

PAEDIATRIC NASAL OBSTRUCTION – A PRACTICAL APPROACH

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INTRODUCTION

Nasal obstruction and subsequent partial upper-airway obstruction can result in troublesome morbidity for the child. It is one of the most common reasons for the presentation of children to the doctor, whether allergologist, paediatrician, general practitioner or ear, nose and throat (ENT) surgeon.¹ This article offers a straightforward and practical approach to managing the most common causes of nasal obstruction in the paediatric population.

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NASAL OBSTRUCTION

In the majority of cases, nasal obstruction is mild with habitual snoring, mouth breathing and rhinorrhoea being the main problems. Chronic mouth breathing, especially during critical growth periods, is also associated with the development of craniofacial abnormalities.² Habitual snoring was thought to be innocuous but it has been shown that snoring children have a higher incidence of neurocognitive disorders.³ Daytime sleepiness, behavioural hyperactivity, learning problems and restless sleep are all significantly more common in habitual snorers.³

Severe nasal obstruction, however, is associated with feeding difficulties, nocturnal enuresis, obstructive sleep apnoea (OSA), emotional problems, hyperactivity and impaired social functioning.^{3,4,5} Upper-airway obstructive events causing apnoeas occur mainly during rapid eye movement (REM) sleep and several hundred of these events might occur each night. These obstructive events are terminated by subcortical arousals which greatly disturb the child's sleep pattern. Despite being relatively sleep deprived, these children often display hyperactivity and irritability during the day.⁶

ANATOMY AND PHYSIOLOGY

The human airway – including the nose – is an extremely sensitive environment prone to a robust inflammatory response to a variety of stimuli. In children, airborne allergens and viral infections are the main offenders. The nose and paranasal sinuses are lined with respiratory epithelium. Characteristic of this epithelium is a vast number of goblet cells and ciliated epithelial cells whose cilia transport the mucus blanket towards the pharynx where it is swallowed. Seromucinous glands are found in the submucosa of the nasal epithelium and, together with the goblet cells, these glands produce most of the nasal

mucus. In addition to the mucus is fluid from the lacrimal glands, fluid leaked or transported across epithelial layers, and condensed water.⁷ Up-regulation of mucous glands through inflammation and infection as well as ciliary dysfunction, whether temporary or long term, results in mucus build-up and obstruction within the nasal cavity.

Unique to the nasal cavity is venous erectile tissue which underlies the mucosa of the inferior and middle turbinates and nasal septum. Engorgement of the capacitance vessels underlying the mucosa in these areas results in swelling of the mucosal lining and a marked increase in nasal resistance. The control of nasal resistance is mediated mainly by the sympathetic nervous system which determines the state of engorgement of the nasal mucosa. Increased sympathetic tone, for example during exercise, will result in a reduction of nasal resistance.

The nose accounts for 50 per cent of the total resistance of the upper airways.^{7,8} The nasal valve region which occurs at the level of the anterior aspect of the inferior turbinate contributes approximately two-thirds of total nasal airway resistance.⁷ Oedema and swelling of the mucosa overlying the anterior aspect of the inferior turbinate and corresponding nasal septum has a profound effect on nasal airflow.⁷ A complex interrelationship therefore exists between anatomical factors, engorgement of nasal mucosa and the amount and viscosity of secretions which determine the degree of obstruction within the nasal cavity itself.

The nasopharynx (post-nasal space) is the space posterior to the nasal cavities. It begins at the choanae (posterior openings of the nasal cavities). The floor is the superior surface of the soft palate. The roof and posterior wall are formed by mucoperiosteum overlying the basisphenoid and basiocciput. The adenoid arises from this mucosa. Laterally

is mucosa overlying the eustachian tube cushions and, posterior to this, lining the lateral pharyngeal recesses.

The adenoid can also cause nasal obstruction, although it is not situated within the nasal cavity. It arises from the posterior pharyngeal wall within the nasopharynx. Along with the palatine and lingual tonsils, it forms part of Waldeyer's ring of lymphoid tissue and is one of the first sites of immunological contact in early childhood. The function of lymphoid tissue in Waldeyer's ring is to produce antibodies. In this way, the adenoid plays a 'gatekeeping' function for the upper airway. The adenoid can be identified in all children by five months of age. It grows rapidly during infancy and reaches its maximum size at around six years of age.⁸ Enlarging in an anteroinferior direction it encroaches on the choanae and the soft palate and causes nasal obstruction. The degree of obstruction does not depend directly on the size of the adenoid but rather on its size relative to the volume of the nasopharynx. Indeed, adenoid-induced nasal obstruction is more common in younger children due to the relatively small volume within the nasopharynx. In addition to physical obstruction of the airway, the presence of hypertrophic adenoid tissue can also impinge on the eustachian tube orifices in the nasopharynx. The adenoid can also serve as a reservoir of infection contributing to both chronic rhinitis as well as to eustachian tube dysfunction and resultant otitis media with effusion (OME). Moreover, severe adenoidal hypertrophy contributes to rhinorrhoea by sustaining both intranasal inflammation and impeding the passage of nasal secretions into the pharynx where they are swallowed.

