Nasal, Septal, and Turbinate Anatomy and Embryology

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This article describes the development and anatomy of the nasal septum and structures of the lateral nasal wall. A clear understanding of the development and anatomic variations of the nasal septum and structures of the lateral nasal wall is vital for successful treatment of nasal obstruction. With knowledge of the specific location and anatomic reason for a patient’s nasal obstruction, clinicians can better identify the specific structure responsible for the obstruction and thus implement a more targeted approach to treatment.

NASOSEPTAL EMBRYOLOGY

The tissue that gives rise to the face and nasal structures derives from three different embryonic sources: the ectoderm, the neural crest, and the mesoderm. The ectoderm provides an overlying cover and, through its interactions with mesenchymal layers, a pattern for developing structures.1,2 Neural crest cells provide the majority of facial mesenchymal tissue.1,2 The paraxial and prechordal mesoderm provides precursors for myoblasts that differentiate into voluntary craniofacial muscles.2

At 4 weeks’ gestation, five identifiable primordial structures surround the stomodeum, a depression below the developing brain and the first sign of a future face. These five structures are the frontonasal prominence, the right and left maxillary prominences, and the right and left mandibular prominances. The maxillary and mandibular

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prominences lie superolaterally and inferolaterally bilaterally respectively. By the end of the fourth week of gestation, paired thickenings of ectoderm appear on the frontonasal prominence superior and lateral to the stomodeum. These oval placodes develop into the nose and nasal cavities (Fig. 1).

During the fifth week, mesenchymes on the periphery of the nasal placodes proliferate to form horseshoe elevations. The lateral and medial limbs are termed nasolateral and nasomedial processes respectively. Mesenchymal tissue surrounding the nasal placodes continues to proliferate and thicken, resulting in a perceived depression of the placodes. These depressions are subsequently called the nasal pits and are the primordia of anterior nares and nasal cavities (see Fig. 1).

From 5 weeks' gestation, the nasal pits continue to deepen toward the oral cavity. By 6 and one-half weeks, only a thin oronasal membrane separates the oral cavity from the nasal cavities. This oronasal membrane subsequently disintegrates, leading to a communication to the nasal cavities posterior to the primary palate. These regions

Fig. 1. Embryogenesis of the face.
of continuity are the primordial choanae. As the palatal shelves fuse and the secondary palate develops, the nasal cavity lengthens, resulting in the junction of the nasal cavity and the pharynx.\textsuperscript{1,2}

Beginning from the fourth to sixth week of gestation, the paired maxillary processes grow medially toward each other and toward the paired nasomedial processes.\textsuperscript{1} By the end of the sixth week, the nasolateral processes begin to fuse with the maxillary processes to form the ala nasi and the lateral border of the nostril bilaterally (see \textbf{Fig. 1}). Along the junctions of the nasolateral and maxillary processes lie the nasolacrimal grooves. Ectoderm within these grooves thickens to form epithelial cords, which then detach and canalize to form nasolacrimal ducts and lacrimal sacs. By late fetal period, nasolacrimal ducts extend the medial corners of the eyes to the inferior meatuses in the lateral wall of the nasal cavity.\textsuperscript{2}

The nasomedial prominences continue to expand but remain unfused until the seventh or eighth week of gestation, when they merge with superficial components of the maxillary processes. The fusion lines between these processes are the nasal fins. As mesenchymes penetrate this articulation, a continuous union is formed, completing most of the upper lip and upper jaw bilaterally (see \textbf{Fig. 1}). The nasomedial processes then merge with each other, forming the intermaxillary segment and subsequently displacing the frontonasal prominence posteriorly. The intermaxillary segment formed from the nasomedial processes is the precursor to several structures, including the primary palate, the tip and crest of nose, and a portion of the nasal septum.\textsuperscript{1}

