Disorders of the Nasal Cavity

Scott R. Schoem

Introduction

One of the myths in otolaryngology is the notion that neonates are obligate nose breathers. This long-believed misconception still permeates our literature. The word "obligate" implies that the neonate has no choice but to breathe through the nose. The appropriate terminology is that neonates are "preferential" nasal breathers since the infant can breathe through the mouth if the nose is blocked [1–3]. However, in certain circumstances such as with bilateral choanal atresia, the child has complete nasal obstruction and must breathe through the mouth. The term cyclical cyanosis refers to this condition whereby the neonate attempts to breathe through the nose, is unable to do so resulting in hypercapnia, opens the mouth to cry, and then breathes orally temporarily resolving the cyanosis. This results in severe breathing and discoordination of feeding problems. Yet, at least one report describes bilateral choanal atresia first discovered in adulthood [4].

Some disorders may be life-threatening and not amenable to surgical correction in the neonatal period, while others are mild and self-limiting. Anatomic, structural lesions may be bilateral or unilateral. Most neonates resolve nonanatomical obstruction with medical management and growth.

Normal Nasal Physiology

The nasal cavity provides several important physiologic functions including air-conditioning, filtration of inhaled particles and microbes, maintaining mucus flow, sinus drainage, and olfaction [5, 6]. The proper physical shape of the nasal interior impacts on these functions. The nasal mucosa contains a rich supply of resistance blood vessels such as arterioles and arteriovenous anastomoses that drain into venous sinusoids. The sinusoids are innervated by sympathetic fibers which release norepinephrine leading to reduced blood flow and venous return into capacitance vessels. This results in decreased nasal congestion. Conversely, parasympathetic fibers release acetylcholine which increases nasal secretions and vasodilation. Sensory C-fibers release neurokinin A, calcitonin gene-related peptide, and substance P that downregulate sympathetic vasoconstriction and increased congestion.

Pathogenesis

On initial evaluation by an otolaryngologist, nasal obstruction in neonates and infants is generally classified as either an anatomic anomaly or congestion due to some nonanatomic problem—medication, allergy, laryngopharyngeal reflux, or some undiagnosed issue. However, the astute clinician should delve much more carefully into the pathogenesis or pathophysiology of neonatal nasal obstruction. Development of nasal obstruction is an extremely complex interplay of still not very well-defined competing sympathetic and parasympathetic triggers. Moreover, mast cells, eosinophils, basophils, and goblet cells release local inflammatory mediators including histamine, kinins, prostaglandins, arachidonic acid metabolites, and mucin that all contribute to nasal congestion [6].

Clinical Presentation and Evaluation

Rarely does a neonate have complete nasal obstruction except in the event of arhinia or bilateral choanal atresia. Usually there is at least some airflow on at least one side. For the baby with bilateral choanal atresia, the child will exhibit cyclical cyanosis marked by failed attempt to breathe nasally, eventual crying with mouth opening, oral air

S.R. Schoem, M.D., F.A.A.P. (✉)
Division of Otolaryngology—Head and Neck Surgery, University of Connecticut, Connecticut Children's Medical Center, Hartford, CT 06106, USA
e-mail: sschoem@ccmckids.org

J. Lioy and S.E. Sobol (eds.), *Disorders of the Neonatal Airway: Fundamentals for Practice*,
DOI 10.1007/978-1-4939-1610-8_4,

exchange with cessation of hypoxia and cyanosis, and resumption of the cycle once the mouth closes. Placement of an oral prop eliminates the problem temporarily allowing for an orderly evaluation.

An initial overall examination includes the assessment of whether the baby has the ability to breathe nasally and if so whether this is unilateral or bilateral. Traditionally, we are taught to pass a 5–6 Fr catheter in the nose on each side to determine patency. However, the catheter often coils up in the nose or bounces against the inferior turbinate invalidating interpretation of the maneuver. Rather than the catheter test, with the mouth closed, try the mirror test by placing a laryngeal mirror in front of the nose on each side to see if nasal airflow fogs the mirror. Alternatively, if the parent has a pocket mirror, place it in front of the nose to see if two fog dots are present. In addition, listen for stertor, which is a low-pitched, inspiratory noise caused by turbulent airflow of nasal, nasopharyngeal or oropharyngeal tissue vibrations. If there is no air movement, then flexible fiberoptic endoscopy usually will help determine the site and degree of obstruction. Flexible endoscopy is very helpful to distinguish pryiform, midnasal or posterior nasal stenosis, a congenital nasal septal deviation, or mass effect either intranasally or in the nasopharynx. Rigid telescope endoscopy in the neonate is much more difficult to accomplish than flexible endoscopy.