Other nasopharyngeal masses that can cause obstruction of the nasal airways include branchial cysts, dermoid cysts, meningoencephaloceles, gliomas and Rathke's pouch cysts. Congenital narrowing of the nasopharynx is more common; this is usually associated with Down's, Treacher Collins, Apert and Crouzon syndromes. These syndromes are all associated with varying degrees of narrowing of the nasopharynx, usually as a result of mid-face hypoplasia.

THE NEONATE

As the neonate is an obligate nasal breather, severe nasal obstruction can result in significant respiratory distress. Fortunately, this is rare with bilateral choanal atresia being the most common cause. Unilateral choanal atresia presents in older children with unilateral nasal obstruction and rhinorrhoea since birth. Choanal atresia can be diagnosed clinically by failure to pass a suction catheter or feeding tube through the nose. The obstruction is relieved with an oral airway until the definitive diagnosis is made, usually by CT scan. CT scanning will also identify other causes of nasal obstruction in neonates, including septal deviation, piriform aperture stenosis, nasal encephaloceles and teratomas.

Babies can suffer from idiopathic rhinitis which is

characterised in the afebrile child with mucoid rhinorrhoea and nasal mucosal oedema that results in stertor and feeding difficulties.⁹ Treatment should begin with saline drops and aspiration.¹⁰ A short course of intranasal steroid drops usually brings relief within a week.⁹ Systemic absorption of steroids poses a risk to the child, however, the risk can be minimised by use of topical steroids with minimal absorption (fluticasone and mometasone) and by using short courses.

OLDER CHILDREN

The development of nasal obstruction in previously unobstructed children commonly occurs with exposure to aeroallergens and viral infections. The effects of viral infections have a major impact on the upper respiratory tract from the age of 12 months.⁴ At about two years of age, as the immune system matures, allergic processes start to predominate in the child. Skin-prick tests are more reliably positive and can confirm the diagnosis of allergic rhinitis (AR). Long-term allergen exposure can lead to a state of chronic inflammation within the nasal cavity. This is responsible for the symptoms of nasal congestion, mucus hypersecretion and nasal hyper-responsiveness to allergens, viruses and other irritants.⁴ In parallel with the period of the *atopic march*, and with exposure to viruses and allergens, the lymphoid tissue of the adenoids and tonsils may become hypertrophic and cause obstructive symptoms. Several studies confirm a link between AR and adenoidal hypertrophy by virtue of allergic inflammation in the nasal mucosa which is in close proximity to the adenoids.^{8,11} The development of severe nasal obstruction in a previously healthy child raises the suspicion of a malignancy and should be appropriately investigated.

HISTORY

Mouth breathing and stertor are both hallmark symptoms of the obstructed nose. Neonates are more likely to present with a history of 'snuffles'. Mouth breathing and cyclical grunting or cyanosis are features that suggest severe obstruction in neonates and warrant detailed investigation. Snoring, mouth breathing and recurrent apnoeas are more likely to arise in toddlers and older children.

It is also important to enquire about cough, wheezing and hearing loss as these commonly coexist in the nasally blocked child, particularly where AR features.⁴ With the advent of high-quality video and audio recorders on mobile phones, video and/or audio recordings of the sleeping child made by the parent or caregiver can provide clear evidence as to the nature and degree of snoring and presence or absence of apnoeas. These are particularly useful at follow-up and may even be emailed to the physician as an aid to treatment efficacy.

EXAMINATION

Examination of the child starts with observation. Mouth breathing, nasal discharge and the facial signs of allergy



Figure 1: Modified stethoscope to allow auscultation of individual nasal airways

(Dennie-Morgan lines, horizontal nasal crease, periorbital hyper-pigmentation) are often evident while the history is being taken. At this stage behavioural observations are also made and hyperactivity and inattention can be evident. The presence of non-resolving OME, particularly in older children without prior OME, may be caused by a nasopharyngeal mass which should be excluded.

