NASAL ATROPHY, ATROPHIC RHINITIS AND OZENA

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It is with a feeling of great humility and much honour for me to be asked to deliver this paper on atrophic rhinitis and ozena. It is not without good reason that I dedicate this paper to the memory of Prof. H. A. E. van Dishoeck. Without the principles in nasal physiology laid down by Donders, Mink, and van Dishoeck, physiologic nasal surgery as we know it today, would be impossible.

Introduction

Trauma is the cause of most septal and nasal deformities (Riggs, 1961). The concept of early nasal injury (Cottle, 1957) causing an acceleration or deceleration in growth or in development to the entire nose, or any of its components, gives the Rhinologist an understanding of the pathology involved.

Developmental anatomy

It is only after the seventh year of life that the bony nasal arch affords adequate protection to the underlying cartilagenous structures. The ethmo-vomerine cartilage gives rise to the perpendicular plate of the ethmoid which is not ossified until the fifth year (Clark). The vomer is ossified in two centers on either side of the posterior inferior part of the cartilage at the eighth week of fetal life. The lamellae unite in the third month to form a groove for the cartilage. The lamellae are almost completely united at puberty.

The palatine bone is ossified in membrane from a single center at the sixth week of fetal life. Ossification spreads. At birth, height of the vertical part is equal to the transverse width of the horizontal part; while in the adult the vertical is twice as large as the horizontal.

The premaxillary and maxillary centers appear during the sixth week und unite during the third month. The nasal bones, vomarian plates and premaxillary wings are well developed in the newborn.

The cartilage of the septum and the upper lateral cartilage are continuous and remain so throughout life (Goss, 1966).

The quadralateral cartilage begins to grow sometime during the eighth year of life from a center located near the wings of the premaxilla.

Early nasal injury

It has been estimated by Cottle that 5 to 7% of all Caucasion newborns suffer lateral displacement of the nose and that approximately 50% show flattening of the lobule. This occurs during normal birth. The distance from

the occiput to the chin and the occiput to the tip of the nose almost approximate each other. Compression of the head and external nose gives rise to injury to the growth centers in the maxilla, premaxilla, vomer, palatine, and ethmoid plate and may result in the arrest of development or of growth of the entire nose and pyriform aperture; acceleration in development and or growth of the entire nasal compound or of any of it's component parts. There may be an arrest of development, an increased development combined with an acceleration or an arrest of growth all occuring in the same nose. The most common is the situation where we see an underdeveloped external nasal pyramid, a short septum, occult retraction of the columella and a large fetal or juvenile type of lobule. The pyriform aperture may continue to grow in size and yet retain its short and increased width which would be normal for the young child or in the mezorrhine and platyrrhine groups. One or both sides may be involved. The soft overlying tissues depict the underlying structures. The configuration of the small or large wide nose may be familial in origin, an arrest due to infection, or the result of ethnic heterogenecity. We frequently see Negro patients who have an internal leptorrhine configuration and a platyrrhine external configuration. Early, the nasal mucosa is usually moist and the expiratory and inspiratory baffles are adequate. However, if this nose suffers repeated unrecognized small nasal injuries such as occurs before the age of five years, or a severe nasal injury (Cottle, 1958) in the adult, involving the structural support of the quadralateral cartilage from the premaxilla to the distal end of the nasal bone, the following changes occur as elucidated by Cottle:

1. External nasal pyramid deformity.
2. Saddling cartilagenous vault.
3. Sagging cartilagenous vault.
4. Ballooning upper lateral cartilage.
5. Pathological returning upper lateral cartilage.
6. Rounding of the nostrils.
7. Widening of the lobule.
8. Widening of the base of the nose.
9. Insufficiency of nasal valves.
10. Insufficiency of inspiratory and expiratory baffles.
11. Increased intranasal air space.
12. Retraction of the columella.

and I wish to add to the list,

13. A nasal septum that is too short in its relationship to overlying pyramid.

Aging

I am sure we have all observed the increased drooping of the tip, widening of the naral openings, and retraction of the columella. The length of the nasal septum remains the same. This disparity increases with the passage of each year. Sooner or later a change in the type and direction of the air current ensues. The laminar flow becomes turbulent. Each inspiration may cause localized or generalized irritation and with each expiration, a loss of moisture and heat occurs. The nasal valve (Gersuny, 1900) widens and ceases to function adequately; as do the baffle systems, and the patient is in "trouble". The nose has an almost fixed pattern in its response to early nasal injury,

acute trauma interfering with the support of the lobule by the septum, and in the process of aging.