The nasal septum grows inferiorly from the nasofrontal prominence to the level of the palatal shelves following fusion to form the secondary palate (\textbf{Fig. 2}). Anteriorly, the septum is contiguous with the primary palate originating from the nasomedial processes. The initial site of palatal fusion occurs posterior to the incisive foramen and extends both anteriorly and posteriorly. The fusion point between the primary and secondary palate is the incisive foramen (see \textbf{Fig. 2}).\textsuperscript{3}

At the end of its development, the nasal septum divides the nasal cavity into two separate chambers. The nasal septum’s components are the quadrangular cartilage, the perpendicular plate of the ethmoid, the vomer, the maxillary crest, the palatal crest, and the membranous septum (\textbf{Fig. 3}).

The tubular vomeronasal organ first appears as bilateral epithelial thickening on the nasal septum. By the fortieth day of gestation, this primordial structure has invaginated along the septum. The structure thus end in a blind pouch and subsequently separates from the septal epithelium. In other species, the vomeronasal organ is lined with chemoreceptors similar to those in the olfactory epithelium. This epithelium projects into the accessory olfactory bulb, which connects to the amygdala and other limbic centers.\textsuperscript{4}

\section*{LATERAL NASAL WALL EMBRYOLOGY}

At 8 weeks’ gestation, a cartilaginous nasal capsule surrounds the nasal cavity and is continuous with the cartilage of the nasal septum. Three soft tissue elevations or pre-turbinate can be identified within the nasal cavity. Even at this early stage, the preturbinates are oriented in size and position comparable with the adult inferior, middle, and superior turbinates (see \textbf{Fig. 2}).\textsuperscript{5}

By 9 to 10 weeks, the cartilage capsule develops into two cartilaginous flanges that penetrate the soft tissue elevations of the inferior and middle turbinate. A small elevation of cartilage located at the entrance to the middle meatus ultimately forms the uncinate process. This cartilage originates from the medial wall of the lateral cartilage capsule. As the uncinate begins to develop, a ridge of bone originating from the
hard palate advances posteriorly to replace the lateral cartilaginous capsule and becomes the posterolateral wall of the nose.\textsuperscript{5}

Around 11 to 12 weeks’ gestation, the primordial ethmoidal infundibulum develops as a space lateral to the uncinate process in the middle meatus. From this space, a short tract running inferolaterally toward the maxillary bone precursor is the initial development of the maxillary sinus. As the primordial maxillary sinus grows, a vertical plate of bone extending from the primitive maxilla lengthens posteriorly to separate the lower part of the orbit from the lateral cartilaginous capsule. Additionally a second vertical bony plate extends cephalad from the hard palate and forms the posteroinferior lateral wall of the nasal cavity.\textsuperscript{5}

Fig. 2. Embryogenesis of the nasal cavity and palate.
By 15 to 16 weeks’ gestation, the inferior, middle, and superior turbinates are well formed. Additionally the primordial maxillary sinus is surrounded by a sleeve of cartilage and has grown from the space lateral to the uncinate, the ethmoid infundibulum, toward the apex of maxilla inferiorly. Posterior protrusions from the ethmoid infundibulum continue to enlarge and will become the posterior ethmoid cells.5

At 17 to 18 weeks’ gestation, the thick cartilage cap of the primitive maxillary sinus leads the continuing extension of the sinus anteriorly, laterally, and inferiorly. This channel runs medial to the nasolacrimal duct near its origin at the orbit. Initial ossification of the cartilaginous precursor of the inferior turbinate also occurs at the angle where the inferior turbinate budded from the lateral cartilaginous capsule. Protrusions posteriorly into the sphenoid bone are visualized.5 Over the next 3 to 4 weeks, ossification progresses to involve the superior aspect of nasolacrimal duct near the orbit and the middle turbinate. As with its inferior counterpart, ossification of the middle turbinate commences at its site of origin from the lateral cartilaginous capsule.

