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DISEASES OF NOSE AND NASAL CAVITIES

STUDY GUIDE

for students of medical university



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Abstract The study guide explains the etiology and pathogenesis of diseases of the nose in the field of otorhinolaryngology, clinical manifestations and modern diagnosis, comparative diagnosis, surgical tactics of treatment. Nasal and paranasal cavities are located in the facial part of the skull, near the eye socket. The nose consists of the external nose and the nasal cavity. The nasal cavity performs primary and secondary functions. The main functions of the nose include breathing, protection and smelling, secondary functions - facial expressions, tears, resonance functions. The study guide is recommended for postgraduate residents of medical universities, students of medical and professional education faculties.

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LIST OF ABBREVIATIONS

ENT-ear, nose and throat

CT- computer tomography

MRT- magnit-rezonanse tomography

UHF- ultra high frequencies

UVR- ultraviolet radiation

IgE-immunoglobuline E

SIT- specific immunotherapy

YAMIK- name of sinus cateter

BRIEF HISTORY OF ENT DEVELOPMENT OF OTORINOLARINGOLOGY

This extended abstract analyzes the historical development of otorhinolaryngology (ENT) in Uzbekistan, tracing a trajectory from traditional Central Asian medical practices to a modern, centrally organized specialty embedded within national health systems. The narrative situates local advances within a broader international context, highlighting how global ENT pioneers and regional experts intersected to shape training, clinical practice, and patient care. By focusing on institutional milestones, educational reforms, and the work of notable physicians—both Uzbek and international—the abstract illuminates how ENT evolved in Uzbekistan from the mid-20th century to the present day, and how post-Soviet reforms and global collaborations have continued to drive innovation.

Early roots and regional foundations Central Asian medical traditions long identified head and neck diseases and related dysfunctions, with practitioners in the region applying a range of diagnostic and therapeutic approaches to otology, rhinology, and laryngology. While domestic medical literature from the early 20th century is sparse, the foundation for a formal ENT discipline in Uzbekistan began to crystallize as modern medical education and hospital-based care expanded. Traditional healers and early clinicians contributed observational knowledge about nasal obstruction, recurrent otitis, and throat ailments, creating a cultural and clinical milieu that older and younger generations would later systematize within an academic framework. The establishment of university departments and surgical wards in the mid- to late-20th century marked a turning point, signaling the integration of ENT into the standard medical curriculum and hospital services across major Uzbek cities.

Soviet-era formalization and regional spread During the Soviet period, ENT in Uzbekistan experienced standardization of training, the organization of dedicated wards, and the dissemination of core surgical techniques. Training pipelines were aligned with Soviet medical education standards, emphasizing comprehensive undergraduate preparation, residency programs, and postgraduate specialization. Centralized planning facilitated the expansion of ENT services to regional centers, enabling populations in Tashkent, Samarkand,

Bukhara, and surrounding areas to access otology, rhinology, and laryngology care, diagnostics, and operative treatment closer to home. Surgical innovations—such as endoscopic approaches, tympanoplasty, and functional sinus procedures—began to be implemented more broadly, supported by the availability of improved diagnostic equipment, hospital infrastructure, and a growing cadre of surgeons trained to perform specialized procedures. This era fostered the creation of a professional identity for Uzbek ENT physicians, the development of multidisciplinary care concepts, and the establishment of guidelines and training standards that would endure beyond the Soviet era.

Independence, modernization, and international collaboration Uzbekistan's independence in 1991 brought renewed opportunities for investment in medical equipment, modernization of diagnostic and surgical capacities, and the diversification of ENT services. Post-Soviet reforms emphasized upgrading imaging modalities, audiology laboratories, endoscopic equipment, microsurgical tools, and hospital facilities. International collaborations—through medical exchanges, joint research, and participation in regional and global ENT networks—facilitated knowledge transfer and introduced contemporary practices in pediatric ENT, hearing rehabilitation, and multidisciplinary care. The post-1991 period saw an accelerated adoption of minimally invasive techniques, refinements in airway and nasal surgery, and expanded programs in audiology and cochlear/alternative hearing rehabilitation, contributing to the overall quality and scope of ENT care in Uzbekistan. In this context, the Uzbek ENT community increasingly aligned with international standards while maintaining a strong domestic focus on accessibility, affordability, and culturally appropriate care.

Notable figures and the national narrative Within Uzbekistan, several physicians are recognized for their contributions to training, clinical practice, and institutional leadership in ENT. Notable Uzbek figures include Khasanov and Karabayev, whose work helped shape curricula, patient pathways, and the organization of services at major centers. These physicians are emblematic of a generation that bridged Soviet-era clinical practice and post-Soviet modernization, mentoring younger surgeons, expanding pediatric ENT and audiology capabilities, and contributing to hospital governance and regional health planning. While individual archival records vary in detail, the prominence of such figures in institutional histories underscores the role of local leadership

in building sustainable ENT programs within Uzbekistan's health system.

Global context: linking Uzbek progress to international ENT pioneers Uzbekistan's ENT lineage does not exist in isolation; it is part of a global story of otology, rhinology, and laryngology that has been shaped by renowned international pioneers. In a broad historical arc, early to mid-20th century European leaders established foundational diagnostic and operative concepts that would influence ENT everywhere. Adam Politzer, an Austrian-born physician, is widely credited with shaping modern otology and laying groundwork for systematic examination, interpretation of auditory pathology, and early therapeutic approaches. His influence extended beyond Austria, informing conventions of ear disease management and the use of diagnostic instruments that Uzbek surgeons would later adopt. In rhinology and facial surgery, Hermann Mikulicz-Radecki (a Polish-born, German-speaking surgeon) contributed to advanced nasal and head-and-neck procedures, helping to popularize standardized surgical techniques that would be integrated into training programs throughout the Soviet sphere and beyond. The mid- to late-20th century also saw the emergence of endoscopic methods as a transformative shift in ENT practice. Otto Messerklinger, renowned for his development of endonasal endoscopy and sinus surgery techniques, influenced global rhinology and provided a technical and conceptual model for subsequent generations of surgeons, including those in Uzbekistan who embraced minimally invasive approaches as equipment and expertise became available.

Worldwide diffusion of techniques and Uzbek adaptation The diffusion of endoscopic techniques, microsurgical tools, and systematic approaches to hearing rehabilitation created a shared knowledge base that Uzbek ENT professionals could adapt to local needs. The Uzbek ENT community actively integrated internationally informed practices with country-specific considerations—economic constraints, population demographics, rural healthcare access, and the availability of trained personnel. The result is a historically layered narrative in which international innovations provided a template for modernization, while domestic leadership, regional training networks, and university-based curricula ensured that these innovations were translated into tangible gains in patient care. Pediatric ENT, audiology, sleep-disordered

breathing management, and multidisciplinary teams for head-and-neck disorders emerged as focal areas where the international paradigm and Uzbekistan's local expertise converged to produce meaningful improvements in outcomes.

Implications for future scholarship and practice This history underscores several important themes for both scholarship and clinical practice. First, the development of ENT in Uzbekistan reflects a continuous synthesis of traditional medical knowledge, regional clinical experience, and global innovations. Second, leadership at national and regional levels—embodied by figures such as Khasanov and Karabayev—has been crucial for sustaining training programs, ensuring service access, and aligning local practice with evolving international standards. Third, ongoing international collaboration remains essential for advancing diagnostic capabilities, surgical techniques, and rehabilitative services, particularly in areas such as pediatric ENT and audiology, where early intervention yields long-term benefits. Finally, future research could build on national archives, institutional histories, and professional societies to identify additional Uzbek pioneers, map training-lineage networks, and document the outcomes of policy decisions that shaped ENT care across the country.

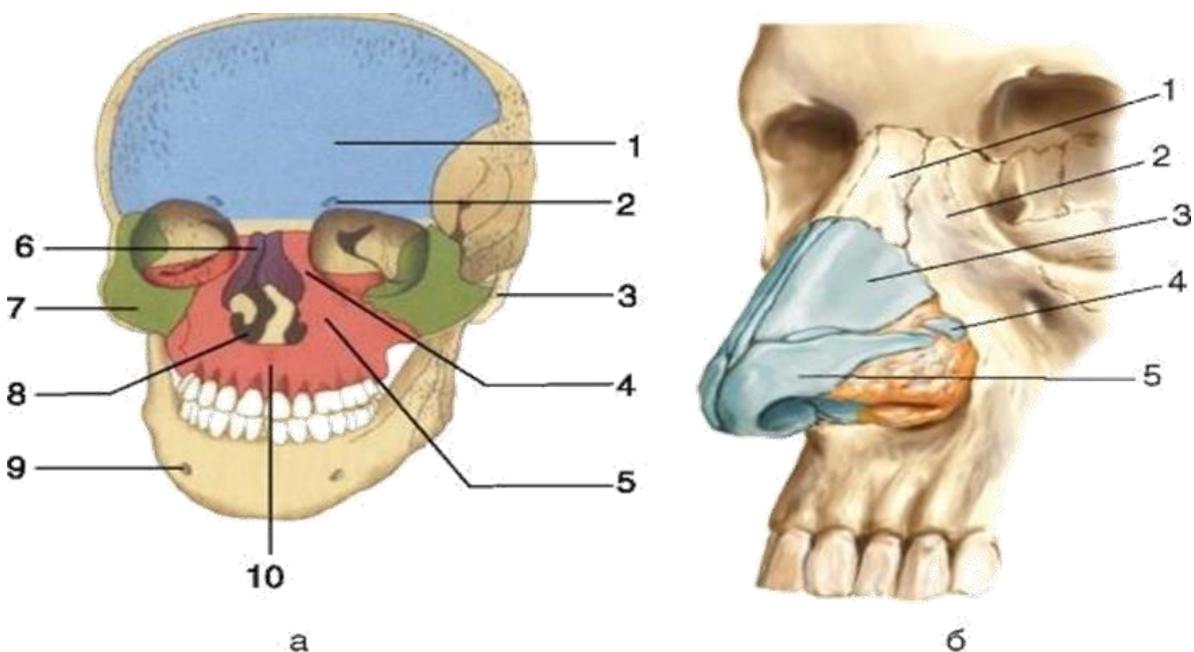
Concluding synthesis The history of otorhinolaryngology in Uzbekistan is a reflection of broader global currents tempered by local adaptation. From early regional traditions to Soviet modernization and post-Soviet modernization, ENT in Uzbekistan has evolved through a dynamic interplay of education, clinical innovation, and international influence. The resulting landscape—characterized by expanded access to diagnostic and surgical services, strong university-based training, and a growing emphasis on pediatric ENT and audiology—speaks to a durable commitment to improving communication, breathing, and quality of life for Uzbek patients. The inclusion of internationally recognized pioneers alongside notable Uzbek physicians demonstrates a shared lineage in which global ideas and local leadership converge to advance ENT care for the people of Uzbekistan.

CHAPTER 1 CLINICAL ANATOMY OF THE NOSE AND NOSE SIDE SPACES

1.1 External clinical anatomy of the nose

The upper respiratory tract comprises the nasal cavity, paranasal sinuses, and larynx. The nose represents the initial segment of the respiratory system and includes two principal components: the external nose and the nasal cavity.

The external nose (*nasus externus*) has the form of a triangular pyramid with its base directed inferiorly. Its superior narrow portion, adjoining the frontal region, is termed the root of the nose (*radix nasi*). Inferior to this lies the dorsum nasi, which extends to the apex. The lateral surfaces form the alae nasi. The inferior free margins of the alae, together with the movable part of the nasal septum, delimit the nostrils and form the entrance to the nasal cavity.

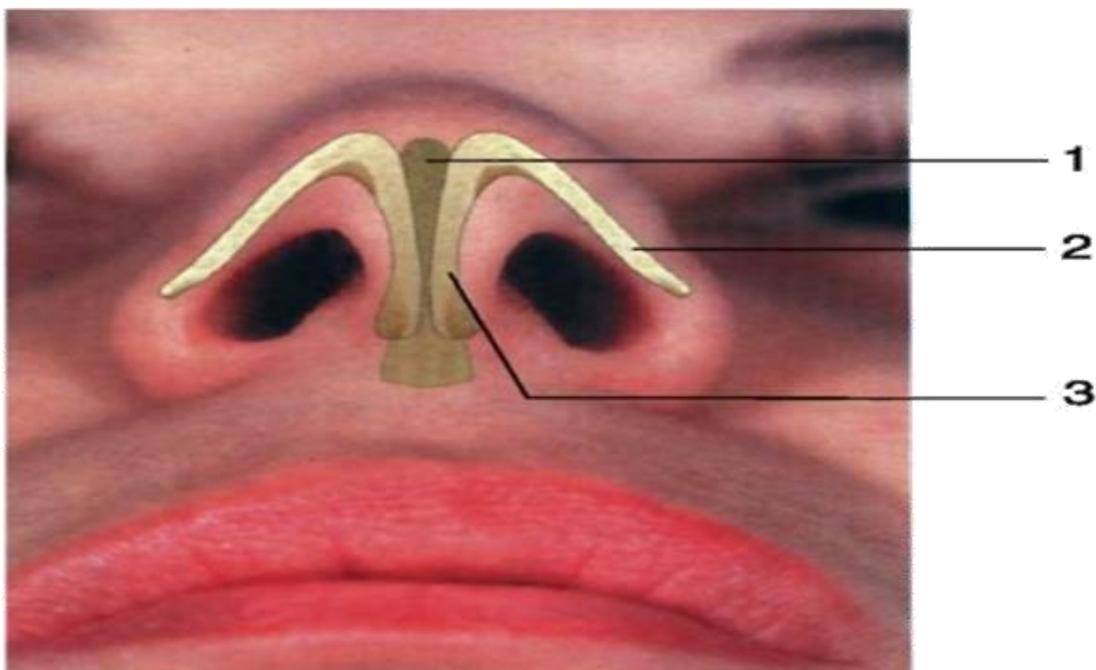


Picture: 1.(a) Bones of the facial skeleton: 1, 2 - frontal bone; 3, 7 – cheek bone; 4 - Frontal growth of the upper jaw; 5, 10 - upper jaw; 6 - nasal bones; 8 – pear-shaped hole; 9 - lower jaw.

1.1(b) External nasal skeleton: 1 - nasal bone; 2 - frontal growth of the upper jaw; 3 - lateral cartilages; 4 – minor wing cartilage; 5 – major wing cartilage.

The osseous framework of the external nose (Pic. 1) consists primarily of paired nasal bones (*ossa nasalia*), which articulate along the midline and form the superior portion of the nasal dorsum. Superiorly, these bones connect with the frontal bone, while laterally they articulate with the frontal processes of the maxillae (*processus frontalis maxillae*), thereby contributing to the formation of the lateral nasal walls.

The cartilaginous portion of the external nose (Pics. 1, 2) includes the paired lateral nasal cartilages (*cartilago nasi lateralis*) and the major and minor alar cartilages (*cartilago alaris major et minor*). The skin covering the root and lateral aspects of the nose is thin and relatively mobile. In contrast, the skin and subcutaneous tissues over the alae and nasal tip are firmly adherent to the underlying structures. This region contains numerous sebaceous glands; obstruction of their ducts or chronic inflammation may lead to pathological processes. Sweat glands are also abundant in this area.

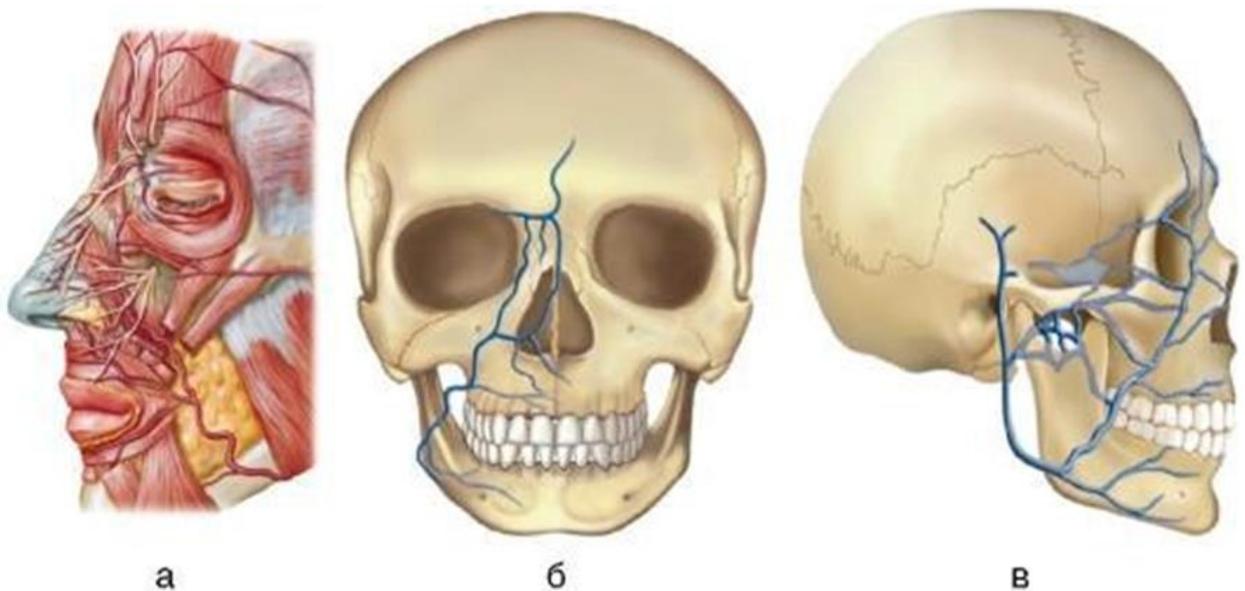


Pic. 2. *Anatomy of the external nasal cavity: 1 – rectangular cavity; 2 – major alar cartilage; 3 - the medial pedicle of the wing-shaped cartilage.*

The nasal vestibule (*vestibulum nasi*) is lined with skin containing a considerable number of hairs, creating favorable conditions for purulent inflammatory conditions such as furunculosis and sycosis. Although the external nasal muscles in humans are relatively rudimentary, they retain functional importance in regulating the size of the nasal entrance through dilation and constriction.

The vascular supply of the external nose demonstrates distinct anatomical features (Pic. 3). Arterial blood is delivered predominantly by branches of the facial artery (*a. facialis*), including its terminal branch, the angular artery, which forms anastomoses with branches of the external maxillary artery and the dorsal nasal artery. At the nasal tip, the arteries form a dense vascular network, which explains both the rapid wound healing observed in this region and the potential for profuse bleeding in cases of trauma.

Venous drainage from the nasal tip, alae, and upper lip occurs via the facial vein (*v. facialis*), which continues as the angular vein (*v. angularis*) and communicates with the cavernous sinus (*sinus cavernosus*) located in the middle cranial fossa through the ophthalmic vein. This anatomical relationship is of significant clinical importance, as infections such as furuncles in the region of the external nose or upper lip may spread intracranially via venous pathways, potentially leading to sepsis or cavernous sinus thrombosis.



Picture: 3. Blood supply and innervation of the external nose: a - facial artery and trigeminal nerve; b, v - facial veins

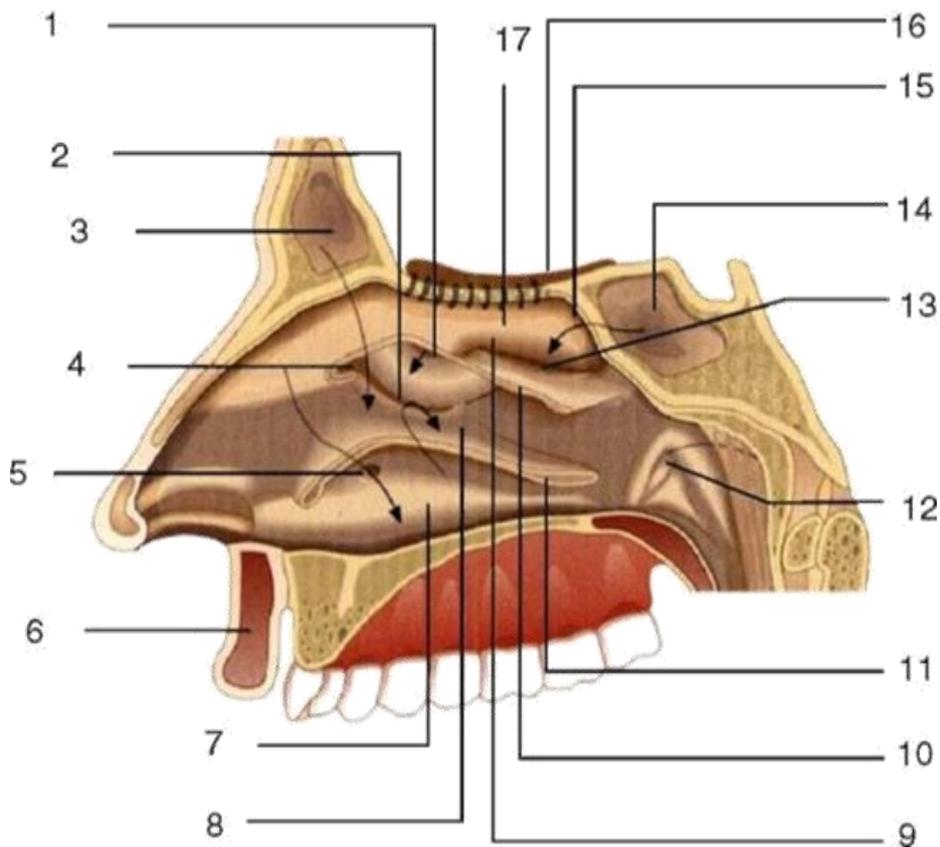
Lymphatic drainage follows vessels accompanying the arteries and veins of this region. These lymphatics penetrate deeper subcutaneous layers near the oral cavity and drain into the submandibular lymph nodes. A portion of the lymphatic vessels also empties into the superficial and deep cervical lymph nodes. This pathway should be considered in cases of regional lymphadenitis.

Motor innervation of the external nose is provided by the facial nerve (*n. facialis*), whereas sensory innervation is supplied by the first and second divisions of the trigeminal nerve (*n. trigeminus*).

1.2. Clinical anatomy of the nasal cavity

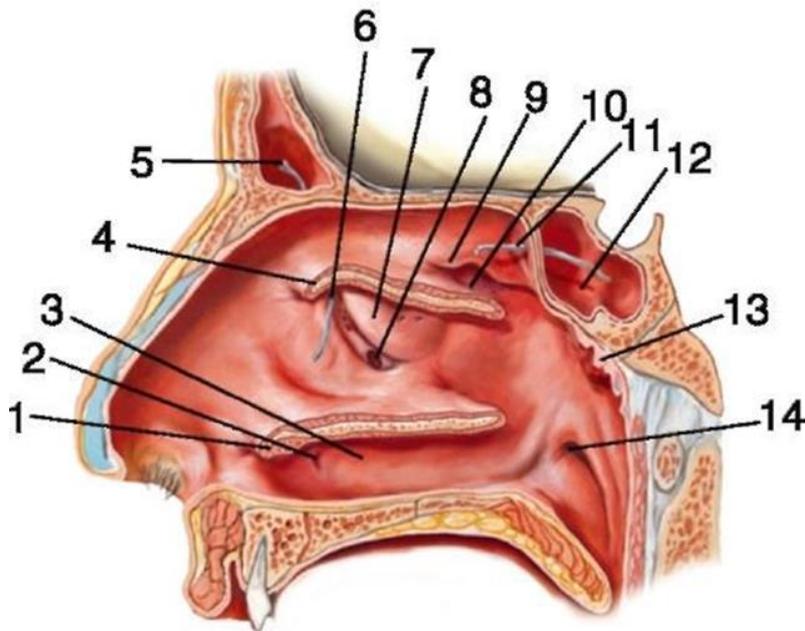
The nasal cavity (*cavum nasi*) is divided by a septum into two symmetrical compartments referred to as the right and left halves. Anteriorly, it communicates with the external environment through the nostrils, and posteriorly with the nasopharynx via the choanae. Each half of the nasal cavity is bounded by four walls: medial, lateral, superior, and inferior.

The cavity begins with the vestibule, which differs histologically from the remaining regions because it is lined with skin. The vestibular epithelium contains numerous hairs that function as a mechanical filter, trapping large particulate matter during nasal respiration.



Picture. 4. The side wall of the nasal cavity: 1 - middle concha; 2 - the opening of the upper jaw cavity; 3 - frontal cavity; 4 - frontal hole; 5 - lacrimal canal; 6 - upper lip; 7 - lower nasal passage; 8 - middle nasal passage; 9 - upper nasal concha; 10 - middle nasal concha; 11 - lower nasal concha; 12 - the opening of the auditory tube; 13 upper nasal passages; 14 - sphenoid cavity; 15 - sphenoid hole; 16 - ethmoid plate; 17 - filtering zone.

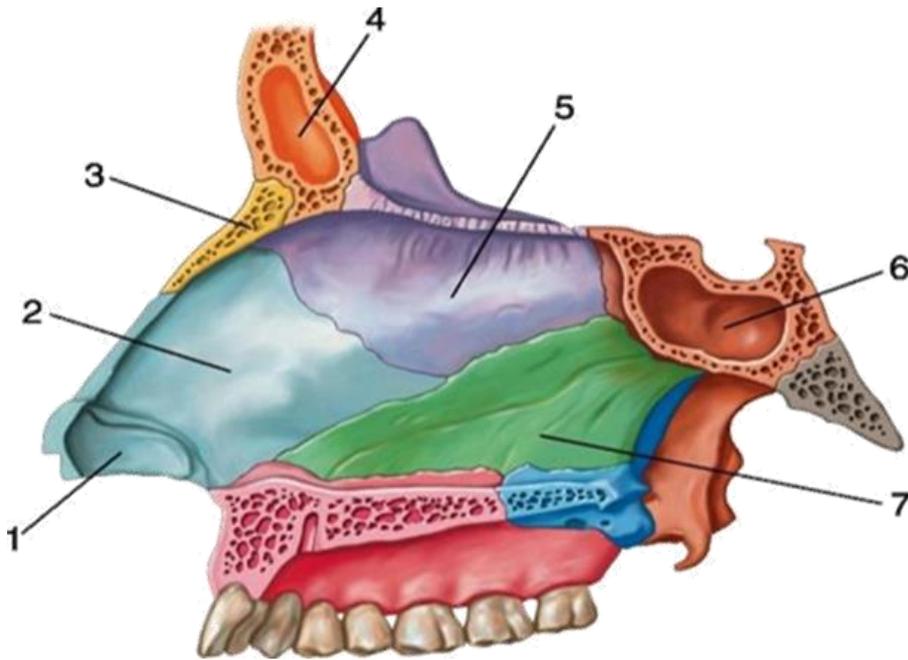
On the lateral wall of the nasal cavity (Pic. 4), three distinct projections are arranged vertically one above the other. These structures are the nasal conchae (*conchae nasales*): inferior, middle, and superior (*concha nasalis inferior, media et superior*). The inferior concha is an independent bone, whereas the middle and superior conchae are components of the ethmoid bone (Pic. 5). Beneath each concha lies a corresponding nasal meatus—*meatus nasi inferior, medius et superior*. The space formed between the medial surfaces of the conchae and the nasal septum is termed the common nasal meatus.



Picture. 5. *The connection of the nasal cavities with the nasal cavity: 1 - lower nasal concha; 2 – opening of tear-nasal channel; 3 - lower nasal passage; 4 – middle nasal concha; 5 – frontal space; 6 - a crack in the frontal cavity; 7 - gular bubble; 8 – cleft of the upper jaw cavity; 9 - upper nasal concha; 10 - upper nasal passage; 11 - base bone crack; 12 – base bone cavity; 13 – larynx lymphoid gland; 14 - laryngeal slit of the auditory tube*

Within the submucosal layer of the conchae, in addition to osseous tissue, there is an extensive venous plexus resembling cavernous tissue. Small-caliber arterioles drain into enlarged venous channels, allowing the conchae to change volume. Under specific physiological stimuli, this vascular engorgement narrows the common nasal passage and prolongs contact between inspired air and the richly vascularized mucosa. In the anterior portion of the inferior meatus, the nasolacrimal duct opens into the nasal cavity, permitting the drainage of tears.

The medial wall of the nasal cavity is formed by the nasal septum (*septum nasi*). Its bony component consists of the perpendicular plate of the ethmoid bone and the vomer. An anterior movable portion is formed by the quadrangular cartilage and the duplication of skin within the vestibular region (Pic. 6). The vomer is an independent, irregularly rectangular bone. Anteriorly, it articulates with the palatine processes of the maxilla, while posteriorly it contributes to the separation of the choanae. The superior border of the quadrangular cartilage supports the dorsum of the nose.



Picture: 6. Nasal septum: 1 - the medial pedicle of the major wing-shaped cartilage; 2 - quadrangular cartilage; 3 - nasal bone; 4 – frontal space; 5 – the perpendicular plate of the glomerular bone; 6 – sphenoid space; 7 – zygomatic bone.

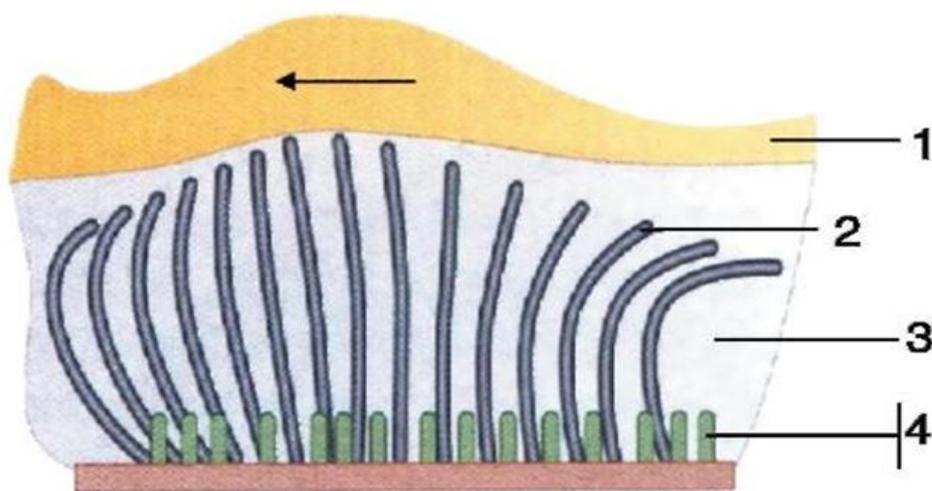
The nasal septum demonstrates a degree of flexibility, which is of surgical relevance: excessive resection may result in collapse of the nasal dorsum. In early childhood, septal deviation is typically absent until approximately five years of age. Subsequently, uneven growth of the bony and cartilaginous components may lead to varying degrees of deviation. In adults—more frequently in males—septal deviation is observed in up to 95% of cases.

The superior wall (roof) of the nasal cavity is formed anteriorly by the nasal bones and centrally by the cribriform plate (*lamina cribrosa*) of the ethmoid bone. This region represents the narrowest and thinnest part of the nasal roof, measuring only a few millimeters in thickness. Due to its fragility, surgical trauma may result in perforation and subsequent cerebrospinal fluid leakage (rhinorrhea). Secondary infection may lead to meningitis. The cribriform plate contains approximately 25–30 small foramina through which olfactory nerve filaments, as well as accompanying vessels, pass into the cranial cavity. This region may also serve as a source of epistaxis.

The inferior wall (floor) separates the nasal cavity from the oral cavity. It is formed by the palatine process of the maxilla and the horizontal plate of the palatine bone. In adults, the width of the nasal floor ranges from 12 to 15 mm, whereas in newborns it measures approximately 7 mm. Posteriorly, the nasal cavity communicates with the nasopharynx through the choanae. In neonates, the choanae are triangular or round, measuring approximately 6×6 mm, and their dimensions double by the age of ten.

In young children, the nasal passages are relatively narrow due to the proportionally large turbinates. The inferior concha lies close to the nasal floor; therefore, even mild mucosal inflammation may result in complete nasal obstruction and impaired sucking.

