Nasal obstruction in infancy

Nasal obstruction is common in infancy, a problem which is compounded by the fact that infants tend to be obligate nasal breathers. Most frequently such obstruction is associated with non-specific rhinitis, episodes of which are fortunately not severe, are of short duration, and respond to conservative measures such as instillation of saline or sodium bicarbonate solutions and aspiration of secretions. This latter manoeuvre is awkward at the best of times but is said to be made easier by using a small bulb syringe. Sometimes addition of topical decongestants is required to reduce mucosal oedema but use of these should be short term and intermittent and if persistent mucosal swelling is the problem, topical nasal steroids such as beclomethasone should be substituted. However, there are infants in whom obstruction is severe enough to present a difficult management problem, others in whom it is so persistent as to be a significant factor in failure to thrive, and the occasional infant in whom it is life threatening. Disorders that contribute to these states are discussed in this article.

Choanal atresia
In the obligate nasal breathing newborn this is a well recognised cause of nasal obstruction, although rarely encountered. It has to be suspected in any neonate unable to breathe until the mouth is opened either voluntarily or by insertion of a laryngoscope blade and suspicion is strengthened by obstruction to passage of a suction catheter through the nose. An oral airway can then be strapped in place as an emergency relief measure until the diagnosis can be confirmed. There is an association with other congenital anomalies and these should be excluded, the most frequent being the anomalies linked as the CHARGE syndrome. Confirmation is obtained either by direct examination, if a suitable fibreoptic endoscope is available, or by radiology. The traditional contrast study after aspiration of the accumulated mucoid secretions will confirm an atresia but, if facilities are available, computed tomography will provide greater detail of both the atresia partition and the dimensions of the posterior nasal cavity, information that aids in planning surgery.

Surgery is undertaken as soon as the infant is fit for a general anaesthetic and can be either performed directly through the nose or by using a transpalatal approach. However Pirsig has reviewed long term results and concludes that the deformity of palatal development seen after transpalatal surgery no longer warrants this approach in infancy. Some restenosis by fibrosis is almost inevitable after surgery and, if it causes significant obstruction, can be managed either by dilatation or by laser ablation of the fibrotic tissue.

Nasal pyriform aperture stenosis
In this more recently recognised condition there is excessive bone growth in the nasal processes of the maxillary bones with consequent stenosis of the nasal pyriform aperture. These babies present with severe nasal obstruction and in them difficulty is experienced in introducing a suction catheter into the nasal cavity. When examined

Computed tomography of congenital nasal pyriform aperture stenosis.
closely the ‘nasal valve’ area at the entrance to the nasal cavity is found to be abnormally narrow and, when probed, felt to be firm and resistant to compression as passage of the probe is attempted. The bony nature of the stenosis is best demonstrated by computed tomography (figure). Surgery is required to relieve the obstruction. Through a sublabial incision the nasal pyriform aperture is exposed, the excess bone identified and then drilled away using a microsurgical otological drill. After redraping of the mucosa in the nasal inlet stenting with Silastic tubes is required for a few days.

**Birth trauma to the nasal cavity**

During ‘moulding’ in the birth canal compressive lateral forces on the midface can cause excessive upward arching of the palate to compress the nasal septum which responds by either buckling laterally or dislocating. Both the buckled septum and associated mucosal oedema cause nasal obstruction. There has been a vogue for septal manipulation in such cases but it has been shown that the majority of buckled septal cartilages straighten spontaneously within 4–6 months. By reducing mucosal oedema using decongestant and steroid drops nasal patency can usually be obtained. Manipulation is only really required when dislocation has occurred and this can usually be recognised because it is associated with some deformity of the cartilaginous nasal skeleton.

**Infection**

Rhinitis is common, most episodes being of viral origin and running a benign course over 5–10 days with initial fever subsiding and some purulence to the secretions after a few days. The associated nasal obstruction can be distressing and the conservative measures outlined may be required but antibiotics are not needed. When purulent secretions persist for longer than 10 days and added symptoms of recurrence of fever and night time cough develop, the possibility of sinusitis has to be considered. Although this is not a well recognised entity in infancy, both the maxillary and ethmoid sinuses are present from birth and can become infected. The diagnosis is clinical since at this age radiology is generally unhelpful. There is also poor correlation between bacteria cultured from nasal secretions and those organisms causing sinusitis. However these are usually *Haemophilus influenzae*, *Streptococcus pneumoniae* or *Branhamella catarrhalis* in the early stages of sinusitis with later appearance of anaerobic organisms. Antibiotic treatment is indicated as orbital and intracranial complications, although not common at this age, can occur. Amoxycillin is the antibiotic generally recommended.