Etiology and pathology

There are many theories regarding the development of atrophic rhinitis. Heredity, injury with an accompanying arrest in bony development associated with overgrowth of the lobule, arrested development of the pyriform aperture and or the face, diphteria, measles, infection of the upper respiratory tract, hormonal imbalance, metabolic disease, allergy and avitaminosis have been considered in the causation of this disease.
The peripheral ganglia (Higbee, 1949) and nerves of the autonomic nervous system in the nose are vulnerable to infection, trauma, changes in barometric pressure and especially cold air. The parasympathetic fibers are most easily affected allowing the sympathetic fibers to overact, resulting in prolonged constriction of the arteries and arterioles in the nasal mucosa. The diminished blood supply, viral and bacterial infection, plus the trauma induced by turbulent aberrant jet air streams lead to degeneration of the subcutaneous tissues, reduced blood supply and atrophy of the nasal mucosa and its overlying tissues.
The nasal mucosa becomes thin, ulcerated and the epithelium is replaced by squamous nociliated epithelium. Perivascular cellular infiltration with generalized fibrosis of the subepithelial tissues and finally atrophy of all the tissues of the nose is present.

Surgery

Extensive submucous resection of the nasal septum without replacement of the septal components rendering effective support of the lobule results in iatrogenic nasal atrophy. Mutilating surgery such as turbinatectomy results in a dry nose. A nose without turbinates suffers from turbulent airflow.

Nasal atrophy

Nasal atrophy refers to the insidious early stages of this chronic degenerative disease complex. In the beginning, thin pale dry patches, areas of crusting, mucoid secretions and increased intranasal space are present. Any part of the septum, turbinate, nasopharynx, pharynx and larynx may be involved. The early stages of this disease may progress very slowly to the stage of fetor and crusting, yet the latter may never occur. The disease process may be limited to a single structure or it may involve the entire nasal compound and contiguous structures. The process of nasal atrophy is relentless. As the years pass by, the observer notes progressive sagging of the dorsum, widening of the base of the nose, retraction of the columella, ballooning of the upper lateral cartilages, insufficiency of the nasal valve, expiratory baffles and a growing dysharmony between the lobule and the underlying pyramid. As the intranasal space increases, the intranasal structures become reduced in size and structure. Finally crust formation, atrophy of the mucosa, subcutaneous tissues, bones, cartilage and skin are present. With this the patient's ability to taste, smell, rest, tolerate cold air, and complaints of obstructed nasal breathing becomes progressively worse.
The frame work of the nasal septum forms the support of the nasal pyramid

in alle directions. A short nasal septum gives rise to occult or manifest columellar retraction; with concomittant widening of the base of the nose.

Medical management

The preoperative and medical management includes the use of (Riggs, 1961) the creation of an adequate expiratory baffle and inspiratory baffle by the use of cotton in the vestibule of the nose or the wearing of nonallergic tape over part of the nasal opening, (Cottle et al., 1957) large doses of ascorbid acid and bioflavanoids (Clark, 1970), vasodilators (Goss, 1966), estrogens, both locally and systemic (Masing, 1965), ringers solution in the form of a nasal spray (Mink, 1907), high protein-low carbohydrate diet (Higbee, 1949), heparin, and (Cottle, 1958) anti-allergic therapy and other supportive therapy. The above regime has proven paliative in many of our elderly patients and in others it sets the framework for a greater success in nasal surgery.

Surgery-historical

Many procedures have been used in an attempt to narrow the nasal chamber. Gersuny (1900) injected paraffin submucosally in 1900. This popularized the use of celluloid, fat, rib, cartilage, ivory, bone, fascia, acrylic resins, etc. Proud (1947) replaced the hard atrophic septal bone with acrylic. Ogura (1954) grafted bone into the intraseptal space removing the hard structures. Réthi (1948) used the septum in the construction of a nasal baffle. Transplantation of Stensons duct was employed by Wittmaack (1919). Dr. Cottle (1957) was probably the first to use cancellous bone as a true graft in the septum in the lateral wall and the floor of the nose after submucous resection of the septal hard structures.