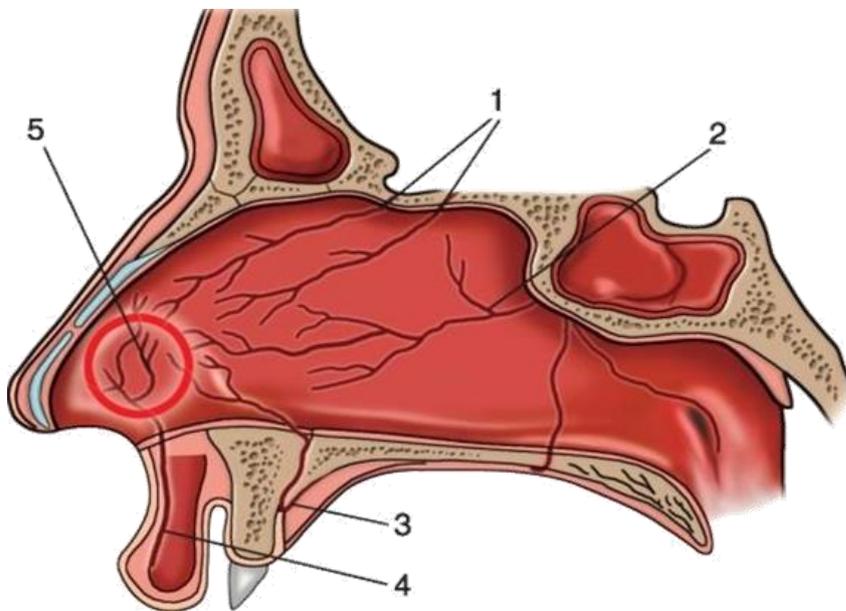
The mucous membrane of the nasal cavity is functionally divided into respiratory and olfactory regions. The respiratory mucosa is firmly attached to the underlying osseous structures and measures approximately 1 mm in thickness, lacking a distinct submucosal layer. It contains ciliated epithelial cells, numerous goblet cells, and basal cells. Each ciliated cell bears approximately 250–300 cilia, which beat at a frequency of 16–25 oscillations per minute toward the posterior nasal cavity and choanae (Pic. 7). During inflammatory conditions, epithelial metaplasia may occur, increasing mucus production. Basal cells are responsible for mucosal regeneration. Under physiological conditions, the nasal mucosa secretes approximately 500 ml of fluid daily; this volume increases significantly during inflammation.



Picture.7. *Mucociliary transport (scheme): 1, 3 - mucus; 2 - cilia (cilia); 4 - microvilli*

Beneath the mucosa of the nasal conchae lies an extensive vascular network resembling cavernous tissue, composed of interconnected arterial and venous channels. The smooth muscle elements within these vessels are innervated by branches of the trigeminal nerve. Stimulation of these fibers results in either vascular engorgement or decongestion, particularly in the inferior conchae. Physiologically, nasal airflow alternates between the two halves of the nose throughout the day, allowing one side to rest while the other predominates.

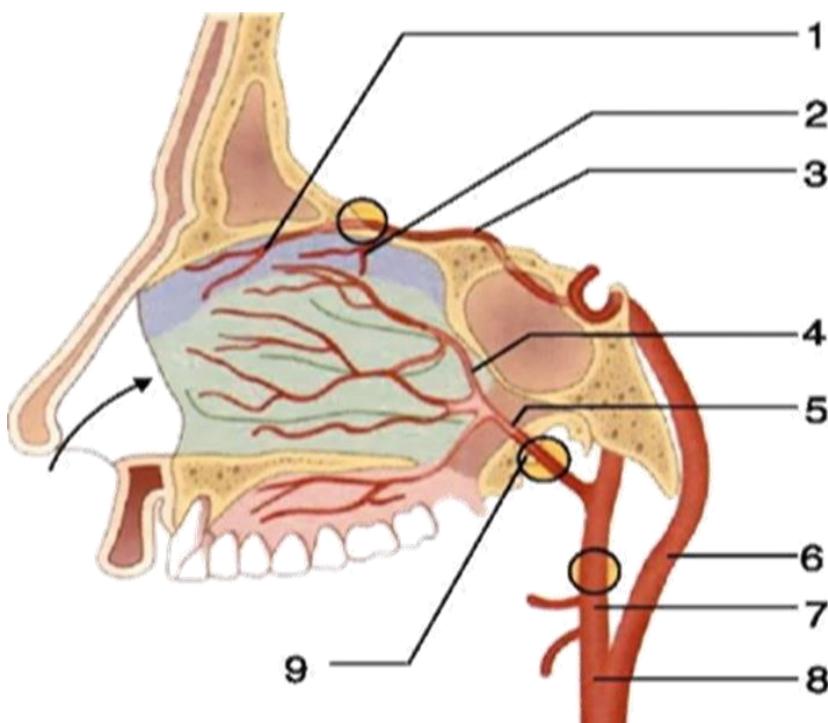
In the anterior portion of the nasal septum, an approximately 1 cm² vascular area with a dense arterial and venous network can be identified. This region, known as Kiesselbach's area (*locus Kiesselbachi*), represents the most common site of epistaxis (Pic. 8).



Picture. 8. Bleeding area of the nasal septum: 1 – anterior and posterior glomerular arteries; 2 – sphenopalatine artery; 3 – palatine artery; 4 – labial artery; 5 – Kiesselbach area

The olfactory region occupies the superior portion of the middle concha, the entire superior concha, and the corresponding upper part of the nasal septum. Axons of olfactory receptor cells form 15–20 delicate nerve filaments that pass through the foramina of the cribriform plate into the cranial cavity and synapse in the olfactory bulb. Subsequent neuronal pathways project to subcortical centers and ultimately to the pyramidal neurons of the cerebral cortex, forming the central component of the olfactory analyzer.

Arterial blood supply to the nasal cavity is derived primarily from the maxillary artery, a terminal branch of the external carotid artery. The sphenopalatine artery (*a. sphenopalatina*) enters the nasal cavity through the sphenopalatine foramen near the posterior end of the middle concha and supplies both the lateral wall and the septum, forming anastomoses with the greater palatine artery and the superior labial artery. Additional supply is provided by the anterior and posterior ethmoidal arteries (*a. ethmoidalis anterior et posterior*), branches of the ophthalmic artery, which itself originates from the internal carotid artery (Pic. 9).



Picture: 9. Blood supply to the nasal cavity: 1 - anterior splenic artery; 2 - posterior iliac artery; 3 - meningeal artery; 4 - palatal artery; 5 - maxillary artery; 6 - internal carotid artery; 7 - external carotid artery; 8 - common carotid artery; 9 - site of embolization of the maxillary artery

Thus, the arterial supply of the nasal cavity is derived from both the internal and external carotid artery systems. For this reason, ligation of the external carotid artery alone does not invariably result in the cessation of severe epistaxis, as collateral circulation from the internal carotid system may maintain bleeding.

The venous vessels of the nasal cavity are situated more superficially than the corresponding arteries and form extensive plexuses within the mucosa of the nasal conchae and septum. One of these

vascular networks corresponds to Kiesselbach's area (*locus Kiesselbachi*), described previously. In addition, larger-caliber venous channels are present in the posterior portions of the nasal septum.

Venous outflow from the nasal cavity proceeds in several directions. Blood from the posterior regions drains into the pterygoid venous plexus (*plexus pterygoideus*), which communicates with the cavernous sinus (*sinus cavernosus*) located in the middle cranial fossa. This anatomical relationship creates a potential pathway for the spread of infection from the nasal cavity and nasopharynx into the cranial cavity.

Venous blood from the anterior regions of the nasal cavity flows into the veins of the upper lip and the angular vein, which communicates with the cavernous sinus via the superior ophthalmic vein. Consequently, inflammatory processes in the region of the nasal vestibule—particularly furuncles associated with hair follicles—may extend intracranially through these venous connections.

Of particular clinical importance is the communication between the anterior and posterior ethmoidal veins and the veins of the orbit, which may facilitate the spread of infection from the ethmoidal labyrinth to the orbital structures. Furthermore, a branch of the anterior ethmoidal veins passes through the cribriform plate, forming anastomoses with the veins of the dura mater and entering the anterior cranial fossa. Owing to the dense venous network and numerous anastomoses in the maxillofacial region, inflammatory processes such as thrombophlebitis may result in severe complications, including orbital vein thrombosis, cavernous sinus thrombosis, and sepsis.

Lymphatic vessels of the nasal cavity direct lymph posteriorly toward the nasopharynx and auditory tubes. These vessels pass around the pharyngeal openings superiorly and inferiorly and drain into the retropharyngeal lymph nodes located between the prevertebral and visceral layers of the cervical fascia. A portion of the lymphatic drainage also reaches the deep cervical lymph nodes. In children, the abundance of lymphoid tissue may contribute to the formation of retropharyngeal abscesses during inflammatory diseases of the nasal cavity, paranasal sinuses, or middle ear.

Metastatic spread in malignant tumors of the nasal cavity and ethmoidal labyrinth demonstrates characteristic patterns determined by lymphatic drainage pathways, typically involving enlargement of lymph nodes situated along the internal jugular vein.

Innervation of the nasal cavity includes olfactory, sensory, and autonomic components.

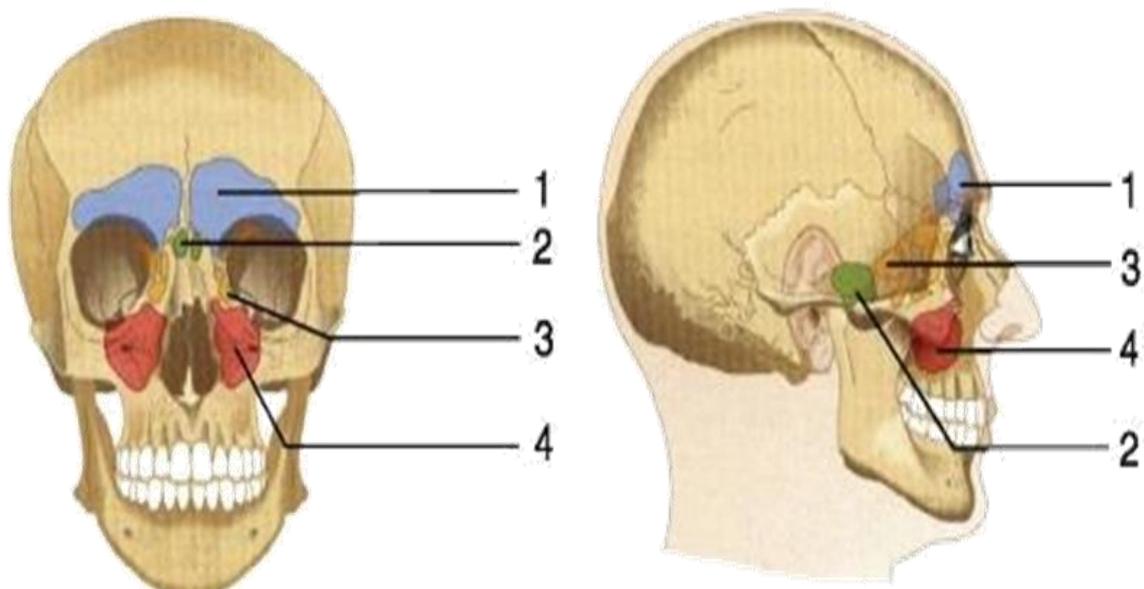
Olfactory innervation is provided by the olfactory nerve (*n. olfactorius*). Its filaments pass through the foramina of the cribriform plate and supply the mucosa of the superior nasal region.

Sensory innervation is mediated by the trigeminal nerve (*n. trigeminus*), primarily through its first and second divisions.

Autonomic (vegetative) innervation is carried by the nerve of the pterygoid canal (*n. canalis pterygoidei*, Vidian nerve). The autonomic nervous system of the nasal cavity regulates vascular tone within the cavernous tissue and controls the secretory activity of the mucous glands, thereby maintaining physiological nasal function.

1.3. Clinical anatomy of the paranasal sinuses

Nasal sinuses (*paranasal sinuses*) - these are air spaces located near the nasal cavity and connected to it through holes. These include the maxilla, the frontal and the occipital cavity, and the occipital cavities (Pic.10).



Picture. 10. Projection of adjacent nasal cavities: 1 – frontal cavities; 2 – sphenoid space; 3 – ethmoid spaces; 4 – upper jaw cavities

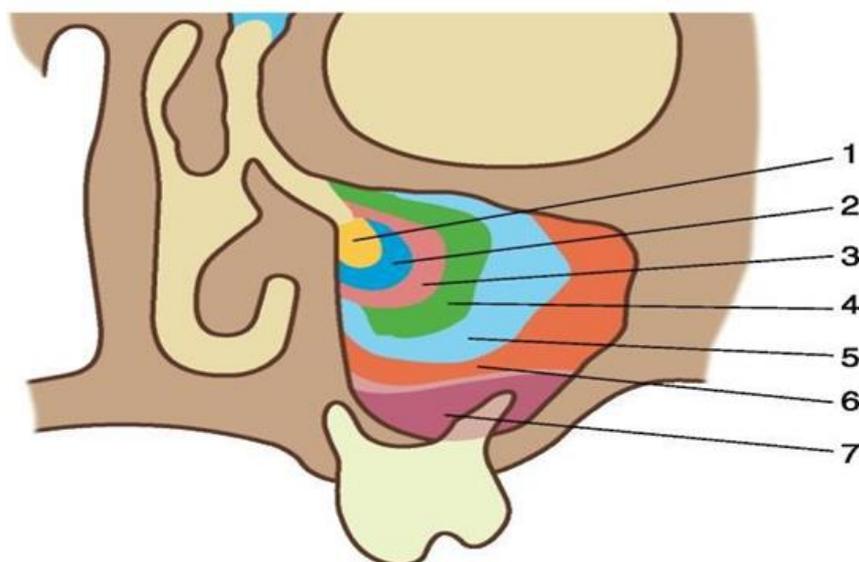
The development of nasal cavities begins at 9-10 weeks of fetal development. In a newborn baby, all paranasal cavities are present, except for the frontal and nasal cavities, which are formed by 6-8 years of age.

Maxillary cavities

The maxillary sinus (*sinus maxillaris*) represents the largest of the paranasal sinuses and is situated within the body of the maxilla. In newborns, this sinus has a slit-like configuration and occupies a narrow space bounded by the anterior wall of the sinus, the inferior wall of the orbit, and the alveolar process (Pic. 11). At birth, its longitudinal dimension ranges from approximately 7 to 14 mm, while its vertical height measures between 5 and 10 mm.

During the first year of life, the cavity gradually assumes a more rounded contour. As the facial skeleton develops and expands, the sinus enlarges correspondingly. By the age of 6–7 years, it acquires a more complex, multifaceted shape characteristic of the adult morphology. Following the eruption and subsequent replacement of the dentition, further expansion of the sinus occurs in parallel with the growth of the maxilla. Final structural formation is typically completed between 15 and 20 years of age.

In adults, the average volume of the maxillary sinus is approximately 15–20 cm³.



Picture. 11. *The size of the upper jaw depending on the age: 1 - 3 months; 2 - 6 months; 3 - 1 year; 4 - 3 years; 5 - 5 years; 6 - 8 years old; 7 - 12 years old*

The anterior wall of the maxillary sinus extends from the inferior orbital margin to the alveolar process of the maxilla. Approximately 0.5–1 cm below the infraorbital rim lies the infraorbital foramen, through which the infraorbital neurovascular bundle emerges. This bundle contains the maxillary nerve—the second division of the trigeminal nerve (*n. trigeminus*)—as well as the accompanying artery and vein.

In the inferior portion of the anterior wall, at the level of the second premolar, a distinct depression known as the canine fossa (*fossa canina*) is identified. This region is commonly regarded as the optimal surgical access point for opening the maxillary sinus. The bone forming the anterior wall is relatively dense and porous; therefore, intraoperative manipulation may be accompanied by persistent osseous bleeding.

The superior wall constitutes the roof of the sinus and separates it from the orbital cavity. Within this wall runs the infraorbital canal, or occasionally a semicanal, transmitting the maxillary nerve and associated vessels. Consequently, pathological processes within the sinus may involve this neurovascular bundle. The inferior wall of the infraorbital canal often projects into the sinus cavity as a bony ridge; in certain cases, the nerve may be covered only by the mucous membrane. Owing to the thinness of the superior wall, inflammatory or neoplastic processes may extend superiorly into the orbit. Anteriorly, the medial margin of the superior wall articulates with the lacrimal bone and contributes to the formation of the superior portion of the nasolacrimal duct.

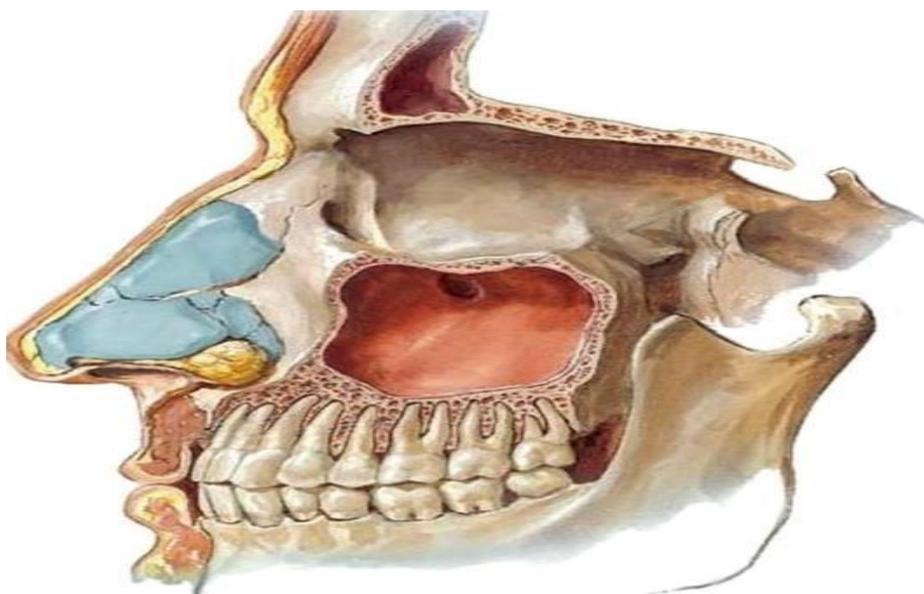
Posterior to this region lies the thin orbital plate (*lamina papyracea*), which separates the orbit from the ethmoidal air cells. The medial wall of the maxillary sinus corresponds anatomically to the lateral wall of the nasal cavity. In its anterior segment, the nasolacrimal canal descends and opens into the inferior nasal meatus. The natural ostium of the maxillary sinus (*ostium maxillare*) is located high on the medial wall, near the roof of the cavity, and drains into the middle nasal

meatus. Due to its superior position, drainage is relatively inefficient when the body is in the upright position.

The posterior wall is inclined and corresponds to the posterior surface of the maxilla, adjacent to the pterygopalatine fossa. In its superior portion, branches of the superior alveolar nerves are present. Posteriorly and superiorly, the wall lies in close proximity to the sphenoid sinus and the posterior ethmoidal cells. Structures located near the pterygopalatine fossa include the maxillary nerve (the second division of *n. trigeminus*), the maxillary artery, and the pterygoid venous plexus, which communicates with the orbital veins and the cavernous sinus of the dura mater. For this reason, pathological processes originating in the maxillary sinus may extend to these adjacent anatomical regions.

The inferior wall (floor) of the sinus is formed by the alveolar process of the maxilla. Typically, the floor of the maxillary sinus corresponds to the region extending from the second premolar to the second molar; in some individuals, it may range from the first premolar to the third molar.

When the sinus floor is positioned relatively low, the apices of the dental roots—and in some cases even the root canals—may project toward or into the sinus cavity (Pic. 12), which has important clinical implications for odontogenic infections and surgical interventions.



Picture.12. *The position of the maxillary cavity in relation to the alveolar septum and orbits.*

The apices of the first and second molars are frequently separated from the maxillary sinus by a very thin osseous lamina, often measuring no more than 0.5 mm. In certain cases, the root apices may protrude into the sinus cavity and be covered solely by the mucous membrane. Under such anatomical conditions, the risk of odontogenic infection spreading into the maxillary sinus is significantly increased. Dental extraction in these situations may result in persistent oroantral communication, through which food particles and oral microorganisms can enter the sinus cavity.

Typically, the right and left maxillary sinuses are symmetrical; however, anatomical variations are not uncommon. One such variation includes partial septation of the sinus cavity into multiple compartments by bony partitions, which may complicate both diagnosis and surgical management of maxillary sinusitis.

The mucous membrane lining the maxillary sinus represents a direct continuation of the nasal mucosa. It is extremely thin and functions as a mucoperiosteum, being firmly adherent to the underlying bone. The membrane is richly vascularized and contains lymphatic vessels, though it possesses relatively few mucous glands. It is also characterized by a considerable absorptive capacity.

The frontal sinuses (*sinus frontalis*) are located within the frontal bone. In sagittal section, each sinus typically exhibits a triangular configuration. The two sinuses are rarely perfectly symmetrical; one may be larger than the other, and in some individuals one or both may be underdeveloped or entirely absent. Hypoplasia or aplasia of one or both frontal sinuses is observed in approximately 5–10% of cases.

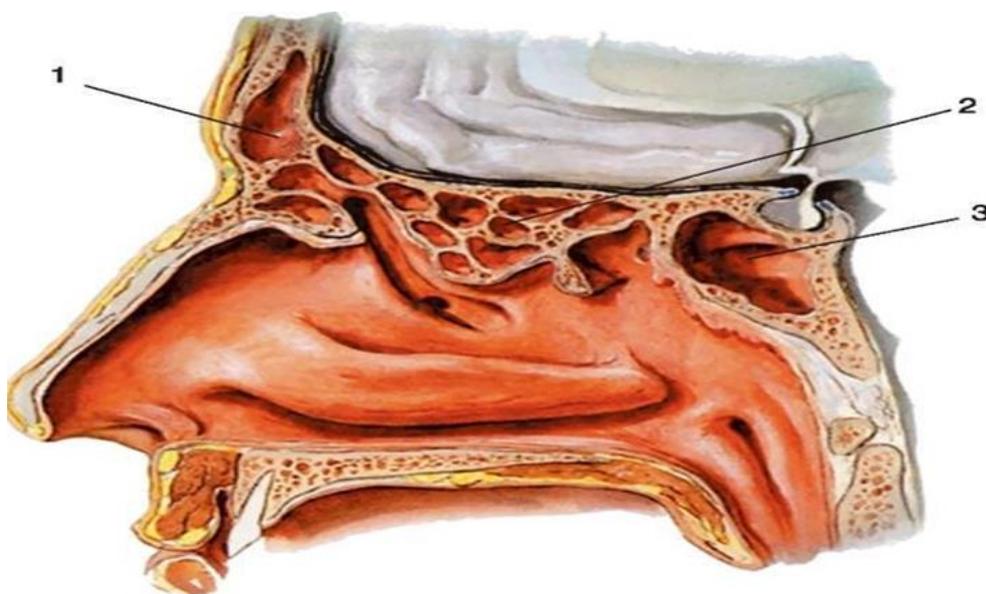
The anterior wall of the frontal sinus is the thickest and is formed by the frontal bone. The posterior wall separates the sinus from the anterior cranial fossa; although thin, it consists of dense and relatively strong bone. The inferior wall forms the boundary between the frontal sinus and the orbit. This wall is particularly thin and, depending on the degree of sinus pneumatization, may extend over a substantial portion of the orbital roof, occasionally reaching toward the lesser wings of the

sphenoid bone. In extensively developed sinuses, close anatomical relationships with the sphenoid sinus may occur, and the cavity may approach the middle cranial fossa near the optic canal.

When both frontal sinuses are present, they are separated by a thin intersinus septum and may occupy asymmetric positions relative to the midline. Each frontal sinus communicates with the nasal cavity via the frontonasal duct, which opens into the anterior portion of the middle nasal meatus. The mucous membrane of the frontal sinus is similar in structure to that of the maxillary sinus and is lined with ciliated epithelium, facilitating the drainage of secretions through the frontonasal canal.

The ethmoidal sinus (*sinus ethmoidalis*) has a complex anatomical organization. It is situated within the ethmoid bone and consists of numerous interconnected air cells (Pic. 13). The number of ethmoidal cells varies individually and typically ranges from 8 to 10 on each side.

The basal (horizontal) plate of the ethmoid bone divides the ethmoidal labyrinth into anterior and posterior groups of cells. Each ethmoidal cell possesses its own opening: the anterior cells drain into the middle nasal meatus, whereas the posterior cells open into the superior nasal meatus.



Picture. 13. Location of cavities: 1 - frontal; 2 - ethmoid; 3 - sphenoid

The mucous membrane lining the cells of the paranasal sinuses resembles that of other sinus cavities. The anatomical and topographical

features of the ethmoidal labyrinth (*labyrinthus ethmoidalis*) allow pathological processes to extend into adjacent structures, including the orbit, cranial cavity, and optic nerve. A thorough understanding of the ethmoidal airway system requires knowledge of the structural components of the ethmoid bone (*os ethmoidale*):

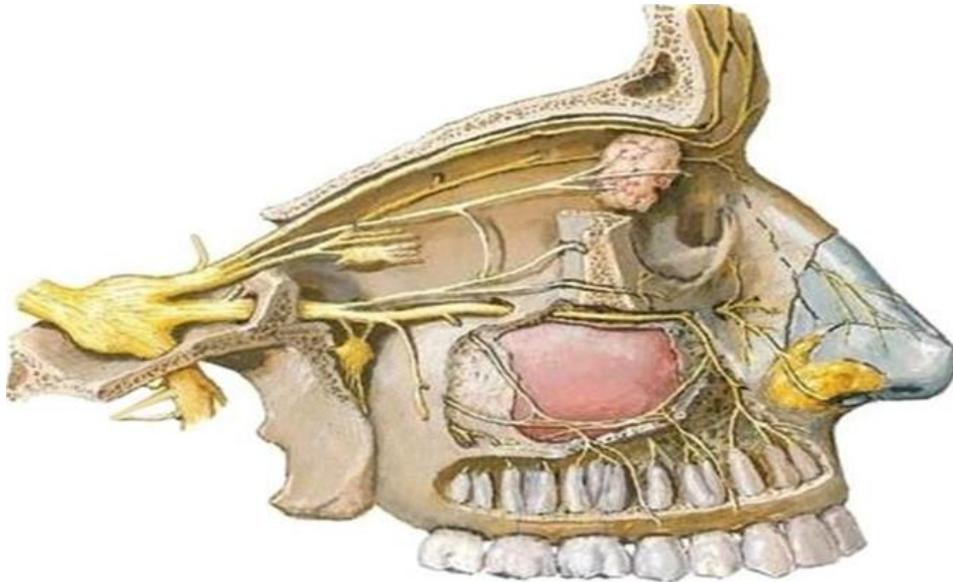
- The perpendicular plate, extending toward the anterior cranial fossa in a shape resembling a “cock’s comb,” which contributes to the bony portion of the nasal septum;
- The cribriform plate (*lamina cribrosa*), perforated with numerous small foramina for the passage of olfactory nerve fibers;
- The orbital plate (*lamina papyracea*), a delicate structure separating the ethmoidal air cells from the orbit;
- The superior and middle nasal conchae (*conchae nasales superior et media*), forming part of the lateral nasal wall.

The sphenoid sinuses (*sinus sphenoidalis*) are paired cavities located within the body of the sphenoid bone, posterior to the nasal cavity (Pic. 13). These sinuses are typically divided into right and left portions by a bony septum. The natural ostium of each sphenoid sinus is situated on the anterior wall and opens into the superior nasal meatus, facilitating drainage toward the nasopharynx.

The superior wall of the sphenoid sinus forms part of the anterior cranial fossa and lies in close proximity to the sella turcica and the pituitary gland, as well as the optic chiasm. The lateral walls are extremely thin and are adjacent to critical structures, including the internal carotid artery, the cavernous sinus (*sinus cavernosus*), the oculomotor nerve (*n. oculomotorius*), the trochlear nerve (*n. trochlearis*), the abducens nerve (*n. abducens*), and the ophthalmic branch of the trigeminal nerve (*n. trigeminus, ramus ophthalmicus*). The posterior wall is typically the thickest and borders the posterior cranial fossa.

Arterial perfusion of the paranasal sinuses is derived from branches of both the internal and external carotid arteries. The maxillary artery (*a. maxillaris*), a branch of the external carotid artery, primarily supplies the maxillary sinus. The frontal sinus receives blood from branches of the maxillary artery, ophthalmic artery (*a. ophthalmica*), palatine arteries, and meningeal arteries. The ethmoidal cells are supplied by the anterior

and posterior ethmoidal arteries (*a. ethmoidalis anterior et posterior*), as well as branches of the lacrimal artery (*a. lacrimalis*) (Pic. 14).



Picture. 14. *Innervation of the mucous membrane of the upper jaw cavity and teeth by the trigeminal nerve. The innervation of the paranasal sinuses is carried out by the first and second branches of the trigeminal nerve.*

The venous system of the paranasal sinuses forms extensive anastomoses with the venous networks of the orbit, nasal cavity, facial region, and intracranial structures. These connections create potential pathways for the spread of infection or inflammatory processes between the sinuses and adjacent areas.

Lymphatic vessels of the paranasal sinuses play a critical role in the dissemination of both infectious agents and metastatic cells. They are closely interconnected with the lymphatic channels of the nasal cavity, as well as with the deep lymph nodes of the larynx and cervical region. Moreover, inflammatory processes may extend to the mucosa of the maxillary sinus (*cavum maxillare*) via lymphatic vessels associated with the dentition, since the lymphatics of the alveolar process form anastomoses with the sinus mucosa.

CHAPTER 2 CLINICAL PHYSIOLOGY OF THE NOSE AND NOSE

The nose performs the following physiological functions: breathing, smelling, protection and resonator (speech).

Respiratory function

The nose (**nasus**) serves primarily as the organ of **respiration**. Under normal conditions, all air that is inhaled or exhaled flows through the **nasal cavities**. During inspiration, the **negative pressure within the thorax** draws air into the lungs, which is then divided between the **right and left nasal passages**.

The **principal stream of airflow** generally moves from the lower part of the **common nasal meatus (meatus nasi communis)**, passes along the **middle nasal concha (concha nasalis media)**, and then turns **posteriorly and downward** toward the **choanae (choanae)**.

While inhaling, a portion of the air is circulated within the nasal cavities, which aids in **warming and humidifying** the incoming air. During exhalation, most of the airflow follows the **inferior nasal concha (concha nasalis inferior)**, while a small fraction enters the **paranasal sinuses (sinus paranasales)**. Additionally, the **mechanical stimulation** of the **nasal mucosa (tunica mucosa nasi)** by the moving air plays a key role in **maintaining respiratory reflexes**.

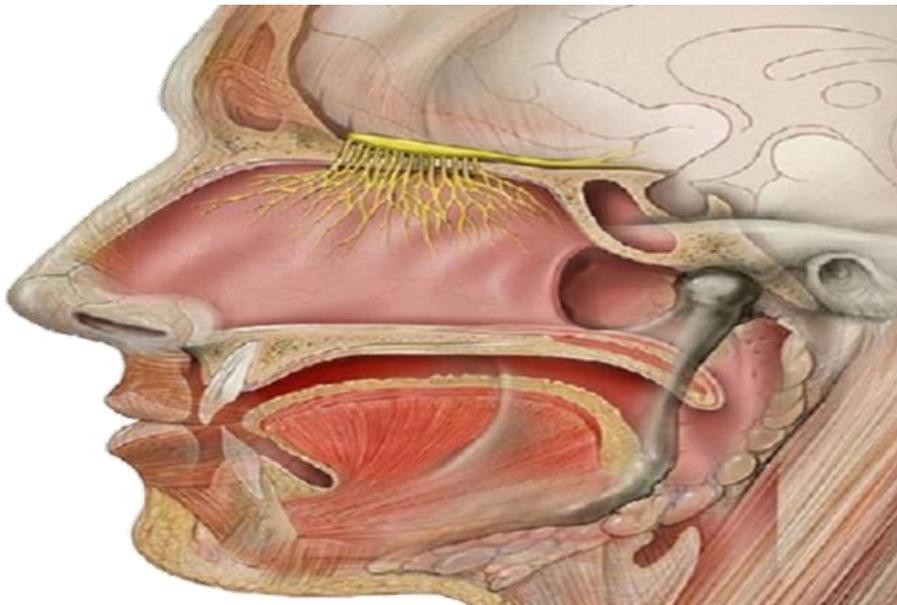
Mouth breathing, however, results in shallower ventilation, reducing oxygen intake and diminishing negative intrathoracic pressure. This, in turn, decreases the amplitude of pulmonary excursions and may contribute to systemic hypoxia. Additionally, ventilation of the nasal cavities and auditory (middle ear) spaces is compromised. In pediatric populations, prolonged impairment of nasal breathing can disrupt normal development of the facial skeleton—particularly the maxillofacial region—predispose to chronic respiratory conditions, and impair the function of distal organs.

Olfactory function

The **olfactory system (analis olfactorius)** belongs to the group of **chemical sense organs**, responding specifically to **odorant molecules**

(**vectores odoris**). During nasal breathing, fragrant substances are transported by the incoming air to the **olfactory region (regio olfactoria)**. This area is located between the lower edge of the **middle nasal concha (concha nasalis media)** and the **nasal septum (septum nasi)**, beginning with a narrow **olfactory cleft**, typically about 3–4 mm in width (Pic. 15).