It is easy to overlook the ‘sniffles’ of congenital syphilis, especially when there are no other obvious stigmata of the disease. In its early stages it is clinically indistinguishable from the above, although later the purulence may be associated with nasal eczematoid and blood staining. It is a diagnosis that should always be kept in mind.

Persistent or frequently recurrent purulent rhinitis should trigger suspicion of cystic fibrosis as it can be the earliest manifestation of this condition. Foreign bodies also have to be borne in mind and are not uncommon in infancy. Classically they present as a unilateral obstruction with purulent discharge often with an associated unpleasant odour.

**Allergic rhinitis**

The voluminous literature on allergic rhinitis is noteworthy for the lack of any definitive article on it in infancy. Although not common it occurs with sufficient frequency to warrant consideration, usually in infants with other allergic manifestations such as asthma and eczema. In early infancy it is related to ingested allergens, most frequently milk but also soya, peanuts, egg white and wheat, but by about 1 year of age sensitivity to inhalant allergens can be demonstrated – house dust mite and pets, particularly cats (E Weinberg, personal communication). In infancy it is difficult to treat. When ingestant allergens can be demonstrated either by skin prick testing or by a radioallergosorbent test, elimination of them from the diet can produce dramatic improvement. The use of oral decongestants for the nasal obstruction is not particularly effective and tachyphylaxis soon arises, but topical decongestants are effective and these can be combined with intranasal steroids. For inhalant allergens a useful topical preparation is made by mixing 20 mg sodium cromoglycate with 10 ml 0·025% oxymetazoline (the late Dr L Shore).

**Rhinitis medicamentosa**

Use of topical nasal decongestants generally produces such a gratifying response from the nasal mucosa that it is sometimes difficult thereafter to avoid overuse and the insidious onset of increasing rebound congestion, a predilection known as rhinitis medicamentosa. In this condition there is probably a combination of reactive vasodilatation as a consequence of fatigue in the vasoconstrictor mechanism in the submucosal vessels and the β sympathomimetic outlasting α sympathomimetic effects of the decongestant. Typically tachyphylaxis occurs in which the need for topical decongestion becomes more frequent as reactive nasal congestion intensifies until a situation of chronic mucosal swelling causes almost continual obstruction. Decongestants have to be stopped and a topical steroid drop can be substituted. In an infant who is still dependent on the nasal airway some form of nasopharyngeal airway may have to be placed for a period until the mucosa has shrunk enough to restore nasal patency.

**‘Upper airway obstruction’**

In infancy it is often difficult to distinguish purely nasal obstruction from the general picture of upper airway obstruction caused by pharyngeal swelling or some mechanical obstruction. It can be functionally separated. The upper airway is a collapsible structure the patency of which is determined by the thickness of its soft tissue walls and the degree of support offered by bony, cartilaginous, and muscular surrounding structures. At this level in the airway there is a negative pressure within the lumen during inspiration and anything obstructing free access of air will increase this. This in turn causes the soft tissues surrounding the space to collapse, further exacerbating the obstruction – particularly noteworthy in ‘floppy’ infants.

There is usually minimal lymphoid tissue in and around the upper airway in early infancy but it soon begins to hypertrophy in response to immunological stimulation. The adenoid is usually the first organ to cause obstructive problems and has to be considered even in early infancy. From about 6 months of age adenoid hypertrophy is by far the commonest cause of nasal obstruction. Evaluation is difficult as interpretable lateral radiographs have to be taken with the infant relaxed during a nasal inspiration. Direct examination of the adenoid using a flexible fibre-optic endoscope passed through the nasal cavity is the best method of assessing the need for adenoidectomy. In floppy infants and those with reduced oropharyngeal dimensions even minimal hypertrophy of the tonsils can produce marked upper airway obstruction and the situation can
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arise when consideration has also to be given to tonsillcotomy in infancy.

