Nasal obstruction

Patients commonly present with a blocked or 'stuffy' nose. There is a long list of differential diagnoses to consider but the symptom must be fully investigated because it may indicate the presence of a significant underlying problem.

LARRY H. KALISH
FRACS, MS, MMED (Clin Epi)
MELVILLE DA CRUZ
FRACS, MS, MD

Dr Kalish is a Rhinologist and Skull Base Surgeon at Concord Hospital, a VMO at Canterbury Hospital and practises at the Sydney Sinus and Allergy Centre, Edgecliff, NSW. Associate Professor da Cruz is Head of the Department of Otolaryngology, Westmead Hospital, Westmead, NSW.

A blocked or 'stuffy' nose is one of the most common patient complaints and it leads to an extensive list of differential diagnoses. It can be self-limited or constant, a stand-alone concern or one of an array of symptoms. Patient perception and acceptance of nasal blockage varies greatly with discordance between presenting complaints and objective findings being common. Nasal obstruction may be the symptom a patient focuses most on, ignoring more telling symptoms. It can also be a symptom that is quickly dismissed by the GP when in fact it heralds a significant underlying problem (Figure 1).

This article presents an organised approach to the blocked nose, highlighting the relevant anatomy, the differential diagnoses and some management paradigms.

Anatomy of the nose

The nose is not just a conduit for airflow but is also a vital component in the filtration and humidification of air, in chemosensation and as part of the nasal reflex (Table 1).

The nasal airway is the entrance to the respiratory tract. Although respiration can occur through the mouth when respiratory demands are high, respiration occurs preferentially through the nose in order to improve humidification and filtration, and to smooth the pattern of respiration by nasal resistance. Resistance of the nasal airway accounts for more than 50% of total airway resistance.

When considering the anatomy of the nose it is best to divide it into sections.

Nasal vestibule

The entrance of the nose is the nasal vestibule, which is the skin-lined part of the nostrils often overlooked during a hasty examination. The internal and external nasal valves define the limits of the nasal vestibule.

The external nasal valve is enclosed:

- inferiorly by the nasal sill overlying the pyriform aperture
- laterally by the lower edge of the lower lateral cartilage
- medially by the medial crura of the lower lateral cartilage making up the columella, which covers the caudal end of the septum.

The internal nasal valve is the narrowest portion of the nasal cavity and, therefore, the compromise of any of the valve's components creates symptoms of nasal obstruction. The internal nasal valve is defined:

IN SUMMARY

- **The nose is a conduit for airflow and a vital component in the filtration and humidification of air, in chemosensation and as part of the nasal reflex.**
- **There is a broad range of differential diagnoses for nasal obstruction.**
- **A diagnosis is usually made after taking a full patient history and conducting a physical examination.**
- **A stepwise approach is required for the management of nasal obstruction.**
- **Management starts with simple measures, progressing to increased medical management and then surgical intervention, if required.**

Table 1. Functions of the nose[1]

Function of the nasal airway	Mechanisms
Airflow	Conduction of environmental air into and out of the respiratory system
Filtration	Mucous, vibrissae and cilia trap and remove airborne viral, bacterial and particulate matter (usually >30 µm)
Heating	Vascular mucosa overlaying the nasal cartilage and turbinates provides radiant heating of inspired air from 31 to 37°C
Humidification	Vascular mucosa increases the relative humidity of inspired air to 95% before reaching the nasopharynx
Chemosensation	Olfactory sensation detects irritants, chemicals and temperature abnormalities of inspired air
Nasal reflex	Nasal sensation may be linked to lower respiratory and vascular reflexes
Endocrine, pheromone detection	Olfactory identification of major proteins may be used in mate selection for production of offspring with more heterogeneous types of human leucocyte antigens

- laterally by the lower edge of the upper lateral cartilage and the head of the inferior turbinate
- medially by the caudal end of the nasal septum.