Examination of breathing – feeding coordination is essential. If the baby is unable to feed properly due to nasal obstruction, or has discoordination of feeding with choking, this may accelerate the need for intervention. A modified barium esophagram or endoscopic evaluation of feeding may assist in the assessment of feeding.

Imaging studies may be very helpful or essential. Computed tomography (CT) is most helpful to diagnose and surgically plan bony anomalies such as pyriform stenosis and choanal atresia. Magnetic resonance imaging (MRI) is best for soft tissue obstructing masses that may have intracranial extension such as encephaloceles and nasal dermoids. Antenatal fetal MRI has been enormously helpful in establishing the extent of head and neck masses causing respiratory obstruction.

Differential Diagnosis and Management

After performing a complete history and physical examination, most diagnoses are evident and straightforward. In general, the care algorithm depends on whether the baby has generalized nasal inflammation or an anatomic/surgical entity.

Nasal Inflammation/Rhinitis

In neonates, placental blood flow transmits medications and drug metabolites that may cause nasal congestion. Several known agents may cause nasal congestion including antihypertensives, beta-blockers, antidepressants, cocaine, and gabapentin. Sometimes, obtaining a truthful maternal medication history is difficult and the clinician needs to proceed with treatment options empirically. Neonatal rhinitis is defined as mucoid or clear rhinorrhea with nasal mucosal edema in the afebrile newborn with stertor, poor feeding, and respiratory distress that responds to the use of steroid drops [7].

After establishing the diagnosis of a non-surgically correctable lesion, treatment starts with nasal saline to decongest the nasal mucosa. If unsuccessful, topical oxymetazoline may be used three times a day for 3–4 days for maximal decongestion while concurrently starting use of topical steroid and continuing with topical nasal steroids.

Birth Trauma

The reported incidence of birth trauma-related external nasal and intranasal septal deviation is reported as 1 %. However, in clinical practice, it seems much less common. Moreover, the clinician should be careful not to label the baby with a deviated nose as birth delivery-related, when the mechanism of action may be positioning of the head in the birth canal during uneventful passage. When the deviation is minor, this usually self-corrects within several weeks after birth. Rarely, the nose is so deviated that a reduction in the first week after birth is required to re-establish normal nasal breathing. Mild anterior nasal septal abnormalities are common, but rarely cause any respiratory distress.

Congenital Anatomic Anomalies

Arhinia

Congenital arhinia (or arrhinia) is a rare entity defined as absence of the external nose, nasal cavities, and olfactory apparatus. Embryologically, arhinia is due to failed fusion of the maxillary process and the lateral nasal process between the third and eighth week of gestation. Cribriform plate fusion abnormalities lead to olfactory agenesis. Tessier defines this malformation as total arrhinia, hemi-arrhinia, and proboscis lateralis [8]. Arhinia may be associated with hypertelorism, microphthalmia, palatal abnormalities, cryptorchidism, and blindness [9]. Many children with arhinia have normal intelligence Fig. 1.

Initial management of arhinia may require tracheotomy to provide a safe airway with deferred surgical planning as a child or adolescent once the degree of cognitive development has been established. For high-functioning children, a craniofacial team approach of otolaryngology, plastic surgery, and potentially neurosurgery is required to establish nasal patency followed by external nasal reconstruction. Both CT and MRI may be complementary for surgical planning.

Anterior (Pyriform) Stenosis

Congenital nasal pyriform aperture stenosis (CNPAS) is an unusual narrowing of the anterior nasal vault. Anatomically, the anterior nasal vault is defined by the nasal bones superiorly, the nasal process of the maxilla laterally and the junction of the horizontal process of the maxilla and the anterior nasal spine inferiorly.

This entity was first described in adults in 1952 [10] and in a neonate in 1989 [11]. CNPAS may be an isolated anomaly or associated with other craniofacial malformations. The most common is the holoprosencephaly spectrum including a single central upper incisor tooth, absent upper labial frenulum, and absence of the corpus callosum [12, 13]. In mild cases, the neonate may have stertor and mild difficulty coordinating breathing and feeding. In more severe cases, the child may have symptoms and signs that mimic atresia with cyclical cyanosis and respiratory distress. Often, patients will pass the mirror test since there is stenosis but not atresia.