For proper **sense of smell**, it is essential that air reaches this olfactory zone. **Short, forceful inhalations** help achieve this by creating small vortices that direct airflow toward the **olfactory epithelium**. The **odorant molecules** then dissolve in the thin fluid layer covering the **cilia of the olfactory receptor cells**, triggering receptor activation. This excitation travels along the **olfactory neurons** to the **olfactory cortical centers**, where it is processed, allowing the perception of odors.



Picture. 15. Odor field

Olfactory perception results from the integrated activity of all components of the olfactory system (*analisis olfactorius*), including the peripheral, intermediate, and central structures. Initial processing of odorants occurs at the peripheral level, while higher-order analysis and synthesis take place in the cortical regions of the olfactory analyzer. Several theories have been proposed to explain the mechanisms of olfaction, including chemical, physical, and physico-chemical models.

The olfactory function plays a critical role in both human and animal life. Although the sense of smell in many animals is markedly

more acute than in humans, its functional significance varies. For instance, a dog's olfactory sensitivity is approximately 10,000 times greater than that of a human, yet its overall reliance on olfaction differs in behavioral contexts.

Olfactory disorders are classified as primary when they arise from pathology affecting the central olfactory structures or the receptor components of the conducting pathways. Disorders are considered secondary when they result from impaired airflow to the olfactory region (*regio olfactoria*). Inflammatory processes, polypoid transformations of the mucosa, and atrophic changes can significantly reduce olfactory sensitivity (hyposmia), and in severe cases, lead to complete loss of smell (anosmia).

Protective function

As air moves through the **nasal cavities (cavitates nasales)**, it is **warmed, filtered, and humidified**. The warming of inhaled air is aided by the **slower airflow**, the extensive surface area of the **nasal mucosa (tunica mucosa nasi)**, and its rich network of blood vessels. Exposure to **cold air** reflexively causes the cavernous vascular spaces in the **nasal conchae (conchae nasales)** to **dilate and engorge**, increasing their volume and narrowing the **nasal passages (meatus nasi)**. This forces air to flow in a thinner stream, enhancing contact with the mucosa and improving the **heating of air**, with efficiency rising as the ambient temperature drops.

Air **filtration** is achieved through several mechanisms. Larger dust particles are trapped by the **nasal hairs (vibrissae nasi)** at the nostrils, while finer particles settle on the **mucosa**, which contains **mucus** rich in **lysozyme and immunoglobulins**. The narrow and curved configuration of the nasal passages promotes particle deposition, with roughly 40–60% of dust and microorganisms being neutralized by the mucus or removed along with it.

The **self-cleaning function** of the nasal mucosa, called **mucociliary clearance (transport mucociliare)**, relies on the **ciliated epithelium**. The epithelial cells' **apical surfaces** are covered with motile **cilia**, which beat at around 10–15 cycles per minute. Coordinated ciliary motion moves **mucus and trapped particles**—including dust,

microbes, and chemical agents—posteriorly toward the **nasopharynx (pars nasopharyngea)**. Transport of mucus from the front of the nasal cavity to the nasopharynx usually takes 10–20 minutes and can be affected by **inflammation, temperature changes, pH variations, or exposure to chemicals**.

Humidification occurs via evaporation of secretions from **mucous and goblet cells**, reflexive transudation of **lymph fluid** from the epithelium, and contributions from **lacrimal fluid (lacrimae)**. On average, the nasal mucosa produces about 500 ml of fluid daily.

Protective **reflexes**, such as **sneezing (sternutatio)** and increased **mucus secretion**, expel irritants from the nasal cavity. The sudden forceful exhalation during a sneeze removes **foreign particles, dust, and other irritants**, preventing them from entering the **respiratory tract** further.

Resonator function

The nasal cavity (*cavum nasi*) and paranasal sinuses (*sinus paranasales*) serve as resonators, amplifying sound and contributing to the timbre of the voice during speech and singing. Unlike the laryngeal resonator, the dimensions of the nasal and paranasal cavities remain relatively constant, which helps maintain the stability of vocal timbre. Resonance is modulated by the position of the soft palate (*palatum molle*). When the mouth is open due to soft palate paralysis (*rhinolalia aperta*), or when nasal airflow is obstructed by congestion, polyps, or enlarged adenoids, a closed-nasal resonance (*rhinolalia clausa*) may occur.

CHAPTER 3. METHODS OF INSPECTION OF THE NOSE AND NASAL SPACES

The study of the condition of the external nose, its cavity or the nasal cavity - begins with the identification of complaints, the collection of anamnesis, and only after that they proceed to the examination of the nasal cavity on all floors and departments. initiates a special investigation that allows Examination of the nasal cavity can indirectly indicate the condition of the adjacent nasal cavities.

Stage I - external examination and palpation.

Clinical assessment of the external nose (*nasus externus*) involves careful evaluation of its shape, alignment relative to the midline, and the position of the nasal dorsum. Palpation is particularly indicated when a fracture of the nasal bones (*ossa nasalia*) is suspected. The procedure is performed by placing the index finger along the nasal dorsum or root (*radix nasi*), while the thumb and middle finger are positioned on the lateral wings of the nose (*alae nasi*). Gentle movement from the root to the tip (*dorsum nasi*) allows the clinician to detect retraction or protrusion of specific regions, assess the mobility of the bony structures, and identify any crepitus associated with fractures.

Examination of the nasal cavity (*cavum nasi*) can be performed without specialized instruments, although the use of a frontal reflector (*speculum frontale*) is essential for adequate visualization. To inspect deeper regions of the nasal cavity, a narrow beam of light is directed through the nostril, illuminating the internal structures. Proper technique involves placing a light source, such as a table lamp, on the patient's right side at the level of the earlobe. The frontal reflector is positioned with its notch aligned to the examiner's left eye. By adjusting the mirror so that the left eye catches the light and directs it through the aperture to the patient's face, a central reference point—sometimes referred to as the "rabbit"—can be visualized. Once the left-eye alignment is achieved, the right eye is opened, allowing binocular vision, which is critical for accurate spatial perception of the nasal structures.

With the patient seated upright directly in front of the examiner, the beam is directed toward the nostrils (*nares*), while the patient gently lifts the tip and dorsum of the nose with the thumb of the right hand, stabilizing the rest of the head with the remaining fingers. This maneuver facilitates optimal visualization of the nasal passage, including the nasal septum (*septum nasi*), the medial surfaces of the alae (*alae nasi*), and the vibrissae (*vibrissae nasi*) located within the vestibule.

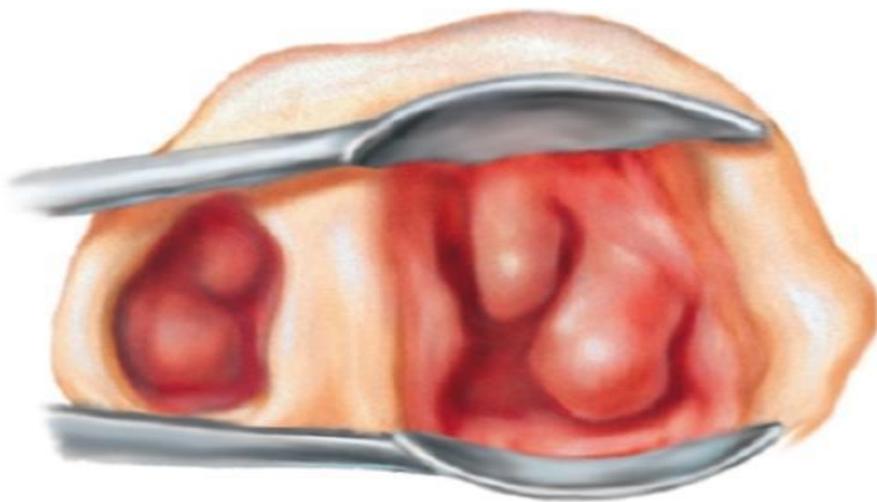
Stage II - anterior rhinoscopy

Anterior rhinoscopy (*rhinoscopia anterior*) is a diagnostic examination of the anterior regions of the nasal cavity (*cavum nasi*), performed using specialized instruments such as a nasal mirror (*speculum nasi*) or nasal dilator (*dilatator nasi*). The posture of both the

patient and examiner remains unchanged during the procedure. The clinician holds the nasal mirror in the left hand while resting the right hand on the patient's head, enabling controlled movement of the head in various directions. The correct positioning of the mirror in the left hand is illustrated in Pic. 16.



Picture 16.Anterior rhinoscopy. Position of the doctor and the patient, examination of the front and back sections of the nasal cavity



Picture. 17.Anterior rhinoscopy

When performing anterior rhinoscopy (*rhinoscopia anterior*), the light beam is directed toward the nostril (*narina*), and the blades of the nasal mirror (*speculum nasi*) are gently inserted in a closed position. Once inside, the blades are gradually opened, elevating the lateral wing

of the nose (*ala nasi*) toward the lateral canthus of the eye. The patient's head should remain in a neutral, upright position.

The initial examination focuses on the anterior portion of the inferior nasal concha (*concha nasalis inferior*), the inferior nasal meatus (*meatus nasi inferior*), the nasal septum (*septum nasi*), and the lower segment of the common nasal passage (*meatus nasi communis*). Slight extension of the patient's head allows visualization of the middle concha (*concha nasalis media*), the middle nasal meatus (*meatus nasi medius*), the upper regions of the common nasal passage, and the majority of the nasal septum (Pic. 17).

To minimize discomfort, the nasal mirror should always be removed in an open position, preventing undue pressure on the nasal mucosa (*tunica mucosa nasi*).

Stage III - back rhinoscopy

Examination of the back of the nasal cavity (posterior rhinoscopy) is a more complicated method. To perform, it is necessary to use a nasopharyngeal glass and a spatula (Pic. 18).



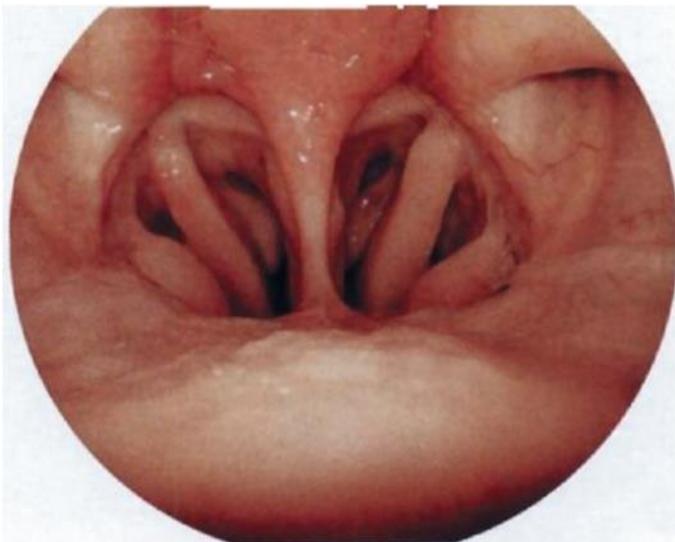
Picture. 18. *Posterior rhinoscopy*

During posterior rhinoscopy (*rhinoscopia posterior*), the clinician holds a spatula (*spatula linguae*) in the left hand to gently depress the patient's tongue without contacting the root (*radix linguae*), thereby minimizing the gag reflex. The right hand holds a nasopharyngeal mirror (*speculum nasopharyngeum*), which is preheated using an alcohol lamp to prevent condensation. While maintaining tongue depression, the

mirror is carefully advanced behind the soft palate (*palatum molle*), and the patient is instructed to breathe through the nose (*cavum nasi*).

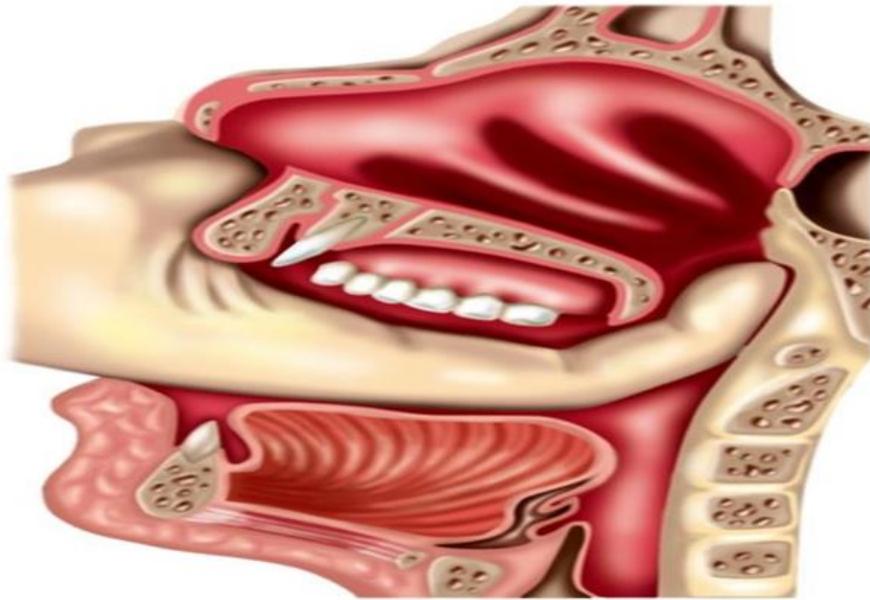
Examination focuses on the lymphoid follicles (*folliculi lymphoidei*) along the lateral wall and the roof of the nasopharynx (*nasopharynx*), the posterior portion of the nasal septum (*septum nasi*), the posterior ends of the nasal conchae (*conchae nasales*), the nasal meatuses (*meatus nasi*), and the openings of the auditory tubes (*tubae auditivae*) (Pic. 19).

In cases where posterior rhinoscopy is not feasible—commonly in infants and preschool-aged children—a digital examination may be performed. In such instances, an assistant stabilizes the child while the clinician stands to the right of the patient.



Picture 19. *Posterior rhinoscopy image*

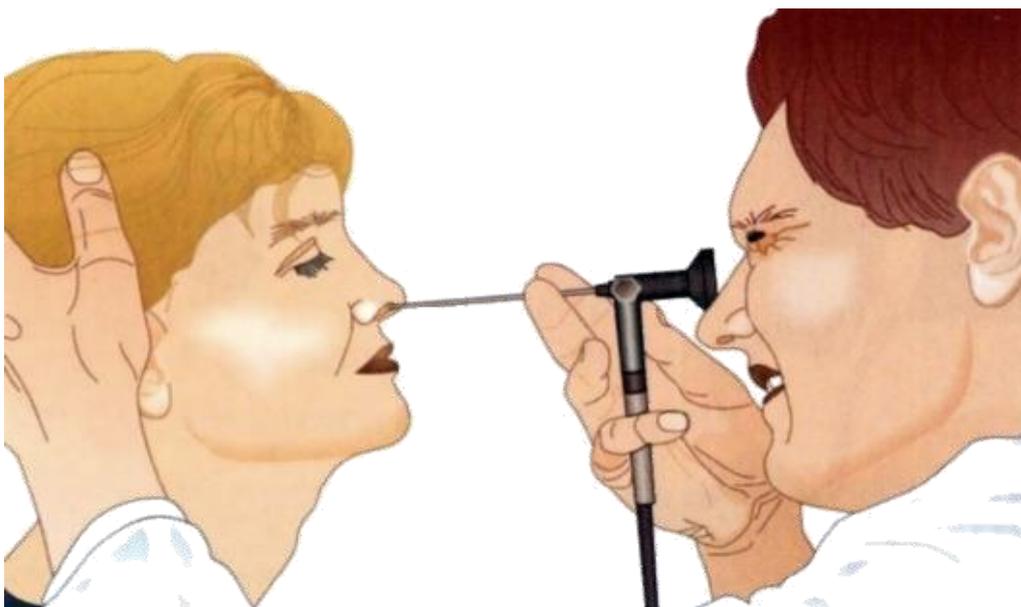
Subsequently, the examiner introduces the index finger of the right hand into the nasopharynx (*nasopharynx*) via the oral cavity (*cavum oris*) for palpation (Pic. 20). This allows assessment of the presence, size, level, consistency, mobility, and extent of adenoids (*tonsilla pharyngea*) or other masses. As previously noted, evaluation of the nasal cavity (*cavum nasi*) provides valuable insight into the condition of the adjacent paranasal sinuses (*sinus paranasales*) and surrounding structures.



Picture. 20. Examination of the nasopharynx with a finger

Stage IV - endoscopic examination of the nasal cavity

Endoscopic examination (*endoscopia nasi*) is regarded as the most informative contemporary diagnostic method for evaluating the nasal cavity (*cavum nasi*). This technique employs both rigid and flexible endoscopes with varying angles of view, ranging from 0° to 90°, enabling visualization of the most anatomically challenging regions of the nasal cavity (Pic. 21). The adoption of this advanced technology has substantially enhanced the diagnostic capabilities for detecting and assessing diseases of the nose (*nasus*) and nasal passages.



Picture. 21. Endoscopic examination of the nasal cavity

Stage V - study of respiratory and olfactory functions of the nose

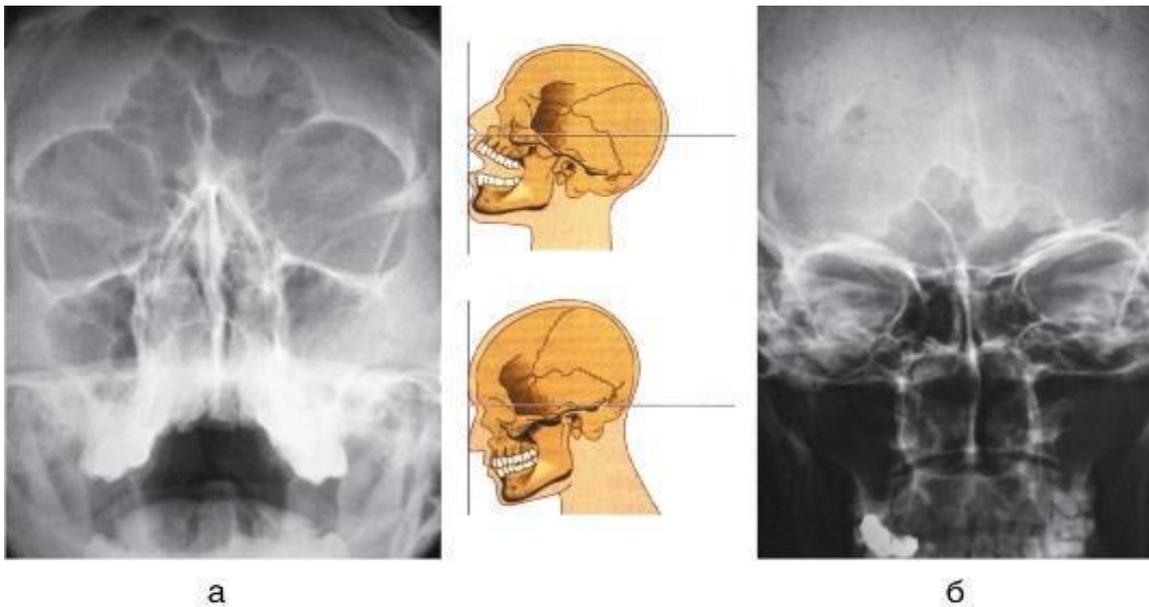
A straightforward approach to assessing nasal airway patency (*permeabilitas cavum nasi*) involves the use of a cotton swab (*cotonis applicator*), which is alternately placed at each nostril (*narina*). The patient is instructed to inhale and exhale, and the ease or difficulty of airflow is observed. Based on these observations, nasal breathing may be classified as normal, obstructed, or absent on one or both sides.

A more advanced method for evaluating nasal airflow is rhinomanometry (*rhinomanometria*), which utilizes specialized instrumentation to quantify resistance and airflow through the nasal passages. Assessment of olfactory function (*functio olfactoria*) can be conducted using an olfactometric set (*apparatus olfactorius*), in which odorants are delivered alternately to each half of the nasal cavity. Typically, substances of increasing concentration—such as *alcoholum vinylicum*, *tinctura valerianae*, *solutio acida aceticum*, or *ammonia*—are used. Olfactometers (*olfactometra*) enable precise dosing of odorants, allowing determination of the detection threshold and overall olfactory sensitivity.

Stage VI – radiography

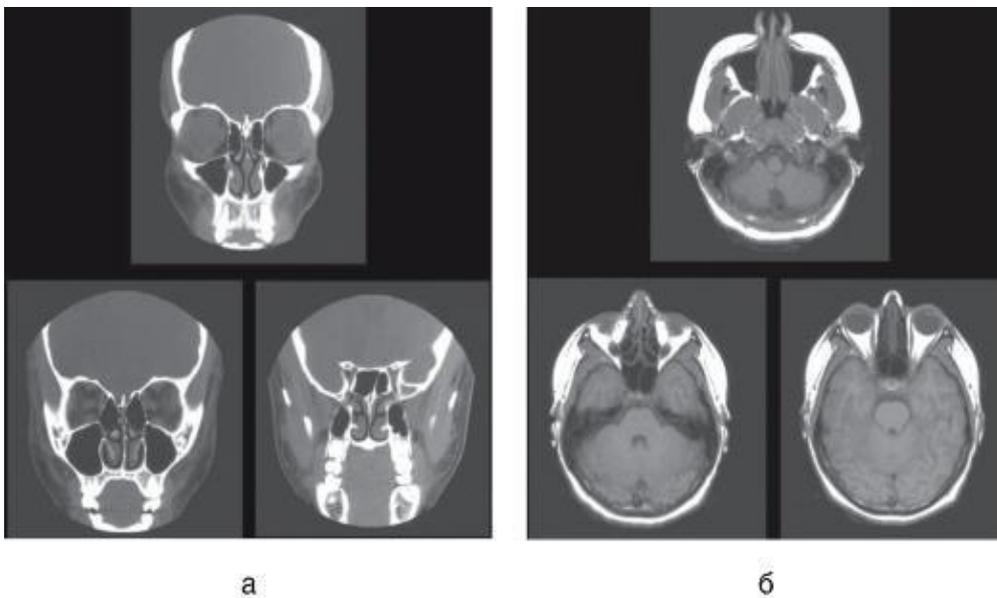
Radiographic examination (*radiographia cavum nasi*) provides an objective assessment of the condition of the nasal and paranasal cavities (*sinus paranasales*). Multiple projections are employed to obtain a comprehensive view. The most commonly used and diagnostically informative projection is the nose–chin view (*proiectio naso–mentalis*), which visualizes all maxillary sinuses (*sinus maxillares*), while the frontal sinuses (*sinus frontales*) are best evaluated using the nose–frontal projection (*proiectio naso–frontalis*) (Pic. 22).

The evaluation of sinus health is based on the degree of radiolucency. Sinuses that exhibit a darkening comparable to that of the eyeball (*bulbus oculi*) are considered normal, with intact pneumatization. Conversely, in pathological conditions, the sinuses appear more opaque or “blackened,” indicating disrupted pneumatization and potential disease processes within the cavities.

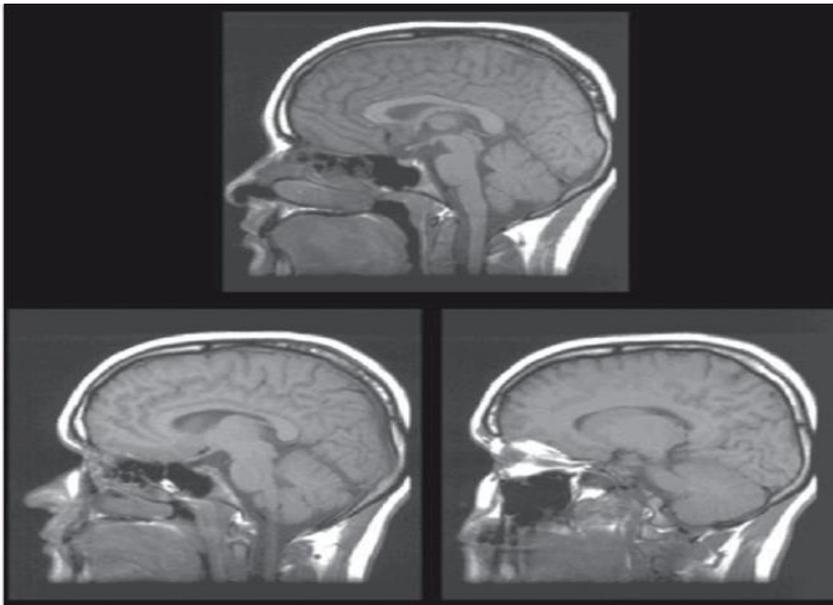


Picture.22. X-ray of nasal cavity: a – nose-chin projection; b - nose-frontal projection

In the last two decades, more advanced methods of research - high-resolution computed tomography (CT) and magnetic resonance imaging (MRT) have appeared. Some time later, these methods completely replaced traditional radiography (Pic. 23).



Picture. 23. Computed tomogram of the paranasal sinuses: a – coronary projection; b - axial projection



Picture. 24. Magnetic resonance imaging of nasal and paranasal cavities

CHAPTER 4 DISEASES OF THE NOSE. DISEASES OF THE EXTERNAL NOSE. ANOMALIES IN NOSE DEVELOPMENT

Developmental anomalies of the nose (*anomaliae nasus*) frequently occur in conjunction with malformations of adjacent structures, particularly the maxillofacial system (*systema maxillofaciale*). A common congenital deformity is the cleft of the hard and soft palate (*schisis palati duri et molle*), which is often associated with a cleft of the upper lip (*labium superius fissum*) (Pic. 25). This congenital condition is typically accompanied by malformations of the external nose (*nasus externus*) and the nasal cavity (*cavum nasi*). The extent of external nasal deformity correlates with the cleft of the lip and palate and is more pronounced in unilateral clefts. In such cases, the affected alar wing (*ala nasi*) is wider, and asymmetry of the external nose is observed. Corresponding changes in the nasal cavity include deviations of the nasal septum (*septum nasi*), primarily in its anterior portion.



Picture. 25. *Congenital cleft of the upper lip, hard and soft palate*

In patients presenting with such congenital anomalies, early surgical intervention (*interventio chirurgica infantia*) is indicated, specifically cheilouranoplasty (*cheilouranoplastica*). This procedure not only restores the separation between the nasal cavity (*cavum nasi*) and oral cavity (*cavum oris*), but also corrects defects of the upper lip (*labium superius fissum*) and reconstructs the nostril (*narina*) morphology. Simultaneously, it helps prevent a range of potential complications involving the nasal passages (*cavum nasi*), nasal cavity, and middle ear (*auris media*). Delay in surgical correction reduces the likelihood of achieving optimal cosmetic and functional outcomes, as the complexity of reconstructive procedures increases with the age of the child.

Nasal congestion

The cutaneous response of the nose (*cutis nasi*) to cold exposure can be classified into three levels: **I – erythema**, **II – blister and scab formation**, and **III – gangrene**.

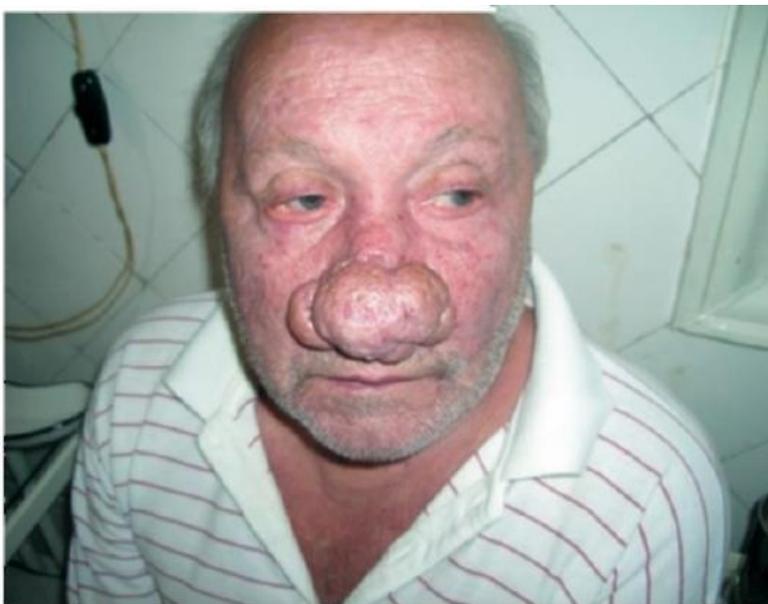
At level I, prolonged vasospasm of the blood vessels in the tip (*apex nasi*) and alar wings (*alae nasi*) leads to sudden pallor of the skin, as occurs in first-degree frostbite. Sensory perception is disturbed, and discomfort may not be immediately felt. Subsequently, the affected area develops erythema, swelling, pruritus, and pain.

At level II, the skin acquires a bluish-red discoloration, and vesicles appear on its surface, which rupture and form crusts. Following the removal of these crusts, epidermal regeneration (*epidermizatio*) occurs.

Level III is the most severe and rare. Tissue necrosis may develop, with the potential for detachment upon minimal manipulation.

Treatment is based on the gradual rewarming of affected tissues. Initially, the nose is gently rubbed with a soft cloth and treated with warm compresses, taking care to avoid damage to the epidermis or secondary infection. After vesicle debridement, the application of ointment dressings promotes *epidermizatio* and prevents infection.

Rhinophyma (*rhinophyma*) is characterized by tumor-like growths of hypertrophied connective tissue, blood vessels, and sebaceous glands in the tip (*apex nasi*) and alar wings (*alae nasi*). Rhinophyma not only alters facial aesthetics but can significantly obstruct nasal airflow due to nodular hypertrophy. This condition is most frequently observed in older males, and surgical correction (*chirurgia plastica*) is indicated for functional and cosmetic rehabilitation (Pic. 26).



Pic. 26.*Rhinophyma*

Nasal furuncle (*furunculus nasi*) is a purulent inflammatory lesion of the hair follicles (*folliculi pili*), typically caused by *Staphylococcus* species. Although the lesion originates at the entrance of the nasal cavity

(*vestibulum nasi*), its clinical manifestations are most prominently observed on the external nose (*nasus externus*). Initially, the lateral surface of the alar wing (*ala nasi*) becomes erythematous. Subsequently, a cone-shaped, reddish elevation appears within the nasal vestibule, accompanied by localized tissue swelling in the surrounding area (Pic. 27).



Picture. 27. *Nasal furuncle*

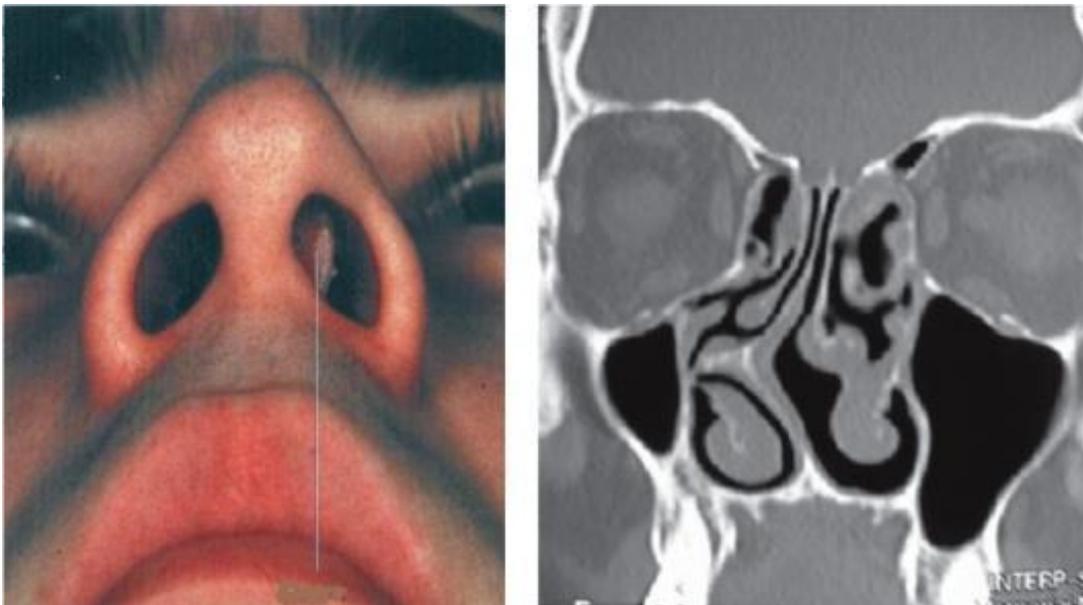
After 3–4 days, the *furunculus* typically ruptures, releasing thick purulent material from its cavity. A residual depression on the *ala nasi* often remains due to necrosis of the surrounding tissue around the *folliculus pili*. The maturation of the *furunculus* is accompanied by significant tension in the tissues of the *ala* and tip of the *nasus*, resulting in sharp pain.

Treatment is determined by the stage of the disease—either infiltrative (*infiltrativus*) or abscessed (*abscessus*)—and the severity of symptoms. Management can be conservative or surgical. Conservative therapy is indicated during the infiltrative stage, when abscess formation is not evident, and involves the administration of broad-spectrum antibiotics (e.g., *penicillinae*, *cephalosporinae*, *macrolida*, *fluoroquinolona*), antihistamines, analgesics, vitamins, and antipyretics. At this early stage, topical application of ointments may also be employed. In severe cases, when there is a risk of *carbunculus* formation or intracranial complications, antibiotic dosage may be increased and anticoagulant therapy added.