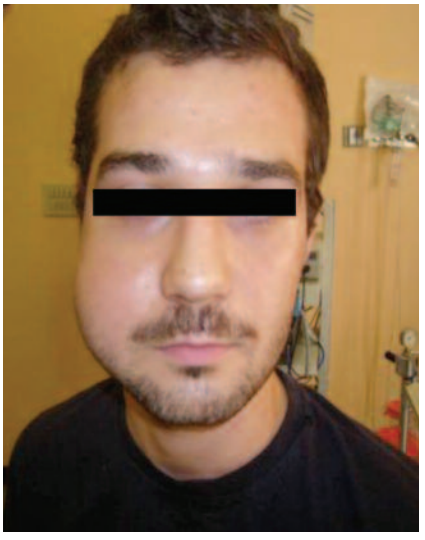


Figure 1. A 16-year-old boy who re-presented with the principle complaint of nasal obstruction and epistaxis and had an associated cheek swelling. He was referred for dental assessment and underwent a biopsy of the cheek mass, which bleed extensively, leading to a referral to a ear, nose and throat specialist. At that time he was diagnosed with a juvenile nasopharyngeal angiofibroma.

The erectile tissues of the nasal septum and inferior turbinate allows them to impinge significantly on the area of this valve. Similarly, collapse of the upper lateral cartilage, if it is anatomically weak or has been surgically detached, can also interfere with airflow at this critical junction.

Nasal vault

The nasal vault extends from the internal nasal valve to the posterior choanae. It is lined by mucosa with a surface area of about 150 cm^2. The functions of the mucosa are to:

- provide heat and fluid exchange for warming and humidifying inspired air
- alter nasal airway resistance through congestion and decongestion of the nasal mucosa blood vessels
- clean and filter inspired air by means of impaction on the moist mucous-coated surface
- sense the environment with specialised (olfactory) and general (trigeminal) sensory nerves.

The medial wall of the nose consists of the septum, which is invariably deviated to some extent. Fullness or deviation in the upper septum does not impact nasal airflow to the same degree that spurs and deviations limit flow along the floor of the nasal passage. The lateral nasal wall has prominent in-foldings that make up the inferior, middle and superior turbinates (or conchae). These are thin bony shelves surrounded by spongy tissue that is rich in capacitance vessels. The vascular spaces expand and contract in response to autonomic innervation. Sympathetic innervation vasoconstricts the feeding arterioles, decongesting the turbinates and opening the nasal passages. Parasympathetic fibres enlarge the venous sinusoids increasing the surface area, stimulating nasal secretions and increasing the nasal resistance.

The inferior turbinate dominates the lateral wall and fills most of the lower air passage, coming close to the septum but with no mucosal contact. Change to this turbinate has the greatest impact on nasal airflow. The middle turbinate is usually evident in the mid-portion of the nasal passage medial to the inferior turbinate. It can be mistaken for a polyp or mass, especially if there are polypoid mucosal changes. It plays an important role in the sensation of airflow and may often be the epicentre of pathological processes.

Nasopharynx

The nasopharynx should always be considered when examining the nasal airway. Obstruction of the nasopharynx, such

as in choanal atresia or from enlarged adenoids or a nasopharyngeal carcinoma, often manifests with nasal blockage early on.

Nasal airflow

Both the nasal resistance of the nose and the sensation of air flowing over the mucosa determine nasal airflow. The turbinates constantly enlarge and contract in response to environmental and certain physiological changes, such as stress or hormonal stimuli. A cyclical variation in nasal congestion, called the nasal cycle, is present in most individuals. Turbinate swelling alternates from side to side over one to four hours.[2] Most people are unaware of this; however, if the threshold of nasal resistance is reached, patients may report nasal obstruction, varying from side to side.