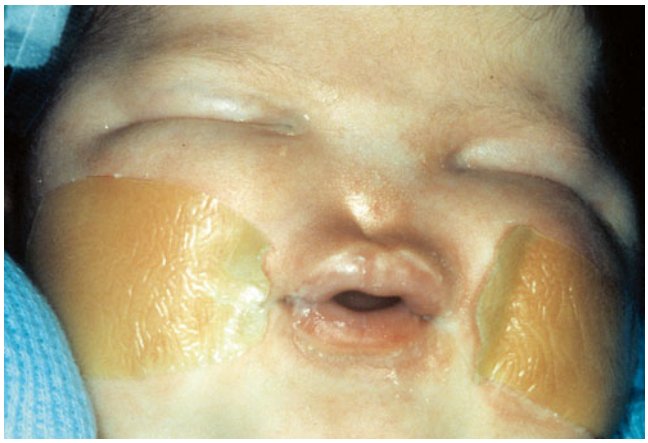

Fig. 1 Photo of arhinia

Flexible nasal endoscopy may demonstrate the inability to pass the infant fiberoptic telescope. CT scan is diagnostic and helps to rule out other midline anatomic abnormalities. Beldon performed CT analysis and determined that the normal term infant should have at least an 11 mm width across the pyriform aperture Fig. 2 [14].

Initial management depends on the degree of respiratory distress. In isolated instances, intubation is rarely necessary. However, in syndromic neonates, establishment of a safe airway may be necessary. If multiple airway anomalies are present, nasal surgery alone may not be sufficient and a tracheotomy is required. When the lesion is isolated, surgery with nasal stenting is very successful.

Mid-vault Stenosis

Rarely, a congenital thickening of the nasal septum or narrowing of the total vault may cause mid-vault stenosis premature infants. Typically, this is self-limited and responds well to topical steroid treatment.

Posterior (Choanal) Stenosis or Atresia

Choanal atresia (CA) is a relatively rare malformation occurring in approximately 1 in 7,000–8,000 births. Unilateral atresia is more common than bilateral. Previous textbooks report that CA is more common in females than males; however, more recent reviews report an equal gender distribution [15, 16]. The purported mechanism leading to CA is either failure of buccopharyngeal membrane breakdown or persistence and misdirection of mesoderm during embryogenesis. In only rare circumstances is the obstruction due to soft tissue alone. In almost all cases, there is some bony component that needs to be removed for successful surgical treatment.

Bilateral CA is diagnosed shortly after birth due to characteristic cyclical cyanosis. The mirror test or inability to pass a 5–6 Fr catheter is suspicious for CA. Flexible

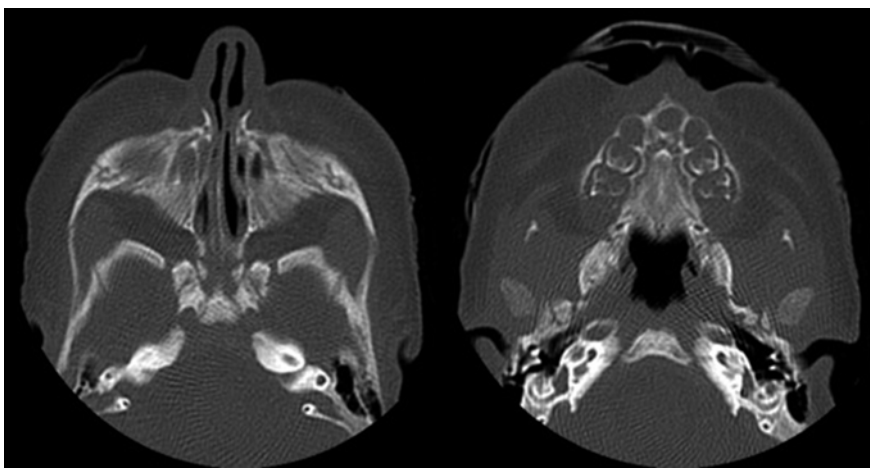

Fig. 2 CT scan of pyriform stenosis and single central incisor associated with holoprosencephaly spectrum