Surgery-new

The addition of rhinoplastic concepts as taught by Joseph, later Fomam, and crystalized by Cottle greatly added to the above procedures. It has been stated that less air enters the nose and therefore the nasal mucosa becomes moist. On closer examination, it is not that simple. The amount of air passing through the nose is controlled by the lower respiratory tract and its associated structures. Cottle (1958) has emphasized the following factors:

1. The direction of airflow is affected by the size, shape and position of the nostrils in their relation to the nasal pyramid.
2. The laminary nature of the air stream is decided by the nasal valve and the function of the lobular cartilages.
3. On inspiration air flow turbulence and direction is influenced by the anterior portion of the inferior and middle turbinate.
4. On expiration the air flow turbulence and stagnation is influenced by the posterior ends of the inferior turbinate, the nasal valves and vestibule, aid in the preservation of moisture and heat.
5. The efficiency of the erectile tissue of the inferior turbinate (and middle turbinate to a lesser degree) determine the normal cyclic response and excursion to the hard septum in response to atmospheric changes, psychic changes and in the control of sleep.
6. Submucosal implants and grafts may stimulate and increase blood supply and increase the secretion of mucosal glands and other structures.

7. Any implanted material may remain in situ or to be replaced by fibrous tissue with its own blood supply.

What can we not do

1. Do not reduce the length of a short nose by resection of the caudal end of the upper lateral cartilage.
2. Do not resect the nasal valve as in the classical Joseph Rhinoplasty.
3. Do not destroy the function of the expiratory baffle system by transplantation and fixing the medial crura.

What can we do?

1. There is no single operation but a regime of rehabilitation that may take ten to fifteen years in the form of multiple consecutive operations. The surgery performed is dependent upon the pathological physiology encountered.
2. Narrow the nose by lateral osteotomy.
3. Make the nose higher by the use of implants, grafts and up fracture.
4. Lengthen the nose by transplanting septal bone to the caudal end of the nasal septum after complete septal mucosal elevation.
5. Remove returning of the upper lateral cartilage.
6. Relieve tension of the atrophic mucosa by push-down, septal imbrication and implanting crushed cartilage to the dorsum and lateral walls (endonasal microplasty).
7. Equalize an uneven pyramid by properly placed lateral osteotomy.
8. Encourage the formation of subcutaneous tissues by the implantation of tissues that will disappear and allow fibroplasia to take place.
9. The use of sharp disection in preparing the septal flaps and septal bone mobilization.
10. Implant cancellous bone in the posterior part of the septum, thereby creating a posterior expiratory baffle in the area of the sphenopalatine ganglion.
11. Treatment of linea nasalis by bone and cartilage overlay and dorsal fixation of the quadralateral cartilage.
12. The use of the long arm U incision, spine repair and basal stitch to narrow the base and bring tissues together.
13. Preserve nasal valve mucosa and construct a proper nasal valve.
14. Treat the nostril and floor of the vestibule by narrowing, alar wedges, and grafts to the floor.
15. Continuous medical management of the entire patient. All implants should create new connective tissue and blood vessels. Use much cartilage and little bone. The postoperative use of a high protein diet, ascorbic acid, heparin in low doses, bioflavanoids and narrowing tapes are of immense value.
16. The secondary replacement of implanted cartilage is indicated. In my hands, at the present time, the use of knee cartilage banked by the orthopedic surgeons has been of inestimable value.

Septal, nasal pyramid and or lobular surgery may be done as indicated. Surgical implants may be done in two or more stages. An autogenous cancel-

lous bone hip graft is the material of choice for grafting in the posterior septum and dorsum. Preserved or refrigerated cancellous bone tends towards early absorption. Cartilage, living and presserved, is much better tolerated. The use of aloplastic implants is not recommended except as a stop-gap. They are inert, self lubricating, move and tend to extrude.

The operation (Cottle, 1958)

The surgery of atrophic rhinitis is performed after narrowing of the external nasal pyramid, lobule, nasal valves, nostrils and base of the nose. A vertical incision is made in the alar labial groove, preserving the rim of the nostril and the skin of the vestibule. This incision leads to the area of the pyriform crest. The next incision is made through the subcutaneous tistues perpendicularly to the first incision. Scraping the soft tissues from the anterior face of the alveolar process with a sharp McKenty exposes the bone and floor of the pyriform crest. Continued gentle scraping of soft tissues exposes the lateral portion of the pyriform aperture and most of the floor. Submucosal elevation of the nasal membrane is now continued medially to the septum and laterally to the attachment of the inferior turbinate.