By 24 weeks’ gestation, the primordial maxillary sinus has invaginated into the woven bone of the maxilla. Laterally, a bony plate separates the channel from the orbit and medially a plate of bone separates the inferior turbinate from the lateral cartilaginous capsule. In addition, the nasolacrimal duct is firmly encased in a tube of bone superiorly near the eye.5

The development of the lateral nasal wall is close to complete by 24 weeks’ gestation. By this time, the superior and middle turbinates have developed and ossified from the ethmoid bone, while the inferior turbinate has emerged from two origins, the maxilla and the lateral cartilaginous capsule. Based on the initial mucosal thickening, turbinate development appears to be a primary process, and meatal ingrowth occurs secondarily.5

Fig. 3. Schematic depiction of a sagittal view of the nasal septum and surrounding structures.
VARIATIONS LEADING TO NASAL OBSTRUCTION

Deviated Nasal Septum

A few large studies have investigated the prevalence of nasal septal deviation and have concluded that a nondeviated septum is present in only 7.5% to 23% of patients, while septal deformities are far more common.6,7 Because septal deflection has a high prevalence and multiple patterns of deformity, clinicians needed a classification system to help them sort and describe cases. Mladina7 developed such a system (Table 1). This system divides septal deformities into seven types. Types 1 and 2 represent a spectrum of septal deformities involving a unilateral vertical ridge in the valve region. A type 2 deformity is severe enough to disturb the function of the valve. In a type 3 deformity, a unilateral vertical ridge is at the level of the head of the middle turbinate. A type 4 deformity has characteristics that combine those of a type 3 deformity with those of either types 1 or 2. A type 4 deformity is often described as an S-shaped deformity. Types 5 and 6 deformities are horizontally based deformities. A type 5 deformity has a horizontal crest that is frequently in contact with the lateral nasal wall (Fig. 4). A type 6 deformity has a prominent maxillary crest contralateral to the deviation and an obvious septal crest on the deviated side. A type 7 deformity combines the characteristics of any of the other six types.8 Although this classification system thoroughly describes anatomic variations of septal deviations, it does not identify sources for these differences.

Another system, which divides septal deformities into anterior cartilaginous deviation and combined (cartilaginous and bony) septal deformity, correlates a cause for nasal septal deflections.6 Anterior cartilage deviation is typically localized to the anterior quadrilateral cartilage and is frequently associated with asymmetry of the external bony pyramid and dislocation of cartilage off the anterior nasal spine. This deformity is more common in newborns delivered vaginally, particularly those delivered from persistent occipitoposterior positions, than from newborns delivered via cesarean. The deformity can also occur, though rarely, in newborns delivered via cesarean secondary to the pressure on the head during internal rotation. The internal rotation stage of delivery forces the face and shoulder against the pelvic wall, which can lead to deformity of the nasal cartilage and distortion of the bony pyramid.9 Combined septal deformity involves all septal components, including the vomer bone, the perpendicular plate of the ethmoid, and the quadrilateral cartilage. Deformities can include a spur at the vomer ethmoid junction or a C- or S-shaped bending of cartilage and a compensatory hypertrophy of turbinate opposite the side of the deviation. There are typically associated deformities of the cheek, external nares, palate, and malocclusion of the teeth. Therefore, a combined septal deformity is part of a greater generalized facial deformity.