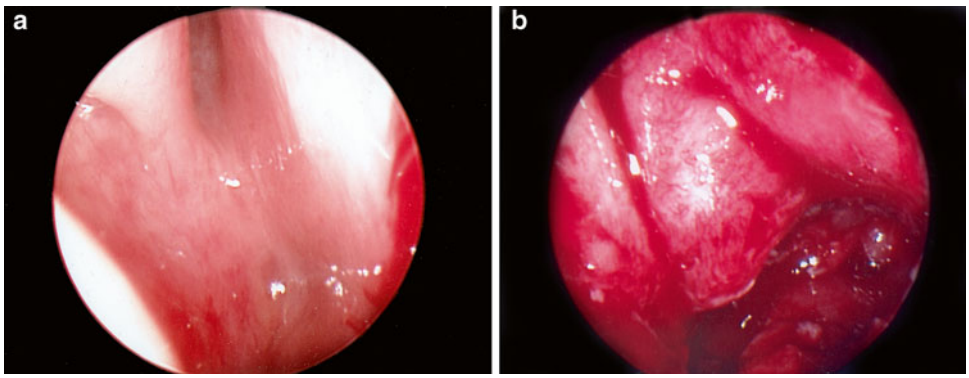

Fig. 3 (**a**) Endoscopic photo of choanal atresia preop. (**b**) Endoscopic photo of choanal atresia postop

fiberoptic endoscopy is diagnostic in most cases Fig. 3. Occasionally, an obstructing posterior nasal or nasopharyngeal growth may mimic CA. Fine cut CT scan is essential to determine the site and degree of obstruction, and image the presence of the nasopharyngeal roof and cavity. Unilateral CA may go unrecognized for years and often presents during childhood or adolescence as inability to breathe through one side of the nose with constant unilateral runny nose. Likewise, choanal stenosis as opposed to atresia may go unrecognized for years.

Every neonate with choanal atresia should be screened for CHARGE syndrome. CHARGE used to be classified as an association. However, with the discovery of the genetic marker CHD7 on Chromosome 8, which encodes the chromodomain helicase DNA binding protein, CHARGE is now classified as a syndrome [17]. CHARGE syndrome is characterized by ocular *C*olobomas, *H*eart defects, choanal *A*tresia, *R*etarded growth, *G*enitourinary hypoplasia, and *E*ar abnormalities. CHD7 analysis in CHARGE syndrome detects mutations in 65–70 % of individuals. Cardiovascular abnormalities occur in 75–85 % and tracheoesophageal fistula is present in 15–20 % of newborns with CHARGE syndrome. Ossicular malformations, cochlear anomalies, and semicircular canal hypoplasia occur in over 80 % of patients [17].

Bilateral CA is a medical urgency; however, it is not a surgical emergency. Use of an oral prop for adequate breathing and an orogastric feeding tube for nutrition allows for the orderly evaluation of other potential anomalies, proper counseling, and surgical planning. With the advent of endoscopic equipment, miniaturization of power-assisted drills and shavers, surgical repair of choanal atresia has evolved over the past 20 years. Whereas previous textbooks discuss both transpalatal and transnasal approaches, the vast majority of current repairs are performed using transnasal techniques. Whether or not to place a short-term stent is still debated in the literature [18–21]. The most difficult cases involve syndromes with severe down sloping nasopharyngeal roofs or nearly absent nasopharyngeal cavities such as Treacher Collins syndrome.

Congenital Masses

Nasolacrimal Duct Cyst (Dacryocystocele)

Obstruction of the nasolacrimal duct is a common anomaly with widely varying reported rates [22, 23]. Many duct obstructions are partial and self-correct by 1 year of age. The duct is usually obstructed at the valve of Hasner just lateral to the inferior turbinate [24]. This may lead to a round mass in the lower nasal cavity. If the proximal valve is obstructed at the common canniculus, this leads to a round facial swelling inferior to the medial canthus of the eye. This may become infected with impressive swelling and erythema mimicking acute unilateral sinusitis. Occasionally, when bitateral cysts are present, this may lead to severe nasal obstruction in the neonate and difficulty coordinating breathing and feeding similar to bilateral choanal atresia. When the clinical picture is uncertain, CT scan with contrast or MRI helps to distinguish between bilateral nasolacrimal duct obstruction, anterior pyriform stenosis, or other mass effects due to tumor.