In a nasally obstructed child, simple anterior rhinoscopy by elevation of the tip of the child's nose is a remarkably useful exercise and it is often possible to see the nature the obstruction using this manoeuvre. In a healthy child, the airway between these structures should be visible as a dark vertical slit. Effacement of the nasal valve region due to septal deviation, septal haematoma, inferior turbinate hypertrophy, foreign body or secretions is usually evident on simple anterior rhinoscopy. A good headlight is an invaluable tool and enhances the clinician's quality of view greatly. The degree of rhinorrhoea is noted as well as the nature of the secretions. If a suction aspirator is available, secretions may be cleared with a fine suction tip to improve visualisation of the anterior 2–3 cm of the nasal cavity. Particular attention is paid to the nasal valve area, where inferior turbinate hypertrophy or nasal septal deviation exerts the greatest effect on the airway. A visibly patent nasal valve area in a child with persistent nasal obstruction suggests that the obstruction lies posteriorly and is usually as a result of adenoidal hypertrophy. Other nasal tumours which are not visible with anterior rhinoscopy include

juvenile nasopharyngeal angiofibromas, lymphomas and sarcomas.

A metallic tongue depressor placed under the child's nose in exhalation has traditionally been used as an office-based examination to quantify the degree of nasal airflow by noting the degree of misting produced on the depressor. A more effective and informative tool is the use of a modified stethoscope where the bell has been removed (see Figure 1). The open end of the stethoscope tubing is held half a centimetre away from the child's individual nostrils. The degree and quality of airflow is clearly heard and the variation between the two nostrils can be appreciated. Transmitted breath sounds from the upper airways can also be heard. Complete cessation of airflow through one or both nostrils is rare. In neonates, this usually suggests choanal atresia. In older children, it is usually due to nasal polyposis. These children should be routinely screened for cystic fibrosis.¹²

Fibreoptic nasendoscopy is routinely used as an office investigation in ENT consultations, particularly in adults. Its usefulness in the paediatric population should not go unrecognised. With a gentle technique it can be performed on children of any age depending on the cooperation of the child and the dimensions of the child's nasal cavity. Topical decongestion and anaesthesia are obtained by nasal spray or the placement of a soaked cotton wool pledget between the anterior aspect of the middle turbinate and nasal septum. A combination of 5% lignocaine HCl and 0.5% phenylephrine HCl is typically used. In the cooperative child, the nasal cavity and nasopharynx may be assessed. The endoscopist is able to 'tour' the nasal cavity and visualise the presence of polyps, foreign bodies and also the presence or absence of mucopus draining from the anterior or posterior sinus drainage pathways. In the nasopharynx the adenoidal pad is clearly visualised and the degree of obstruction assessed.¹³ This obviates the need for a post-nasal space X-ray in these patients.

INVESTIGATIONS

Skin-prick testing remains the most important investigation for the child with the obstructed nose in whom allergy is suspected. If adenoidal hypertrophy is suspected then a post-nasal space X-ray is indicated (see Figure 2). Both these tests are reliable and cost-effective and are therefore helpful in guiding treatment.^{4,13,14} CT scanning is seldom required in children. Usual indications for this are the evaluation of masses within the nasal cavities, nasal polyposis and recalcitrant infective rhinosinusitis where endoscopic sinus surgery is being considered. Endoscopic sinus surgery is, however, rarely indicated in children.

MEDICAL TREATMENT

Nasal saline irrigation is surprisingly well tolerated in the majority of children, irrespective of age.^{10,15,16,17} Nasal

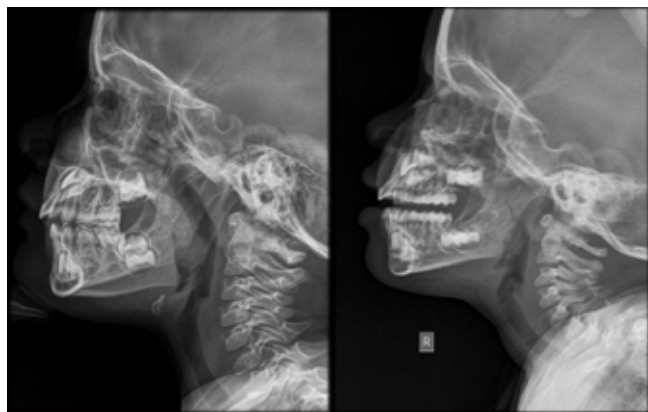


Figure 2: Comparative X-rays of the post-nasal space demonstrating obstruction of the nasopharyngeal airway due to adenoidal hypertrophy in the image on the right

saline lavage may be sufficient on its own in neonatal rhinitis and viral upper respiratory tract infections.⁹ Nasal saline irrigation has also been shown to significantly reduce nasal symptoms in children with AR or acute sinusitis.¹⁵ Marked rhinorrhoea may result in the dilution of the active ingredient of intranasal steroid (INS) sprays and failure of the drug to be deployed at a mucosal level. Therefore, saline nasal irrigation may be a useful adjunct in nasal obstruction with associated rhinorrhoea. It is beneficial in patients with AR where it maintains the effectiveness of INS sprays at lower dosages.^{15,17}