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<td>Mladina classification for nasal septal deviation</td>
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<th>Septal Deformity</th>
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<td>Type 1</td>
<td>Unilateral vertical ridge in the valve region</td>
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<td>Type 2</td>
<td>Similar to type 1 but more severe obstruction and disturbance of nasal valve</td>
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<tr>
<td>Type 3</td>
<td>Unilateral vertical ridge at the level of the head of the middle turbinate</td>
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<td>Type 4</td>
<td>Combination of type 3 with either type 1 or 2</td>
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<td>Type 5</td>
<td>Horizontal septal crest in contact with the lateral nasal wall</td>
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<tr>
<td>Type 6</td>
<td>Prominent maxillary crest contralateral to the deviation with a septal crest on the deviated side</td>
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<tr>
<td>Type 7</td>
<td>Combination of previously described septal deformity types</td>
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The maxillary molding theory, in giving possible explanations for septal deformities, considers anterior septal deviation and combined septal deformity to be variations in a single spectrum of deformities stemming from stresses and strains on the skull of the fetus. During pregnancy, the fetus is subjected to various torsions and pressures and the skull bones are malleable to these forces. Skull bones are not elastic. Once they are displaced, these bones will continue to grow in their altered alignment. Depending on the severity, direction, and location of the pressure, local deformities may develop, including anterior septal deviation. If the force is great enough, the septum can be compressed against the solid skull base, resulting in a splaying of the cartilage at the vomer ethmoid junction and creating a C- or S-shaped deformity.

Torsional strains can cause unequal parietal bone molding. This unilateral pressure can cause medial displacement of the maxilla and subsequent malocclusion of the teeth and elevation of the palate on the side of the pressure. Palatal elevation causes a tilting of the vomer away from the compressing forces, leading to septal deviation. Although forces during parturition are probably responsible for most septal deformities, a genetic component may be involved in posterior deformities.

The maxilla has solid articulations posteriorly with the skull. Therefore, when external forces are applied, the resulting deformities are typically located anteriorly. Given this anatomic arrangement, it appears that posterior deformities have a genetic component or a normal component to maxillary complex development, whereas anterior deformities are more often related to extrinsic forces.

**Inferior Turbinate Hypertrophy**

Inferior turbinate hypertrophy is a common cause of surgically correctable nasal obstruction. No clear developmental reasons have been given for this condition. Three different variations are often encountered and include bony, soft tissue, and mixed hypertrophy. Bony turbinate hypertrophy is usually caused by a prominent (broad) inferolateral turn of the turbinate. Very large but normally shaped obstructing inferior turbinates are also described. However, these are not as prevalent as the prominent inferolateral turn. Soft tissue hypertrophy is very common and represents the majority of cases of inferior turbinate hypertrophy. The common underlying pathophysiology in soft tissue hypertrophy is chronic rhinitis and other conditions that cause chronic mucosal inflammation (Fig. 5). Mixed inferior turbinate hypertrophy involves anatomic bony hypertrophy in the setting of chronic rhinitis (Fig. 6). Although very uncommon,
pneumatization of the inferior turbinate (Fig. 7) may cause inferior turbinate hyper-
trophy and leads to nasal obstruction.

**Paradoxical Middle Turbine**

Paradoxical middle turbinate refers to an inferomedially curved middle turbinate edge with its concave surface adjacent to the septum (Fig. 8). This anatomic variant alone is not pathologic, but can lead to significant narrowing of the middle meatus and cause ostiomeatal complex obstruction with resultant rhinosinusitis. Although not frequently seen, when associated with a bulbous middle turbinate, paradoxical middle turbinate can potentially lead to nasal obstruction. This finding usually occurs bilaterally.

**Concha Bullosa**

A concha bullosa represents pneumatization of the middle turbinate. This anatomic variant is usually found bilaterally with variable asymmetry (Fig. 9A). However, unilateral conchae bullosa are not uncommon (Fig. 9B). Although a concha bullosa is not a pathologic entity, it can lead to nasal obstruction when overly pneumatized.
Unilateral concha bullosa is often associated with a deflection of the nasal septum away from the side of the concha. The degree of septal deflection usually parallels the size of the concha bullosa. Since the middle turbinate is part of the ethmoid complex, concha bullosa is typically seen in patients with highly pneumatized ethmoid sinuses.