Surgical treatment and timing varies depending on surgeon preference and severity of presentation. Unilateral dacryocystoceles rarely need urgent surgical correction. However, bilateral obstruction may require surgery within the first few weeks of life. Treatment ranges in escalation from duct probing and balloon dilation for simple duct obstruction to intranasal marsupialization and silastic stenting for large obstructing cysts [25].

Infantile Hemangioma

Infantile hemangiomas are the most common benign neoplasms of infancy. The most common site is in the head and neck primarily in the central facial region. Occasionally, a

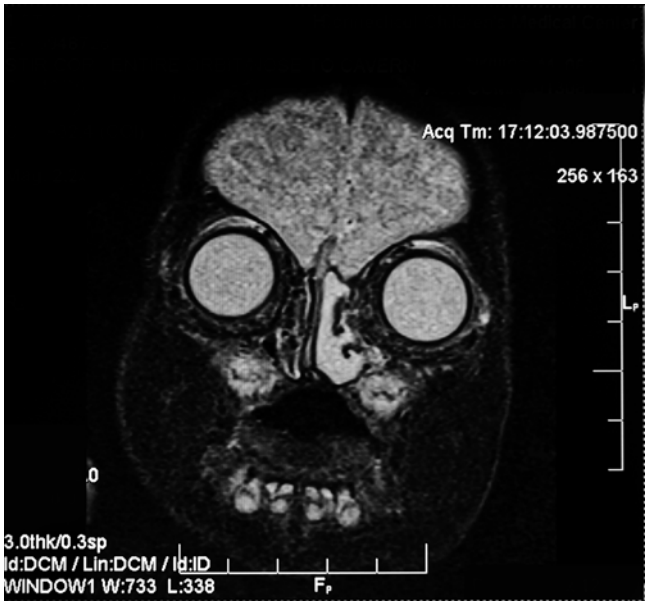

Fig. 4 CT scan of left intranasal glioma: note distinct separation from dura

large nasal and facial hemangioma may proliferate quickly in the intranasal region leading to obstructed breathing. The traditional treatment for many years had been intralesional injection of steroids or systemic steroids with laser ablation during the proliferative phase. However, in recent years, propranolol treatment has supplanted older modalities [26, 27]. Propranolol has been identified on molecular analysis to block endothelial cell proliferation, migration, and formation of the actin cytoskeleton with alteration in vascular endothelial growth factor receptor- 2 [28]. Neonates and infants should be referred to a center familiar for multidisciplinary evaluation and management [29].

Meningocele, Meningoencephalocele, and Glioma (Heterotopic Glial Tissue)

Meningoceles, meningoencephaloceles, and gliomas are often grouped together. A meningocele represents downward displacement into the nose of only the meninges due to small defects in the floor of the intracranial cavity. A meningoencephalocele results from a larger defect that allows for herniation of both meninges and brain tissue. This may extend outward at the glabellar region or only downward with a cribiform plate defect with a large intranasal mass that resemble a polyp. By definition, a nasal glioma is sequestered or displaced neural tissue with fibrous and vascular connective tissue that increases the firmness of the mass. There may be a connecting fibrous stalk to the skull base in 10–15 %; however, it is rare to have any meningeal connection. Therefore, clinically, a meningoencephalocele is soft, compressible, pulsatile; whereas a glioma is firm, noncompressible, and non-pulsatile.

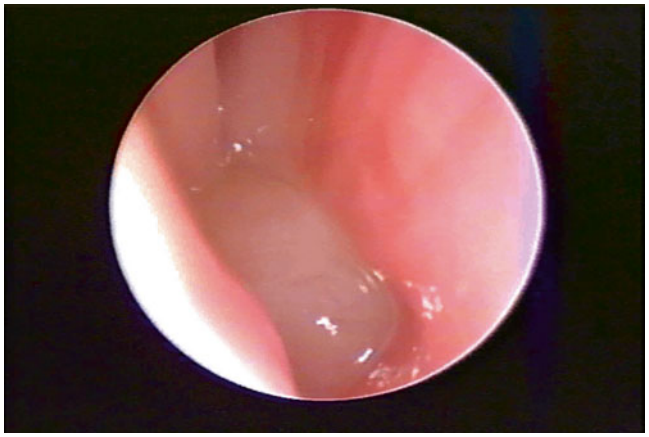

Fig. 5 Endoscopic photo of nasopharyngeal glioma

CT scan helps to identify the bony defect in the skull base. MRI is much more useful to demonstrate the characteristics of the soft tissue mass and locate the site of intracranial extension Fig. 4. On T1-weighted MRI, the intranasal component is isointense or hypointense and on T2-weighted images is hyperintense relative to brain tissue. Gliomas are 60 % extranasal, 30 % intranasal, and 10 % combined (dumbbell growth).

