

Pathogenesis and Anatomical Features of Children With Congenital Cleft Lip and Palate.

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Annotation: For the treatment of children with congenital cleft lip and palate (CCLP), it is necessary to periodically study the dynamics of physical development. The development of the craniofacial region in children with CCLP after surgical manipulations is a widely discussed topic. The study of the growth of the facial skeleton and, in general, the entire physique of a child can be a theoretical and methodological basis for the development and improvement of morphometric methods of diagnostics and reconstruction in medicine and predict the appearance of secondary defects in the craniofacial region.

Key words: craniofacial region, congenital cleft lip and palate.

Topicality.

Congenital cleft lip and palate (VRGN) can be unilateral, bilateral, complete, partial: the defect captures the upper lip, hard and soft palate; and the cleft palate can also be complete (i.e. the defect captures the hard and soft palate) and partial, i.e. within the soft palate (clinical and anatomical classification of congenital cleft lip and palate, Ad.A. Mammadov, 1998)

With a **unilateral complete** cleft of the upper lip and palate, the defect divides the alveolar process into two fragments - a large and a small one. Typical of a unilateral cleft palate, when the nasal septum is attached to one of the maxillary horizontal palatine plates and there is a connection between the coulter (vomer) and the horizontal non-bub plate of one of the sides.

With a bilateral cleft, the latter divides the lip and palate into three fragments: one central (intermaxillary bone), and two lateral - left, right.

The intermaxillary bone can be pushed forward or backward from the normal dentofacial arch in different stages. Lateral fragments may, depending on the position of the intermaxillary bone, be shifted forward, downward, to the sides, to the center. This is also due to impaired embryonic development and the absence of circular pressure of the upper lip on the alveolar process, the anterior part of the dentition.

Research methodology:

Modern scientific literature was used as a material.

Results of the study and their discussion:

"A healthy mother is a healthy child", the State programme "Year of a Healthy Child", large-scale work is being carried out in the republic aimed at further strengthening the reproductive health of the population, strengthening the material and technical base and personnel potential of medical institutions providing medical assistance in the field of protecting motherhood and childhood, introducing modern methods of diagnosis, treatment and prevention of diseases of women, children and adolescents.

With a cleft of only the palate, the hard and soft palate is involved in the pathological process of development. However, there may be options where only the soft palate can be involved in isolation (within the uvula, up to the level of the boundaries of the hard and soft palate).

The cleft of the hard palate can be within its 1/2, and up to the incisor opening (complete). Cleft palate embryologically in its development is not associated with congenital cleft of the upper lip. Often, cleft palate is present in Pierre-Robin syndrome, which includes micrognathia, macroglossia and cleft palate. This is accompanied by impaired respiratory function, swallowing and, accordingly, nutrition.

The latent form of cleft is one of the forms of palate pathology in which the main cause of speech impairment can be anatomical and functional disorders associated with the defeat of the speech-producing structures of the articulatory apparatus.

In some cases, foreign authors refer to the hidden cleft as a submucosal species and call it submucous cleft palate (Fara M. and Weatherley W., 1980; Stark R., 1980) a cleft palate. Here in Russia it is customary to call it a hidden form of cleft palate. In our opinion, both are correct, and we also refer to it as an innate crevice of the sky of a hidden form.

In cases of diagnosing a hidden cleft palate, we note that the muscles of the soft palate are split, the nasal and oral mucous membranes are inseparable, the area of the soft palate looks whitish or partially translucent. A bone defect in the hard palate along the midline can have a variety of shapes and lengths: triangular-sharp at the apex or horseshoe-shaped, sometimes reaching the incisor. With the preserved pharyngeal reflex, the latent cleft palate tends to "visible" increase the defect in the hard palate. The bulge of the muscles of the soft palate, in its central part, is smoothed when closing. A light source placed in the nasopharynx, behind the soft palate, shows a true defect (the study is diaphanoscopy). Palpation confirms the actual size of the bone defect of the posterior edge of the horizontal palatine plate.

It should be pointed out that indentation on the uvulae (uvulae) is not a reliable sign of the latent form of cleft, since the prevalence of microsignals of VRGN in the healthy population is more common than 1% (Fara M. and Weatherley - W. 1980; Stark R., 1980). However, in a patient with a hidden cleft of the soft and hard palate, the uvula is rarely not split and, this phenomenon suggests to the researcher the presence of a congenital hidden cleft palate.

One of the microsignia of various pathological conditions of the maxillofacial region are anatomical disorders in the tissues of the ChLO.

It seems to us that knowledge of the pathogenesis of the latent form of cleft palate and its timely diagnosis at the level of a micropediatrician, pediatrician, ENT doctor, psychoneurologist, starting from the maternity hospital, will improve the quality of care for this category of patients, especially at an early age. However, we have to deal with the fact that pediatricians "miss" such a pathology and only at an older age, when a speech disorder is clearly determined in the form of pronunciation "like cotton wool in the mouth", patients have to turn to a speech therapist, and then to a maxillofacial surgeon.

When considering the embryonic aspect of the formation of congenital cleft lip and palate (Veau V., 1938; Smutz M.K., 1981; Enlow D.H., 1990; McCarthy J.G., 1990; Smith L.F. et al., 1991) it should be noted that the primary palate is formed approximately by 4-6 weeks of intrauterine development and contributes to the initial separation between the oral and nasal cavities.

