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Anatomy, Physiology, and Fundamentals of Nasal Reconstruction

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ABSTRACT
In the past 50 years, significant progress has been made in nasal reconstruction. The concept of "filling the hole" has been abandoned by nasal reconstructive surgeons, who now have various methods to obtain both an aesthetically beautiful and functionally effective nose. 1 As the core and frequently most prominent part of the face, the reconstruction of the nose is among the most complex. Reconstruction of the nose necessitates a comprehensive understanding of its complex, three-dimensional structural and topographic anatomy. Also crucial to this sort of surgery is the interaction between the nose and the surrounding facial tissues, as well as how these tissues can be used to reconstruct a nose that is aesthetically pleasing and allows the patient to breathe normally.
Understanding the anatomy of the nose is the first step in nasal reconstructive surgery. The facial plastic surgeon must comprehend the intricate interrelationships between the underlying nasal support structures, the exterior skin, the function of the nasal lining, and the placement and features of the nose.

Keywords: Nasal reconstruction , Nasal anatomy, Skin physiology, Wound healing , Facial lines

Nasal Anatomy

Skin

The thickness of the skin varies depending on the position of the nose. [1]In actuality, this variation is Gonzalez-Ulloa and colleagues were the first to identify the nasal components.[2] The area with the most sebaceous glands is the caudal section of the nose, including the nasal tip and ala. This nasal skin becomes gradually thinner until it reaches the rhinion, where it is at its thinnest,[3] and again as it transitions from the tip to the columella and alar rim. [4,5]

Soft-tissue envelope

Soft-tissue envelope consists of four layers: superficial fatty layer, fibromuscular layer, deep fatty layer, and perichondrial/periosteal layer.[4] The dermis is tightly related to the superficial fatty layer. This layer is immediately followed by the fibromuscular layer. This structure is known as the nasal superficial musculoaponeurotic system (SMAS) and is

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continuous with the SMAS that covers the rest of the face. The nasal mimetic muscles are contained inside this layer.

The deep fatty layer encases the neurovascular system and supplies the skin-soft-tissue envelope.[6] Between this layer and the perichondrium/periosteum is the avascular plane used during rhinoplasty to degloss the nose.

Lining

The nasal vestibule is bordered with a thin skin strip (stratified squamous keratinized epithelium). This is a epithelium goes farther into the nose, it loses its keratinizing tendency and transforms into nasal mucosa (pseudostratified columnar ciliated epithelium). This epithelium, referred known as the respiratory epithelium, lines the sinonasal cavity, excluding the region covered by the olfactory epithelium. The high vascular supply of the nasal mucosa gives it an excellent candidate for repairing the inner lining of full-thickness nasal lesions using flaps.[8]

Life support

The blood supply to the nose is provided by branches of the external carotid artery (through the facial artery and infraorbital artery) and the internal carotid artery (by the ophthalmic and anterior ethmoidal artery) .

The facial artery has two terminal branches in the external carotid artery system: the angular artery and the superior labial artery. It gives off the lateral nasal branch, which supplies blood to the lateral section of the caudal nose. Following the rim of the pyriform hole, the angular artery gives off 7 to 14 branches that perforate through the soft-tissue envelope to supply the nasal skin. [9] The superior labial artery courses medially to the columella, where it gives off septal branches to supply the anterior portion of the nasal septal mucosa, and it terminates as the columellar artery, which runs between the medial crus of the lower lateral cartilage (LLC) and is commonly severed during the transcolumellar approach for open rhinoplasty. The infraorbital artery originates from the infraorbital foramen along with the infraorbital nerve and augments the blood supply with branches that give rise to the lateral nasal artery and the dorsal nasal artery.