Surgery on a meningoencephalocele requires a neurosurgeon and otolaryngologist. Surgery is typically via anterior frontal craniotomy [30]. Glioma surgery is typically performed via the intranasal approach and usually does not require intraoperative neurosurgical involvement; however, a neurosurgeon should be available on a standby basis for any unexpected encounter or complication Fig. 5 [31, 32].

Nasal Dermoid Sinus and Cyst

Derived from ectoderm and mesoderm, a dermoid cyst or sinus may form due to a remnant in the prenasal space posterior to the nasal bones and anterior to the nasal and septal cartilages. The typical sinus tract has a pit and related small hair tuft on the nasal dorsum. This may present as an infected midline mass or with keratin discharge. The tract is usually well-defined on imaging. However, in some cases, the distal extent may project anteriorly all the way to the nasal tip altering surgical planning or increasing the likelihood of recurrence. CT scan helps to define bony skull-base defects and widening of the crista galli, which is associated with potential intracranial extension. MRI is very useful to determine the extent of the tract and mass, and serpentine extensions and intracranial extension.

If no imaging evidence of intracranial extension, surgical correction may be limited to a nasal approach with neurosurgical standby. Various options include the endoscopic-assisted closed approach for cysts without sinus tracts, external rhinoplasty, and the direct, open approach [33–35]. Even if the tract has intracranial extension, in selected cases

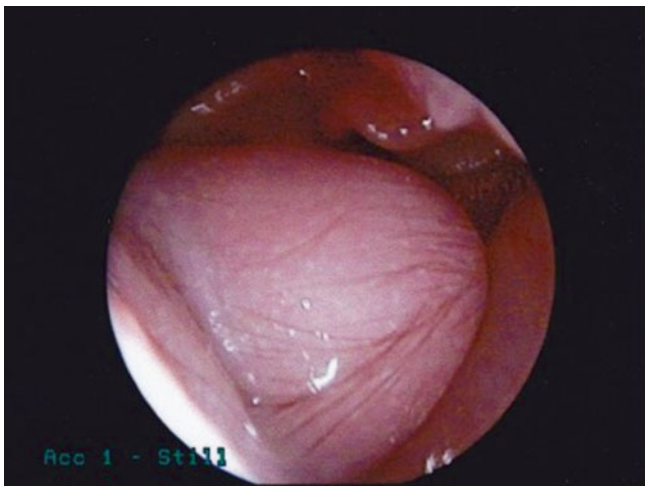

Fig. 6 Endoscopic photo of nasopharyngeal teratoma (courtesy of Tulio Valdez, MD)

the endonasal endoscopic approach may be sufficient; [36] however, neurosurgical standby is recommended for patient safety.

Rare Tumors

In rare cases, neonates are born with unrecognized nasal and nasopharyngeal tumors causing varying degrees of obstruction. These may be benign or malignant teratomas, or other unusual and aggressively growing fibrous tissue tumors Fig. 6 [37–39]. More often, these tumors are noted on fetal ultrasound during gestation with polyhydramnios due to impaired fetal swallowing. Fetal MRI delineates the site and degree of obstruction enabling an orderly evaluation and plan for management.

Multidisciplinary Considerations

The *EX*-utero *I*ntrapartum *T*reatment (EXIT) approach to management has been highly successful in many cases to provide safe initial airway management followed by tumor excision after stabilization [40]. Maternal-fetal circulation may be prolonged for up to almost 1 h providing oxygen to the baby while establishing airway control. The EXIT procedure using a combined team approach has become the best modality for optimal care of known obstruction lesions discovered during pregnancy [41].