Children with nasal obstruction and rhinorrhoea due to AR – with or without adenoidal hypertrophy – should be placed on an INS spray.⁸ The benefits of INS sprays are well documented in patients with AR with or without asthma.¹⁸ INS sprays have also been shown to be effective in reducing adenoidal hypertrophy.^{19,20,21,22} In addition, INS sprays have been effective in reducing the severity of OSA.^{20,23,24} Lastly, in patients following adenoidectomy, INS sprays have been shown to reduce adenoidal regrowth and nasal obstruction scores at least until one year post-operatively.²⁵ The safety of long-term INS spray use is a concern, especially in patients with asthma who also receive treatment with inhalational steroids (IHS). Studies have confirmed that with INS and IHS there is a small, transient reduction in growth velocity in that resolves with continued time on treatment.²⁶ Moreover, this effect does not occur in all children and, in the case of asthma, growth can be suppressed by poor control of the disease itself. The aim of treatment with INS is to start with a high dose in order to control the inflammation and then to reduce to the minimum effective dose to maintain control. At the same time steroid-sparing strategies should be employed where possible such as allergen avoidance and use of add-on therapies such as antihistamines and leukotriene inhibitors.²⁶

SURGICAL TREATMENT

In patients in whom INS sprays fail to reduce obstructive adenoidal tissue, adenoidectomy appears to be a safe and reliable procedure to undertake. In addition to the

improvement of the nasal airway, adenoidectomy is shown to have positive effects on the resolution of OME and an improvement in polysomnographic scores in patients treated for OSA.²⁷ In patients with OSA, current recommendations suggest additional tonsillectomy irrespective of tonsil size.⁶ Besides relief of nasal obstruction caused by adenoidal hypertrophy, adenotonsillectomy also improves several asthma outcomes.^{28,29}

Gross inferior turbinate hypertrophy prevents the delivery of INS sprays to much of the nasal mucosa posterior to the nasal valve area. In these refractory cases where medical treatment has failed, inferior turbinoplasty is extremely useful. The aim is to reduce and effectively normalise the size of the turbinate without impairing the warming and humidifying function of the nose. Surgery is focused on the anterior third of the inferior turbinate in the nasal valve area. This allows improved nasal airflow without compromising the physiological function of the nose. A variety of techniques are employed in an effort to reduce the bulk of the inferior turbinate. Patients experience improvement in nasal patency irrespective of the technique used, however, patients with AR experience the greatest improvement of nasal patency.³⁰ Medial-flap turbinoplasty, a mucosa-preserving technique, where both turbinate bone and mucosa are judiciously excised from the critical nasal valve area, appears to be the most effective method in order to ensure long-term relief of symptoms.^{31,32} Turbinoplasty is, however, not commonly indicated in the paediatric population.

Septal deviation seldom causes severe nasal obstruction as there is usually sparing of a functional nasal airway on one side. Symptoms tend to be worse if there is coexistent AR. Corrective septal surgery is controversial in children due to concerns that disruption of nasal growth centres can lead to changes in midfacial growth and final midfacial contour. Although surgery is deemed safe, most authors suggest deferring surgery until at least the early teenage years in cases where symptoms are not severe.³³

CONCLUSION

Nasal obstruction in the child is often multifactorial with links between AR and adenoidal hypertrophy. It is essential to attempt to extinguish local inflammation within the nasal cavity with anti-allergic medication in order to maximise the nasal airway and reduce rhinorrhoea. INS sprays have the added benefit of a reduction in adenoid size and reducing regrowth of adenoidal tissue following adenoidectomy. If medical treatment fails in patients with proven adenoidal hypertrophy, surgery to reduce bulky adenotonsillar tissue will improve upper-airway patency. In cases of refractory inferior turbinate hypertrophy, inferior turbinoplasty will improve both the nasal airway and the delivery of intranasal steroids to the greater nasal cavity.

DECLARATION OF CONFLICT OF INTEREST

The author declares no conflict of interests.

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