Cartilage and bone structure

The skeletal framework is a pyramidal structure composed of the nasal bones and the temporal bones.On either side of the maxilla is an ascending process.The cephalic portion articulates superiorly and caudally with the frontal bone to produce the cranial section of the pyriform aperture . Superiorly, the nasal bones are thicker and gradually get thinner till their free edge inferiorly. [10]

The typical length of the nasal bones is 25 millimetres, however their length can vary considerably. They merge at the midline and provide an internal projection that supports the ethmoid bone's perpendicular plate. The nasal bones articulate inferiorly with the overlapping cephalic section of the ULC, which is medially attached to the cartilage of the nasal septum. This region of dense fibrous tissue connecting the nasal bones, perpendicular plate of the ethmoid bone, ULCs, and cartilage of the nasal septum is known as the "keystone area." [11,12]This region gives vital support to the nose's midvault.

Nasal valves

As the region of the nose with the smallest cross-sectional area of the total airway, the nasal valves regulate airflow. 18 Typically, they are referred to separately as the exterior and

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internal nasal valves. The nasal vestibule, internal nasal valve, and turbinated cavity of the nasal channel contribute to the resistance of the nasal airway. In contrast to the nasal vestibule, which contributes only approximately a third of the nasal resistance, and the nasal channel with its turbinates, which contribute just marginally, the nasal valves are the most resistant portions in the nasal cavity. [19]

The area between the head of the inferior turbinates, the nasal septum, and the ULC corresponds to the internal nasal valve.[20] The angle between the nasal septum and the ULC, with a usual range of 10 to 15, is a crucial component of this valve. [18] The area of the internal nasal valve is the flow-limiting portion of the nasal airway, accounting for approximately fifty percent of the total airway resistance from the nasal vestibule to the alveoli. [21] According to Poiseuille's rule, the resistance of the nasal passages is inversely related to the radius of the nasal passages (resistance 5 [viscosity length]/radius4). [22,23] This indicates that modest alterations in the size of the nasal valve can have exponential impacts on airflow resistance.

As previously stated, the Bernoulli principle plays an important role in the physiology of the nasal valve. As air travels across the constricted nasal valve, both velocity and pressure increase. This negative pressure in the vicinity of the nasal valves leads additional collapse. 24 Not only the internal nasal valve, but also the external nasal valve or nasal ala, may collapse as a result of the higher negative pressures produced by inspiration.

Viscoelasticity

One of the features of viscoelastic skin is stress relief. When a continuous force is exerted on. There is a decrease in skin tension during a time of stretching the skin to a specific length and then maintaining it at that length.[22]

Creep is another viscoelastic feature of the skin.This phenomenon happens when strain is applied to a segment of skin over time, resulting in an increase in skin length. The parallel alignment of collagen fibres, the fragmentation of elastin, and the displacement of extracellular fluid within the dermis have been related to skin creep. The process of tissue expansion employs skin creep, not to generate new skin, but to recruit skin nearby to the defect, allowing it to be closed with minimal tension. [19-22] Additionally, there are biological and mechanical types of creep. Mechanical creep occurs when the skin expands beyond its typical extensibility in a shorter period of time than biological creep. When utilising a tissue expander to recruit extra skin, it is typical to observe the second type.

Anisotropy

There are vast differences in facial skin tension amongst the various anatomic units. For example, the eyes and cheeks have significantly thinner skin.Tension greater than that on the nose and forehead. However, the skin is under tension in all directions at any one moment. This tension is always there and is altered by muscle contractions (which can either raise or decrease the tension). This tension is the force responsible for separating a wound's edges. The relaxed skin tension lines (RSTLs) were first defined by Borges and Alexander44. They are a series of imagined face lines along which the skin has the least amount of stress for closure [10-14]. These lines are typically not apparent while the skin is at rest, but can be seen as furrows when the skin is pinched together. [12-16] Parallel incisions heal without stress or tension on the wound closure. [14-17] If a wound is created perpendicular to the RSTLs, the edges may begin to spread or separate. Borges46 argued that no other aspect (not even surgical skill) is as crucial to the creation of an acceptable scar as the orientation of the wound being parallel to the RSTLs. If an incision must be made in an antitension line, he

advised that it be made in a zigzag pattern, more closely matching the RSTLs. RSTLs are always either parallel, concentric, or intersect at a sharp angle. Individuals do not differ greatly in their direction. These lines tend to be radial near face openings, such as the nose and mouth. Additionally, they do not coincide with the glabella, the upper portion of the crow's feet, or the mentolabial fold.