Future Considerations

In the past decade, the newest frontier is development of operative teams for intrauterine surgery. Intrauterine surgery is a rapidly developing field. Fetal surgery for spina bifida and congenital diaphragmatic hernia has become a therapeutic alternative for expectant mothers in selected cases [42–44]. The potential exists for expansion of fetal surgery to other embryologic anomalies and tumors.

References

1. Bergeson PS, Shaw JC. Are infants really obligate nasal breathers? Clin Pediatr. 2001;40:567–9.
2. Miller M, Martin R, Carlo W, Fouke J, Strohl K, Fanaroff A. Oral breathing in newborn infants. J Pediatr. 1985;107:465–9.
3. Trabalon M, Schaal B. It takes a mouth to eat and a nose to breathe: abnormal oral respiration affects neonates' oral competence and systemic adaptation. Int J Pediatr. 2012; 2012:207605.
4. Panda N, Simhadri S, Ghosh S. Bilateral choanal atresia in an adult: is it compatible with life? J Laryngol Otol. 2004;118:244–5.
5. Wang D, Lee H, Gordon B. Impacts of fluid dynamics simulations in study of nasal airflow physiology and pathophysiology in realistic human three-dimensional nose models. Clin Exp Otorhinolaryngol. 2012;5:181–7.
6. Leong S, Chen X, Lee H, Wang D. A review of the implications of computational fluid dynamic studies on nasal airflow and physiology. Rhinology. 2010;48:139–45.
7. Nathan C, Seid A. Neonatal rhinitis. Int J Pediatr Otorhinolaryngol. 1997;14:59–65.
8. Tessier P, Ciminello F, Wolfe S. The arrhinias. Scand J Plast Reconstr Surg. 2009;43:177–96.
9. Graham J, Lee J. Bosma arhinia microphthalmia syndrome. Am J Med Genet. 2006;140:189–93.
10. Douglas B. The relief of vestibular nasal obstruction by partial resection of the nasal process of the superior maxilla. Plast Reconstr Surg. 1952;9:42–51.
11. Brown O, Myer C, Manning S. Congenital nasal pyriform aperture stenosis. Laryngoscope. 1989;99:86–91.
12. Hui Y, Friedberg J, Crysdale W. Congenital nasal pyriform aperture stenosis as a presenting feature of holoprosencephaly. Int J Pediatr Otorhinolaryngol. 1995;31:263–74.
13. Tavin E, Stecker E, Marion R. Nasal pyriform aperture stenosis and the holoprosencephaly spectrum. Int J Pediatr Otorhinolaryngol. 1994;28:199–204.
14. Belden C, Mancuso A, Schmalfuss I. CT features of congenital nasal pyriform aperture stenosis: initial experience. Radiology. 1999;213:495–501.
15. Harris J, Robert E, Kallen B. Epidemiology of choanal atresia with special reference to the CHARGE association. Pediatrics. 1997;99: 363–7.
16. Hengerer A, Brickman T, Jeyakumar A. Choanal atresia: embryologic analysis and evolution of treatment, a 30-year experience. Laryngoscope. 2008;118:862–6.
17. Bauer P, Wippold F, Goldin J, Lusk R. Cochlear implantation in children with CHARGE association. Arch Otolaryngol Head Neck Surg. 2002;128:1013–7.
18. Schoem S. Transnasal endoscopic repair of choanal atresia: why stent? Otolaryngol Head Neck Surg. 2004;131:362–6.
19. El-Ahl M, El-Anwar M. Stentless endoscopic transnasal repair of bilateral choanal atresia starting with resection of vomer. Int J Pediatr Otorhinolaryngol. 2012;76:1002–6.
20. De Freitas R, Berkowitz R. Bilateral choanal atresia repair in neonates—a single surgeon experience. Int J Pediatr Otorhinolaryngol. 2012;76:873–8.
21. Bedwell J, Choi S. Are stents necessary after choanal atresia repair? Laryngoscope. 2012;122:2365–6.