The primary palate is the triangular-shaped space of the front of the hard palate extending from the incisor to the lateral incisors of the upper jaw. It includes the area of the alveolar process, in the key four upper incisors.

The two middle nasal and maxillary rollers join to form the upper lip (Smith L. F., Calhoun K.N., 1991). The nasal rollers merge at deeper levels and form the maxillary arch, which becomes the primary palate, so that in the embryonic period the cleft of the upper lip almost invariably accompanies the cleft of the primary palate (Smutz M.K., 1981; Enlow D.H., 1990; McCarthy J.G., 1990; Smith L.F., Calhoun K.H., 1991).

By about 9 weeks of embryogenesis of the fetus, after the development of the primary palate ends, **the secondary palate** begins to develop (Smutz M.K., 1981). It is formed by the palatine rollers of the upper jaw, which usually connect together with the nasal septum, since the tongue is pushed down during its development. Thus, the pathogenesis of the secondary palate is associated with the insufficiency of this fusion. The secondary palate consists of a hard and soft palate, forming the upper wall of the oral cavity and the base of the nasal cavity.

Violation of the growth of the upper jaw prevents the formation of contact of the palatine plates. This can sometimes lead to the emergence of "Pierre-Robin" syndrome, including: micrognathia, cleft palate, macroglossia (Gotlin R.J., Pindborg J.J., Cohen M.M., 1976).

Violation of functional ability occurs in the muscles of the soft palate (NZ), BSG, ZSH. In connection with this violation, children develop compensatory functional changes aimed at compensating for the insufficiency of the muscular activity of these structures and manifested in the location of the root of

the tongue closer to the oropharynx. This ultimately leads to a violation of the contact of the tip of the tongue with the alveolar process, causing changes in sound pronunciation. Hypernasal speech is observed, children cannot form fricative sounds (Sykes J. and Senders G., 1995).

In the overwhelming number of patients with ARGN, an eccentric, sphincter mechanism of ntK closure is noted (Ad.A. Mammadov, 1986). According to Kaplan E. N., (1975), the sphincter mechanism of closure is nothing more than a compensatory mechanism of activity of NTK, and it appears as a result of the insufficiently effective function m. levator veli palatini (MLVP) - muscles that raise the soft palate.

Патологическое прикрепление м. levator veli palatini (MLVP) - мышцы, поднимающие мягкое небо при сквозной и скрытой (submucosae) расщелинах неба были детально описаны Veau V. (1938), Ruding R. с соавт. (1964), Braithwaite F. и Maurice D. (1968), Kriens O. (1969), Hoopes J. с соавт. (1969), Millard D. с соавт. (1970), Fara M. и Dvorak J. (1970), Edgerton M. и Dellon A. (1971).

The muscles that raise the soft palate m. levator veli palatini (MLVP) are normally attached to the Eustachian tube and the mouth of the carotid canal, but in pathology they are fixed to the posterior edge of the hard palate and in the tendon structures of the tensile muscle m. tensor veli palatini (MTVP) - instead of attaching in the midline area of the soft palate.

Патологическое прикрепление мышц ML VP при сквозной расщелине неба и при скрытой (подслизистой) (submucosae) были детально описаны Veau V., 1938; Ruding R. с соавт., 1964; Braithwaite F. и Maurice D., 1968; Kriens O., 1969; Hoopes J. с соавт., 1969; Millard D. с соавт., 1970; Fara M. и Dvorak J., 1970; Edgerton M. и Dellon A., 1971.

Palato-pharyngeal insufficiency with latent cleft palate is associated with pathological anatomy (congenital underdevelopment of the muscle structures of NTK), location (pathological attachment of muscles) and impaired muscle function (sometimes associated with impaired conductivity of V, VII and IX, X, XII pairs of cranial nerves. In this regard, the most important information about the mechanism of closure and the state of the muscle structures of NTK can be given by transnasal endoscopy, electrodiagnostics and X-ray diagnostics, electromyography, other types of objective examination in the system of complex diagnostics and treatment.

Taking into account the above data of the anatomical and functional features of the congenital cleft palate, its types and essence, I would like to dwell on the historical reference of the dynamically developing surgery of this defect.

Conclusion.

Thus, the morphogenesis of the craniofacial region is a complex process associated with a strictly regulated interaction of mesenchymal and epithelial cells, which requires further research.

Analysis of the studied scientific and medical literature showed that in published sources the morphometric characteristics of the craniofacial region of children of the I and II period of childhood with congenital cleft lip and palate are not presented in sufficient volume. With the help of this article, a number of problems can be solved: to determine the morphometric parameters of the craniofacial region in children I and II the period of childhood in healthy children, taking into account gender (boys and girls), to assess the compliance of these parameters with the "principle of the golden ratio"; to identify the features of changes in the parameters of the dentition system during the period of tooth change in children with congenital cleft lip and palate in a comparative aspect; allow in the early periods to determine the secondary defect and the state of the bite in children with congenital cleft lip and palate, depending on age, as well as to determine the optimal timing of surgical treatment.

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