The inferior concha attachment begins one millimeter or so behind the pyriform crest and is between thirty-five millimeters (lleptorrhine) and thirty millimeters (platyrrhine) above the floor of the nose. The attachment has a conelike appearance. The inferior turbinate is then separated with a curved seven millimeter chisel. The lateral wall should be scarified. The implant material should consist of large pieces of crushed cartilage. Sharp edged blocks of cartilage and bone are not retained well by the atrophic mucosa and tend to extrude. Enough implanted material is used to allow the turbinate to caress the septum.

Implants in the septum should be placed posteriorly in sufficient quantity to create a posterior baffle. Anteriorly, implants are limated to the lateral wall. Bank sclera and bone has been used in the treatment of flaccid nasal septum. A dorsal graft of autogenous cancellous bone or bank sclera may be needed to replace atrophic nasal bones.

Septal perforations are treated first by meticulous sharp dissection under good observation and secondly by suturing of the flaps, rotation and grafting with buccal mucosa. A 0.30 millimeter buccal graft is easily attained and is adequate.

The implantation is easily repeated as needed. Each successive procedure is more easily performed aided by a thicker mucosa that is more easily handled.

SUMMARY

The surgery of atrophic rhinitis is unique. There is no routinely successful operation, but, rather a series of steps dependent upon the pathology involved. Nasal atrophy, atrophic rhinitis and ozena form a continuium of a combined process of arrested development and nasal injury. The concept of early nasal injury at birth or in infancy may result in arrested development of the external nasal pyramid with concomitant under development with an acceleration in growth of the lobule. A dysharmony results. An arrest in the development of the pyriform aperture may also occur rendering the intranasal space too large.

The upper lateral cartilage septum compound is susceptible to slight injuries resulting in ballooning of the upper lateral cartilage, sagging of the cartilagenous vault, rounding of the nostrils and retraction of the columella. The septum which supports the lobule now becomes too short. The nasal valve and both the inspiratory and expiratory baffles become insufficient. No longer is moisture and heat adequately retained. The laminary airflow becomes turbulent and the process of nasal atrophy ensues.
Both medical and surgical treatment are indicated. The short wide nose must be made higher and longer. All nasal areas are treated. The nose is made narrower, higher, and longer by the use of septal transplants and posterior implants. Anteriorly, implants are placed in the lateral pockets (Cottle). Implants should have the ability to absorb and form new fibrous tissue in order to increase the thickness of the nasal mucosa. Implants are replaced as needed and ultimately the patient's entire problem is resolved. Multiple surgeries are indicated. It may take as long as ten to fifteen years before we are awarded with a completely medically and surgically rehabilitated patient.

RÉSUMÉ

La chirurgie de la rhinite atrophique est un problème tout à fait spécial. Il n'y a pas d'opération de routine qui réussisse, mais plutôt une série d'actes qui dépendent des cas pathologiques.
L'atrophie nasale, la rhinite atrophique et l'ozène forment une transition d'un processus associé de lésion nasale et d'arrêt du développement.
Le concept d'un traumatisme nasal précoce à la naissance ou dans l'enfance peut provoquer un arrêt du développement de la pyramide nasale et une accélération dans la croissance du lobule, constituant une disharmonie. L'arrêt dans le développement de l'orifice piriforme peut aussi survenir et rendre l'espace intranasal trop large.
Le cartilage septo-latéral (cloison et cartilages triangulaires) est exposé à des lésions plus discrètes qui se manifestent par un bombement du cartilage latéral et de la valve, l'affaissement de la voûte cartilagineuse, la dilatation des narines et la rétraction de la columelle. La cloison qui supporte la pointe peut devenir ainsi trop courte. La valve nasale, et les rétrécissements normaux à l'inspiration et à l'expiration deviennent insuffisants; dès lors, l'humidité et le réchauffement ne peuvent plus être maintenus, et le courant aérien laminaire devient turbulent, déclenchant le processus d'atrophie nasale. Les traitements médicaux et chirurgicaux sont indiqués.
Un nez court et large doit être rendu plus haut et plus long. Toutes les zones nasales doivent être traitées et révisées. Le nez est rétréci, raccourci allongé, et projeté par l'inclusion d'implants provenant de la cloison postérieure notamment.
En avant, les implants sont introduits dans des loges latérales (Cottle) et doivent avoir la possibilité de former un nouveau tissu fibreux pour augmenter l'épaisseur de la muqueuse nasale. On pourra répéter, selon les besoins, cette introduction d'implants sous muqueux jusqu'à ce que l'état du malade soit nettement amélioré, de sorte que plusieurs interventions sont indiquées. Cela peut prendre 10 à 15 ans pour obtenir un résultat médical et chirurgical complet.

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