Choanal Atresia

Choanal atresia is a congenital obstruction of the posterior nasal apertures. This abnormality can occur unilaterally or bilaterally (Fig. 10) with a female-to-male ratio nearing 2:1. The incidence of choanal atresia is estimated to be 1 in 5000 to 7000 live births with the unilateral anomaly occurring more frequently. When the unilateral atresia is present, the right side is affected twice as often with a corresponding septal deviation on the affected side. Traditionally, choanal atresia has been described as bony, membranous, or mixed membranous-bony with the bony entity being the most common, accounting for approximately 90% of cases. Recently, the mixed...
membranous–bony atresias have been found to be the most common defect, occurring in 70% of cases. Choanal atresia can be an isolated finding but is associated with other anomalies in 50% of cases. The most commonly described association is with CHARGE syndrome (CHARGE stands for cluster of characteristics that include coloboma of the eye, heart defects, atresia of the choanae, retardation of growth or development, genital or urinary abnormalities, and ear abnormalities and deafness). Association with Crouzon syndrome and other syndromes have also been described.

Since the initial description of choanal atresia by Roederer in 1755, several theories have been developed to explain the etiology of the atresia plate. The four basic principles that have come to be accepted are:

- Persistence of the buccopharyngeal membrane from the foregut
- Abnormal persistence or location of mesoderm-forming adhesions in the naso-choanal region
- Abnormal persistence of the nasobuccal membrane of Hochstetter
- Misdirection of neural crest migration with subsequent mesodermal flow

Fig. 9. (A) Coronal CT showing bilateral conchae bullosa (arrows). (B) Coronal CT demonstrating markedly enlarged unilateral concha bullosa (arrow) with contralateral septal deflection.

Fig. 10. Axial CT demonstrating bilateral choanal atresia (arrows).
Other theories that have been proposed but not commonly accepted include:

Resorption of the floor of the secondary nasal fossa
Incomplete dorsal extension of the nasal cavity

Most believe that the latter two hypotheses are likely to result in nasal stenosis as opposed to choanal atresia.

The development of choanal atresia can only be understood with a fundamental knowledge of embryologic development. The first 12 weeks of gestation are critical for facial development. The neural crest cells migrate to preordained locations in the branchial arch mesenchyme at the end of fourth week of gestation. In the next 2 weeks, cellular proliferation and migration within the various facial processes occur, leading to the development of identifiable structures, including the columella, philtrum, and upper lip. Additionally, the nasal processes proliferate around the nasal pits, while the nasal pits are furrowing deeper within the mesenchyme. As the nasal pits migrate posteriorly, they ultimately meet the frontal portion of the stomodeum with only a sheet of tissue separating them. This thin sheet of tissue is termed the *nasobuccal membrane* and typically ruptures to create a nasal cavity with the primary choanae. As development continues, the primary of primitive choanae undergoes further alterations as fusion of septal elements and palate moves the choana more posteriorly to form the secondary choana.19

**Pyriform Aperture Stenosis**

Congenital pyriform aperture stenosis (CPAS) was first described by Brown and colleagues20 in 1989. Although its exact cause is unknown, this very rare disorder is thought to be secondary to overgrowth of the nasal process of the maxilla during the fourth to eighth week of gestation.20,21 CPAS is usually bilateral, although unilateral presentation can occur.20 The pyriform aperture represents the narrowest most anterior bony part of the nasal airway and small changes in the diameter at this site can significantly affect nasal airway resistance and cause nasal obstruction (**Fig. 11**). It is deemed stenotic if the maximum transverse diameter of each aperture is less

![Axial CT demonstrating a narrowed pyriform aperture (arrows).](image-url)
than or equal to 3 mm or when the combined aperture width is less than 8 mm.\textsuperscript{20} CPAS is associated with a central maxillary incisor in about 63\% of cases.\textsuperscript{22} There may also be a relationship between CPAS and holoprosencephaly since a central maxillary incisor is also commonly found in the latter, and a case report of monozygotic twins reported that one twin presented with holoprosencephaly and the other with CPAS.\textsuperscript{23,24}

REFERENCES