

MODERN VIEWS ON THE PHYSIOLOGY OF DENTAL HARD TISSUES AND THEIR FLUORINATION

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Annotation:

Hypersensitivity of the teeth is a common disease. The disease causes discomfort in eating and causes pain. This article gives you a brief overview on tooth sensitivity.

Keywords: hyperesthesia, fluorination, enamel prizm, metobolism, odontoblasts.

The effectiveness of the methods of treatment and prevention of hyperesthesia of hard tissues of teeth, caries is largely based on knowledge of the mechanisms of metabolism in hard tissues. Research in the field of morphology and physiology of the tooth allowed M. Brannstrom to formulate the hydrodynamic theory of dentin sensitivity, which is now widely used and recognized in dental science and practice [2].

In enamel and dentin, two types of liquid are distinguished: water of crystallization, which forms a hydratin shell of crystals, and water that can move freely [4].

Mineral and organic substances enter the enamel and dentin of an erupting tooth from saliva and pulp. Enamel and dentin are impregnated with a liquid - dental liquor [8]. It has been proven that cerebrospineal fluid moves centrifugally from the pulp, slightly changing its chemical composition when moving from dentin to enamel. The physiological role of the liquor is ensured by both the constituent components and the possibility of its movement. The dentine fluid contains total nitrogen - 64.3 mg%, residual nitrogen - 49.4 mg%, protein - 124 mg%, which is qualitatively close to the protein composition of blood plasma and interstitial fluid. The dentine fluid has a high percentage of alkaline phosphatase, and the concentration of sodium and potassium is 700 and 800 mg%, that is, higher than in blood plasma. The sugar content in dentinal fluid is 45 mg% [11]. In the dentinal cerebrospinal fluid, 92 mg/l of calcium, 42 mg/l of phosphorus, 27.7 mg/l of chlorides were found. Dentinal fluid is alkaline and contains enzymes, antibodies, proteins, carbohydrates, hormones, enzymes, fats and salts [5].

When centrifuging the crowns of the teeth, a light yellow, extremely adhesive liquid was obtained in the amount of 0.00424 m in 24–48 hours, which easily coagulates



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when exposed to air, organic solvents or heat. The liquid is homogeneous, has no cells and organelles. It has been established that the dentinal fluid is not a derivative of the cytoplasm, but is formed extracellularly, that is, the plasma, passing through the fenestra of the vessels, turns into dentinal fluid at the odontoblast level, without passing through the processes of odontoblasts [3].

Free water occupies up to 20% of the dentin volume and is able to move in the dentinal tubules at a speed of 4 mm/h. The centrifugal flow of dentinal cerebrospinal fluid is provided by intrapulpal pressure [11]. Measured by direct and indirect methods, the pressure inside the pulp is about 24 mm Hg. and directed outward.

Theoretically, it is calculated that the dentinal tubules are emptied 10 times during the day. In addition to pulpal tissue pressure on dentinal cerebrospinal capillary forces develop in the dentinal tubules. They are so significant that they are theoretically capable of raising a liquid column by 7 m. It is possible that they explain the pain sensitivity of exposed dentin [7].

According to I.K. Lutskoy (1998), the reaction of tooth enamel to certain external stimuli is not always clear enough. The ability to explain the sensitivity of enamel, by analogy with dentin, by hydrodynamic mechanisms requires the formulation of initial provisions. This means that a more detailed study of the ways in which information is transmitted from the enamel surface to the pulp and vice versa is required.

Free spaces in enamel are represented by various kinds of micropores [I]. On the surface of the enamel of impacted teeth, they are found in the form of cup-shaped depressions. In some cases, large pores corresponding to the diameter of the enamel prism are determined [4].

The passive transport of K+, Na+, Ca2+, C1-, F- ions through the tooth enamel was studied, taking into account its porous structure. Using the method of low-temperature nitrogen adsorption, data on pore radii were obtained. Their size allows large molecules and ions to penetrate the enamel. It has been proven that due to the porous structure of enamel, a connection is established between the internal environment of the tooth and the electrolyte solutions surrounding it [10]. Chlorine-containing electrolytes form electric potentials of positive polarity, and fluorine-containing ones - negative. Some of the fluoride ions are adsorbed by the tooth wall, forming fluorite (CaF2) and fluorapatite, and some of the ions penetrate the entire depth of the enamel, displacing hydroxyl ions and hydrophosphate anions (HPO42') - this results in alkalization of the internal solution NaCl (pH changed from 5.41 to 7.41). In addition, phosphate anions appear in the internal solution. The share of micropores (r < 1.5 nm) in enamel is 5.44*10'4 cm3/g. (Less than 10% of the volume of all pores). Therefore, tooth enamel should be referred to as mesoporous objects





(1.5 nm < r < 50.0 nm). The main volume of micropores falls within the range of their radii 3 - 15.0 nm. Pore sizes allow large molecules to penetrate the tooth wall, but the transport speed is low [6].

An increase in the number of micropores in the enamel contributes to hypersensitivity, which is typical for initial caries, acid necrosis, when the enamel porosity can increase up to 25% [4]. This happens because a larger volume of fluid is involved in the process of movement, a significant increase in the speed of movement, a more significant mechanical irritation transmitted to cells - odontoblasts and nerve endings.

In the clinic, modern methods of treating increased sensitivity of enamel and dentin are based on stopping the hydrodynamic mechanism, namely: reducing the activity of the response of dental cerebrospinal fluid to external stimuli in one way or another. In particular, varnishes are used to seal microspaces (micropores in enamel, tubules in dentin), or preparations that reduce the volume of microspaces by increasing the mineralization of enamel.

A less effective way is to grind the neck of the tooth. E.G. Absi, M. Addy and D. Adams (1995) in an in vitro study determined that grinding of the dentine surface on teeth with severe hyperesthesia using grinding heads can lead to blockage of part of the dentinal tubules. However, the combination of this procedure with the treatment of teeth with water or acid-containing solutions significantly reduces the effect obtained [1].

The results showed that Gluma Desensitaizer was able to bind a large amount of protein. The study using electron microscopy revealed the formation of partitions in the dentinal tubules, which block the movement of dentinal fluid. In experimental studies, the effectiveness of the Optibond Solo dentin adhesive (Kerr, USA) and the Seal&Protect desensitizer (Dentsply) was compared in relation to the hypersensitive surface of erosion of hard tooth tissues. Using scanning electron microscopy, it was found that the Optibond Solo film was gradually covered with cracks throughout the study, passing through the entire thickness of the polymer coating. When using Seal & Protect, no cracking of the coating was observed, but peeling of the coating was observed in the peripheral areas. This study showed that the Seal & Protect desensitizer was more resistant to abrasion than Optibond Solo [9].

Thus, the analysis of the literature shows that the method of deep fluoridation against the background of known methods is a fairly effective way to prevent and treat caries, to combat hyperesthesia of hard dental tissues. At the same time, many questions related to the mechanism of action of deep fluoridation, its effectiveness, rational methods of use, indications and contraindications remain open. According to our





data obtained as a result of a survey of dentists in Moscow, only about 11% of pediatric dentists and 0.5% of adult dentists use deep fluoridation in their practice today. That is why we tried in our work to determine the place of deep fluoridation in modern dental theory and practice.

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