dysfunction is associated with gastrointestinal disorders, has a significant effect on proliferative processes, motor and secretory activity of the gastrointestinal tract [7,9]. Thyroid hormones are necessary for the normal functioning of all organs and systems. The presence of thyroid pathology affects the condition of the mucous membrane of the gastroduodenal zone in two ways: directly, affecting metabolic and regenerative processes, and indirectly, interacting with GIG and modulating their effects. Thyroid hormones have a powerful catabolic effect on all organs and systems. Excessive secretion of thyroid hormones causes dissociation of oxidative phosphorylation processes in tissues, an increase in the activity of oxidative processes is accompanied by a decrease in energy accumulation in macroergic phosphorus compounds. The body is dominated by the processes of dissimilation — increased breakdown of proteins, increased utilization of fat from fat depots, reduced glycogen levels in muscles, liver, disturbed potassium balance, vitamin metabolism, increased permeability of the vascular wall, which complicates the exchange and leads to tissue hypoxia. The result of metabolic shifts is a decrease in body weight, the

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development of dystrophic changes in organs [10]. At the same time, hypermotor activity of the entire digestive tract is noted, and in particular of the stomach, the emptying of which occurs faster than normal at the initial stages of thyroid hyperfunction. The secretion of gastrin, the acidity of gastric juice increases, the enzymatic activity of the mucous membrane increases. With an increase in the severity and duration of the disease, there is a marked decrease in the motor evacuation function of the stomach and the above indicators. With a decrease in the function of the thyroid gland, all types of metabolism are suppressed: oxygen utilization by tissues, oxidative reactions are inhibited and the activity of various enzyme systems decreases, gas exchange, basic metabolism, thermoregulation is disrupted. Protein metabolism changes: protein breakdown and synthesis slows down, glycosaminoglycan metabolism is disrupted, mucin glycoprotein, hyaluronic and chondroitinseric acids accumulate in tissues. Their excess changes the structure of connective tissue, enhances its hydrophilicity and binds sodium, which forms a myxedema in conditions of impaired lymph outflow. Lipid metabolism is disrupted — the content of cholesterol, triglycerides, betalipoproteins increases [9].

The electrogastrogram shows an increase in the amplitude of oscillations when the rhythm of gastric peristalsis slows down. The secretory and acid-forming function of the stomach changes, the volume of gastric juice decreases, the acid-enzyme-forming functions of the stomach, the level of gastromucoproteins increases. A significant role in the development of destructive inflammatory processes in the gastrointestinal mucosa belongs to hormonal factors that cause disorders of the motor and secretory functions of the stomach and duodenum and disruption of metabolic processes in the mucous membrane. Thyroid dysfunction is associated with gastrointestinal disorders. Gastritis, peptic ulcer, pancreatitis, hepatitis, functional biliary disorders, cholelithiasis, constipation, diarrhea, dysbiosis — this is not a complete list of conditions related to thyroid pathology [11].

The hepatobiliary system plays an important role in maintaining the endocrine status. It has been proven that the liver can act as a regulator of the level of free thyroxine in the blood, changing the rate of excretion and reabsorption of hormones [14]. The study of the hepatobiliary system in various thyroid pathologies showed the dependence of bile formation and bile secretion on the functional activity of the thyroid gland. Thyroid hormones regulate the biliary function of the liver. The molecular mechanisms of action of thyroid hormones on hepatocytes are known. They penetrate through the cytoplasmic membrane of the hepatocyte, bind to specific protein receptors localized in the nucleus, and selectively activate the cellular genome. The liver performs specific functions related to the transport and metabolism of thyroid hormones. The realization of the effects of thyroid hormones depends on the liver function, as well as on the peripheral violation of their action.

Peripheral tissue deficiency of thyroid hormones plays a leading role in the discrepancy between the clinical picture of the disease and laboratory tests. A decrease in thyroid function can directly affect the structure and function of the liver. In some cases, with hypothyroidism, cholestatic jaundice is noted, due to a decrease in the excretion of bilirubin and bile. The decrease in bile outflow can be partially explained by an increase in the ratio of cholesterol/phospholipids in membranes and a decrease in their fluidity, which can affect tubular membrane transporters and enzymes [10]. It was found that the liver is involved in the pathological process in 70-80% of patients with increased thyroid function [14].

With hyperthyroidism, the thyroid gland secretes an excessive amount of thyroxine, the main consumer of which is the liver. With an excess of thyroxine intake in the liver cells, there is a continuous process of increased production of bile and cholesterol. At the same time, there are two types of liver pathology: hepatitis and cholestatic [15].

It is known that the intestine not only absorbs iodine contained in food, but also reabsorbs iodine, which is released in the liver as a result of the deioding of thyroid hormones and released with bile into the duodenum. As a result, 80-90% of the iodine contained in the body is reutilized and used in the repeated biosynthesis of thyroid hormones. Therefore, it is quite understandable that a disorder of the hepatobiliary system can affect the metabolism of these hormones and lead to a weakening of iodine reutilization and the development of relative iodine deficiency. And this is especially important also because the increase in thyroid pathology is associated with a deficiency of iodine — the most important component of the molecule of the main thyroid hormone — thyroxine [16].

## CONCLUSION.

The pathogenetic mechanism of hepatitis disorder appears to be relative perivenullary hypoxia caused by an increase in the liver's oxygen demand

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without an increase in hepatic blood flow. Progressive liver damage is possible in conditions of severe hypoxia. With a cholestatic disorder, there is an increase in the level of alkaline phosphatase, the level of gammaglutamyltranspeptidase and bilirubin, indicating cholestasis. The liver regulates the amount of thyroid hormones and with the help of enterohepatic circulation. The liver plays a central role in the deioding of thyroid hormones with the formation of their more active or inactivated forms. Deficiency of thyroid hormones inhibits higher nervous activity, weakens conditional and unconditional reflex activity. It also leads to a decrease in the motor activity of the gastrointestinal tract, which is clinically manifested by constipation, flatulence.

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