22. Cunningham M, Woog J. Endonasal endoscopic dacryocystorhinostomy in children. Arch Otolaryngol Head Neck Surg. 1998;124:328–33.
23. Guery D, Kendig E. Congenital impotency of the nasolacrimal duct. Arch Ophthalmol. 1979;97:1656–8.
24. Shashy R, Durairaj V, Holmes J, Hohberger G, Thompson D, Kasperbauer J. Congenital dacryocystocele associated with intranasal cysts: diagnosis and management. Laryngoscope. 2003; 113:37–40.
25. Leonard D, O'Keefe M, Rowley H, Hughes J. Neonatal respiratory distress secondary to bilateral intranasal dacryocystoceles. Int J Pediatr Otorhinolaryngol. 2008;72:1873–7.
26. Leaute-Labreze C, Dumas de la Roque E, Hubiche T, Boralevi F, Thambo J, Taieb A. Propranolol for severe hemangiomas of infancy. N Engl J Med. 2008;12:2649–51.
27. Sans V, Dumas de la Roque E, Berge J, Grenier N, Boralevi F, Mazereeuw-Hautier J, Lipsker D, Dupuis E, Ezzedine K, Vergnes P, Taieb A, Leaute-Labreze C. Propranolol for severe infantile hemangiomas: follow up report. Pediatrics. 2009;124:e423–31.
28. Stiles J, Amaya C, Pham R, Rowntree R, Lacaze M, Mulne A, Bischoff J, Kokta V, Boucheron L, Mitchell D, Bryan B. Propranolol treatment of infantile hemangioma endothelial cells: a molecular analysis. Exp Ther Med. 2012;4:594–604.
29. Parikh S, Darrow D, Grimmer J, Manning S, Richter G, Perkins J. Propranolol use for infantile hemangiomas: American Society of Pediatric Otolaryngology vascular anomalies task force practice patterns. JAMA Otolaryngol Head Neck Surg. 2013;139:153–6.
30. Mahapatra A, Agrawal D. Anterior encephaloceles: a series of 103 cases over 32 years. J Clin Neurosci. 2006;13:536–9.
31. Rahbar R, Resto A, Robson C, Perez-Atayde A, Goumnerova L, McGill T, Healy G. Nasal glioma and encephalocele: diagnosis and management. Laryngoscope. 2003;113:2069–77.
32. Yokoyama M, Inouye N, Mizuno F. Endoscopic management of nasal glioma in infancy. Int J Pediatr Otorhinolaryngol. 1999;51:51–4.
33. Holzmann D, Huisman T, Holzmann P, Stoeckli S. Surgical approaches for nasal dermal sinus cysts. Rhinology. 2007;45:31–5.
34. Locke R, Kubba H. The external rhinoplasty approach for congenital nasal lesions in children. Int J Pediatr Otorhinolaryngol. 2011;75:337–41.
35. Winterton R, Wilks D, Chumas P, Russell J, Liddington M. Surgical correction of midline nasal dermoid sinus cysts. J Craniofac Surg. 2010;21:295–300.
36. Re M, Tarchini P, Macri G, Pasquini E. Endonasal approach for intracranial nasal dermoid sinus cysts in children. Int J Pediatr Otorhinolaryngol. 2012;76:1217–22.
37. Klein A, Schoem S, Altman A, Eisenfeld L. Inflammatory myoblastic tumor in the neonate: a case report. Otolaryngol Head Neck Surg. 2003;128:145–7.
38. Katona G, Hirschberg J, Hosszu Z, Kiraly L. Epipharyngeal teratoma in infancy. Int J Pediatr Otorhinolaryngol. 1992;24:171–5.
39. Hansen J, Soerensen F, Christensen M. A five week old girl with inspiratory stridor due to infantile hemangiopericytoma. Eur Arch Otorhinolaryngol. 2006;263:524–7.
40. Hirose S, Farmer D, Lee H, Nobuhara K, Harrison M. The ex utero intrapartum treatment procedure: looking back at the EXIT. J Pediatr Surg. 2004;39:375–80.
41. Osborn A, Baud D, Macarthur A, Propst E, Forte V, Blaser S, Windrim R, Seaward G, Keunen J, Shah P, Ryan G, Campisi P. Multidisciplinary perinatal management of the compromised airway on placental support: lessons learned. Prenat Diagn. 2013; 18:1–8.
42. Adzick N. Fetal surgery for myelomeningocele: trials and tribulations. Isabella Forshall Lecture. J Pediatr Surg. 2012;47:273–81.
43. Diemert A, Diehl W, Glosemeyer P, Deprest J, Hecher K. Intrauterine surgery—choices and limitations. Dtsch Arztebl Int. 2012;109:603–38.
44. Adzick N. Fetal surgery for spina bifida: past, present, future. Semin Pediatr Surg. 2013;22:10–7.