Surgical intervention is indicated during the abscess stage. The *furunculus* is incised, purulent-necrotic tissue is removed, and the cavity is drained to prevent further complications.

Diseases of the nasal cavity - nasal septum curvature.

As mentioned earlier, the **cavum nasi** is separated by a **partition**, generally creating two asymmetrical compartments referred to as the **nasal hemicavities**. Frequently, especially in male individuals, **deviations of the septum nasi** are observed, often taking the form of **spurs or ridges**. Such **anatomical variations** can markedly **narrow the meatus nasi communis**, thereby disrupting normal nasal airflow and resulting in **impaired nasal breathing** (Pic. 28).



Picture. 28. Obliqueness of the nasal septum

In most instances, **minor deviations of the septum nasi** do not substantially compromise nasal airflow. Only when a patient reports significant **difficulty breathing** through one or both **nasal hemicavities** should the septal deviation be regarded as **pathological** and potentially warrant **surgical intervention**. In neonates, **septal deviations** are exceedingly uncommon; thus, most curvatures arise during the **development of the facial skeleton** or secondary to **external factors**, such as trauma, compression from a hypertrophied **concha nasi**, tumor growth, or other mechanical influences.

The **extent of septum nasi deviation** can vary considerably: it may present as a pronounced displacement forming an **S-shaped**

configuration contacting the **nasal conchae**, be restricted to the upper segment, or involve the **osseous portion** of the septum. Clinically, such deviations can manifest as **unilateral nasal obstruction**, **chronic rhinitis**, **hyposmia** or **anosmia**, and occasionally **noisy nasal respiration**. Evaluation is typically conducted via **anterior rhinoscopy**, which reveals the degree of **septal displacement**, **narrowing of one hemicavity**, and **compensatory enlargement** of the contralateral side.

It is noteworthy that a **deviated septum** may coexist with a **cleft palate**. The primary **morphological variants** of septal deformities include **curvature**, **ridge**, and **spur** formations.

Treatment indications are primarily surgical and include:

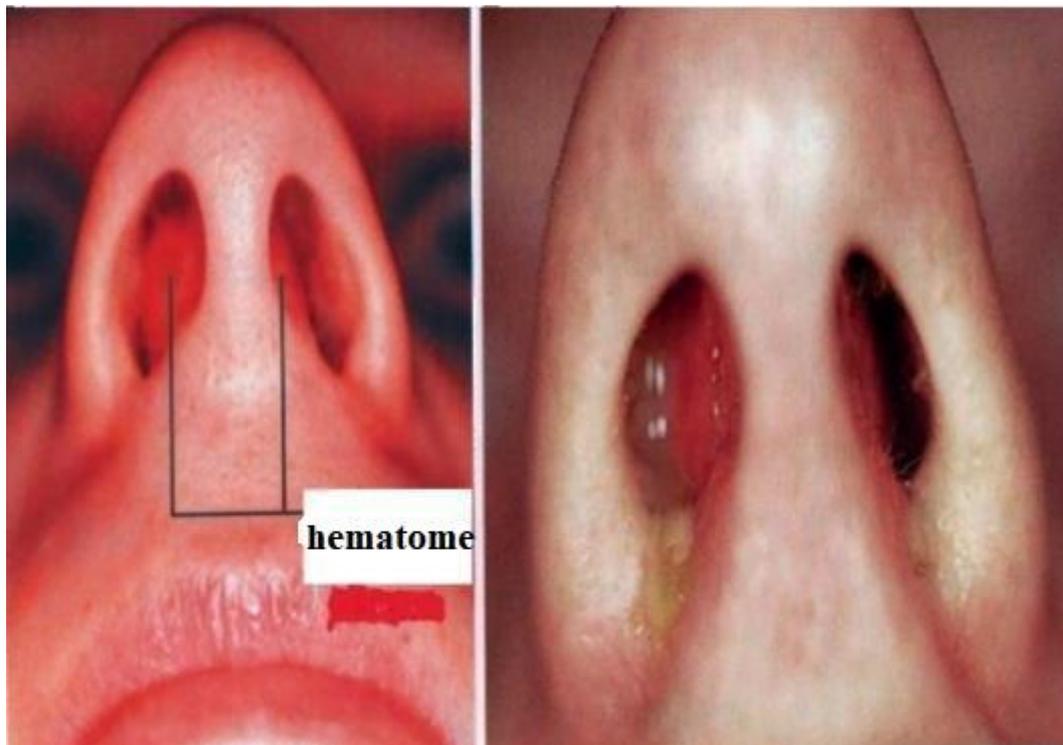
- **Marked obstruction** or inability to breathe effectively through the nasal passages;
- **Hindrance to intranasal procedures**, such as polyp excision, ear catheterization, or **endonasal access** to the **maxillary sinus**.

The surgical correction is termed submucosal resection of the *septum nasi* (*septoplasty*). It is usually performed under local anesthesia. The procedure involves incision of the mucosa on both sides of the deviated portion, elevation of the mucoperichondrial and mucoperiosteal flaps, and removal of the deviated cartilage or bony portions, such as the *lamina perpendicularis* or the *vomer*. The remaining septal components are then repositioned along the midline, and the mucosal flaps are approximated using tampons. To prevent postoperative septal perforation, excised cartilage fragments are often reimplanted in their original location. Following a successful procedure, normal nasal airflow is restored.

Hematoma and Abscess of the Nasal Septum

These complications typically arise after trauma to the external nose, often associated with fracture of the nasal bones. Blood accumulation separates the epithelium from the underlying mucosa on one or both sides of the *septum nasi*, forming a *septal hematoma*. Clinical signs include nasal obstruction and altered voice resonance. If

the hematoma becomes infected, it can progress to an abscess, which may exacerbate symptoms with headache, fever, and malaise (Pic. 29).



Picture. 29. *Nasal septum hematoma and abscess*

Treatment. Early intervention is crucial. Initial management involves prompt puncture and thorough opening of the *septum nasi* hematoma cavity, with drainage established on both sides. Anti-inflammatory therapy should be prescribed. Both *hemi-cavities* of the nose must be firmly packed with tampons to compress the separated mucosa against the underlying *cartilago quadrangularis* and the adjacent septal structures.

In cases where an abscess has formed, wide surgical opening of the abscess cavity is required. If a significant period has elapsed since abscess formation, the patient should be warned that dissolution of the *cartilago quadrangularis* may lead to posterior nasal collapse. The abscess cavity must be evacuated, its contents aspirated, and systemic antibacterial therapy administered as indicated. Hospitalization of the patient is mandatory for close monitoring and further management.

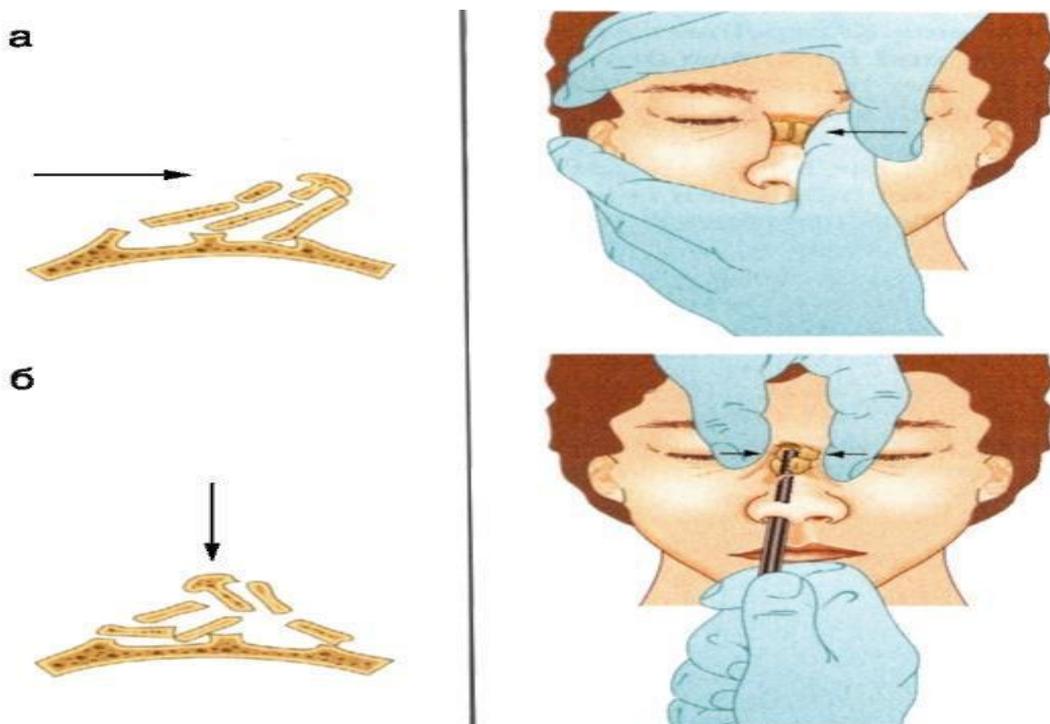
Fracture of nasal bones

Fractures of the nose can be classified as **open fractures**, with disruption of the skin integrity, and **closed fractures**, which occur without displacement. In addition to the *ossa nasalia*, trauma can also affect the *frontales* of the *maxilla (os maxillare)*. Depending on the direction of impact—lateral or frontal—the resulting deformation of the external nose varies (Pic. 30).

In some cases, the lateral slope of the nose corresponds to the side of impact, whereas in others, the opposite slope is displaced. A frontal impact may result in depression of the *dorsum nasi*, giving a “flattened” appearance.

Management depends on the general condition of the patient and the type of fracture. Care may be provided on an outpatient basis, or hospitalization may be required. **Clinical assessment** includes inspection, palpation of the *dorsum* and lateral walls of the nose, and *anterior rhinoscopy* to determine the fracture characteristics. Lateral radiography of the *ossa nasalia* is recommended.

In cases of **open fractures**, primary surgical treatment of the wound is required, along with administration of **anti-tetanus serum** to prevent tetanus infection.



Picture: 30. Fracture of the nasal bones: a - lateral displacement of the bone pieces, which requires repositioning of the finger; b - crushed fracture requiring instrumental reposition

The optimal time for repositioning the *ossa nasalia* is 1 day after the injury or 5–6 days later. This timing accounts for the development of edema in the surrounding soft tissues, which can make it difficult to assess whether the repositioned fragments are correctly aligned or whether contusions are present.

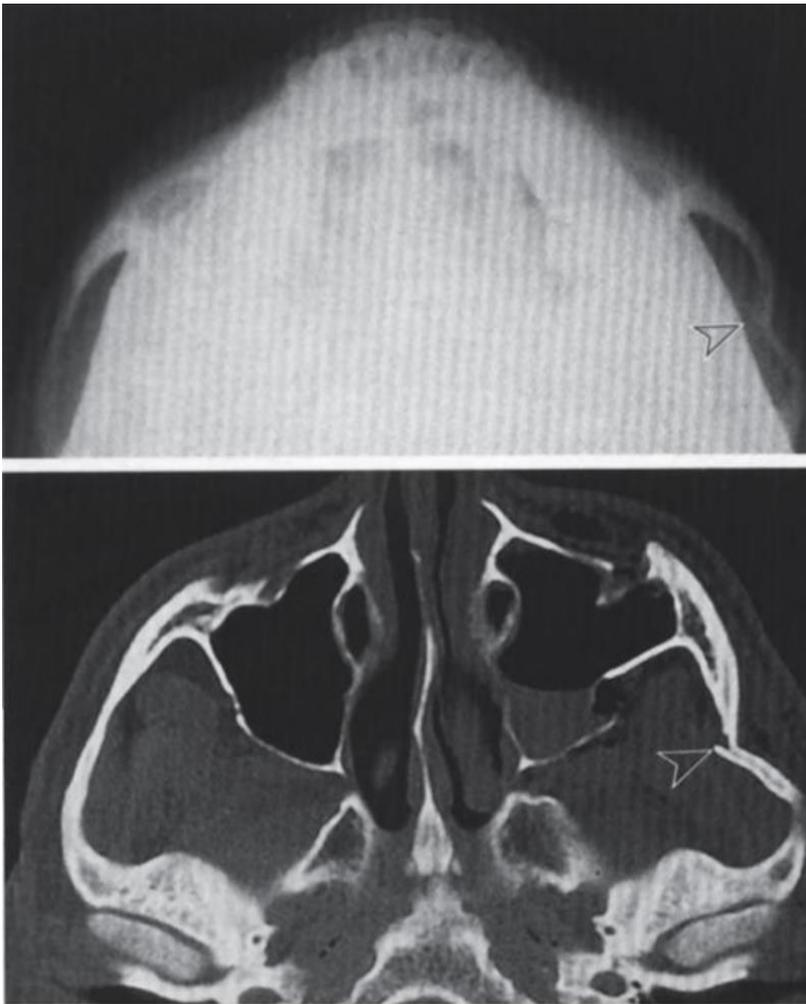
Before repositioning, local anesthesia is administered. A 2% solution of procaine (novocaine) or articaine (ultracaine) is injected subcutaneously near the *vena nasalis*, approximately 2.0 ml at the *radix nasi*. The *mucosa nasi* is anesthetized using a 10% lidocaine solution applied by spray or local application.

Fracture correction is performed with the patient lying down. Sunken fragments are elevated using a tool inserted into the *meatus nasi communis*; a Kocher clamp is often used, with a piece of rubber tubing placed on its tips to avoid damaging the soft tissues. Specialized Y. N. Volkov elevators, shaped to match the contour of the nasal cavity, may also be used (see Pic. 4.7).

Protruding parts of the lateral nasal wall are stabilized with the thumbs of both hands, placed over the patient's face, which allows the necessary force to reposition the bone fragments.

Immobilization of the bony fragments is typically achieved by firm tamponade of the nasal cavity with gauze pads after the swelling has subsided.

Fractures of the *ossa nasalia*, *paranasal sinus walls*, and *maxilla (os maxillare)* are often combined injuries (Pic. 31).



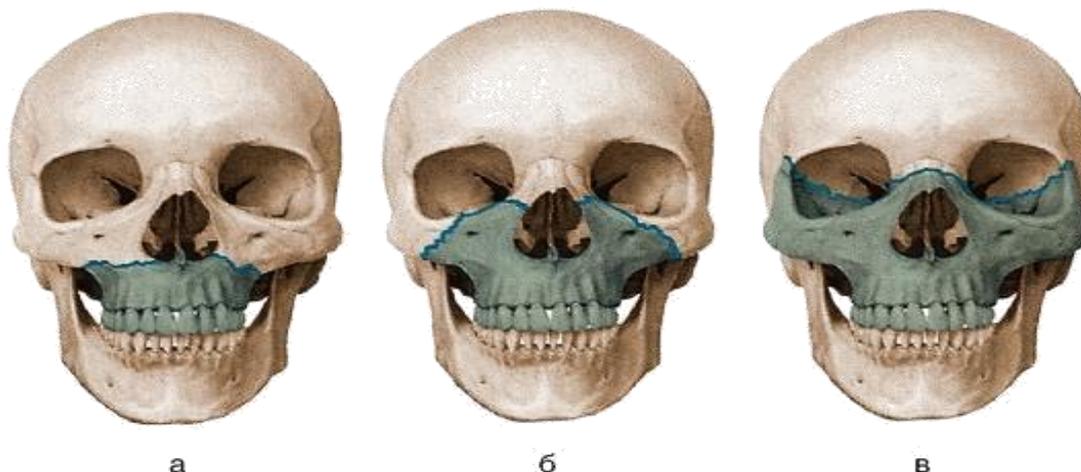
Picture.31. *Fractures of the upper jaw bone, upper jaw cavity and cheek bone.*

Currently, all authors in maxillofacial surgery use the term “midface fracture” (*fractura mediae faciei*). This category includes the *ossa nasalia*, the walls of the *orbita*, the *ossa zygomatica*, and the *maxilla*. The nature, direction, and force of the trauma determine the pattern of fractures in the *maxilla*, *zygoma*, and *orbital walls*.

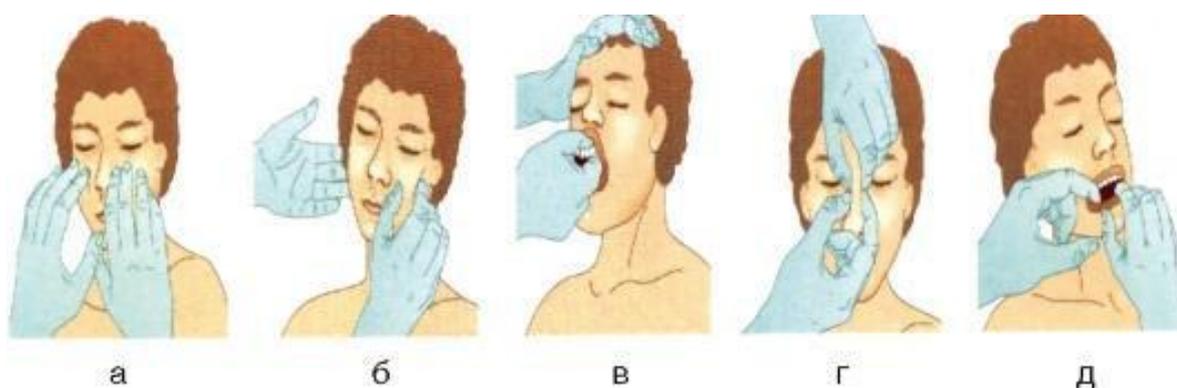
In some cases, damage to the facial bones extends to the anterior and middle cranial fossae (*fossa cranii anterior et media*). Fractures of the *maxilla* account for 2–5% of all facial bone fractures (Pic. 32).

The L. Le Fort classification remains the most widely used system for describing these injuries. The bones of the middle face contribute to the formation of the *orbita*, and therefore fractures in this region often involve damage to the orbital walls and the *bulbus oculi*.

Fractures of the middle facial bones are complex and highly variable, requiring an individualized treatment approach for each patient.



Picture. 32. Typical lines of fractures of the upper jaw.: a - Lefor I; b - Lefor II; c - Lefort III



Picture. 33. Palpation in fractures of the middle area of the face: a - orbits; b - cheek bone; v – upper jaw bone; g - nose; d – lower jaw

Bleeding from the nose

Epistaxis (nosebleed) can occur in patients of any age and may be encountered by doctors of any specialty. It can be caused by local or systemic factors and may be traumatic (local) or symptomatic (local or systemic). Bleeding can occur spontaneously or under the influence of objectively determined causes.

1. General/systemic causes:
 - 1.1. Cardiovascular diseases: atherosclerosis, uncontrolled hypertension
 - 1.2. Blood disorders: thrombocytopenic purpura, hemophilia
 - 1.3. Congenital vascular anomalies: Osler-Weber-Rendu syndrome

(hereditary telangiectasia causing weak arterial walls)

1.4. Infections: influenza or other febrile illnesses increase vascular permeability and impair blood coagulation

2. Local causes:

2.1. Trauma to the nasal mucosa

2.2. Atrophic processes of the mucous membrane

2.3. Vascular formations of the nasal cavity and nasopharynx

In most cases (about 80%), bleeding occurs from Kisselbach's area, located on the anterior part of the nasal septum. Bleeding from the posterior and superior parts of the nasal cavity is usually more abundant, as the vessels there are larger.

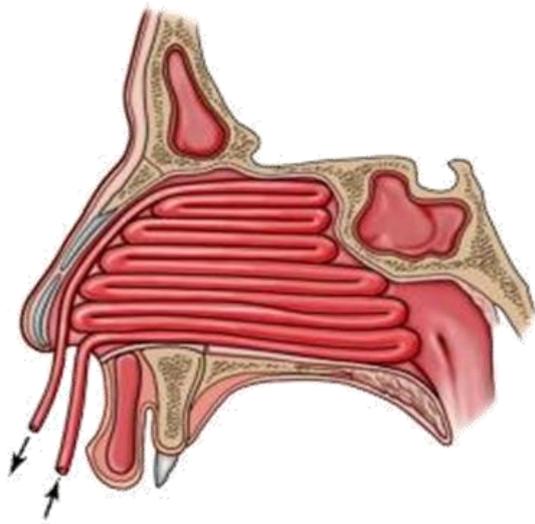
Clinical management

Mild anterior bleeding. If bleeding is not severe and localized to the front, it is usually sufficient to compress the wings of the nose against the septum for 15–20 minutes.

Bleeding from a visible vessel. If the source of bleeding is identified (e.g., in Kisselbach's area), a cotton swab soaked in an adrenaline or ephedrine solution is inserted into the nasal passage, and the nasal wing is pressed. If the bleeding vessel is visible on rhinoscopy, it can be cauterized using electrocautery, crystalline silver nitrate, or electrocoagulation. Local anesthesia should be applied beforehand.

Severe or persistent bleeding. An anterior nasal tamponade is performed according to proper procedure. Required tools include nasal dilator forceps, a gauze swab, and a forehead reflector. The patient should be seated. The clinician lowers the tampon 4–5 cm from the edge, grasps it with forceps 40–50 cm long, and places loops starting from the lower part of the nasal cavity, pressing them tightly together.

Proper tamponade prevents significant blood loss, reduces painful repeated interventions, and stabilizes the patient's condition.



Picture. 34. *Stop nosebleeds with anterior tamponade*

So that the tip of the tampon does not fall into the nasopharynx, does not irritate its mucous membrane, and does not trigger vomiting, the tip of the tampon should be left outside the patient's nostril. It should be remembered that bleeding from a previous tamponade can be stopped only if the tampon is firmly fixed. In such cases, the tampon is considered less hygroscopic and does not absorb blood effectively. The tampon can remain in the nasal cavity for up to 48 hours. It is recommended to moisten the tampon with petroleum jelly to prevent damage to the mucous membrane when removing a dry gauze.

Posterior tamponade is a complex procedure, used not only for severe nosebleeds arising from the previously mentioned causes, but also in cases where surgical procedures themselves involve significant blood loss, such as removal of neoplasms in the nasal cavity or larynx. It is often applied at the end of a series of surgical interventions in the nose.

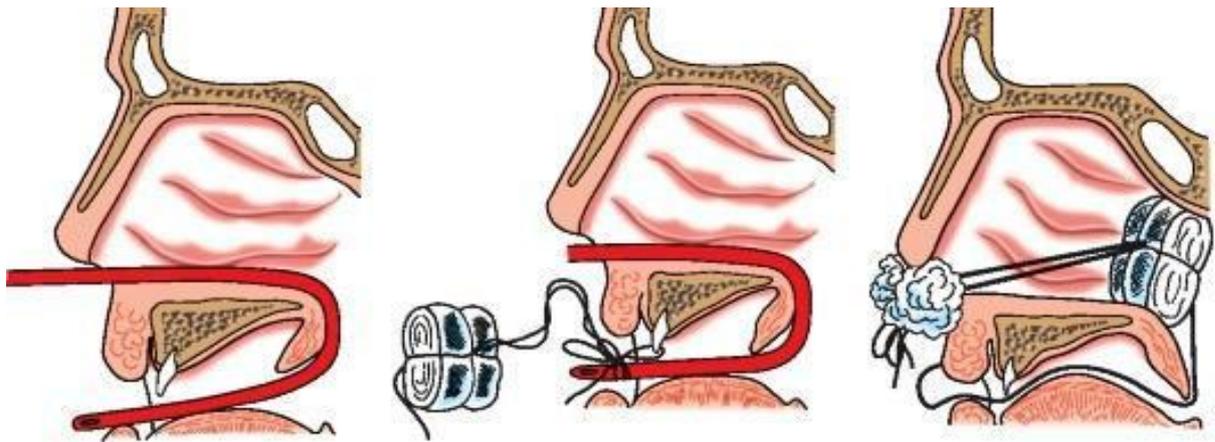
Posterior tamponade consists of several stages. The size of the patient's nasopharynx is determined to select an appropriate tampon. Usually, the volume of the nasopharyngeal cavity is compared with the phalanges of the patient's thumbs. The strength of the threads used to pull the tampon, as well as the strength of the tampon itself, is checked.

In cases of persistent bleeding, a thin rubber catheter is passed halfway along the inferior nasal passage on the affected side until it emerges behind the soft palate, which is clearly visible when the patient

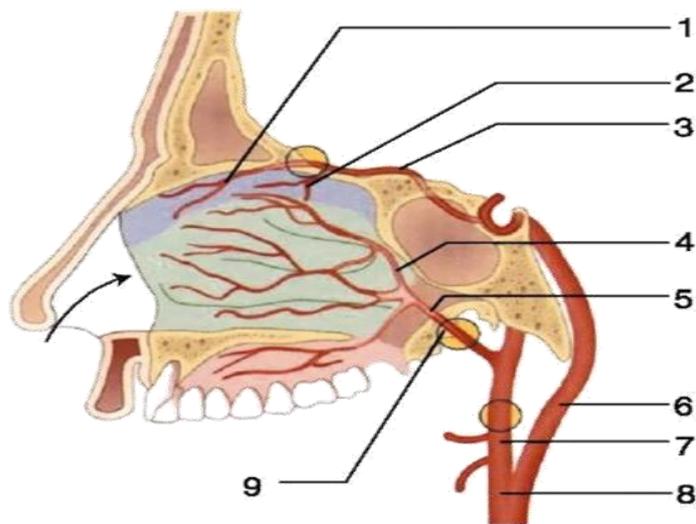
opens their mouth. The tip of this catheter is grasped with forceps and pulled out through the mouth by 4–5 cm. Two threads used to pull the tampon are tightly tied to the end of the rubber catheter, with each thread at least 20 cm long.

The catheter is then withdrawn through the mouth, bringing the attached threads with it. If the threads are shorter than 20 cm, they will be insufficient to securely fix the tampon. A gauze pad of the same density is then inserted into the other side of the nose. Using the left hand, the tampon is raised and inserted into the nasopharyngeal cavity, while the index finger of the right hand presses it into position, tightly against the larynx and nose. Before tying the knot in front of the nasal passage, an anterior tamponade may sometimes be applied first.

In most cases, posterior tamponade effectively stops severe nosebleeds. To prevent serious complications involving the middle ear, the tampon should remain in the nasopharynx for no more than 48 hours, as blood-soaked tampons can transmit infection through the auditory tube and promote the growth of pathogenic flora. The tampon is removed from the nasopharynx by pulling the third thread, which is brought out through the oral cavity and secured to the cheek with adhesive tape. Before reinserting it into the nasal cavity, the posterior tampon must be cut, and the anterior tampon is removed from the nasal cavity.



Picture. 35. On the back bumper



Picture:36. *Places of embolization of external carotid artery and adductor veins to stop nosebleeds: 1 - anterior glomerular artery; 2 - posterior iliac artery; 3 - anterior meningeal artery; 4 – pterygoid-facial artery; 5 – maxillary artery; 6 - internal carotid artery; 7 - external carotid artery; 8 - common carotid artery; 9 - the place of embolization of the maxillary artery.*

Ligation of the external carotid artery. In cases where posterior tamponade cannot stop nosebleeds, it may be necessary to perform ligation of the external carotid artery. Ligation of the external carotid artery is a serious surgical procedure, and its indications must be clearly defined. For practical purposes, it is important to know the topography of this area, including the relative locations of blood vessels, nerves, muscles, and fascia.

First, it should be noted that the division of the common carotid artery into external and internal carotid arteries occurs at the level of the upper edge of the thyroid gland. This serves as an important landmark for making a skin incision, starting from the junction with the sternocleidomastoid muscle near the mastoid process. To better identify the anterior edge of the sternocleidomastoid muscle, the patient's head is tilted back and sharply turned to the opposite side.

An incision about 6–7 cm long is made along the anterior edge of the muscle. After cutting the skin and subcutaneous fat, the anterior edge of the sternocleidomastoid, wrapped in a fascial sheath, is exposed. The fascial sheath is cut along the anterior edge of the muscle throughout the length of the incision. The sternocleidomastoid muscle is then pushed back, revealing the inner sheet of the muscle sheath, which is divided

along the incision line where the large jugular vein is located. After moving the jugular vein aside, both carotid arteries are visible behind it: the external carotid artery is deeper, while the internal carotid artery is more superficial.

The external carotid artery is identified by the fact that it gives off branches to the neck region, which distinguishes it from the internal carotid artery. Ligation is performed above the point where the first branch, the superior thyroid artery, separates.

A modern method for stopping severe nosebleeds is embolization of the blood vessels. Angiography is performed, and under the guidance of an electronic-optical converter, the bleeding vessel is embolized through a special catheter.

Foreign bodies of the nasal cavity.

A variety of **extraneous objects**, including **ectopic teeth**, can occasionally be found within the nasal cavity. When these objects become lodged in the **common or inferior nasal passages**, they may provoke a **chronic inflammatory response**, sometimes mimicking serious systemic illnesses. For instance, **living foreign bodies adhering to the mucosa** can lead to persistent **epistaxis**, which may be misattributed to conditions such as **tuberculosis or neoplastic processes**.

Smaller retained objects, over time, may undergo **mineral deposition on the nasal mucosa**, eventually giving rise to the formation of **nasal calculi (rhinoliths)** (Pic. 37). They also cause purulent forms of chronic rhinitis.



Picture. 37. X-ray of a foreign body in the nasal cavity (rhinolith) and its condition after removal.

The diagnosis of foreign bodies is based on the data of anterior and posterior rhinoscopy performed with the use of vasoconstrictor drops and X-rays to fully visualize different parts of the nasal cavity.

Living foreign bodies are removed by injecting a concentrated sodium chloride solution into the nasal cavity, and inanimate (local anesthesia) foreign bodies are removed using special hooks so that the upper part of the trachea does not move into the nasopharynx.

Rhinitis

Rhinitis is an acute or chronic inflammation of the nasal mucosa, caused by environmental factors with pathogenic or influencing properties (microorganisms, allergens, thermal, mechanical, and other effects), or it may develop in the presence of certain diseases in the body. It is an infectious disease. Under the influence of these factors, the inflammatory process manifests as swelling of the mucous membrane, hypersecretion, an increase in connective tissue or epithelial metaplasia, as well as atrophic changes in the mucous membrane. This leads to the following symptoms: difficulty breathing through the nose, discharge, itching in the nasal cavity, paroxysms, dryness in the nose, crusting, and impaired sense of smell.

There are many classifications of rhinitis, but no universally accepted system exists. The latest proposed etiopathogenetic classification of rhinitis is presented below.

Classification of rhinitis

I. Infectious:

- Acute:
- viral
- bacterial
- Chronic:
- nonspecific
- specific

II. Allergic:

- intermittent (seasonal)

- persistent (year-round)

III. Vasomotor:

- medication-induced
- hormonal
- food-related
- cold-induced
- psychogenic
- idiopathic

IV. Hypertrophic

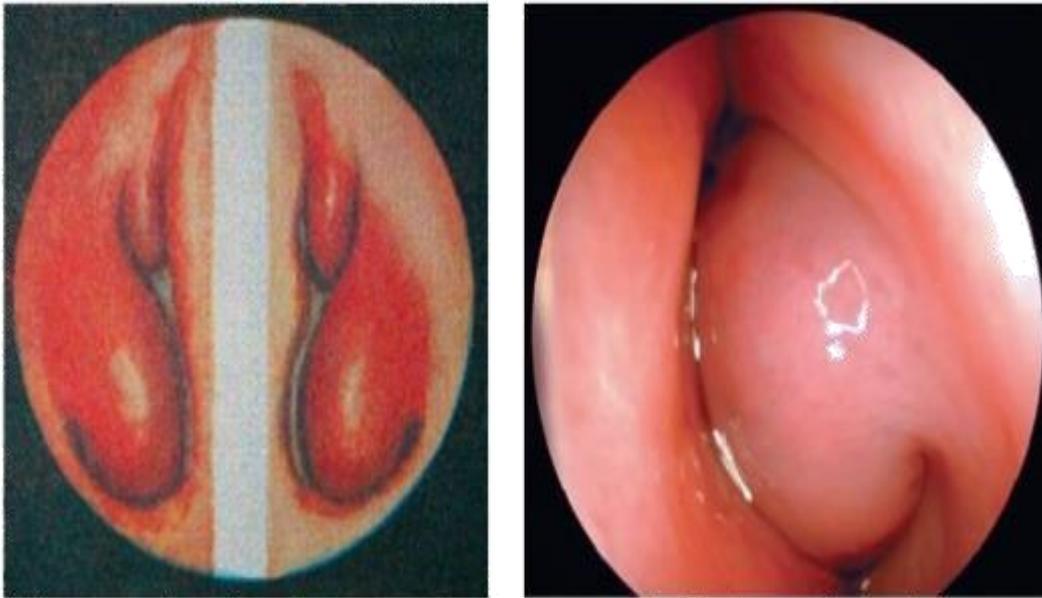
V. Atrophic:

- "empty nose" syndrome
- ozena

Acute rhinitis is the most common disease that every person experiences several times in their life. Acute rhinitis can occur as an independent pathological process in the nasal cavity, or it can be accompanied by a cold or many other infectious diseases. The cause of acute rhinitis is often exposure to cold in various parts of the body, frequently located far from the nasal cavity. For this reason, influenza is considered to be associated with reflex mechanisms that trigger a reaction of the nasal mucosa in response to cold exposure in certain body areas.

