## GENESIS OF SEPTAL DEFORMITIES

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Force, external, whatever its source, is an obvious factor in the genesis of septal deformities, but do not exclude the prolonged progressive influence of internal forces such as acceleration, deceleration, arrest, and the unbalanced interplay of a multiplicity of growth centers.

Without reservation it is an accepted fact that aberrant development results from a distortion of normal developmental processes, but other situations and factors contribute to this pathogenesis such as intrauterine position and pressures, birth canal presentation, ethnic measurements, anatomical differences in the osseous and cartilagenous septum of the newborn child and adults growth of the vomer and premaxilla, eruption of the central and lateral incisor teeth and expansion of the maxillary sinus.

Studies on the development of the nasal sac reveal that it undergoes the typical embryonic process of a gradual increasing differentiation at the expense of a progressive loss by the embryo to constitute the organ from adjacent tissue.

The septum develops separately as the external nose is being formed. Whenever its rate of growth is accelerated beyond that of the nose, the cartilagenous septum must bend to accommodate itself to the nasal space delineated by the nasal ridge, ethmoid, body of sphenoid and maxilla.

In the fetus, newborn, and early child the bony elements of the external nose and septum are not well developed whereas the cartilagenous and soft tissue elements predominate. Overwhelming force, when applied to cartilage, receives a cushioning effect but the end result may be inevitable, noticeable deformity, due to displacement, bending or fracture of the cartilage, trauma to the mucous membranes, local and dissecting hematomas, changes in potential of growth, acceleration, deceleration, and total growth arrest.

Injuries to the nose may be so remote or trivial as to be unrecognized, undiagnosed or untreated. These may occur during birth and early infancy so that growth processes may be unbalanced.

Prenatal injuries must not be confused with congenital anomalies and deformities. In a congenital defect the pathology is directed toward the area that failed to develop and grow, such as a columella and septum inclined toward an undeveloped side of nostril or tip, while in injuries, it is usually contralateral.

Deformities of the septum commonly occur during the process of birth, especially in primipara. There is flattening and a lateral displacement of the nose with contralateral displacement of the septum in 6% of white births. Cartilagenous injury alone occurs in 60%.

Obstetric statistics by DeLee state that 95% of all deliveries are vertex

presentation divided as 70% L.O.A., 25% R.O.A. and 5% other. In a L.O.A. and R.O.A. 45 degree rotation is required while an occiput posterior rotates 135 degrees, a transverse 90 degrees. The distance from the tip of the nose to occiput is frequently the same as chin to occiput. Pressure of the external nose against the sacral prominence, coccyx, smyphysis pubis, and floor of perineum is evident.

In newborn nasal deviation, 80% show a deviation of the external nasal pyramid to the right with the cartilagenous septum displaced from the vomerine groove to the left. The infants septum is almost entirely cartilage which is extremely resilient and tends to rebound to normal.

The force of growth in embryological tissue in reverse ratio to its chronological age directs tissue to assume the normal anatomical and physiologic pattern. This potentiality of tissue to develop its normal, even after injury, we call totipotence.

To understand the genesis of septal deformities we must consider growth and ossification centers.

The frontal bone has two centers of ossification above the superciliary arches. By the eighth week ossification is spreading quickly through membrane bone. Shortly after birth the frontal bones articulate with each other, fusing and obliterating at the metopic suture line. This gives firm anchor to the infants square nasal bones at the frontal nasal synchondrosis. This becomes a pyramid strong point for the cantilever positioning of the nose when it is reinforced, as osseous material is deposited at the lower end of the metopic suture to complete the formation of the frontal spine by age of twelve years. The spine, nasal crests of nasal bones plus perpendicular plate of ethmoid form a very strong point.

Rostrum of the sphenoid ossifies from the chondrocranium of the median part of the basisphenoid of the presphenoid center and grows between the two leaves of the developing vomer. As the rostrum wedges between the ala of the vomer another strong point is established.