Blood Supply to Local Skin Flaps

Understanding the vascular supply to the skin and soft tissue is crucial for the reconstructive surgeon's operational planning. When planning regional flaps for nasal defect repair can be divided into random and axial pattern flaps based on their blood supply. Random skin flaps obtain their blood supply through musculocutaneous and septocutaneous vessels, which run deep to the muscles or in the muscular fascia, respectively, and then perforate through the muscles or muscular septae to feed the dermalsubdermal plexus of the skin with blood.

Axial pattern flaps, on the other hand, receive their blood supply from direct cutaneous veins that run along the axis of the flap.The flap's axis of direction. The survival of these flaps depends on the length of the true axial vessel. [20] When elevating these flaps, they should be positioned and aligned so that their long axis runs parallel to the blood artery of interest. Any skin region beyond the distribution supplied by this artery becomes a random flap and again depends on the dermal-subdermal plexus for nutrition. This axial blood supply's robust and stable vascular supply makes these flaps more receptive to primary thinning and contouring.

Additionally, wound strain might influence the blood supply to a skin flap. It has been demonstrated that the blood supply is inversely proportional to the distance from the flap's base, therefore a flap stretched under severe tension can develop distal necrosis. [17] This is one reason why the closures of local skin flaps should be repaired without strain.

Wound Healing Phases

Inflammatory phase (0-5 days)

The process of wound healing begins soon after tissue injury and once hemostasis is achieved. For the wound's blood to clot,Local vascular vasoconstriction, fibrin deposition, and platelet aggregation must occur simultaneously. Fibrin deposition is dependent on the extrinsic coagulation pathway, and platelets begin to deposit on the artery walls after being exposed to the circulating collagen and tissue components in the wound bed. The release of histamine, leukotrienes, and prostaglandins from the endothelium causes the initial vasoconstriction to transition into a phase of vasodilation after 10 to 15 minutes.[22]

The invasion of neutrophils, which begins to dominate the wound bed, lends its name to the inflammatory phase. These cells patrol the wound and assist avoid local infection by debriding and digesting foreign particles and germs. These cells reach their peak between days 1 and 2 and then begin to fall when monocytes and macrophages migrate into the area, reaching their peak between days 4 and 5.

Phase of proliferation or granulation (6-14 days)

The subsequent phase of wound healing involves the restoration of the epithelium, the synthesis of collagen, and the promotion of the formation of new blood vessels. Reepithelialization is supposed to occur approximately 12 hours after the original wound damage.[17-19] Reepithelialization occurs as epithelial cells migrate along the fibrin scaffold to re-cover the wound bed. 40 Adnexal structures engage in the reepithelialization process in superficial wounds.

During this phase of wound healing, epithelial cells play a major role, and fibroblasts, which are essential for the synthesis of elastin, proteoglycans, and collagen, become active.In the early stages of a wound, fibroblasts produce an abundance of collagen type III, which is later converted to type I collagen. Around days 7 to 14.53, fibroblasts also transform into myofibroblasts, which are essential for the contraction of wounds.

In conclusion, the wound generates various angiogenic growth factors, such as vascular endothelial growth factor, which stimulate angiogenesis.[20-24] This development of new blood vessels is required to promote the growth of granulation tissue in the wound bed.

Remodeling or maturation phase (15 dayse-1 year)

The last phase of wound healing is called remodelling or maturation. This is also the most time-consuming phase of the process and can last up to one year. This is why many surgeons wait one year before trying surgical wound or scar correction.[25] Two weeks after the formation of the lesion, the collagen arrangement shifts from the disordered fibrils of the juvenile scar to the parallel, thicker collagen fibril arrangement of a mature scar. In addition, type III collagen, which was initially present in the wound bed, is replaced by type I collagen, which is the primary component of the scar.[26-28] As the arteries regress, neovascularization ceases and the erythema that was initially evident in the wound due to the vasculature turns white.

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