In most cases, acute rhinitis appears immediately or shortly after cooling of the legs or lumbar region. Thus, the cold factor serves as a trigger for the onset of acute rhinitis. At the beginning of the disease, a serous fluid is released from the nose, which, upon examination, is confirmed to be a transudate completely free of protein components. This is considered the first stage of acute rhinitis.

With epithelial maceration and the appearance of small wound surfaces due to the large volume of nasal discharge, the body activates protective mechanisms, including leukocytes and macrophages. As a result, nasal secretions change from a serous to a purulent appearance. Therefore, based on clinical presentation and morphological features, acute rhinitis is divided into three stages or phases.



Picture. 38. Acute rhinitis

Stage 1 (initial dry phase) generally persists for a few hours and, in rare cases, up to one or two days. Patients typically report **nasal dryness, burning sensations, warmth, and discomfort during breathing**, often accompanied by a general feeling of malaise. Examination of the frontal nasal cavity reveals **hyperemia of the mucosa, pronounced dryness, and absence of secretions** on the surface (Pic. 38).

Stage 2 (serous secretion phase) begins approximately one to two hours after the onset of the first stage. It is characterized by **nasal obstruction, headache, copious watery discharge, sneezing, generalized weakness, and a slight rise in body temperature**.

Stage 3 (mucopurulent phase) may persist from several days to multiple weeks. During this phase, **nasal blockage continues, secretions thicken and become purulent**, and the patient experiences ongoing malaise and headache.

Management. Most cases are treated on an outpatient basis. Hospitalization is considered when **fever is significant or the patient's general condition is compromised**. At the onset of the condition, **antipyretic therapy and supportive measures** are recommended, including **even body warming, rest, and administration of anti-inflammatory agents** such as acetylsalicylic acid. Additional supportive interventions—like **warming foot baths or mustard compresses**

applied to muscles—may also be employed. Nasal drops containing **menthol or preservative oils** are advised to protect the mucosa from excessive environmental exposure and to facilitate nasal airflow.

Topical vasoconstrictors (e.g., naphazoline, naftizin, sanorin, xylometazoline variants such as galazolin, thizin xylo, ximelin, or otrivin) may be used, but **only briefly and in controlled doses**, to avoid development of tolerance.

In the **mucopurulent stage**, treatment can include **sulfonamide powders and ointments applied locally**. Additionally, **antihistamines or hyposensitizing agents**—such as loratadine (Claritin), desloratadine (Erius), or fexofenadine (Telfast)—may be prescribed.

It is important to recognize that **acute rhinitis should not be underestimated**, as potential complications may extend beyond the nasal cavity and paranasal sinuses to involve adjacent structures, including the **middle ear**.

The clinical course may culminate in **complete recovery** or progress to a **subacute phase**, which in some cases transitions into a **chronic condition**, displaying pathological features distinct from the original acute catarrhal inflammation.

Acute rhinitis in infectious diseases (measles, scarlet fever, diphtheria)

In these conditions, **secondary acute rhinitis** commonly develops and exhibits certain distinctive characteristics, particularly in **diseases with toxin-producing bacteria**, where careful differentiation of symptoms is essential to prevent severe outcomes.

Measles. During the early prodromal stage, nasal inflammation often signals the onset of the illness, so clinicians should maintain high vigilance for any flu-like symptoms in pediatric patients. Fever associated with the disease is typically accompanied by abundant **mucous nasal discharge**. Examination of the nasal passages frequently reveals **small red spots on the lower turbinate**, appearing against a background of generalized mucosal hyperemia. These lesions are transient, present only during the prodromal phase, and therefore serve

as an important diagnostic clue. Management focuses on **clearing nasal secretions** effectively. Patients should be instructed on proper techniques for nasal evacuation to prevent retrograde movement of secretions into the middle ear and avoid **mucosal injury**. The application of **oil-based preparations and levomenthol (1% menthol oil)** is recommended to soothe the mucosa.

Scarlet fever. In milder presentations, particularly in the era of **broad-spectrum antimicrobials**, the disease often manifests without notable nasal specificity and follows the conventional progression of **acute rhinitis** in three stages. The characteristic **maculopapular rash** first appears on the neck and face, then spreads over the trunk and limbs. Management is conducted in specialized infectious disease facilities, and specific nasal treatment is limited to standard supportive care. **Antibiotics** are administered as indicated, and **local nasal therapy** may be applied.

Diphtheria. In this condition, nasal inflammation can emerge as a primary manifestation or secondarily from laryngeal involvement, predominantly affecting **young children**. The early stage may present without the classic **mucosal pseudomembranes**, resembling a typical acute rhinitis. However, there are characteristic signs: **unilateral nasal discharge**, often **bloody**, **reddening of the skin over the upper nose and lip**, and **cracks or erosions** in these areas. Definitive diagnosis relies on **bacteriological confirmation** of the causative organism. Treatment consists of administration of **specific antitoxin**, **systemic antibiotics**, and **topical ointments** applied to eroded or fissured areas of the skin.

Chronic rhinitis

Chronic non-specific inflammation of the nasal mucosa is a widespread disease that affects all functions of the nasal cavity (respiratory, olfactory, protective, resonant). Currently, a convenient clinical classification is used, according to which several forms of chronic rhinitis are distinguished.

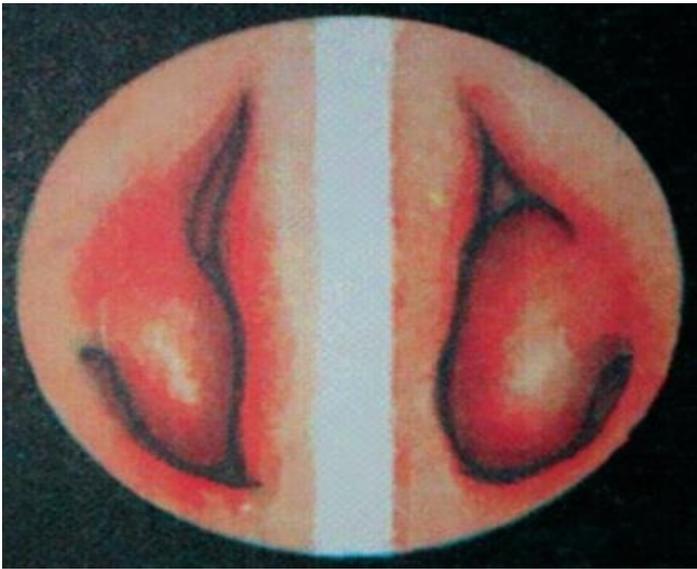
- Catarrhal rhinitis.
- Hypertrophic rhinitis: - limited; - scattered.
- Atrophic rhinitis:
 - Normal form;
 - Ozena (smelly tumov)

Each of the forms of chronic rhinitis has its own characteristics, which makes it possible to distinguish the diseases.

Chronic catarrhal rhinitis

Chronic catarrhal rhinitis (*rhinitis catarrhalis chronica*) Diffuse humid hyperemia of the mucous membrane is characterized by uniform swelling of the shells.

Symptoms. Constant or periodic congestion in both halves of the nose, which is especially noticeable when lying on the side and on the back. Congestion is accompanied by a large amount of mucus discharge from the nose. Fatigue, headache, dry mouth are observed.



Picture. 39. Chronic catarrhal rhinitis

Anamnesis. The disease occurs after acute catarrhal rhinitis for many reasons (irrational treatment, presence of adenoids). The duration of the disease is different - it can last from several months to many years and even decades.

Clinical appearance. The general condition is satisfactory, body temperature does not rise. Difficulty breathing through one or both halves of the nose. Anterior rhinoscopy reveals a hyperemic mucous membrane of the scrotum, the scrotum is enlarged due to swelling of the submucosa and dilated blood vessels in paresis (Pic. 39).

There is mucus in the common and lower nasal passages, which sometimes has a stretchy appearance. Characteristic symptoms of chronic catarrhal rhinitis are periodic swelling of one or the other half of the nose. Usually, patients use vasoconstrictor drops as a therapeutic tool for a long time and have a temporary effect.

Diagnostics. A test with epinephrine (adrenaline) is used to distinguish it from other forms of chronic rhinitis (true hypertrophy). If the patient has chronic catarrhal rhinitis, then in 2-3 minutes after rubbing the mucous membrane of the shells, their size will be significantly reduced and the nasal passages will expand, breathing will be restored.

Treatment. It is necessary to make sure that there are no adenoids in the nasal part of the larynx. If they are, then the treatment begins with the removal of adenoids. Only one local therapy is used - ointments that help to dry the mucous membrane, reduce secretion and reduce the size of the nasal cavity. Astringents are also used for local treatment: 3-5% colloidal silver solution (collargol), 1-3% solution of silver proteinate (protargol), application of 3-5% solution of silver nitrate to the mucous membrane; physiotherapy with ultra-high frequencies (UHF), ultraviolet radiation (UVR) is recommended.

Complications. They appear in the middle ear (otitis media), nasal cavities (sinusitis). In chronic catarrhal rhinitis, it helps to cause dental diseases (caries, gingivitis) due to nasal breathing disorders, drying of the mucous membrane, enamel nutrition disorders.

Chronic hypertrophic rhinitis(*rhinitis chronica hypertrophica*)It is characterized by hyperplasia of the mucous membrane of the shells, often the periosteum and bone tissue of the shells are involved in the process. Most often, the growth and thickening of the mucous membrane occurs in the lower shell, less often - in the middle part, in the areas of the location of cavernous tissues. Narrowing of the nasal passages due to changes in the size of the shells occurs mainly in the general condition (Pic. 39). This leads to difficulties in breathing and exhalation, the development of signs of dampness in the shells, and a large amount of mucus secretion.



Picture. 40. *Chronic hypertrophic rhinitis*

Symptoms are similar to chronic catarrhal rhinitis. A distinctive feature is the lack of reaction to the application of epinephrine (adrenaline) solution on the surface of the shells - there is no significant reduction in the size of the shells.

Treatment. In mild cases, the surface of the shells is smeared with anti-wrinkle preparations [collargol, silver proteinate (protargol)], and finally various types of surgical treatment are resorted to - ultrasonic dissection, submucosal vasotomy, cutting a certain part of the shells with special instruments removal - conchotomy. These operations can be combined with the lateral displacement of the shells to the lateral wall of the nose - lateroposition. All these measures are mainly aimed at reducing the size of the lower shells to ensure the unhindered passage of air through the nasal cavity. Currently, due to the introduction of endoscopic methods in rhinosinus surgery, the efficiency of intranasal operations has increased significantly. Endoscopes allow to perform all types of intranasal operations under constant visual control, including those performed in cases where vision is difficult in the back of the nasal cavity.

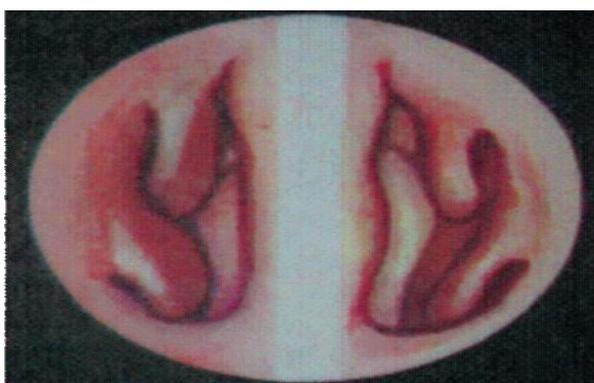
Chronic atrophic rhinitis

Chronic atrophic rhinitis (rhinitis atrophica) is a severe form of the disease of the mucous membrane of the nasal cavity, which can be accompanied by atrophic manifestations in the nasal part of the larynx, larynx and larynx. At the basis of the disease, there are changes in the mucous membrane of the nasal cavity caused by many reasons, these reasons include - congenital specificity of the mucous membrane, high level of air pollution (during the production of flour, cement) or

occupational harmful habits caused by the presence of certain substances (chemical in nature) that have a drying effect on the mucous membrane.

Symptoms. Severe dryness in the nose, the formation of crusts, difficulty in breathing through the nose, purulent discharge and impaired sense of smell can be caused.

Clinical appearance. Rhinoscopy reveals a characteristic picture - the mucous membrane is pale and thinned, the nasal passages are very wide, yellow sticky discharge accumulates on the surface of the crusts, forming large crusts (Pic. 41).



Picture. 41. *Chronic atrophic rhinitis*

Treatment. It is mainly symptomatic and focuses on moistening the mucous membrane of the nose and removing crusts. To remove crusts, the nasal cavity is regularly cleaned with isotonic sodium chloride solution 1-2 times a day, sea water (aqua maris, physiomer) and others are dripped. Medicinal products with vegetable oils and iodine are used for the same purpose (mineral drops cause further drying of the mucous membrane), they affect the mucous membrane and stimulate the secretion of the glands.

Iodine + [potassium iodide + glycerol] (Lugol) is used as a means of this medicine.

Drugs containing vitamin A are used as drugs that stimulate regenerative processes. Biogenic stimulants (aloe tree leaves, FIBS), vitamin therapy, and iron preparations are prescribed from the common agents.

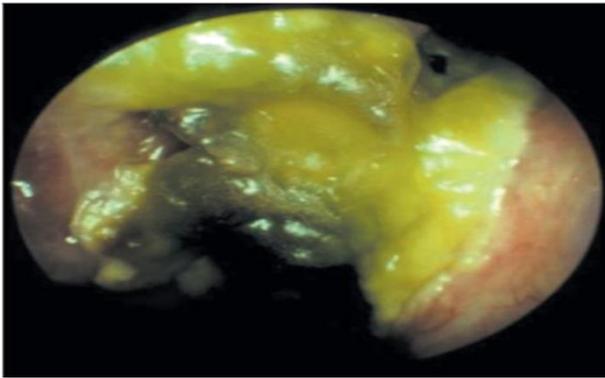
Ozena

A form of atrophic rhinitis (ozena). In modern times, this condition is encountered very rarely, largely due to improvements in general living standards.

Etiology. Multiple hypotheses have been proposed to explain its development, including **social, anatomical, inflammatory, infectious, neurodystrophic, and endocrine** factors. According to the **infectious theory**, the dominant role is attributed to the nasal microflora; in particular, *Klebsiella ozaenae* has been isolated in approximately 80% of cases.

The disease is characterized by progressive atrophy affecting not only the **nasal mucosa** but also the **bony structures of the turbinates**, leading to a marked widening of the nasal passages. This expansion can make the posterior nasopharyngeal wall visible through the nasal cavity. Despite this increased space, the nasal passages are frequently obstructed by large, greenish-yellow crusts with a pronounced malodor. Patients often fail to notice the odor themselves, as the extensive atrophic changes significantly impair their **olfactory function**.

Treatment. Management is primarily symptomatic and focuses on regular **nasal irrigation** with isotonic solutions or commercially available sea-water preparations (e.g., *Aqua Maris*, *Physiomer*), careful removal of crusts, and administration of **antibacterial agents** such as streptomycin, gentamicin, or chloramphenicol (levomycin), along with **iron supplementation**. Surgical interventions are also employed in certain cases, aiming to artificially narrow the widened nasal cavity and improve airflow and mucosal function.



Picture. 42. Ozena

Allergic rhinitis

Allergic rhinitis is one of the most common allergic diseases today. Allergic rhinitis is an IgE-mediated eosinophilic inflammatory disease of the nasal mucosa, characterized by nasal itching, sneezing, nasal discharge, and nasal congestion. In some patients, there is long-term nasal obstruction and thick nasal discharge from the back of the nasopharynx, which the patient may not notice—these are hidden symptoms. Sometimes, symptoms are clear, including reddened and affected eyes, nasal itching, sneezing, and so on. As a result, the disease often goes undiagnosed in childhood because parents may mistake allergic rhinitis for acute rhinitis or ignore the child's persistent nasal congestion. Moderate to severe allergic rhinitis can impair quality of life. Symptoms can disrupt sleep, which subsequently affects daytime functioning and activity. It can also be complicated by secondary diseases associated with rhinitis, such as sinusitis, pharyngitis, and otitis media. Rhinitis that is not completely treated in childhood (40–60% of cases) may progress to bronchial asthma as the child grows, which is a serious complication and worsens quality of life. Allergic rhinitis is often triggered by pollen and clinically presents as severe runny nose,

cough, watery eyes, and itching, collectively called rhinoconjunctival syndrome.

One of the most common names for this condition is “hay fever,” a term dating back to the 19th century when the English physician D. Vostok first described this clinical manifestation in 1819. The prevalence of allergic diseases is increasing worldwide, driven by many factors including diet, environment, and heredity. Environmental pollution is a major factor, particularly affecting urban populations, as demonstrated in numerous clinical studies.

Features of allergic rhinitis include:

- onset at an early age (7–20 years);
- an allergic predisposition of the body. Individuals with this predisposition are often called “messengers” in childhood, indicating a tendency toward future allergic problems—diathesis, food intolerance, frequent allergic reactions such as rashes, gastrointestinal upset, and runny nose after eating honey, sunflower seeds, watermelon, melon, or halva;
- heredity plays a major role in its development;
- in most patients, symptom onset coincides with the beginning of the flowering season, occurring at roughly the same time every year;
- symptoms immediately decrease or disappear when exposure to pollen ends.

Identifying effective allergens does not require knowing the flowering calendar of every plant. By comparing the start of flowering periods with the onset of allergic rhinitis symptoms, it is possible to roughly determine the allergenic group. Of the thousands of plants on Earth, only about fifty have pollen with allergenic properties.

The ability of plant pollen to trigger allergies depends on several factors:

- allergenic properties associated with protein and non-protein substances in the pollen; a conductivity factor in pollen promotes pollination and helps pollen penetrate the epithelial layer of the human nasal mucosa;

- particle size: pollen with a diameter up to 25 microns can penetrate deep into the respiratory tract and trigger allergic disease; pollen over 30 microns is trapped by protective barriers, including the nasal mucosa and ciliated epithelium of the upper respiratory tract;
- distribution in human habitation areas: the more widespread a plant, the more likely it is to cause allergic reactions (e.g., timothy grass in central Russia and Ukraine, wormwood in Central Asia, ambrosia in the North Caucasus);
- pollen mass and dispersibility: wind-pollinated plant pollen is often responsible for hay fever due to its spherical shape and small size, whereas insect-pollinated pollen rarely causes allergic rhinitis;
- concentration of pollen in the air: when pollen exceeds 25 particles per cubic centimeter, the number of individuals with symptoms sharply increases. Symptoms are reduced after rain and vary by time of day depending on pollen concentration.

Allergens can be divided into three groups:

1. environmental allergens (plant pollen);
2. domestic allergens (house dust, animal dander, insects, mold);
3. occupational allergens.

Mechanism of development: Allergic rhinitis involves a hypersensitivity reaction in the nasal mucosa, triggered by allergen interaction with IgE, leading to histamine release and other immunologically active substances that produce clinical symptoms.

Clinical appearance: Patients' complaints vary widely. The most typical manifestation is rhinoconjunctival syndrome. It begins with irritation of the inner corner of the eye, followed by redness of the conjunctiva and sclera, dilation of superficial vessels, eyelid swelling, lacrimation, and photophobia. Initially, conjunctival discharge is clear, but it can become thick and purulent due to secondary infection. Patients report sharp, stabbing, or pressing eye pain. Both eyes are usually affected, but severity can differ. Visual acuity remains unchanged.

Simultaneously, patients experience severe nasal and pharyngeal itching, paroxysmal sneezing with watery nasal discharge, and nasal obstruction. The inflammatory process often extends to the mucosa of

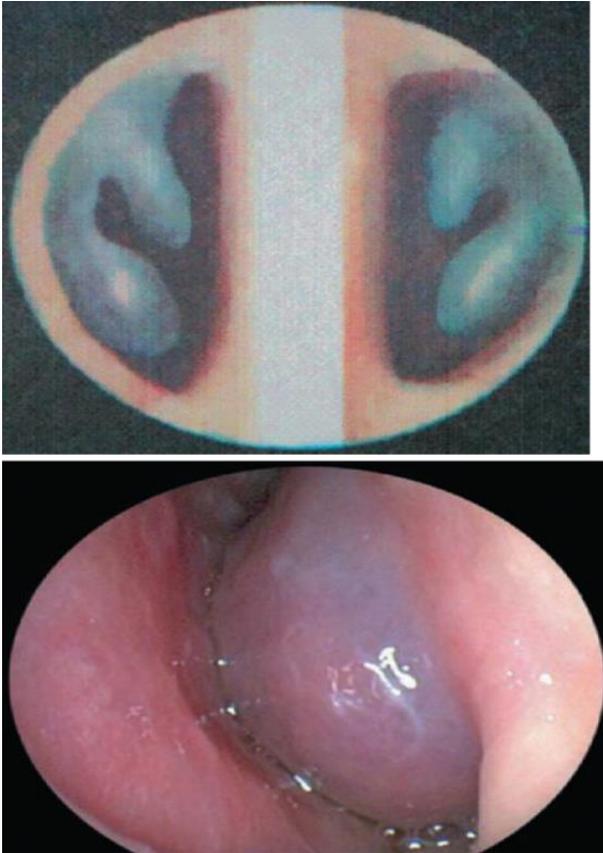
the larynx, nose, and auditory tubes, causing ear pain, a dull voice, and increased sensitivity to triggers such as cold, strong odors, and dust. Nighttime breathing may be impaired, preventing sleep. Symptoms are often worse at night, though some patients improve when lying down. Condition generally improves on rainy, cloudy, or calm days.

Acute allergic inflammation can also affect the respiratory tract, conjunctiva, digestive system, skin, cardiovascular system, and nervous system. General symptoms often include weakness, headache, fatigue, dizziness, drowsiness, memory loss, malaise, and fever.

One severe manifestation of pollinosis is bronchial asthma, with reversible symptoms including cough, wheezing, chest tightness, and shortness of breath. Signs of pre-asthma include acute cough, chest heaviness, occasional chest pain with breathing, and noisy respiration. A key indicator is sticky cough, which is not typical for isolated rhinitis. Rhinoconjunctival syndrome usually precedes asthma attacks by several days. Seasonal allergic bronchial asthma typically recurs annually in accordance with the flowering period of the triggering plants. Symptoms develop sequentially: first rhinitis and rhinoconjunctivitis (sometimes with skin involvement), followed by asthma during peak pollination. In some patients, asthma occurs only upon direct pollen exposure. Symptoms resolve in reverse order, beginning with asthma, then rhinitis and conjunctivitis. Relief is commonly observed on rainy days due to decreased airborne pollen.

Patients with pollen-induced asthma are sensitive not only to specific allergens but also to non-specific triggers such as cold, emotional or physical stress, and weather changes. Allergic rhinitis is often accompanied by inflammation of the nasal mucosa and the maxillary sinus. This mucosa is susceptible to secondary infections, which may lead to purulent sinusitis. Some allergens cross-react with foods or herbal preparations, exacerbating allergic attacks, conjunctivitis, urticaria, or angioedema even “off-season.” For example, patients allergic to tree pollen may react to apples, walnuts, apricots, or brandy; grass pollen allergy may trigger reactions to wheat or rye products; melon, watermelon, seeds, and halva can trigger reactions in other patients. Products such as propolis, derived from bees, can also cause cross-reactions.

Rhinoscopic findings include clear mucosal swelling, with areas of cyanosis interspersed with pale regions.



Picture. 43.*Allergic rhinitis*

Laboratory diagnostics. Blood eosinophilia is a standard indicator of allergic disease. In allergic rhinitis, the microscopy of the discharge from the nasal cavity, stained by the Romanovsky method, is more clearly visible. In this disease, the amount of eosinophils in the nasal smear increases and makes up more than 10% of the total number of cells. Skin tests - used to identify causative allergens. Usually, the tests are applied by injection or scarification of the skin of the wrist and instillation of a drop of allergen from a special kit. The appearance of an allergic reaction at the site of application of the allergen is considered a positive test (Pic.44).



Picture. 44. *Skin tests in the diagnosis of allergies*

Treatment. Treatment strategies for allergic rhinitis include:

1. elimination of allergens;
2. pharmacotherapy;
3. specific immunotherapy (SIT).

Eliminating the offending allergens helps reduce the severity of clinical symptoms, decrease the number of complications, and indirectly improve the patient's quality of life. The main recommendations include following certain rules. Since the highest concentration of pollen in the air is observed in the morning and afternoon, patients should limit outdoor activities during these times. Windows should be kept closed as much as possible, and high-quality air purification systems should be used. If this is not possible, a damp cloth or gauze can be hung over the windows. When going outside, dark sunglasses should be worn. After returning indoors, it is recommended to wash the body and hair thoroughly and change clothes.

Sometimes, if feasible, the patient may relocate to another climate during the flowering period—coastal regions generally have lower pollen concentrations.

Pharmacotherapy is widely used in the treatment of allergic rhinitis; however, without eliminating the underlying cause of allergy, it remains purely symptomatic. Medications include antihistamines

[loratadine (Claritin), desloratadine (Erius), fexofenadine (Telfast)]; local vasoconstrictors in the form of nasal drops; mast cell stabilizers (sodium cromoglicate); and local glucocorticosteroids [mometasone (Nasonex*)].

In the treatment of pollinosis, the most modern and effective method is specific immunotherapy (SIT), the goal of which is to reduce the patient's specific sensitivity by administering the allergen repeatedly, starting from a microdose.

Vasomotor rhinitis
The classification of **vasomotor rhinitis** is determined primarily by its etiological basis. Three forms are distinguished: **medicinal**, **reflex**, and **idiopathic vasomotor rhinitis**.

Etiology. This chronic condition arises from abnormal vasodilation or hyperreactivity of the nasal mucosa in response to non-specific exogenous or endogenous stimuli, rather than an immunological reaction. Common triggers include:

- Cold exposure;
- High humidity;
- Hot or spicy foods;
- Alcohol consumption;
- Tobacco smoke;
- Environmental pollutants;
- Strong odors;
- Emotional stress;
- Certain medications;
- Hormonal fluctuations.

The pathogenesis of **vasomotor rhinitis** remains incompletely understood. It is hypothesized that the principal mechanism involves dysregulation of the efferent neural pathways controlling vascular tone within the nasal turbinates.

Drug-induced rhinitis often develops in individuals using **vasoconstrictor medications** for extended periods (>7–10 days), leading to tachyphylaxis and the need for increasing doses to maintain nasal patency. Additionally, many antihypertensive drugs, certain

neuroleptics, and oral contraceptives can enhance nasal mucosal blood flow. Chronic use may damage **adrenoceptors** in the nasal mucosa.

Hormonal rhinitis commonly occurs during pregnancy. Elevated estrogen levels may inhibit acetylcholinesterase, resulting in increased acetylcholine and subsequent swelling and hypersecretion of the nasal mucosa. A similar pattern can emerge during puberty. Alterations in nasal secretions and congestion are sometimes linked to thyroid hypofunction, although the precise relationship between thyroid hormone levels and rhinitis remains unclear.

Reflex rhinitis. Various stimuli may alter autonomic nerve activity, affecting vascular tone and glandular secretion. **Food-induced rhinitis** is a typical example, primarily affecting men over 50, and is thought to involve **vagal reflexes**. Alcoholic beverages, such as beer and wine, may provoke nasal congestion through vasodilation. **Cold-induced rhinitis** is another form of reflex vasomotor rhinitis, mediated by nonspecific nasal hyperreactivity.

In some cases, it is challenging to categorize vasomotor rhinitis precisely. When a clear link between symptoms and triggers cannot be established, the condition is classified as **idiopathic rhinitis**. Structural nasal anomalies, particularly sharp ridges or spines on the nasal septum contacting the lateral nasal wall, can exacerbate local mucosal swelling and contribute to the development of the disease.

Clinic. **Vasomotor rhinitis** presents with either intermittent or persistent symptoms, most prominently **nasal obstruction** and **rhinorrhea**. Sneezing is less frequent. Examination often reveals hypertrophy of the **inferior turbinates**, which exhibit poor contractility. The mucosa may appear congested, hyperemic, cyanotic, or even pale, and occasional bleeding can occur. Enlarged turbinates may obscure posterior septal regions, which are often the true sites of vasomotor dysfunction on anterior rhinoscopy.

During pregnancy, **nasal congestion** typically begins in the second trimester, persists throughout gestation, and resolves postpartum. **Food-induced vasomotor rhinitis** is exemplified by copious, watery nasal discharge triggered by specific foods, often hot or bitter. **Cold vasomotor rhinitis** manifests as abrupt sneezing and rhinorrhea,

followed by persistent nasal discharge, triggered by cold exposure to the extremities, face, or environment, and occasionally by bright sunlight.

Diagnostics. Assessment should exclude allergic causes, including a thorough history, blood and nasal secretion analysis for **eosinophilia**, and, if indicated, **allergen skin testing**.

Differential diagnosis. Atypical allergic rhinitis may mimic vasomotor rhinitis; careful evaluation is required. True food-induced vasomotor rhinitis must be distinguished from allergic reactions to food in children or cross-reactive plant allergens (e.g., apples, peaches, plums, apricots, pears, cherries).

Treatment. Avoiding or eliminating triggers is central but often challenging. Pharmacotherapy typically begins with **local glucocorticoids**, though their effect is less pronounced than in allergic rhinitis. Gradual tapering of daily glucocorticoid doses is recommended, alongside discontinuation of **vasoconstrictor drugs**. If these measures fail, a short course of systemic glucocorticoids may be indicated.

Minimally invasive surgical interventions on the **inferior turbinates** are employed in refractory cases. Techniques include **mucosal conchotomy**, **laser surgery**, **osteconchotomy**, **vasotomy**, and **ultrasonic dissection**. Correction of nasal septal deformities is addressed surgically at the onset of therapy. Adjunctive measures such as **physiotherapy** (e.g., electrophoresis with calcium chloride or zinc sulfate) and **acupuncture** are applied empirically.

Tumors of the nasal cavity

Benign tumors include **papillomas**, **fibromas**, and **angiomas**, most commonly arising from the nasal mucosa or extending from adjacent structures (e.g., **septal angiomas** or **skull base angiofibromas**). Patients frequently report **nasal obstruction**, epistaxis, or occasionally purulent discharge due to secondary inflammation. Detection via rhinoscopy varies with size and location; polyps at **Kisselbach's area** are typically the easiest to identify. Treatment is always surgical, with subsequent **electrocautery** or **cryotherapy** at the excision site.

Malignant tumors are less common but include **carcinomas and sarcomas**, predominantly affecting men aged 60–70. Tumor appearance depends on histology: malignant lesions often exhibit an irregular, ulcerated surface with dense, gray tissue. Progressive nasal obstruction, purulent or bloody discharge, facial neuralgia, and headaches are common. Regional lymph node metastases initially involve the **laryngeal nodes**, later spreading to deeper cervical nodes, which delays diagnosis. Biopsy is mandatory due to the non-specific appearance of the tumor. Early recognition of malignant nasal tumors remains a significant clinical challenge, and any substantial mass should be evaluated for **potential malignancy**.

Treatment.

For malignant tumors of the nose, surgery, radiation and chemotherapy methods are often used together.

Specific diseases of the nasal cavity

Tuberculosis of the nasal cavity

Nasal tuberculosis is an extremely rare condition and may go unrecognized in the early stages due to the absence of distinctive clinical features. The disease can occur in primary form, resulting from direct inoculation of *Mycobacterium tuberculosis* into the nasal mucosa—such as during manipulation or removal of crusts—or in secondary form, arising from hematogenous spread from preexisting pulmonary tuberculosis.

The most common sites of involvement in the nasal cavity include the anterior portion of the nasal septum, the anterior end of the inferior turbinate, and the lower regions of the nasal cavity. Patients typically present with nasal discharge, pruritus, and crust formation. As the disease progresses, ulceration develops on the mucosa, which may extend posteriorly toward the pharynx, accompanied by extensive granulation tissue formation.