The ethmoid ossifies in the cartilagenous nasal capsule from three centers, lamina papyracia, cibriform plate, and perpendicular plate. Sometime during the first four years after birth a nucleous appears in the ethmovomerine cartilage to form the perpendicular plate.

The nasal crest of the palatine bones is ossified in membrane lining the interior surface of the cartilagenous nasal capsule beginning by the eighth week.

The maxillae appearing by the eighth week in membrane bone ossify from two centers to develop a midline shallow bony through to receive the septal cartilage.

The ploughshare vomer ossifies from two centers at the eighth week in membrane investing the cartilagenous septum of the nasal capsule. The two lamella unite below by the third month to form a shall bony through in which the cartilage lies. As the lamina extend upwards with growth the cartilage enclosed between them is absorbed and the lamina fuse, thus forming a V-shaped groove to receive the septal cartilage and the rostrum of the sphenoid. The vomer develops as two plates and ossifies from posterior to anterior. Any difference in nutrition between the plates of the vomer will cause an inequality in growth with a consequent tendency to buckle. This

tendency is transmitted to the septal cartilage at the anterior surface of vomer plates and becomes accenuated.

The septum also includes the premaxilla, medial crura, membranous septum, colunella, and anterior nasal spine.

The septum at birth is mostly cartilage with bony parts of premaxilla and vomer. At this time crests of the palatine and superior maxilla bones do not reach or support the lower edge of the septum. Because of double origin the vomer, premaxilla, and anterior nasal spine of maxilla all form V's. The septal cartilage sets in a V of the anterior edge of the vomer. The anterior tip of the vomer rests in a V of the premaxilla wings. The premaxilla is anchored in a V of the anterior nasal spine. The dorsal border of the cartilagenous septum forms a V called the supraseptal groove to receive the crests of the nasal bones. One must realize that these areas of V contact do not resist force without dislocations.

By the age of four the upper border of the vomer is gutter shaped and the lower border of the perpendicular plate of the ethmoid forms a gutter. Between these gutters is an offshoot of cartilage from the posterior inferior angle of the septal cartilage call the caudal prolongation.

After age eight, the growth centers of cartilage are primarily just posterior and dorsal to the premaxillary wings and secondarily midway along the posterior border. In the adult the septal cartilage is thin where it rests on the premaxillary wings called the processus lateralis ventralis. It is thick opposite the middle turbinate and called the tubercle of the septum.

With acknowledgement to Dr. Wexler, whose laboratory experiments indicated that interference with growth centers alters the progression of development and resulted in faulty growth. Subsequent deformity depends on intensity, type, site, and susceptibility of the area to growth changes.

Experiments involving trauma or resection of the septovomeral joint, cartilagenous septum, vomer, and premaxilla, resulted in deformities of growth, septum deformity, dislocations, and disturbance of eruption and direction of teeth.

In the anterior human palate the premaxilla are inconspicuous. They are ankylosed to and covered by the superior maxilla at fetal three months. There is one osseous center in each premaxilla and they unite by age one.

The forepart of the nose supported by septal cartilage rests along with the apex of vomer on a fragile portion of the premaxilla. The premaxilla which harbors the central and lateral upper incisor teeth is a pedestal which positions the septum by a joint capsule held together by fibrous tissue interlaced around, across and through the joint capsule. Injury to this joint is more likely if the joint is filled with fatty tissue. The meeting place of the septum, vomer, and premaxilla is one centimeter in size and is called the center of the septum Mosaic. After age of six this area grows more rapidly in proportion than any other part. At age fifteen the premaxilla fuses with the vomer.

If the central incisors fail to erupt then the premaxillary wing on that side forces by excessive growth a dislocation of the septum at the joint. This can carry with it the lower cartilagenous vault and the anterior vomerine elements. If the lateral incisors fail to erupt, only a mound of growth arises in the floor of the pyriform aperture and seems to have no effect on septal dislocation. The history of the eruption of central incisor teeth is to be read in the nose.