Adenoectomy and adenotonsillectomy are not straightforward procedures in infancy particularly when the obstruction necessitating surgery has been severe or prolonged. Many are unable to maintain their airway in the postoperative period and require either intubation or a nasopharyngeal tube. As a general rule infants undergoing these procedures should be managed in a high care area with suitable monitoring for at least the first postoperative night and may need to remain under observation for an extra day or two before discharge.

There are also significant lymph nodes within the retropharyngeal space and inflammatory adenitis can cause significant enlargement that may present as ‘nasal obstruction’, usually associated with a fever. If suspected a lateral radiograph will confirm widening of the retropharyngeal soft tissue shadow, although these are notoriously unreliable when infants are crying or swallowing at the time they are taken. The best method of examination is digital palpation. In the early stages intravenous antibiotics may produce resolution – airway support is frequently required – but if there has been progression to abscess formation drainage is essential.

The tendency for nasal obstruction to exacerbate upper airway obstruction is also a factor that has to be considered in laryngeal disorders such as laryngomalacia and vocal cord palsy, both inspiratory prolapso disorders. Maintenance of a clear upper airway may enable other interventional procedures to be avoided.

Craniofacial anomalies

Craniofacial anomalies are associated with very variable nasal airway patency both as to cause and severity of obstruction. If obstructed the potential sites that have to be evaluated are the ‘nasal valve’ area anteriorly, hypertrophic turbinates in narrowed nasal cavities, posterior choanal stenosis and reduced dimensions to the nasopharynx that may be compounded by adenoid hypertrophy. A combination of endoscopic and radiological examination with both good lateral plain films and computed tomograms should enable determination of which areas are predominantly affecting the airway. For many some form of surgical procedure can then be devised to attempt to increase patency. These are not always successful and the potential for developing cardiac complications from chronic upper airway obstruction may mean that tracheostomy has to be considered, although there are some parents who prefer a long term nasopharyngeal airway but these have potential to cause nasal cartilage deformity.

Midline nasal masses

The commonest ‘tumour’ involving the nose is the haemangioma. Although these are usually external, as they expand during the first year of life, any intranasal component may cause nasal obstruction necessitating surgical intervention. Otherwise the majority completely involute over the next three to four years without the need for any treatment.

Within the nasal cavity the commonest tumour is the encephalocele which arises when the embryological foramen caecum fails to close and cranial contents herniate through the defect. They are classified as encephaloceles when the content is both dura and brain tissue and as gliomas when either only a fibrous connection extends intracranially or complete separation has occurred. Computed tomography delineates those cases in which the roof of the nasal cavity is intact, when the mass can be excised intranasally, from those in which there is a defect.8 When a defect is present local policy varies. Some neurosurgeons advocate an intranasal approach for all cases, surgery that has devastating consequences for the sense of smell and frequently precipitates epilepsy, while others prefer primary intranasal excision only proceeding to intracranial surgery when a true encephalocele is demonstrated.

Even more rare are neoplastic masses in the infant nasal cavity. At this age these are usually teratomatous malformations,9 true neoplasms usually presenting at a slightly older age.

Conclusion

Nasal obstruction in infancy can be very distressing for parents but severity is often underestimated by physicians particularly at its worst at night when the infants are sleeping but at its best when they are in our consulting rooms. It is a complaint to which, in general, we need to be more sensitive because significant nasal obstruction can pose a very real danger in infancy. The severity of the obstruction when it is at its worst needs to be determined and in particular whether or not it is associated with episodes of obstructive apnoea – a very definite indication for urgent intervention. At this age arousal mechanisms are often poorly developed and infants can very rapidly desaturate down to the levels at which cardiac arrhythmias arise.

Although most nasal obstruction in infancy is not of this order of severity and generally responds to simple conservative measures, when some of the conditions outlined need to be considered diagnosis requires a combination of history, examination, and investigation. Here the problem lies in trying to find the skills and equipment needed. The infant nose is a highly specialised area of practice and time invested in persuading a colleague in the ear, nose, and throat department to develop such an interest will be well rewarded and thereafter the more such cases are referred the greater the expertise that will develop in their management.

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