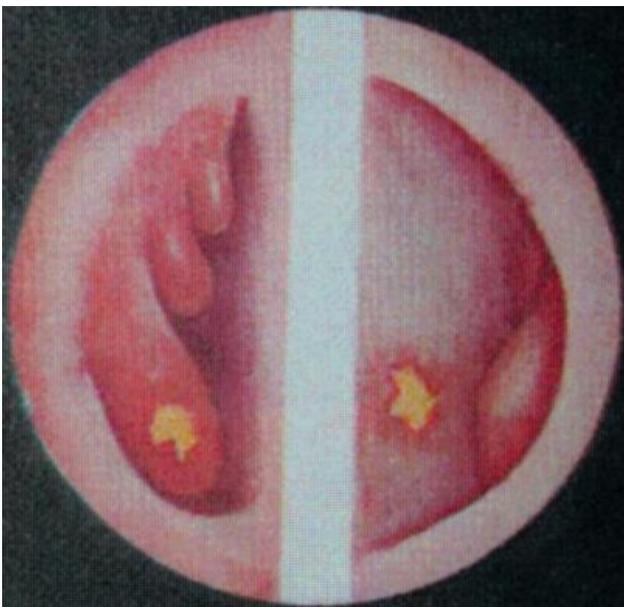
Treatment involves systemic antituberculous therapy combined with local interventions, including ultraviolet light exposure, and administration of specific antitubercular medications to control the infection and promote mucosal healing.

Ulcers of the nasal cavity

The clinical presentation within the nasal cavity varies depending on the stage of the lesion. During the primary stage, dense infiltrates of a brown-red hue may appear on the mucosa at the entrance to the nose or within the nasal cavity itself. Within the cavity, these infiltrates are typically reddish, raised above the mucosal surface, firm, and tender upon palpation.

It is essential for all healthcare providers, particularly dentists, to recognize the early manifestations of such lesions in the external nose and nasal cavity. Awareness of these signs helps prevent inadvertent manipulations or procedures that could exacerbate the condition, and it emphasizes the need to defer nonurgent interventions until a definitive diagnosis requiring targeted therapy is established.

Regional lymphadenitis may also develop, with enlargement of lymph nodes in the preauricular, submandibular, and cervical regions. This clinical feature often prompts patients to seek dental consultation, as awareness of lymph node involvement is critical when assessing lesions of the nasal cavity and planning appropriate management.



Picture. 45. *Tuberculosis of the nasal cavity*

Secondary (papula) and tertiary (gumma) lesions are common. Gums can be located in the mucous membrane, bone, periosteum, and integument, and necrosis of bone tissue occurs with the formation of sequestrations (Pic. 45). In the third stage of the wound, it is necessary

to consult a dermatovenerologist, as well as to conduct a mandatory blood test - Wasserman's reaction.



Picture 46. Nasal sores (bumps)

Treatment.

General, anti-ulcer.

Diseases of the nasal cavity

Inflammatory processes affecting the paranasal sinuses are collectively referred to as sinusitis. The specific terminology depends on the sinus involved: inflammation of the maxillary sinus is termed maxillary sinusitis; of the frontal sinus, frontal sinusitis; of the ethmoidal labyrinth, ethmoiditis; and of the sphenoid sinus, sphenoiditis. Frequently, multiple sinuses are affected simultaneously, giving rise to pansinusitis when all sinuses on both sides are involved, or hemisinusitis when all sinuses on one side are affected.

Sinusitis is further classified based on the nature of the inflammatory process into purulent, catarrhal, allergic, and polyposis forms. The duration of the condition distinguishes acute, subacute, and chronic sinusitis. The disease often develops following viral infections, particularly influenza, which can inflame the nasal mucosa, impair ciliary activity, and facilitate the migration of nasal microflora into the sinus cavity. Obstruction or narrowing of the sinus ostium and impaired

drainage are central to the pathogenesis of sinusitis, particularly in rhinogenic forms.

The anatomical proximity of the maxillary sinus to the upper dentition explains why inflammation in this sinus can arise not only from rhinitis but also from dental pathology, leading to a distinct clinical entity known as odontogenic sinusitis. This form requires specialized diagnostic and therapeutic approaches. Odontogenic sinusitis is most commonly observed in adults, correlating with the developmental expansion of the maxillary sinus during facial growth. At birth, the sinus volume is approximately 0.15 cm³, reaching 1.5 cm³ by 2–3 years of age, with a well-defined contour. In early childhood, the sinus is deep, the anterior wall is 0.5–0.8 cm thick, and tooth buds occupy the cavity, limiting surgical access. At this stage, molars are closest to the sinus; by ages 5–6, canines, second premolars, and first molars come into direct contact with the sinus mucosa. After age six, rapid sinus growth occurs in association with the eruption of dental sacs in the alveolar process.

The relationship between tooth roots and the sinus wall varies with sinus size. In large sinuses, premolar and molar roots may be separated from the mucosa by a thin bony layer or may even protrude into the sinus cavity. In smaller sinuses, bone thickness ranges, for example, from approximately 1.3 mm for the second molar to 7.6 mm for the first premolar. Although frontal, ethmoidal, or sphenoid sinusitis is typically unrelated to dental causes, severe odontogenic sinusitis may occasionally extend to these adjacent sinuses.

Pathological changes in the sinus mucosa are closely linked to the duration and nature of the inflammatory process. Acute inflammation is characterized by mucosal edema, nasal obstruction, and marked hyperemia. Normally thin, the maxillary sinus mucosa can thicken up to 20-fold during inflammation, narrowing the sinus cavity and potentially blocking secretion outflow, which exacerbates headache and general malaise. In the acute stage, mucosal thickening compresses local vessels, initially producing hemorrhagic exudate and eventually purulent discharge.

In chronic sinusitis, as well as in chronic processes affecting adjacent nasal cavities, pathological features vary according to the underlying etiology. Allergic or inflammatory swelling, polyp

formation, and cyst development may predominate. In chronic purulent forms—whether maxillary sinusitis, frontal sinusitis, or ethmoiditis—mucosal thickening, persistent hyperemia, nasal congestion, fibrosis, and mucosal hyperplasia are characteristic findings, reflecting long-standing inflammatory remodeling.

Sinusitis

In the clinical picture of sinusitis, there are many common features and signs that allow us to distinguish inflammation of each sinus, regardless of the location of the inflammatory process in a particular sinus. There is acute sinusitis, which lasts no more than 12 weeks, and chronic sinusitis, which lasts more than 12 weeks.

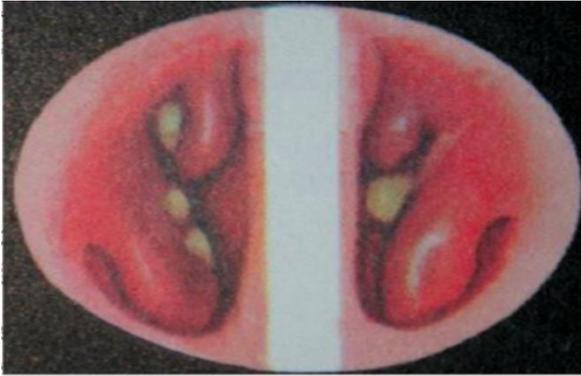
Acute inflammation of the upper jaw cavity.

Acute maxillary sinusitis is characterized by sudden inflammation of the mucosa lining the maxillary sinus and represents the most common pathology among the paranasal sinuses.

Patient complaints typically include generalized or localized headache over the affected sinus, a sensation of cranial heaviness, pain radiating to the upper teeth, and occasionally discomfort in the cheek region. Nasal discharge is observed in approximately half of cases, particularly in unilateral involvement, and may be purulent or mucous in character.

Anamnesis often reveals a temporal association with precipitating factors such as exposure to cold, acute or chronic rhinitis, upper respiratory tract infections, or influenza.

Clinical findings commonly include fever, significant elevation of body temperature, generalized weakness, and malaise. Rhinoscopic examination typically demonstrates accumulation of mucous or purulent secretions within the common nasal passage, often forming a "purulent corridor" beneath the middle nasal concha. The mucosa of the affected nasal cavity, particularly in the middle nasal meatus, appears erythematous and edematous.



Picture. 47. *Purulent discharge from the nose in acute sinusitis*

Roentgenography - the image obtained in the nose-chin projection reveals a strong darkening of the maxillar cavity (Pic. 48).



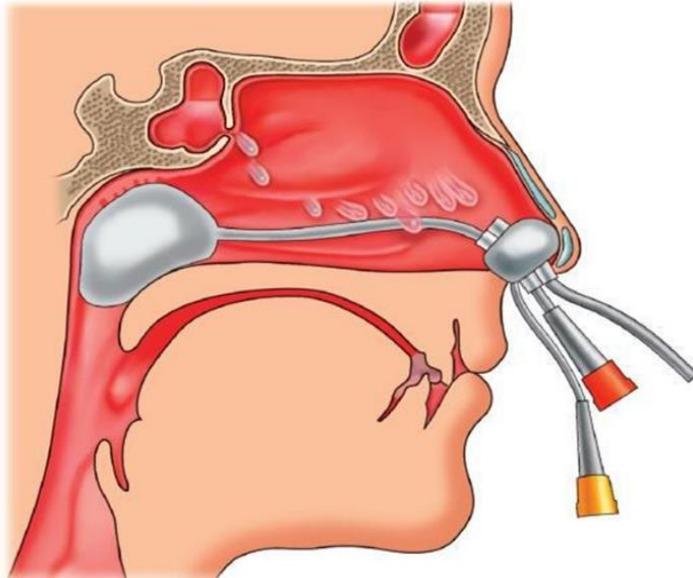
Picture. 48. *X-ray in acute sinusitis*

In addition to ordinary radiography, CT examination is also used to determine the pathology of the paranasal sinuses. serves (Pic. 49).



Picture. 49. Computer tomography of paranasal sinuses in acute sinusitis

Treatment is conservative. By instillation of vasoconstrictors (naphazoline, naphthizine, sanorin, xylometazoline, galazolin), by expanding the anastomosis connecting the affected sinus with the nasal cavity, it is possible to improve the discharge from the maxillary cavity. When using drops, you should try to direct them to the area of the middle nasal passages. Vasoconstrictor drops can be replaced by drugs with mucolytic and secretolytic effects [acetylcysteine + tuaminoheptan (rinofluimucil*), sinupret*]. Evacuation of exudate from the nasal cavity and sinuses in the Proets method has a positive effect. In recent years, a non-functional method of removing purulent discharge from the paranasal sinuses - the method of removal with the help of a YAMIK sinus catheter - has become widespread. (Pic. 50). This device, which creates negative pressure in the nasal cavity, allows to remove pathological discharge from the paranasal sinuses and introduce medicinal substances into them.



Picture. 50. Sinus catheter "YAMIK"

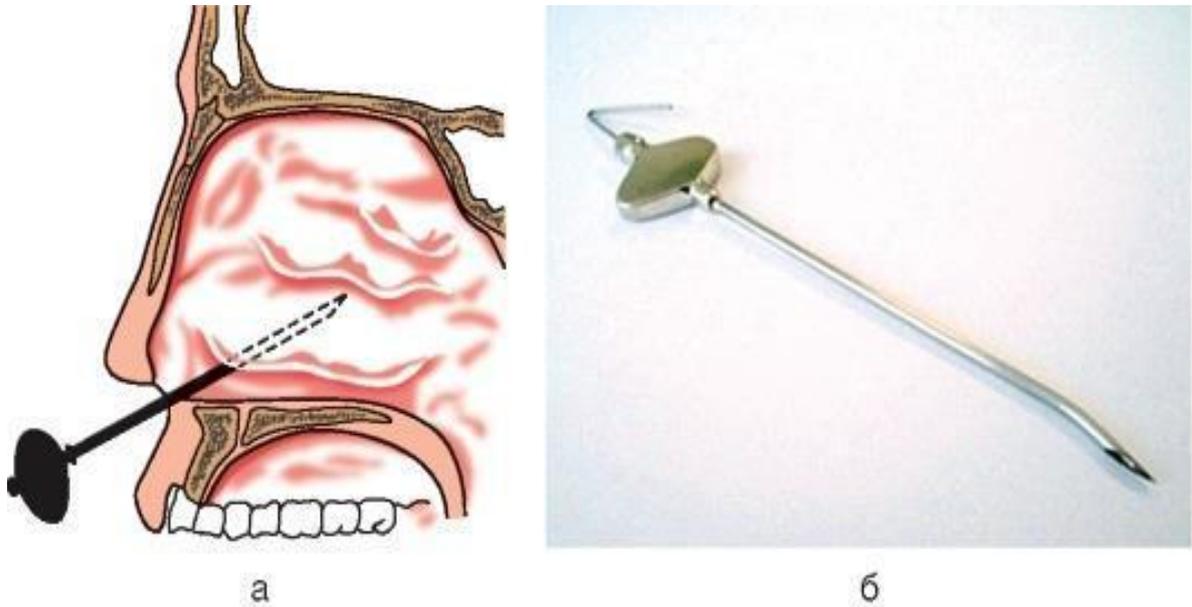
General treatment consists of using antibacterial (antibiotics), hyposensitizing [clemastine (Tavegil), loratadine (Claritin), desloratadine (Erius)] drugs. Topical glucocorticoids are also used [mometasone (nazonex)]. Physiotherapy procedures include: UHF, UVR, electrophoresis and others. In cases of prolonged duration of the procedure, persistent headache and other symptoms of intoxication, it is necessary to puncture the maxillary sinus for diagnostic and therapeutic purposes. The piercing of the maxillary sinus is performed through the lower nasal passage. This method is very simple, most importantly, it does not cause complications if it is done correctly.

Puncture is performed in the following sequence.

- Anesthetizing the mucous membrane of the lower nasal passages by applying 10% lidocaine solution.
- Inserting a cotton swab soaked in epinephrine (adrenaline) into the middle nasal passages to widen the sinus opening.

Under visual control (after 5 minutes), a special needle (Kulikovsky needle) is inserted from the area where the bony wall of the

lower nasal passage is the thinnest, i.e. at the junction of the lower nasal concha with the side wall, the tip of which is lateral to the orbit is directed to the corner (Pic. 51).



Picture. 51. *Maxillary puncture (a) and Kulikovsky needle (b)*

Following penetration of the sinus wall, the needle is attached to a syringe to aspirate the cavity contents. The nature of the discharge is assessed, and samples are collected for microbiological analysis and determination of antibiotic sensitivity. Subsequently, the sinus is irrigated with antiseptic solutions until the effluent becomes clear. Once cleansing is complete, therapeutic agents, including antibiotics and enzymatic preparations, are instilled into the sinus via the needle. It is essential that the patient remains in a lateral position for approximately 30 minutes to prevent spillage of the instilled solution through the anastomosis.

While sinus puncture is generally a safe and effective procedure, potential complications may arise, including:

- inadvertent passage of the needle from a pus-filled sinus into the orbit, potentially resulting in orbital phlegmon;
- penetration of the needle tip through the anterior sinus wall into the soft tissues of the cheek;
- occurrence of air embolism, leading to circulatory collapse or shock.

Therefore, indications for maxillary sinus puncture must be strictly justified, and contraindications carefully considered. These include hematologic disorders, drug allergies, and the patient's mental and emotional state. Since each puncture represents a stressful event, in cases requiring repeated or multiple interventions, it is advisable after the initial puncture to insert a polyethylene catheter into the created opening. A properly positioned catheter, invisible externally, allows prolonged irrigation of the sinus and repeated administration of therapeutic agents, minimizing patient discomfort and reducing procedural risk.

Chronic inflammation of the maxillar sinus

Chronic sinusitis (sinusitis maxillaris chronica) is a chronic inflammation of the mucous membrane of the maxillary sinus. It is often considered a continuation of the acute process. Chronic sinusitis is a long-term disease, which especially occurs when allergic conditions are associated with the formation of polyps. Acute sinusitis can progress to chronic sinusitis due to:

- recurrence of acute episodes;
- deviation of the nasal septum;
- hypertrophy of the nasal turbinates;
- presence of adenoids;
- reduced immune protection;
- pathogenicity of microflora;
- living and working conditions;
- inappropriate treatment.

Dental diseases, as well as surgical interventions, perforation and infection of the lower wall of the sinus, and inflammatory processes in the alveolar process of the maxilla, play an important role in the development of chronic sinusitis. Sinusitis of this etiology is called odontogenic sinusitis.

Most often, purulent, purulent-polypous, and polypous forms of chronic sinusitis are distinguished, while premural hyperplastic types are rare.

Symptoms. Patients with chronic sinus disease commonly present with difficulty breathing through one or both nasal passages, accompanied by unpleasant odors and nasal discharge, which may be purulent, serous, or mucous in nature. Frontal headaches and facial pain are frequently reported, often worsening in the afternoon or when the head is bent forward. This pattern arises because, during upright posture, purulent secretions fail to drain efficiently through the ostium on the medial sinus wall. The accumulated exudate stimulates sensory receptors within the sinus mucosa—primarily branches of the trigeminal nerve—resulting in persistent headache and referred pain to the upper teeth. Patients often report generalized fatigue and weakness.

A detailed patient history typically reveals a wave-like course of the disease, with alternating periods of exacerbation and remission. Chronicity is especially pronounced in mixed, polyposis-purulent, or allergic forms of sinusitis, which may persist for several years.

The clinical presentation and overall patient condition are influenced by the degree of nasal obstruction, the presence of comorbidities such as bronchial asthma, the intensity of purulent inflammation within the sinuses, and the severity of systemic intoxication. These factors collectively determine the patient's symptom burden and functional impairment.

Rhinoscopy often reveals purulent discharge in the middle nasal passage and polyps filling the middle and common nasal passages (Pic. 52).

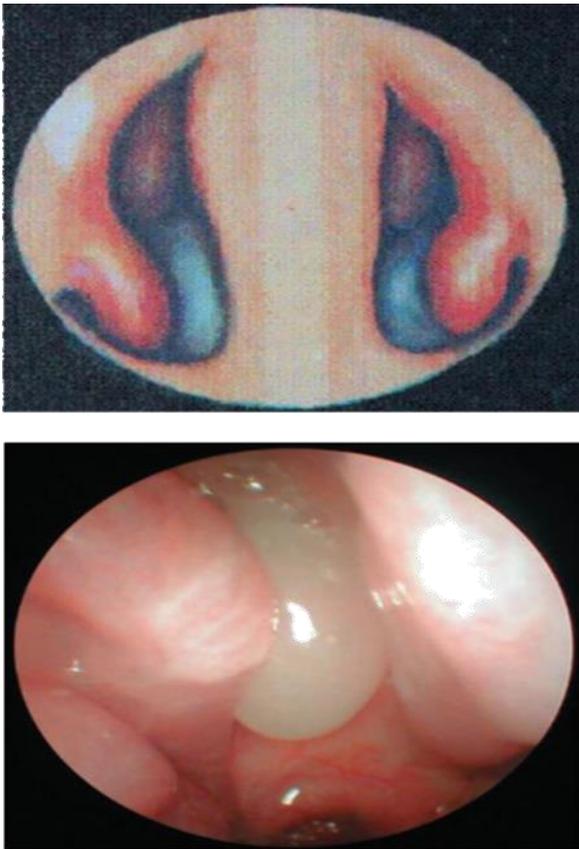


Picture. 52. *Computed tomography of the paranasal sinuses in polyposis sinusitis*

The color of the mucous membrane depends on the nature of the process: in purulent forms, it appears obviously red, while in allergic forms, it takes on a cyanotic hue.

Polyps identified during prior rhinoscopic examination typically originate from adjacent paranasal sinuses, most commonly the maxillary sinus and the ethmoidal labyrinth. Maxillary sinus polyps tend to grow posteriorly, and when they reach substantial dimensions—up to 10 cm in length—they may extend through the choanae into the nasopharynx, a condition referred to as choanal polyps. Histologically, these growths are benign and are characterized by a smooth, blue-gray surface. They are pedunculated, and the stalk exhibits mobility when gently manipulated with a probe.

Radiography not only helps to detect sinus pathology but also clarifies the characteristics of the process: uniform opacification is typical of purulent forms, mucosal thickening of the sinus wall indicates catarrhal forms, and patchy shadows suggest polyposis sinusitis. Naso-mental (chin) projections are usually taken. CT scanning of the paranasal sinuses provides more detailed and informative diagnostic data for chronic sinusitis.



Picture. 53 rinoendoscopy in polyposis sinusitis

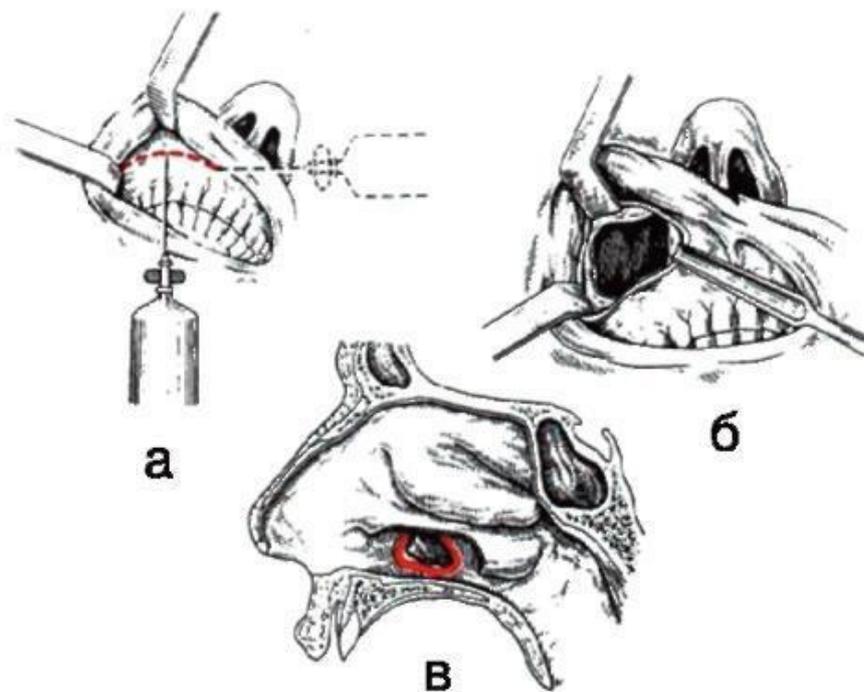
Puncture of the sinus – allows to clarify the nature of inflammation, to take material for bacteriological studies, to remove persistent purulent secretions with a pungent, putrid smell.

Diagnostics. It is organized taking into account all the received information: **chronic purulent, chronic polyposis, chronic polyposis-purulent sinusitis** are distinguished.

Complications. The progression of sinus inflammation may lead to complications involving adjacent anatomical structures. In some cases, the inflammatory process extends to the orbit, with potential involvement of the second branch of the trigeminal nerve. Infection can also track along the superior wall of the sinus, while teeth in close proximity to the inferior maxilla may become affected.

Therapeutic management predominantly involves surgical intervention. Initial approaches often employ a puncture technique, which may include removal of nasal polyps and other localized procedures within the maxillary sinus. However, if repeated punctures or irrigation over a period of two weeks fail to achieve resolution, a more

radical surgical procedure becomes necessary. This involves complete excision of pathologically altered sinus mucosa. A critical component of radical surgery is the creation of a wide fistula between the maxillary sinus and the inferior nasal meatus. This fistula allows postoperative irrigation of the sinus and the targeted administration of therapeutic agents, including antibiotics. Enzymatic preparations may also be introduced to facilitate the dissolution and removal of adherent purulent crusts from the sinus walls, ensuring effective postoperative cleansing and recovery.



Picture. 54. *Radical operation in the maxillary cavity: a, b, v - stages of the operation*

Currently, radical surgical methods are rarely used in the treatment of chronic sinusitis. The most modern surgical method of treating chronic sinusitis is a functional endoscopic operation in the maxilla using an endoscope and special microsurgical instruments (Pic. 55,56).



Picture. 55. Endoscopes for operations of the paranasal sinuses



Picture 56. Endoscopic surgery of the paranasal sinuses

Endoscopic sinus surgery

The advantages of endoscopic sinus surgery over the traditional method is that it does not require a surgical incision. Endoscopic surgery is performed using special equipment - an endoscope. This equipment is a metal tube in which there are optical fibers. The endoscope has a lens at one end and an eyepiece at the other end through which the doctor looks. The endoscope allows you to see the pathological process that

occurs in the sinus. Another advantage of the endoscopic method is that it allows treating the cause of sinusitis directly. With its help, the doctor can directly see the pathological focus and remove normal tissue without cutting, which significantly reduces unnecessary injury, accelerates the postoperative recovery period, and reduces the time of the operation itself. and reduces postoperative complications. This method is characterized by the absence of an external scar, slight swelling and pain intensity after surgery. The goal of endoscopic surgery is to widen the sinus openings. Usually, the paranasal sinuses open into the nasal cavity through a thin bony canal lined with mucous membrane. Due to inflammation, the lining of this tube swells and thus blocks the exit from the sinus. Endoscopic surgery makes it possible to expand the bony canal of the sinus, so even if the patient later has inflammation of the nasal mucosa and sinus outlet, or their allergic swelling, there will be no obstacles to the opening of the adjacent nasal cavities. This greatly facilitates the subsequent treatment of sinus inflammation. In addition, special tools for endoscopic operations facilitate the removal of pathological tissues in the sinus cavity - polyps, granulations, etc. In the near future, the improvement of the endoscopic method of surgical intervention for diseases of the paranasal sinuses is a computer navigation system.

It allows creating a three-dimensional image of the nasal passages on the monitor screen, which makes it easier for the doctor to make a diagnosis and perform an operation. Endoscopic surgical interventions in the nasal cavity and paranasal sinuses are performed only if conservative treatment is ineffective. Endoscopic sinus surgery is rarely accompanied by postoperative bleeding, intracranial and intraocular complications.

Odontogenic sinusitis

Odontogenic sinusitis may present with subtle clinical manifestations, largely due to direct communication between the sinus and the oral cavity. In such cases, purulent material may drain through this connection, partially alleviating nasal obstruction and reducing headache severity compared with rhinogenic sinusitis, thereby complicating timely and accurate diagnosis. The condition is commonly precipitated by the introduction of foreign substances from the oral cavity into the sinus, including dental filling materials, fragments of

dental instruments, displaced tooth roots, or other debris. Additionally, chronic dental pathologies such as root granulomas of the upper molars, subperiosteal abscesses, and periodontal infections can initiate or exacerbate sinus inflammation.

Clinically, patients with odontogenic sinusitis often report mild frontal headache, purulent discharge through an alveolar fistula, the escape of air into the oral cavity during nasal exhalation, and occasional retrograde entry of food particles into the sinus following mastication. An unpleasant odor originating from the affected sinus is frequently noted.

Accurate diagnosis relies heavily on a thorough medical and dental history, emphasizing prior dental pathology or interventions. Rhinoscopic examination commonly identifies the causative tooth—either decayed or previously treated—and the residual sinus opening following extraction. Purulent material may be observed draining through this communication into both the oral cavity and nasal passages, with a characteristic fetid odor that is highly indicative of odontogenic origin.



Image:57 Oroantral fistula

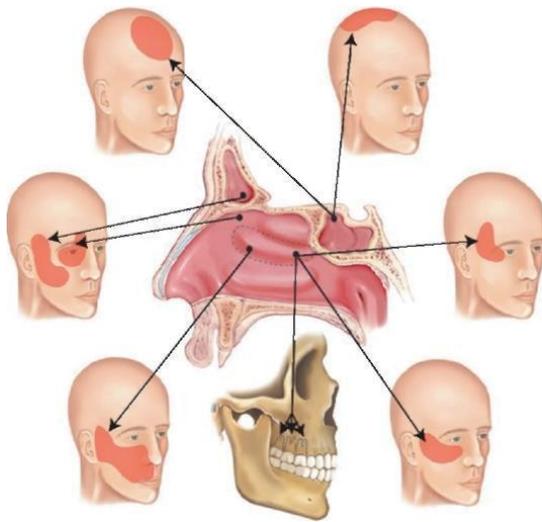
The leading diagnostic methods for detecting odontogenic sinusitis are x-rays of the nasal cavities, as well as orthopantomography, which allows assessing the condition of the roots of the teeth associated with

the sinus. Sometimes, iodolipol can be introduced through a puncture needle or fistula, and a contrast examination of the sinus cavity can be performed. In the x-ray of the adjacent nasal cavities, compared to other walls, the thickening of the lower wall of the sinus bordering the alveolar septum is determined. CT scan of the nasal cavities is the most modern diagnostic method.

Treatment. The primary diagnostic tools for identifying odontogenic sinusitis include radiographic imaging of the nasal cavities and orthopantomography, which enables evaluation of the dental roots in close proximity to the sinus. In certain cases, contrast studies can be performed by introducing iodinated lipol through a puncture needle or existing fistula, allowing detailed visualization of the sinus cavity. Radiographically, thickening of the inferior sinus wall adjacent to the alveolar septum is often observed, while computed tomography (CT) of the nasal and paranasal sinuses represents the most advanced and precise diagnostic modality currently available.

Treatment of odontogenic sinusitis is primarily surgical and aims to address both the infected tooth and the affected sinus. Standard management involves removal of the causative dental source and restoration of sinus patency. In select cases, prolonged sinus drainage may be sufficient, particularly when the oral-sinus communication can be effectively irrigated with iodine solution to promote closure. Repair of the oroantral fistula, when present, requires careful plastic reconstruction, a technically demanding procedure.

Management also entails treatment of the offending teeth, which may involve root resection or removal of foreign bodies from the sinus. Diseased sinus mucosa must be excised, and during the postoperative period, continuous drainage is necessary to evacuate residual secretions. Establishing an anastomosis between the sinus and the inferior nasal meatus facilitates ongoing drainage and promotes healing. Contemporary approaches increasingly employ minimally invasive endoscopic surgery of the maxillary sinus, which allows precise removal of pathologic tissue while preserving surrounding structures. Similar principles apply to the treatment of inflammation in other paranasal sinuses, including the frontal sinus, ethmoidal labyrinth, and sphenoid cavity.



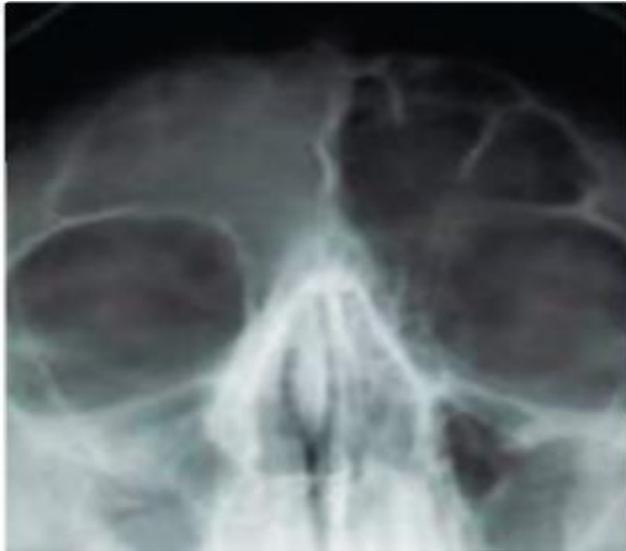
Picture: 58. Localization of headache in different forms of sinusitis. Acute inflammation of the frontal sinus

diseases, not directly related to tooth-jaw pathology. But since it has many similarities with sinusitis, diseases of these sinuses can confuse the clinical picture, and therefore it is necessary to know the features of ethmoiditis, frontal sinusitis, sphenoiditis (Pic. 58). In addition, these diseases themselves can cause complications inside the eyeball and severe brain box.

Acute inflammation of the frontal cavity (frontal sinusitis, acute frontitis) is an acute inflammation of the mucous membrane of the frontal cavity. Acute frontal sinusitis frequently develops secondary to an upper respiratory tract infection or influenza. Inflammatory edema leads to thickening of the sinus mucosa, which impairs the drainage of sinus contents into the middle nasal meatus via the frontonasal duct. This obstruction can result in active exudation and, in severe cases, suppuration, culminating in empyema of the frontal sinus. During acute episodes or exacerbations of chronic frontal sinusitis, periostitis may develop in the region separating the inferior wall of the sinus from the orbital cavity. Clinically, this can manifest as upper eyelid swelling, complete closure of the eye, and, in some instances, formation of a purulent fistula at the medial canthus.

Clinical signs. Patients typically report pronounced frontal headache and a sensation of pressure or heaviness localized over the frontal sinus region, reflecting the underlying inflammatory process. In previous rhinoscopy, there may be purulent or mucous discharge in the

middle nasal passage. The radiograph reveals the same darkening of one or both forehead cavities (Pic. 59).