Marked inequality and delay in eruption of one central incisor to the other results in enlargement of premaxillary wing plus vomer spurs. On the side of the delayed eruption, the premaxillary wings overgrow, thus tipping the quadrilateral cartilage out of its bed along the vomer - ethmoid suture.

The paraseptal cartilage is a support for the paired specialized epithelial organ assisting in sensory perception of flavors located in the organ of Jacobsen which is effective in some vertebrates, but is without function in man.

As the nasal cartilagenous septum develops by the inevitable extension of the nasal pits, the septal cartilage is bracketed by an anterior and posterior island of developing cartilage which fuses with the processus laterales ventralis of the septal cartilage in the 21 millimeter human embryo.

At birth in the human they separate. The paraseptal cartilage can cause deformities by growth, chondromas, spurs or an actual impacting ridge since the paraseptal cartilage may extend past the apex of the vomer.

Seven septums may be formed; right lateral paraseptal wing, right medial paraseptal wing, vomer, cartilagenous septum, vomer left medial paraseptal wing and left lateral paraseptal wing.

If one attempts to arrange septal deformities in relation to abnormal potentials there could be several groups:

Type I with etiology as an enlarged premaxillary wing results in a dislocation or a ridge of cartilage on the opposite side of the enlarged premaxillary wing.
Type II with basic pathogenesis of bilateral enlargement of the premaxillary wings results in a bend or dislocation of the septum to either side.

Type III is represented as a fractured premaxillary wing with septal convexity and influences growth of right wing of vomer.

Type IV lists the problem of complete hypertrophy of premaxilla with extreme enlargement resulting in a bend of the septum to either side.

Type V groups the problems due to an elevated position of the anterior nasal spine, which is due to failure of maxillary sinus to expand, resulting in diminished space for development of anterior nasal spine and septal cartilage. Deformities of the septum complex are frequently the result of trauma changing to deflection, depression, and disatrophy.

In type I classification force is applied on a line from distal tip of nasal bones to anterior nasal spine. This is a frequent injury and results in bend, angulation or fracture of the septal cartilage.

In type II force is applied from above downward which reduces the distance between the cartilagenous dorsum and the vomer ridge. This results in displacement of the cartilage from the vomer groove with additional possibilities of fractured cartilage and oblique extension to involve the perpendicular plate of the ethmoid.

In type III excessive force results in a dislocation of the cartilage off the vomer groove. This results in a buckling fracture of the septum, plus dislocation from the premaxilla plus dislocation from the vomer, with resultant displacement of the septal cartilage into the nasal vestibule.

In type IV force causes a compression fracture of cartilage with duplication.
In type V trauma causes multiple fractures of septal cartilage with subsequent

hemmorrhage and organization of the clot. Fibrous infiltration occurs followed by contracture, scar, and deformity. Scar may also result in atrophy and perforation.


## SUMMARY

Septal deformities are congenital and developmental, traumatically acquired before growth changes are completed, traumatically acquired after growth.
Many combinations of injury, growth and arrest may occur with associated changes in teeth, face, palate and pyriform aperture, resulting in deformity, obstruction, dysharmony, and assymetry.
No hypothesis yet formulated is universally accepted by all which can explain adquately the asymmetries and deflections in all individuals.

## RÉSUMÉ

Les déformations du septum nasal sont congénitales ou acquises. Parmi celles-ci, les unes résultent d'un traumatisme subi avant la croissance; les autres sont la conséquence d'un traumatisme survenu après le développement du nez.
Plusieurs types de lésions peuvent intervenir en association avec des modifications dentaires, faciales, palatines ou de l'orifice piriforme; elles entraînent des déformations, une obstruction respiratoire, une dysharmonie ou une asymétrie de la face.
Aucune des hypothèses formulées jusqu'à présent n'est capable d'expliquer de façon adéquate les déformations observées chez l'ensemble des individus.


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