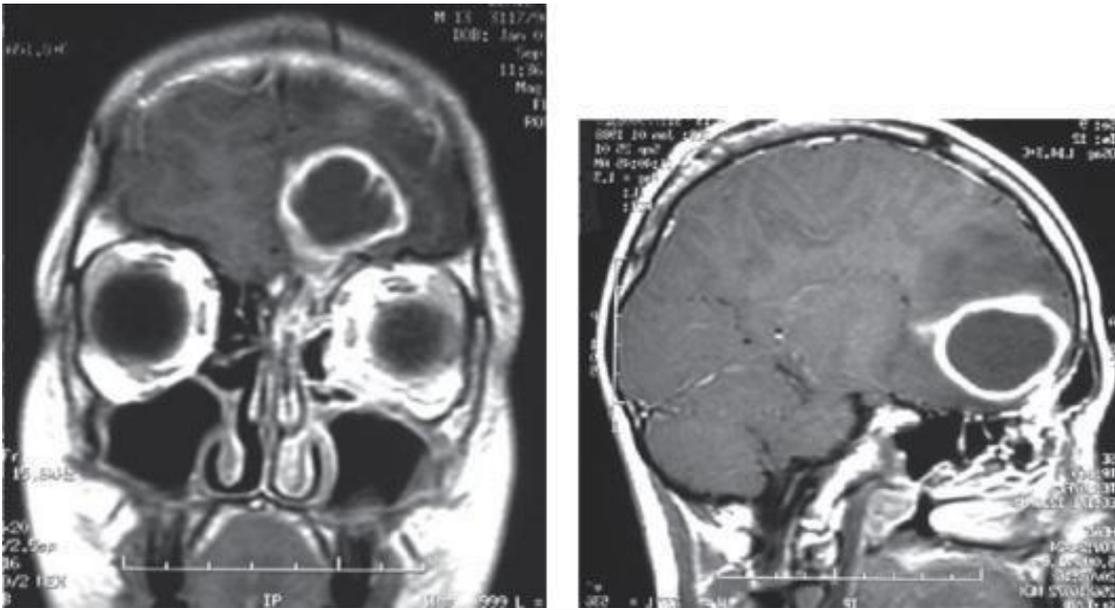


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Picture 59 X-ray of paranasal sinuses in acute frontitis

Treatment. It is usually carried out conservatively, aimed at improving sinus drainage and Pichting infection through local and general anti-inflammatory therapy. Initial management of frontal sinusitis prioritizes conservative measures aimed at restoring the patency of the frontonasal duct. Therapeutic interventions commonly include intranasal instillation of vasoconstrictive agents, such as naphazoline or xylometazoline, as well as systemic administration of antihistamines, including loratadine, desloratadine, or fexofenadine. Supportive therapy may also involve antipyretics, antibiotics, and sulfonamide compounds to control infection and inflammation.

In cases where acute frontal sinusitis is complicated by intracranial involvement, such as periostitis, prompt surgical intervention becomes necessary. The procedure typically involves opening the frontal sinus, reconstructing its bony walls, excising diseased mucosa, and establishing a functional anastomosis with the nasal cavity to ensure adequate drainage and prevent recurrence.



Picture:60 Rhinosinusogenic abscess of the frontal cavity of the brain (MRT)

Chronic inflammation of the frontal sinus

Chronic inflammation of the frontal sinus, referred to as chronic frontal sinusitis or chronic frontitis, represents a persistent inflammatory condition of the sinus mucosa. In cases of prolonged or refractory inflammation, trepanopuncture of the frontal sinus is considered a standard therapeutic intervention. This procedure is typically performed via the anterior wall of the sinus using a specialized instrument known as a trepanator (Pic. 61). The trepanator enables penetration of the relatively thick anterior sinus wall, allowing the insertion of a metal cannula through which the sinus cavity can be thoroughly irrigated. Additionally, this access permits the administration of therapeutic solutions, including antibiotics and enzymatic preparations, directly into

the sinus over an extended period.



Picture: 61 Trepanopuncture of the forehead

The most modern method of surgical treatment of chronic frontal sinusitis is functional endoscopic surgery.

Inflammation of the sinus

Acute ethmoiditis represents a frequent inflammatory condition affecting the mucosa of the ethmoidal air cells. Among paranasal sinus infections, it ranks second in prevalence, following maxillary sinusitis. Inflammation of the ethmoidal cells is often observed in conjunction with concurrent involvement of the maxillary and frontal sinuses. Common predisposing factors include acute rhinitis, viral upper respiratory infections, influenza, and other infectious conditions affecting the nasal passages.

Symptoms. Nasal congestion, purulent or mucous discharge from the nose, heaviness at the root of the nose, headache in the forehead.

Clinical picture. Previous rhinoscopy reveals swelling of the mucous membrane, accumulation of purulent secretions in the area of the middle nasal passage. Endoscopic examination allows careful examination of the middle nasal passages and the presence of pathological separation.

On x-rays of the paranasal sinuses, especially on CT, darkening of the cells of the glomerular labyrinth is visible.

Treatment.

In uncomplicated cases of acute ethmoiditis, management is predominantly conservative. Local therapy focuses on reducing mucosal edema and restoring adequate drainage of the affected ethmoidal sinuses. Vasoconstrictive nasal drops are commonly used to achieve decongestion, while combination medications containing mucolytic and antibacterial components, such as acetylcysteine and tuaminoheptane (e.g., Rinofluimucil), provide additional therapeutic benefit. Adjunctive physiotherapy, including ultrahigh-frequency (UHF) treatments and therapeutic laser applications, has been shown to enhance clinical outcomes.

In selected cases, a YAMIK sinus catheter may be employed to gain direct access to the sinus cavity, allowing for targeted administration of pharmacological agents—a method particularly effective in ethmoiditis. Systemic therapy typically includes antibiotics, antihistamines or other hyposensitizing agents, and supportive symptomatic care to address inflammation, infection, and patient discomfort.

Complications from the organ of vision may occur: retrobulbar neuritis of the optic nerve, inflammation of the orbital tissue (Pic.62), these conditions are considered an indication for surgical treatment.



Picture: 62. *Orbital complication - orbital tissue tumor. Chronic ethmoidal cavity inflammation*

Chronic ethmoiditis (*ethmoiditis chronica*) is a long-lasting inflammation of the mucous lining of the ethmoidal labyrinth cells.

It usually develops as a consequence of untreated or inadequately managed acute ethmoiditis. Factors such as frequent infections, the presence of adenoid hypertrophy, or a deviated nasal septum can contribute to the persistence of inflammation and the development of chronic disease. In many cases, chronic ethmoiditis leads to the formation of polyps within the middle nasal passages and the nasal cavity. These polyps vary in size and number and may partially or completely obstruct the nasal airway. Polyps arise from swollen mucosa, and their tissue typically shows diffuse neutrophilic infiltration.

Clinical presentation is often subtle. General symptoms may be mild or unnoticed, though patients usually experience some degree of impaired nasal breathing and a reduced sense of smell. Rhinoscopic examination reveals one or more gray-blue polypoid masses within the nasal cavity (Pic. 4.35).

Treatment is most commonly surgical, involving nasal polyp removal (polypotomy) or endoscopic ethmoidotomy. Endoscopic access to the ethmoidal cells is performed using a rigid endoscope. To reduce the risk of postoperative recurrence, local glucocorticoid therapy is prescribed for 3–6 months, typically using mometasone (Nasonex), fluticasone (Flixonase), or beclomethasone (Aldecin).



Picture 63 Nasal polyps

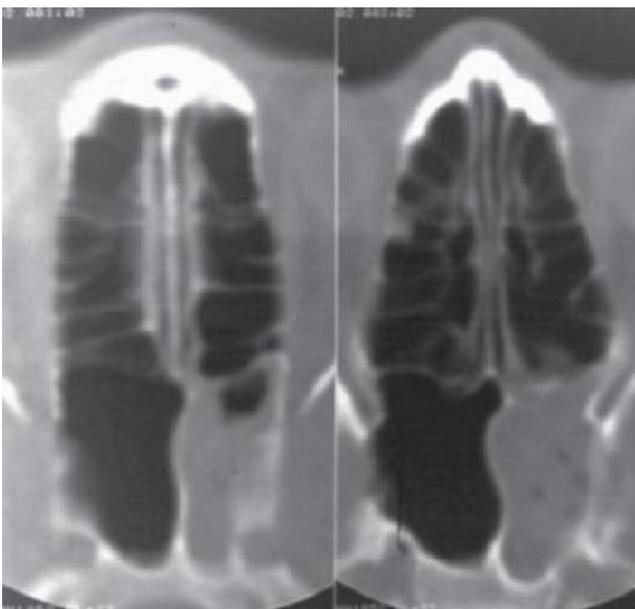
Acute and chronic inflammation of the parotid cavity

Sphenoiditis refers to inflammation of the sphenoid sinus mucosa, which can be either acute or chronic. This condition is less frequently encountered than other sinus inflammations. Patients often report a distinctive **belt-like headache**, sometimes described as a sensation of wearing a tight helmet, and in some cases, pain radiates to the nape of the neck.

Discharge from the sphenoid sinus typically drains posteriorly, making it detectable only through **posterior rhinoscopy**. In chronic cases, the inflammation may extend toward the **optic nerve** due to the extremely thin superior wall of the sphenoid sinus, potentially leading to **visual disturbances**.

Diagnosing sphenoiditis is generally more challenging than inflammation in other sinuses. **Radiographs** taken in axial and lateral projections play a critical role, while **CT scans or MRI** are considered the most reliable imaging techniques for confirming the diagnosis (Pic. 64).

If left untreated, **purulent sphenoiditis** can result in severe complications such as **meningitis, cavernous sinus thrombosis, or optic nerve damage**. Therefore, prompt and thorough diagnostic evaluation is essential whenever sphenoiditis is suspected.



Picture: 64 *Computed tomographic examination in acute sphenoiditis*

Treatment. Management of acute sphenoiditis is primarily conservative. Local therapeutic measures are employed to reduce mucosal edema, restore sinus drainage, and facilitate the clearance of inflammatory exudates from the sphenoid cavity. In cases accompanied by systemic symptoms such as fever or intoxication, general supportive treatment is indicated. Vasoconstrictive agents may be administered to decrease nasal mucosal congestion, and specialized techniques such as the Proets method or the placement of a YAMIK sinus catheter can be utilized to enhance sinus ventilation and drainage. Adjunctive pharmacological therapy typically includes broad-spectrum antibiotics, antihistamines, and analgesics to address infection, allergic components, and pain.

Surgical intervention is reserved for cases of chronic or refractory sphenoiditis. A range of approaches exists for accessing the sphenoid sinus, including both endonasal and external techniques. In recent years, endoscopic endonasal procedures employing microsurgical instruments have become the preferred method due to their precision, minimal invasiveness, and improved postoperative outcomes.

Features of sinusitis in children

In pediatric populations, the mucosa of the maxillary sinuses and the ethmoidal labyrinth exhibits significantly greater thickness compared to adults. This anatomical feature underlies the heightened secretory activity of the sinus mucosa in children, which partially accounts for the increased incidence and distinctive manifestations of inflammatory sinus conditions within this age group. Epidemiological data indicate that approximately 18% of all pediatric illnesses involve sinusitis.

Despite the relative frequency of these conditions, accurate diagnosis by clinicians is not always achieved. In children, exudative forms of sinus inflammation are most common, typically presenting as bilateral processes. Young children are particularly vulnerable to respiratory infections, in which viral activity leads to pronounced desquamation of the nasal epithelial lining, thereby facilitating the spread of pathogens into adjacent paranasal sinuses. Additionally,

hypertrophic adenoidal tissue in young children can further predispose to inflammatory involvement of the maxillary and ethmoidal sinuses.

Acute sinusitis in this population generally responds well to conservative management. However, delays in diagnosis or inadequate therapeutic intervention may result in serious complications, including orbital abscess formation or phlegmonous involvement of surrounding tissues.

Cysts of the maxillary cavity

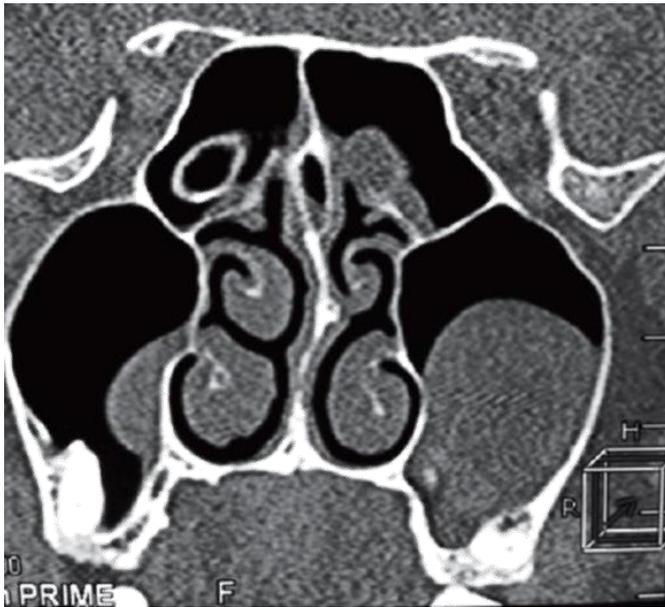
Beyond inflammatory disorders of the sinus mucosa, cystic lesions account for approximately 6–13% of all pathologies affecting the maxillary sinus, with a subset associated with odontogenic conditions. These cases necessitate careful differential diagnosis and timely therapeutic intervention. Although cysts can originate in any of the paranasal sinuses, their occurrence is most prevalent in the maxillary sinuses. When linked to dental origins, they are generally categorized as either radicular or follicular cysts.

Radicular cysts typically develop secondary to inflammation at the apex of a tooth root. Over time, the inflammatory process promotes granuloma formation, which gradually enlarges, erodes the alveolar bone, and may extend into the sinus cavity. The internal contents of these cysts may be either serous or purulent. Follicular cysts, in contrast, are relatively rare and arise from developmental anomalies of the dental follicle.

Chronic inflammation of the sinus mucosa remains the most common underlying factor for cyst formation in the maxillary sinus. Retention cysts, a specific type, result from obstruction of glandular ducts or impaired function of the ciliated epithelium lining these ducts. Such cysts frequently present as multiple lesions within the sinus, which may originate from any sinus wall, although they are often located on the inferior, alveolar wall—resembling the distribution seen in odontogenic sinusitis.

In most instances, maxillary sinus cysts are clinically silent and are discovered incidentally during radiographic examinations conducted for unrelated indications. Radiographically, these cysts typically manifest as

well-circumscribed, spherical opacities occupying variable portions of the sinus cavity.



Picture. 65 *Left maxillary sinus cyst*

While some *maxillary sinus* cysts remain asymptomatic, there are specific signs that may indicate their presence. Patients can experience a persistent, unexplained headache in the forehead, not attributable to other nasal or laryngeal conditions. Occasionally, this is accompanied by partial nasal obstruction. Many cysts are discovered incidentally during examinations of the paranasal sinuses. The most informative diagnostic tools include CT and MRI scans performed via a nasal approach. In addition to radiographic imaging, diagnostic puncture of the sinus plays a crucial role in confirming the diagnosis. In cases where the cyst is large, a needle can be inserted into the sinus to penetrate the cyst wall, allowing its fluid to be aspirated. The aspirated material is typically white-yellow and serous in nature.

Treatment of small, asymptomatic cysts is generally unnecessary. Surgical intervention is recommended for larger cysts that exert pressure on sinus walls, cause headaches, or interfere with nasal breathing. If aspiration of the cyst relieves the headache, complete removal of the cyst is advised. This involves opening the sinus and excising the cyst membrane entirely. In some cases, cysts may become suppurated, producing symptoms similar to acute sinusitis. Treatment in such cases includes removal of the purulent cyst and creation of a communication with the nasal cavity, similar to a radical *maxillary sinus* operation. The

most advanced approach for *maxillary sinus* cysts is endonasal endoscopic excision.

Ultimately, the choice of surgical method and timing depends on the patient's symptoms, the size of the cyst, and its etiology.

Tumors of the nasal cavity

Benign and malignant tumors of the paranasal sinuses are common, so oncological awareness of doctors working in peripheral areas is important. In some cases, the participation of a dentist is required for diagnosis and complex treatment.

Benign tumors

Benign neoplasms of the maxilla and ethmoid labyrinth, including osteomas, angiofibromas, and inverted papillomas, are diagnosed based on radiographic findings in conjunction with clinical manifestations such as headache and epistaxis. Surgical intervention is the treatment of choice for these lesions. Malignant tumors of the paranasal sinuses typically exhibit slow growth, often extending beyond the sinus walls, while bony erosion remains uncommon and may only be detected through radiographic imaging. Pain associated with the expansion of paranasal sinus tumors arises from stimulation of the nerve endings innervating the sinus mucosa, often presenting clinically as facial pain, or prosopalgia.

Dangerous tumors

Cancers and sarcomas of the nasal cavity are relatively uncommon, accounting for **1–3% of all malignant tumors**. Among the paranasal sinuses, the *maxillary sinus* is most frequently affected, followed by the *frontal sinus* and other cavities.

The most common malignant tumor of the paranasal sinuses is **carcinoma**, predominantly keratinizing types, while adenocarcinomas occur less frequently. Sarcomas in this region include round cell and germ cell types, as well as fibro-, chondro-, and angiosarcomas.

Tumors of the paranasal sinuses tend to invade adjacent structures rapidly, including the **skull, pterygoid-palatal fossa, orbit, and oral**

cavity. Metastasis usually occurs later, initially affecting regional lymph nodes of the jaw and subsequently the neck, detectable by palpation.

Clinical picture. Symptoms develop gradually. Patients often report progressive nasal obstruction, headaches of varying intensity, and a sensation of heaviness in the affected area of the face. Nasal discharge may start as mucous, then become mixed with blood, and in advanced stages, epistaxis can occur, sometimes accompanied by ear fullness and otalgia. The tumor in the nasal cavity is typically firm, occasionally polypoid. Extension into the *ethmoid sinus* and orbit can lead to exophthalmos and displacement of the eyeball. Malignancies of the maxillary sinus are predominantly primary in origin, with metastatic involvement of the sinus being uncommon. Metastatic dissemination from maxillary sinus tumors generally occurs at a later stage, as these neoplasms often remain localized for an extended period. Regional cervical lymph nodes are typically involved during advanced stages of the disease.

Consistent with other localized malignancies, maxillary sinus tumors are classified into stages as follows:

- **Stage I:** Tumor confined to the sinus without evidence of metastasis.
- **Stage II:** Tumor extends to the bony walls of the sinus; regional lymph node involvement may occur but is not always clinically apparent.
- **Stage III:** Tumor penetrates the sinus walls, invading adjacent structures such as the orbit, nasal cavity, or hard palate, with metastases to mandibular and cervical lymph nodes.
- **Stage IV:** Tumor extends into the facial soft tissues, cheek, and pterygopalatine fossa, accompanied by regional and distant metastases.

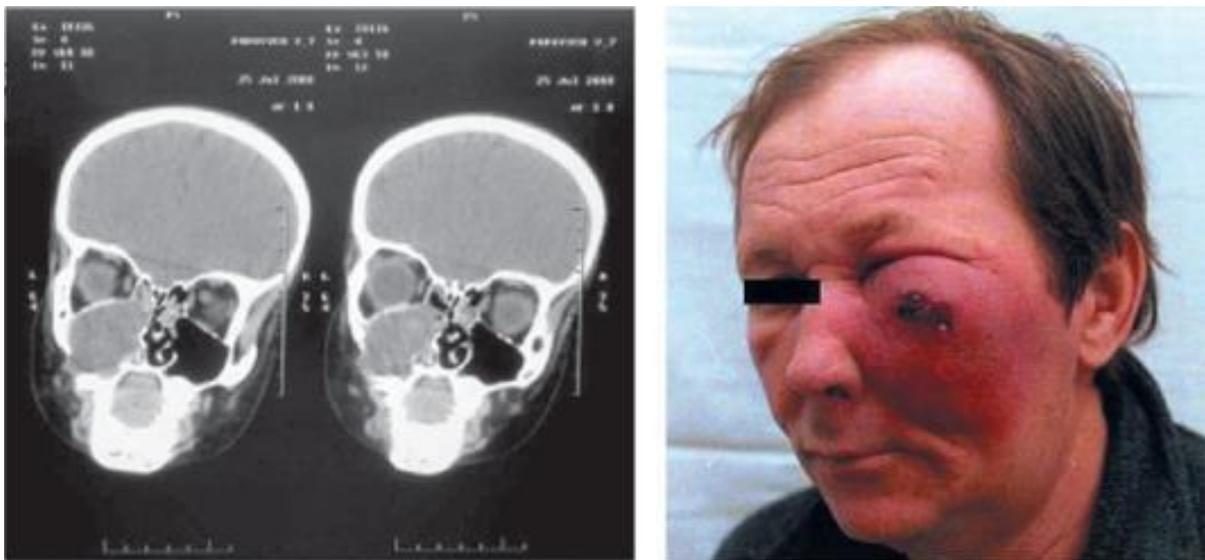
These tumors often remain clinically silent for prolonged periods, which significantly complicates early detection and timely intervention.

Symptoms vary depending on tumor location. Tumors on the **floor of the sinus** near the nasal cavity can cause pain in the upper teeth. Those on the **roof or posterior walls** often produce mandibular

neuralgia. Tumors extending to the alveolar process may present with **palatal softening and loosening of teeth** on the affected side.

Persistent **headache and orbital pain**, worse at night, are significant warning signs. As the tumor infiltrates soft tissues and approaches the medial sinus wall, **unilateral nasal obstruction** develops. The tumor may occupy part of the nasal cavity, cause nosebleeds, and provoke secondary inflammation of the nasal mucosa.

Diagnostic evaluation relies heavily on computed tomography, which is essential for determining the extent and anatomical distribution of the tumor. Definitive diagnosis is established through histopathological analysis, which also allows for precise classification of the tumor type. Material is obtained easily if the tumor extends into the nasal cavity; otherwise, a sinus puncture is required. Detection of atypical cells and bleeding from the puncture site confirms malignancy. The prognosis is generally serious and largely depends on **early detection, tumor location, and radical surgical treatment**.



Picture: 66 illustrates cancer of the maxillary sinus, characterized by homogeneous opacification of the sinus with extension of the process into the orbital region.

Management of such tumors typically involves a combination of surgery, radiotherapy, and chemotherapy. The primary objective of surgical intervention for sinus neoplasms is complete tumor excision. In certain instances, the surgical scope must be extensive to include adjacent anatomical structures, such as the orbit or the maxilla. Tumors

located along the inferior wall of the sinus generally present a more favorable localization; in these cases, tumor progression and invasion into neighboring structures tend to be limited, allowing for less radical surgical approaches. When orbital contents must be excised or when maxillary resection is performed, customized postoperative prostheses are designed to restore the tissue defect and address associated facial cosmetic deformities.

Trigeminal neuralgia

Many diseases of the paranasal sinuses and the nasal cavity can be accompanied by pain syndrome localized in different areas of the face. If the pain is associated with the inflammatory process of the sinus mucosa, for example, sinusitis, then the patient's treatment tactics will be aimed at sanitizing the sinus with conservative or surgical means. At the same time, in the practice of otorhinolaryngologist, dentist, neuropathologist, there are patients who complain of severe pain in different parts of the face, and only the joint work of these specialists helps to carry out diagnostics and complex treatment. It can occur due to pathology of the ENT organs and diseases of the dentoalveolar system, the manifestation of which is neuralgia of the second and third branches of the trigeminal nerves. Until now, there is no consensus on the causes of trigeminal neuralgia: occlusion pathology, dental diseases, effects of neurotropic viruses, atherosclerotic changes in blood vessels, bone canals through which the branches of the trigeminal nerve pass. narrowing, acute respiratory diseases. In the occurrence of trigeminal neuralgia, the role of its second branch, inflammation of the mucous membrane of the maxillary sinus and the nasal cavity is known. The clinical presentation is very typical - the pain is always paroxysmal, its duration lasts from a few seconds to a minute. The pain is very strong, burning. Patients compare it to an electric shock. Pain can appear without any reason, and in some cases - it is observed with the tension of facial muscles (laughing, yawning, etc.). Sharp pain covers the area of the face innervated by one of the branches of the trigeminal nerve, but sometimes spreads to the areas supplied by other branches of this nerve. Neuralgia of the second and third branches of the trigeminal nerve is common.

With neuralgia of the second horn, localization of pain in the nasolabial fold, wing of the nose, upper lip is characteristic. With

neuralgia of the third branch, pain is felt in the lower lip, jaw, tongue, cheek and lower jaw teeth. A common condition for neuralgia is a "trigger" zone, pressure or even a light touch, which causes severe pain. Such zones are located on the skin of the face, on the mucous membrane of the oral cavity, and in the throat. During the period of remission, these "trigger" zones disappear, and the nature of their exposure does not pass with a pain attack. An attack of severe pain is accompanied by lacrimation, a large amount of saliva, hyperemia of the skin of the face. A complete anamnesis, accurate examination by a dentist, neurologist and otorhinolaryngologist, distinguishing the pain associated with the usual inflammatory process in the nasal cavity and paranasal sinuses from the tumor, trigeminal neuralgia, and rational treatment orders. It should be noted that the use of drugs for trigeminal neuralgia does not bring relief; this serves as another diagnostic sign.

Treatment. Conservative treatment is carried out in the course of inpatient treatment through repeated courses using drugs such as carbamazepine (finlepsin, tegretol), trimethadione (trimetin). Both dentists and otorhinolaryngologists perform surgical treatment. Such treatment is based on the resection of the branch of the trigeminal nerve as far as possible from the site of the excretory opening, in order to separate the peripheral and central parts of the nerve, The receptor section in the "trigger" zone should not be affected by the irritation that leads to the onset of an attack.. Otorhinolaryngologists approach the infraorbital nerve through the maxillary cavity, open the wall of the canal along the entire length and divide the nerve, which is then resected. They also act with neuralgia of the third branch of the trigeminal nerve - the channel is opened through the oral cavity, the nerve fiber is pulled as far as possible and resected.

Surgical treatment does not allow to get rid of painful attacks in any case, recurrence of pain after resection can occur at different times, because the nerve trunk again reaches the excretory hole and approaches the skin receptors. In cases where the basis of neuralgia of the second branch of the trigeminal nerve is an inflammatory process of the maxillary cavity, they resort to surgical cleaning of the sinus.

The condition of the ENT organs with cleft lip and throat

The frequency of cleft lip and cleft palate takes one of the first places among congenital defects (Pic. 67). Up to 5,000 children with this developmental anomaly are born in the country every year. This fact has long attracted the attention of specialists, mainly dentists who have

developed complex programs for the rehabilitation of patients. Optimum conditions were determined, surgical intervention techniques were developed to achieve good cosmetic and functional results. But the changes in the ENT organs, accompanied by such anomalies, were successfully performed. Cheilouranoplasty after, it does not disappear by itself and requires the active intervention of an otorhinolaryngologist in the treatment process, because in most cases cleft lip and palate, nasal cavity, throat, larynx and middle ear pathologies are detected.



Picture:67 *Cracked lip and hard palate*

Often, a sore throat is accompanied by a curvature of the nasal septum due to chronic catarrhal rhinitis.

The progressive enlargement of the inferior nasal conchae can be regarded as a compensatory mechanism, which serves to limit the penetration of food particles into the upper regions of the nasal cavity. However, this compensatory hypertrophy simultaneously contributes to a persistent impairment of nasal breathing, compelling the patient to rely predominantly on oral respiration. This, in turn, exerts additional stress on the mucous membranes of the oral cavity, pharynx, and larynx, leading to structural and functional alterations. In such cases, alongside the characteristic manifestations of chronic pharyngitis, clinicians frequently observe hypertrophy of the palatine tonsils and recurrent or chronic tonsillitis.

A number of deviations from the norm were also noted in the nasal part of the throat. Because the upper nasal septum is not developed, the forehead is enlarged. The laryngeal tonsil, which is located in the dome

of the nasal part of the larynx, is mainly hypertrophied, which also maintains the tendency of the nasal mucosa to stick together and make breathing through the nose difficult.

More than 50% of patients with cleft lip and palate have inflammatory diseases of the middle ear along with permanent hearing loss. First of all, it leads to a violation of the ventilation and drainage function of the auditory tube caused by the deformation of the hard palate, the muscles that tighten and lift the soft palate, adenoids.

Thus, the treatment of patients with cleft lip and palate is not limited to repairing the lips, restoring the integrity of the palate, but should be combined with a qualified otorhinolaryngological examination in the pre- and post-operative period, because the function of the nasal cavity is automatically restored after uranoplasty. no recovery. In cases where a decision is made to remove a hypertrophied pharyngeal tonsil in cleft patients, adenotomy should be done 7-8 months before uranoplasty.

SELF-STUDY QUESTIONS

1. Modern methods of examination of the nose and nasal cavity

Currently, endoscopic functional microsurgical techniques are extensively utilized in nasal and sinus surgery. The guiding principle of these tissue-preserving approaches is the maximal conservation of the nasal mucosa and intranasal structures, achieved through limited, targeted interventions in critical regions of the lateral nasal wall. The



primary objective of such surgery is to restore physiological airflow and the patency of the drainage pathways while minimizing alterations to the native anatomical structures.

Picture. 68. Workplace of an otolaryngologist

For the examination of the nasal cavity and paranasal sinuses, as well as for diagnostic and surgical interventions in these regions, endoscopic systems produced by manufacturers such as **Karl Storz**, **Richard Wolf**, and **Karl Zeiss** are commonly employed. These include rigid and

flexible endoscopes, as well as long-working-distance surgical microscopes. Endoscopes with diameters of 2.7 mm and 4 mm, and angular fields of view of 0°, 30°, 70°, 90°, and 120°, facilitate detailed inspection of the anatomically complex nasal cavity, paranasal sinuses, and nasopharynx. In compliance with modern standards, ENT facilities should be equipped with advanced endoscopic technology supporting endomicroscopy, endophotography, video recording, and electronic data storage (see Pic. 5-1).

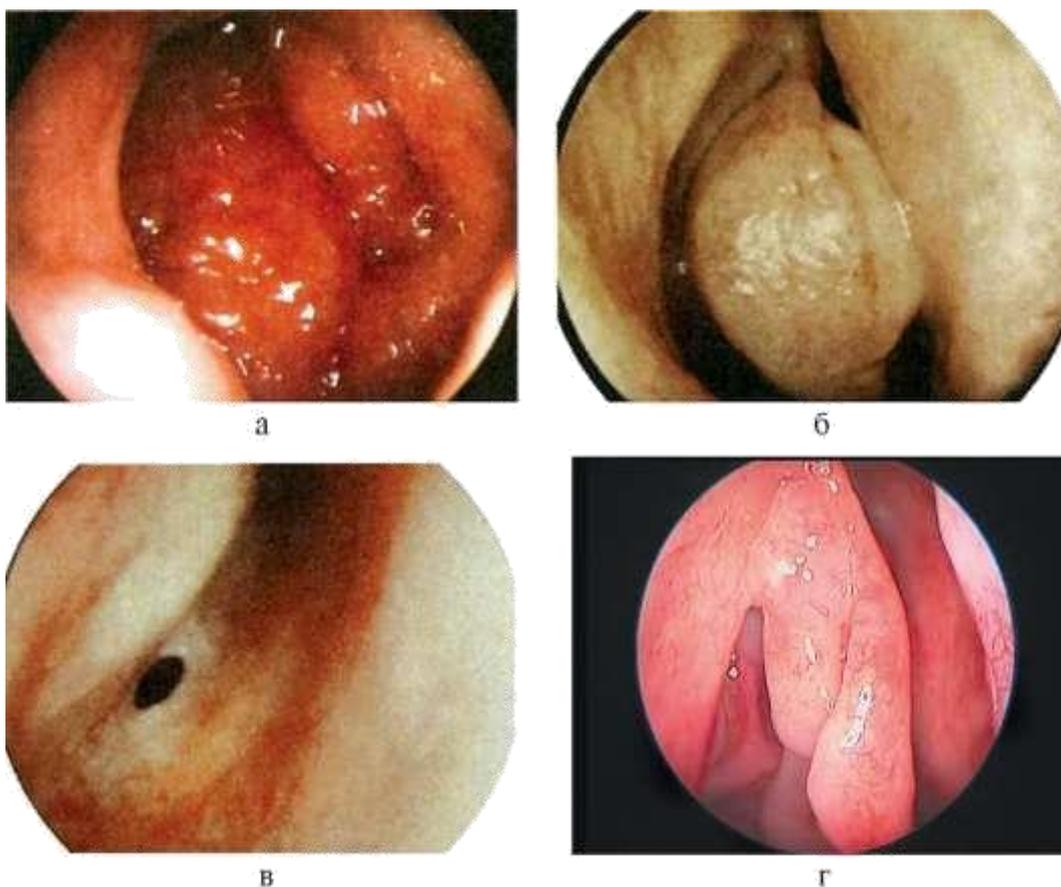
The indications and methodology of diagnostic endoscopy for the nasal cavity and paranasal sinuses differ significantly from traditional diagnostic and surgical techniques. The primary aim of diagnostic endoscopy is to detect early pathological changes in the mucosa and to perform minimally invasive interventions, thereby preserving the integrity of the mucosa, nasal conchae, and other anatomical structures. The indications for diagnostic endoscopy are broad and encompass nearly the full spectrum of nasal and sinus pathologies, including:

- Impaired nasal breathing;
- Persistent rhinorrhea;
- Olfactory dysfunction;
- Recurrent epistaxis;
- Nasal cavity tumors;
- Polypoid sinusitis;
- Eustachian tube dysfunction;
- Headache of unclear etiology;
- Preoperative evaluation and postoperative monitoring of ongoing therapy;
- Need for photographic or video documentation.

Prior to endoscopy, nasal cleansing is required, and in some cases, topical decongestants and local anesthesia of the mucosa are applied. The rigid endoscope with a 0° field of view remains the most commonly utilized instrument for routine examination.

Endoscopic examination of the nasal cavity consists of three main stages.

- **Comprehensive panoramic examination of the nasal cavity and passages:** The endoscope is initially introduced to provide a general overview of the nasal passages. It is then advanced along the lower portion of the nasal cavity toward the nasopharynx. During this process, the mucosa of the lower nasopharynx is carefully evaluated. In some cases, the opening of the nasolacrimal duct may be visualized. If the patient has a history of sinusitis requiring prior puncture, assessment of the lower nasal passage and maxillary sinus is indicated, and any potential leaks can be observed. Upon withdrawing the endoscope, the posterior extremities of the lower nasal passages, the opening of the auditory (Eustachian) tube, the nasopharyngeal dome, and the presence of adenoid tissue are systematically assessed (see Fig. 69 ad).
- **Examination of the middle nasal concha and passage:** The endoscope is then guided toward the middle nasal concha to evaluate the middle nasal passage. In certain cases, gentle medial subluxation of the middle turbinate may be necessary to improve visualization. Structures such as the uncinate process, ethmoidal bulla, semilunar hiatus, and infundibulum are carefully inspected. Hyperplasia of the mucosa overlying the middle nasal concha and obstruction of the osteomeatal complex are also evaluated (see Fig. 69 e). Occasionally, the natural ostium of the maxillary sinus can be distinguished, though it is often obscured by the free edge of the uncinate process and not directly visible in standard inspection.



Picture. 69. Endoscopic examination of the nasal cavity: a - grade III adenoid vegetations; б - osteomeatal complex, hyperplasia of the anterior part of the middle nasal concha; c - opening of the sphenoidal sinus; d - a polyp in the osteomeatal area blocking the middle nasal passage.

- **Final stage – assessment of the upper nasal passages and olfactory fissure:** During the concluding phase of endoscopic examination, attention is directed to the upper nasal passages, including the olfactory fissure. Occasionally, the excretory openings of the posterior ethmoidal cells and the superior nasal concha can be visualized. To enhance the detail of inspection, endoscopes with viewing angles of 30° and 70° are recommended.

Indications for diagnostic maxillary sinus endoscopy (sinusoscopy) include:

- Precise evaluation of suspected maxillary sinus injuries;
- Removal of foreign bodies;

- Performance of various therapeutic interventions (therapeutic sinusoscopy).

The anterior wall approach is most commonly employed due to its technical simplicity. The procedure is performed under local infiltration anesthesia targeting the infraorbital branch of the trigeminal nerve and the soft tissues surrounding the canine fossa.

Using a specialized 4 mm trocar, the anterior wall of the maxillary sinus is carefully penetrated with rotational movements, typically at the level between the roots of the third and fourth upper teeth. Subsequently, endoscopes with viewing angles ranging from 0° to 70° are introduced through the created access, allowing comprehensive examination of the sinus walls, evaluation of the mucosal condition, tissue sampling for biopsy, and the opening or removal of cystic formations.

Upon completion of the procedure, the trocar sleeve is removed using the same gentle rotational technique. The puncture site is generally left unsutured, and patients are advised to avoid forceful nose blowing for 5–6 days to prevent complications.

Methods of operations

Several techniques are employed in endoscopic endonasal surgery. The most widely adopted approach is the Messerklinger method, in which surgical intervention proceeds in a stepwise manner, sequentially opening the paranasal sinuses from anterior to posterior. In contrast, the Wiegand technique initiates the procedure in the deeper regions of the nasal cavity, beginning with the sphenoidal sinus, followed by the posterior and middle ethmoidal cells, then performing infundibulotomy, and ultimately addressing the anterior ethmoidal cells, thereby advancing from posterior to anterior.

According to Wiegand, this posterior-to-anterior approach is more radical, as it typically involves complete opening of the ethmoidal labyrinth cells and establishing communication with the maxillary sinus beneath the inferior nasal concha in nearly all forms of sinusitis.

A defining characteristic of endoscopic surgery is the continuous visual control afforded by the endoscope, which ensures high precision and functional effectiveness of the interventions. This approach minimizes intraoperative bleeding and allows preservation of healthy, unaffected mucosa, thereby optimizing surgical outcomes.

2. Balloon sinusoplasty

Balloon sinusoplasty is recognized as the most effective and safe method of surgical treatment of acute and chronic sinusitis. Access is performed endonasally under endoscopic observation, which allows to preserve the natural anatomy of the sinuses and minimally injure the mucous membrane. Through a natural fistula with the nasal cavity, the nasal sinus is examined and the fistula expands without disturbing its anatomy and integrity. The accuracy of delivery of a special balloon to the affected sinus is controlled by the surgeon using a light source located at the tip of the guiding catheter. The light in the operating room is turned off for a few minutes to better visualize the exact beat in the sinus. A balloon is inflated with the help of fluid, and the blocked or narrowed anastomosis expands.

As a result of balloon sinusoplasty, adequate ventilation and drainage of the nasal sinus is ensured and its functions are restored. The absence of incisions in the nasal cavity dramatically shortens the recovery period, reduces the risk of postoperative and intraoperative complications, and also prevents sinus hyperventilation that occurs after classical endoscopic interventions. Minimally invasive surgery is performed in an outpatient setting when seeking medical help early. Balloon sinusoplasty can be used by the doctor as a separate method for the treatment of acute or chronic sinusitis or in combination with traditional methods of functional paranasal sinus endoscopy.

3. Allergic rhinitis and their modern treatment methods

The procedure involves subcutaneous administration of small, gradually increasing doses of the allergen and is performed during periods of disease remission, typically repeated three times.

Nonspecific hyposensitization is indicated in cases where the allergen remains unidentified or in instances of polyallergy. This approach aims

to alleviate allergic manifestations using pharmacological agents. Commonly employed medications include:

- Oral antihistamines;
- First-generation antihistamines such as diphenhydramine and chloropyramine (suprastin);
- Second-generation antihistamines, including astemizole (Gismanal), loratadine (Claritin), terfenadine (Histadine), fexofenadine (Telfast), cetirizine (Zyrtec), ebastine (Kestin), and desloratadine (Erius).

Additional agents recommended may include pipolfen, fenkarol, tavegil, intal, zaditen, 5% aminocaproic acid, 10–30% sodium thiosulfate, calcium supplements, 5% ascorbic acid, atropine, and belladonna derivatives (Patients diagnosed with vasomotor rhinitis should undergo a comprehensive clinical assessment prior to initiating treatment. Any latent infectious foci should be identified and addressed, particularly involving the teeth, palatal folds, paranasal sinuses, stomach, liver, gallbladder, and intestines.

Management of allergic rhinitis encompasses several strategies:

- Implementation of personal protective measures to minimize allergen exposure;
- Administration of specific immunotherapy (SIT);
- Application of nonspecific hyposensitization therapy;
- Surgical interventions to eradicate purulent or structural foci;
- Local corticosteroid therapy and reflexotherapy.

A fundamental principle in the pathophysiological management of allergic rhinitis is the removal or avoidance of the allergenic trigger. Patients are advised to temporarily relocate during peak pollen seasons, utilize respiratory masks or home air filtration systems, avoid keeping pets and aquariums, remove carpets and soft toys, maintain rigorous household hygiene, and regularly launder clothing and linens.

Specific immunotherapy can only be initiated after precise identification of the causative allergen in an allergology clinic. belloid, bellaspon).

In mild forms, local antihistamines such as azelastine (Allergodil) or levocabastine (Histimet) may be applied. Topical therapy may involve diphenhydramine, hydrocortisone, or 1% cromoglycate (Intal) ointments, while aerosol formulations like becotide are also used. Physiotherapeutic interventions, including electrophoresis with diphenhydramine, cromoglycate, or calcium chloride, phonophoresis with hydrocortisone or splenin ointments, aeroionotherapy, UHF therapy, magnetotherapy, and helium–neon laser treatment, may provide adjunctive benefit.

Topical corticosteroids are particularly effective for patients with prominent nasal obstruction. These agents penetrate cellular membranes, inhibit histamine release from mast cells, reduce vascular permeability, and exert anti-inflammatory effects. Contemporary intranasal corticosteroids include fluticasone (Flixonase), beclomethasone (Aldecin/Becanase), mometasone (Nasonex), and budesonide (Rhinocort), typically administered two to four times daily over extended periods of four to six months.

In severe allergic rhinitis, systemic corticosteroids such as prednisolone or methylprednisolone may be prescribed for short-term use. While vasoconstrictive agents are frequently utilized to relieve nasal obstruction, their efficacy is temporary, and prolonged use beyond two weeks may exacerbate disease progression.

Surgical interventions should ideally be scheduled during disease remission and outside peak pollen seasons. Bilateral hypertrophy of the nasal conchae can be addressed via galvanocaustic, cryogenic, or ultrasonic techniques, with additional procedures such as submucosal vasotomy, lower conchotomy, polyp removal, or correction of nasal septal deviations performed as indicated. Six to ten days prior to surgery, nonspecific hyposensitization is recommended, followed by extended intranasal corticosteroid therapy postoperatively.

Reflexotherapy, including acupuncture and electroacupuncture, may complement standard management. In selected cases, 0.5–2 ml of novocaine solution is injected into the mucosa of the middle nasal concha.

Patients are advised to organize daily activities to allow for adequate rest and exposure to fresh air and to consider recuperative stays at health resorts, such as Aq-Tash or Zomin, to support overall recovery.

4. Ozena

Nasal congestion or ozena (ozaena) is a severe form of atrophic rhinitis, and the atrophic process in the nasal cavity spreads not only to the mucous membrane, but also to the bone tissue. The patient's nose has a peculiar smell. Mumps is more common in young women.

Etiology. There are social, anatomical, inflammatory, focal, infectious, neurodystrophy, and endocrine theories of the origin of measles. Among them, the theories of infection and neurodystrophy are widespread.

According to the theory of infection, the microflora in the nasal cavity is the primary cause of the development of sinusitis. Under the influence of microbial toxins, tissue proteins are broken down, releasing foul-smelling gases such as indole, skatole, and hydrogen sulfide.

Klebsiella (Klebsiella ozaenae), a member of the family of enterobacteria, is often found in the nasal cavity of patients with the common cold, and there are three types of it:

- 1) Klebsiella rhinoscleromatis - causes scleroma disease;
- 2) Klebsiella ozaenae - causes ozena;
- 3) Klebsiella pneumoniae - causes diseases such as sepsis, zotiljam, acute intestinal diseases, pyelonephritis, peritonitis, meningitis.

In addition, corynebacteria and protean microbes were also detected in the ozena.

According to the theory of neurodystrophy, the development of the dystrophic process in the nasal cavity is caused by disturbances in the central autonomic nervous system and the endocrine system or sympathetic innervation.

Signs. The patient complains that the mucous membrane of the nasal cavity dries up, a large number of crusts form in it, a bad smell comes from the nose (the patient himself usually does not feel this smell), it is difficult to breathe through the nose, he does not feel the smell. Due to the bad smell coming from the patient's nose, the surrounding people try to stay away from him, which has a negative effect on the patient's psyche. Hyposmia first occurs as a result of pods covering the olfactory area, and later due to atrophy of olfactory receptors. Sometimes the patient develops anosmia.

Frontal rhinoscopy shows that the nasal cavity is filled with liver or yellow-green crusts, which spread to the nasopharynx and lower respiratory tract. After the peels are removed, the nasal cavity expands so much that even the back wall of the nasopharynx, the laryngeal openings of the auditory tube, and the tubules of the tube are visible.

It should be distinguished from simple atrophic rhinitis and rhinitis developed in the wound.

In simple atrophic rhinitis, the atrophy process spreads only to the mucous membrane, and the patient's nose does not smell bad. In the case of rhinitis developed in the wound, the patient's nose has a bad smell, crusts accumulate in the nasal cavity, it is determined that there is a gum ulcer and a hole in the bone part of the nasal septum, and the Wasserman reaction is positive. In most cases, acne occurs in women of puberty. In addition, ozena should be distinguished from tuberculosis, wound, scleroma, Wegener's granulomatosis.

Treatment. Symptomatic treatment measures are carried out step by step. In the 1st stage, the nasal cavity is cleaned of crusts. It uses 2% sodium bicarbonate, 1% hydrogen peroxide, 0.1% potassium permanganate, 1% dioxidine. The nasal cavity must be washed daily with the following solutions: isotonic solution of sodium chloride with iodine; 1/2 teaspoon of the mixture (10 ml of salicylic acid, 20 g of sodium chloride, 20 g of sodium bicarbonate) per 1 glass of water.

Suppositories made from Solodov's chlorophyll-carotene paste also have a bactericidal and deodorizing effect and are effective in treatment. Suppositories are inserted into the nostrils once a day for 3-4 weeks after cleaning the inside of the nose. Treatment is repeated 3-4 times a year.

At the 2nd stage, a cotton swab soaked in iodine-glycerin (or 1% Lugol's solution), Vishnevsky, Kalanchoe ointments and 5% synthomycin emulsion is inserted into the nose for 2-3 hours.

Rp: Succi Kalanchoes 20.0

Furasolidone

Novocain aa 0.125

Mfuguentum

Nose in the form of cotton embroidery
to insert into.

In the 3rd stage, the patient is prescribed physiotherapeutic procedures: aeroionotherapy, inhalations with drug aerosols. Chamomile decoction,

trypsin, ribonuclease, collagenase, streptomycin sulfate, and tetracycline hydrochloride solutions are used for inhalation.

In the 4th stage, general therapy is given to the patient; antibiotics against ozena klebsiella (streptomycin sulfate, kanamycin, gentamicin, levomycetin succinate), S and V drugs, 1% nicotinic acid are injected into the muscle. Injection of 0.5 - 1% - 5.0 ml of novocaine solution under the mucous membrane of the nasal concha also gives a good result (15 - 20 procedures in total).

Iron-containing drugs (ferrumlek, ektofer), autohemotherapy treatment are recommended to the patient, endocrine system pathology treatment measures are carried out.

Surgical procedures performed in the nasal cavity are performed with the aim of narrowing the nasal cavity. For this, autograft (bone, rib cage) or synthetic grafts (lafsan, Teflon, kapron, acrylic plastic, polyurethane, polyphosphazene) are placed under the mucous membrane of the nasal septum or side wall of the nose, methods of partially or completely closing the nostrils are used. Before surgery, the above-mentioned local measures should be carried out in the nasal cavity for 12-14 days.

5. Neoplastic diseases of the organs of the nose and nasal cavities

Benign tumors of the nose include papilloma, fibroma, hemangioma, angiofibroma, adenoma, chondroma, osteoma, and lipoma.

Signs. On the side where the tumor is located, the patient has a sharp difficulty in breathing through the nose, hyposmia or anosmia, sometimes bleeding from the nose, in the late stages of the facial skeleton, headache, displacement of the eyeballs, decreased visual acuity. Nasal endoscopy, examination and palpation with a probe, radiography, computed tomography, angiography of the carotid artery system, and histological examination help to make a diagnosis.

Bleeding polyp resembles an angiofibroma in its structure and has a wide leg, is usually located in the upper part of the nasal septum, often does not increase in size, sometimes increases and protrudes from the nasal cavity. Polyp bleeds often, blood flows in small quantities. The disease often occurs in pregnant women and patients with high blood pressure.

Diagnosis is not difficult and is based on patient complaints, initial causes of the disease, rhinoscopy and histological examination.

Treatment: The polyp is removed intermittently (the unremoved part may recur). A broad-legged polyp is excised together with the

surrounding tissue, the edge of the wound is treated with the help of galvanocoustics, ultrasound or laser, and the cause is treated.

Nasal fibroma is rare and is usually located in the nasal passage, nasopharynx and external nasal area. Diagnosis is not difficult. The tumor is treated surgically.

Nasal osteoma is more common in patients aged 15-25 and grows slowly. Usually, osteoma grows on the wall of the forehead and cranial cavities, sometimes from the maxillary cavity. A small osteoma is invisible to the eye and does not bother the patient. In cases where cosmetic, functional or other disorders do not develop, the patient is under the supervision of a doctor for a long time. When the osteoma grows relatively quickly, it is removed during surgery. Sometimes, if located in the cerebral wall of the frontal cavity, osteoma, despite its small size, can cause severe headaches. After ruling out other causes, the osteoma is removed. In some cases, the osteoma can grow and spread to the cavity of the skull, nose, and eye socket.

Signs. The structure of the patient's facial skeleton is disturbed, headaches occur, visual acuity decreases, nasal breathing and olfactory functions are disturbed.

The disease should be distinguished from chondroma, osteosarcoma and exostoses.

Treatment: Osteoma is treated using a surgical method.

6. Specific diseases of the organs of the nose and nasal cavities

Scleroma is a chronic infectious disease, manifested by the formation of specific infiltrates in the upper respiratory tract. The disease is caused by *Klebsiella scleroma* (Frisch-Volkovich bacillus).

Distribution. Large endemic foci of scleroma are located in Ukraine, Belarus, Italy, Germany, Yugoslavia, Czech Republic, Slovakia, Indonesia, Brazil, Guatemala, Mexico and Egypt. A total of 10,000 patients with multiple sclerosis have been recorded on earth. Most often, scleroma occurs in men and women aged 16-30 years, sometimes older. The disease is widespread among the rural population (87.5-85%).

Etiology. Frisch-Volkovich bacterium (*Klebsiella rhinoscleromatis*) differs from other capsular bacteria. The detection of antimicrobial antibodies in the Bordet-Jangou serological reaction of a patient with scleroma and the change in the titer of the serological reaction after

treatment with streptomycin confirm that the development of scleroma was caused by the Frisch-Volkovich bacillus.

Scleroma first appears in the nasal cavity, its mucosa and mucous membrane with catarrhal changes, with the first symptoms in 3-5 years. Illness of family members, relatives, spouses, people who moved to an endemic area confirms that scleroma is an infectious disease. Frisch-Volkovich bacillus in the infiltrative form of scleroma and in the period of relapse is spread by droplet or contact route (through hands, towels, handkerchiefs, dishes, bed linen).

Clinical signs. At the initial stage of the disease, the patient complains of thick discharge from the nose, flu, difficulty in breathing through the nose, dryness of the nasal cavity, accumulation of crusts. Later, the patient has a hoarse voice, cough, and shortness of breath. Disorders of breathing develop slowly, so the patient adapts to it and does not turn to the doctor. In addition, the patient complains of general weakness, rapid fatigue, headache, loss of appetite.

The process of scleroma is often located in the anterior part of the nasal cavity, choanae, vocal cavity of the larynx, bifurcation of the trachea and bronchi. The process develops simultaneously in 2-3 parts of the respiratory tract, sometimes in all parts.

Scleroma process develops in the nasal cavity in 93-95%, in the larynx in 62-73%, in the nasopharynx in 62-67%, in the trachea in 13-27%, in the larynx in 5.4-13.9%, in the bronchi in 4-16% of cases. It is symmetrically positioned and prone to injury, fragmentation, and scarring.

Group 1 - etiotropic therapy - therapy against the causative agent of scleroma;

Group 2 - pathogenetic therapy - elimination of specific changes in the respiratory tract;

Group 3 - sanogenetic therapy - increasing the body's resistance to disease.

Control questions and assignments

- 1 What are the most common causes of nosebleeds?
- 2 Where is the most common source of nosebleeds?
- 3 How is nosebleed classified according to the amount of blood loss?
- 4 Show clinical signs of significant blood loss.
- 5 What are some ways to stop a nosebleed?
- 6 How can the external and internal carotid arteries be distinguished when ligating the external carotid artery?
- 7 Name the main provoking factors of acute rhinitis.
- 8 How are the different forms of chronic rhinitis classified?
- 9 How does the catarrhal form of chronic rhinitis differ from hypertrophic rhinitis?
- 10 Why should conchotomy be rarely performed for chronic hypertrophic rhinitis?
- 11 How can we explain the difficulty of nasal breathing in a patient with atrophic rhinitis, who has a sharp increase in the damage of the nasal passages?
- 12 Show the main clinical signs of Ozena. List the shapes
- 13 What are the similarities and differences between allergic and vasomotor rhinitis? When is surgical treatment indicated for vasomotor rhinitis?

Test questions

1. What are the absolute indications for radical surgery on the paranasal sinuses?
 - A. rhinogenic intracranial complication
 - B. chronic maxillary sinusitis
 - C. acute hemisinusitis
 - D. polypous rhinosinusitis
 - E. sinus suppuration

2. Through which nasal passage is a puncture of the maxillary sinus performed?
 - A. middle
 - B. upper
 - C. common
 - D. inferior
 - E. puncture is performed through the anterior wall

3. Into which nasal passages is the nasal cavity divided?
 - A. inferior, medial, posterior, anterior;
 - B. posterior, anterior, middle, middle, common;
 - C. anterior, middle, upper, common;
 - D. respirator, olfactor
 - E. lower, middle, upper, total;

4. What are the nasal meatus in the nasal cavity?
 - A. lower, upper
 - B. lower, middle, upper
 - C. inferior, middle, superior
 - D. lateral, inferior, middle, superior.
 - E. medial, inferior, middle, superior.

5. What opens into the upper nasal meatus?
 - A. lacrimal nasal canal
 - B. frontal sinus, posterior commissures of the lattice labyrinth
 - C. frontal sinus, sphenoid sinus
 - D. posterior ethmoid sinus and the sphenoid sinus
 - E. posterior commissures of the lattice labyrinth

6. What opens into the middle nasal meatus?
 - A. lacrimal nasal canal
 - B. frontal sinus, maxilar sinus, anterior ethmoid sinus
 - C. frontal sinus, sphenoid sinus
 - D. posterior ethmoid sinus and the sphenoid sinus
 - E. posterior commissures of the lattice labyrinth

7. What happens to air as it passes through the nasal cavity?
 - A. warms
 - B. is dried
 - C. decontaminated, warmed
 - D. is moistened, warmed, purifie
 - E. cleaned, moistened, dried

8. What makes the air warm in the nasal cavity?
 - A. the abundance of blood vessels, the presence of cavernous tissu
 - B. slowing the speed of air movement in the nasal cavity
 - C. the presence of cylindrical flashing epithelium, slowing the rate of airmovement in the nasal cavity
 - D. slow air velocity in the nasal cavity, abundance of blood vessels, presence of cavernous tissue
 - E. presence of nasal septum and cylindrical flickering epithelium, slowing of air velocity in the nasal cavity

9. What are some methods of viewing the nasal cavity?
 - A. anterior rhinoscopy, posterior rhinoscopy
 - B. direct rhinoscopy, indirect rhinoscopy
 - C. direct rhinoscopy, indirect rhinoscopy, anterior rhinoscopy, posterior rhinoscopy
 - D. direct rhinoscopy, indirect rhinoscopy, middle rhinoscopy
 - E. anterior rhinoscopy, posterior rhinoscopy, upper rhinoscopy

10. What anatomical structures border the nasal cavity?
- A. anterior cranial fossa, orbit, oral cavity
 - B. posterior cranial fossa, orbit, oral cavity
 - C. posterior cranial fossa, anterior cranial fossa, orbit, oral cavity
 - D. orbit, oral cavity
 - E. orbit, oral cavity, middle cranial fossa
11. What are the choanas limited by?
- A. vomer, the inner plate of the wing process and the body of the basilar bone, the horizontal plate of the palatine bone
 - B. palatine palatine arches
 - C. quadrangular cartilage, scaphoid
 - D. horizontal lamina of the palatine bone, condyle
 - E. inner plate of the wing process and body of the sphenoid bone, horizontal plate of the palatine bone
12. What opens into the middle nasal passage?
- A. the lacrimal nasal canal;
 - B. the maxillary sinus, frontal sinus, anterior ethmoid sinus;
 - C. maxillary sinus, frontal sinus, middle commissures of the ethmoid sinus;
 - D. maxillary sinus, frontal sinus, anterior commissures of the ethmoid sinus;
 - E. maxillary sinus, frontal sinus, commissures of the ethmoid sinus;
13. What are some reflex phenomena in the nasal cavity?
- A. sneezing, lacrimation
 - B. sneezing, lacrimation, rhinorrhea
 - C. tinnitus, sneezing, rhinorrhea
 - D. rhinorrhea, sneezing
 - E. sneezing, lacrimation, rhinorrhoea, runny nos
14. What factors can cause nasal septal deformity?
- A. frequent inflammatory processes, congenital
 - B. physiological, traumatic, compensatory
 - C. congenital, acquired

- D. birth trauma, uneven skull growth
- E. traumatic, mixed

15. How much moisture does the nasal cavity mucosa produce during the day?

- A. about 800 ml
- B. about 3000 ml
- C. about 500 ml
- D. about 200 ml
- E. about 50 ml

16. Forms of vasomotor rhinitis?

- A. acute, chronic
- B. catarrhal, hypertrophic, atrophic
- C. allergic, neurovegetative
- D. purulent, catarrhal, polyposis.
- E. catarrhal, purulent, polyposis-purulent

17. A complication of the allergic form of vasomotor rhinitis?

- A. nasal breathing disorders, chronic runny nose
- B. intracranial
- C. orbital
- D. acute purulent sinusitis
- E. formation of polyps in the nasal cavity

18. What are causes of nasal furuncle?

- A. infectious factor (staphylo-, streptococcus)
- B. gastritis
- C. trauma
- D. stress, trauma
- E. deformation of the external nose, atrophic processes in the nasal cavity

19. Symptoms of nasal furuncle?

- A. disturbance of nasal breathing, nasal discharge, headache, periodic pain in the mustache
- B. headache, impaired nasal breathing, purulent discharge from the nasal cavity

- C. sharp nasal pain, cone-shaped infiltrate, elevated ESR, leukocytosis, increased body temperature, enlarged and painful regional lymph nodes
- D. increased body temperature, nasal pain, nasal breathing disorders, nasal discharge
- E. nasal breathing difficulties, nasal rhinorrhea, paroxysms of sneezing

20. Types of acute rhinitis?

- A. acute catarrhal rhinitis, acute catarrhal rhinopharyngitis, acute traumatic rhinitis
- B. acute catarrhal rhinitis, acute catarrhal rhinopharyngitis.
- C. acute rhinitis stages 1, 2, 3
- D. acute runny nose, allergic rhinitis, neurovegetative rhinitis
- E. acute catarrhal rhinitis, hypertrophic rhinitis

21. What plays a role in the etiology of acute catarrhal rhinitis?

- A. reduction of local and general reactivity of the organism, activation of microflora at the nasal cavity, general and local hypothermia
- B. occupational factors, infectious diseases
- C. infectious diseases, microtraumas
- D. bacteria, viruses
- E. infectious diseases, allergens, decreased local reactivity of the organism

22. Treatment of acute rhinitis in stage 3?

- A. abortive method
- B. antibiotic therapy, antiseptic, vasoconstrictive nasal drops
- C. nasal vasoconstrictor drops, UVO, UHF, alkaline oil inhalations, antibiotic
- D. antibiotics, sulfonamides, vasoconstrictive nasal drops
- E. immunomodulators, antiseptics, vasoconstrictive nasal drops

23. What are forms of chronic rhinitis?

- A. mural, central, posterior
- B. catarrhal, neurovegetative
- C. vasomotor, catarrhal, neurovegetative
- D. catarrhal, hypertrophic, atrophica, sicca, caseosa

- E. upper, middle, lower
24. What are complaints of patients with chronic catarrhal rhinitis?{
- A. periodic difficulty in nasal breathing, nasal discharge, headache
 - B. difficult nasal breathing, cough, headache
 - C. nasal discharge, sneezing, difficulty in nasal breathing.
 - D. rhinorrhea, general weakness, drowsiness
 - E. difficult nasal breathing, nasal pain, headach
25. What treatment methods are used for chronic hypertrophic rhinitis?{
- A. chemocautic, galvanocautic, cryotherapy, conchotomy
 - B. conservative, surgical
 - C. antibiotics, sulfonamides, vasoconstrictive nasal drops
 - D. abortive, antibiotic therapy, vasoconstrictive nasal drops
 - E. vasoconstrictive nasal drops

Situational issues

1. A 30-year-old patient complains of headache, general weakness, elevated body temperature, difficulty breathing through the nose, and purulent discharge from the right half of the nose. Anterior rhinoscopy shows hyperemia and swelling of the mucous membrane of the left half of the nose, and pus in the middle nasal passage. Posterior rhinoscopy shows There is no hyperemia or swelling of the mucous membrane. The task is to determine which paranasal sinuses may be affected in the patient.

- A. Right maxillary, frontal, anterior and middle cells of the ethmoid labyrinth
- B. Right maxillary, frontal and main
- C. Right all paranasal sinuses
- D. Right frontal and maxillary
- E. Right maxillary, frontal and posterior cells of the ethmoid labyrinth

2. A 40-year-old patient complains of pain in the forehead, nasal congestion, and purulent nasal discharge. He has been ill for 5 days. He associates the disease with a previous runny nose. Anterior rhinoscopy shows hyperemia and swelling of the mucous membrane in the area of the left middle nasal passage, with a purulent streak there. Radiologically, there is darkening of the left frontal sinus. The task is to establish a diagnosis.

- A. Aggravation of left-sided purulent frontal sinusitis
- B. Left-sided acute purulent ethmoiditis
- C. Left-sided acute purulent frontal sinusitis
- D. Aggravation of chronic left-sided purulent ethmoiditis
- E. Left-sided acute purulent sphenoiditis

3. A 37-year-old patient complains of viscous mucous discharge from the nose that is difficult to remove, and difficulty breathing through the nose. He has been ill for 5 days. Pain in the cheeks appeared 5 days ago. Anterior rhinoscopy shows hyperemia and swelling of the mucous membrane in the middle nasal passages, and mucous discharge there. X-ray, both maxillary sinuses are dark in color. The task is to establish a diagnosis.
- A. Aggravation of chronic bilateral catarrhal maxillary sinusitis
 - B. Bilateral chronic catarrhal maxillary sinusitis in remission
 - C. Aggravation of chronic bilateral catarrhal ethmoiditis
 - D. Aggravation of bilateral chronic catarrhal frontal sinusitis
 - E. Bilateral chronic catarrhal ethmoiditis in remission
4. A 37-year-old patient complains of severe difficulty breathing through both halves of the nose, heaviness in the head. Has been ill for 7 years. Anterior rhinoscopy - polyps in the middle nasal passages, purulent discharge there. Radiologically - darkening of the ethmoid labyrinth cells on both sides. The task is to establish a diagnosis{
- A. Chronic bilateral polypous ethmoiditis
 - B. Chronic bilateral polypous-purulent ethmoiditis
 - C. Chronic bilateral purulent ethmoiditis
 - D. Chronic bilateral polypous-purulent frontal sinusitis
 - E. Chronic bilateral polypous maxillary sinusitis
5. A 49-year-old patient complains of constant headache and foul-smelling discharge from the left side of the nose. Has been ill for 7 years. Anterior rhinoscopy shows hyperemia and swelling of the mucous membrane of the left middle nasal passage, with purulent discharge. Radiologically, there is darkening of the left frontal sinus. The task is to establish a diagnosis.
- A. Left-sided chronic purulent frontal sinusitis in the acute stage
 - B. Left-sided chronic purulent frontal sinusitis and ethmoiditis in remission
 - C. Left-sided chronic purulent frontal sinusitis and ethmoiditis in the acute stage

- D. Left-sided chronic purulent frontal sinusitis and sphenoiditis in remission
- E. Left-sided chronic purulent frontal sinusitis in remission
6. A 45-year-old patient complains of purulent discharge from both halves of the nose, difficulty breathing through the nose, headache, heaviness in the cheeks, and an increase in body temperature to 37.3 °C. He considers himself ill for a week. During anterior rhinoscopy, the mucous membrane of both halves of the nose is hyperemic, edematous, and there are stripes of pus in the middle nasal passage in both halves of the nose. Radiologically, there is homogeneous darkening of the left and right maxillary sinuses. The task is to establish a diagnosis.
- A. Aggravation of chronic bilateral purulent maxillary sinusitis
- B. Bilateral acute purulent ethmoiditis
- C. Bilateral purulent frontal sinusitis
- D. Bilateral acute purulent maxillary sinusitis
- E. Aggravation of chronic bilateral purulent ethmoiditis

answers to tests questions

1	A	6	B	11	A	16	C	21	A
2	D	7	D	12	B	17	E	22	C
3	D	8	D	13	B	18	A	23	D
4	C	9	A	14	B	19	C	24	A
5	D	10	A	15	C	20	A	25	A

answers to situational issues

1	A	4	B
2	B	5	E
3	A	6	D

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