There is often a lack of correlation between the subjective impression of airflow and the actual measured airflow. As nasal resistance is increased the sensation of poor airflow is also increased until a critical point is reached when the patient feels completely obstructed; however, intranasal receptors can be confounded by various chemical or physical means. Inhalation of volatile oils or cold air gives a sensation of improved airflow, even if nasal resistance is unchanged, and similarly anaesthesia to the nose can create a false impression of obstruction without measured change in resistance.[3] This is most evident in a patient who has had extensive surgery to remove all midline structures of the nose, such as tumour removal, and radiotherapy. The patient may appear to have a large nasal passage and demonstrable airflow, but without the ability to sense the airflow, the patient will still report complete nasal obstruction.

Differential diagnoses

There is a broad range of differential diagnoses for nasal obstruction (Table 2) that needs to be considered during any evaluation. Diagnosis is often determined by taking a full patient history and conducting a physical examination.

History

While taking a patient's history, it is best to try and characterise the nasal obstruction and then determine its duration, the side of the nose affected and any contributing factors. The character of the obstruction may include a blocked or stuffy sensation but may also present with non-nasal manifestations such as breathing through the mouth, sore dry throat, facial fullness, loss of taste or reduced sense of smell. The obstruction can be acute, recurrent or chronic. Acute congestion usually has a sudden onset, is self-limited and of short duration. Recurrent congestion includes episodes that persist beyond viral symptoms but have intermittent periods of normal airflow. Chronic nasal congestion means that patients never experience relief from their symptoms.

The season of onset is also important. Contributing factors are key in determining the cause of nasal obstruction. Allergic symptoms include sneezing, a scratchy throat requiring clearing, nasal pruritis and associated eye symptoms including itchy, watering red eyes. Symptoms caused by infectious disease include purulent rhinorrhoea, facial pain, fever, referred toothache and a general foul smell or halitosis. Nonallergic symptoms are dominated by clear rhinorrhoea and congestion. Inflammatory and neoplastic causes should be considered in any chronic cases of nasal obstruction with associated epistaxis or blood in nasal secretions, nasal pain or pain relating to the orbits. A patient history of smoking, drug use (especially cocaine) and nasal trauma, including iatrogenic causes and that from certain occupations (for example, wood workers and smelter workers have a higher incidence of nasal cancers), is important when considering inflammatory and neoplastic causes.

Table 2. Causes of nasal obstruction[4]

Primary causes

Physiological
- Nasal cycle

Nonphysiological

Allergic:
- Seasonal
- Perennial

Nonallergic:

Infective
- Acute or chronic
  - Viral, bacterial, fungal, parasitic, protozoal

Noninfective
- Mechanical
  - Septal deformity
  - Hypertrophic turbinates
  - Foreign body
  - Choanal atresia
- Hyper-reactive
  - Hormonal
  - Drug induced
  - Chemical irritants
  - Emotional
- Inflammatory
  - Polyps
  - Sarcoid
  - Wegener's granulomatosis
- Tumours
  - Benign
  - Malignant
- Disturbance of airflow
  - Overpatency
  - Septal perforation

Secondary causes

Postnasal space
- Adenoid enlargement
- Nasopharyngeal mass

Oropharynx
- Enlarged tonsils, soft palate, tongue base
- Sleep apnoea

Lower respiratory tract
- Asthma
- Chronic obstructive pulmonary disease

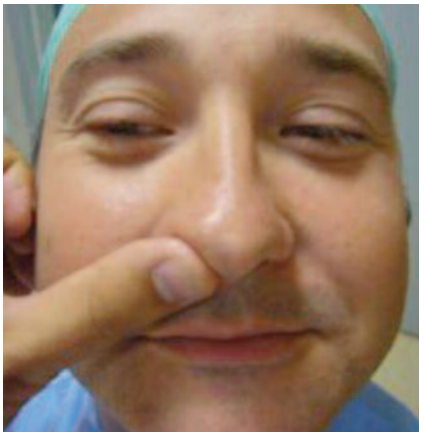


Figure 2. An example of how to obstruct the right nostril during a clinical examination without distorting the left nostril.

Medications can alleviate or contribute to nasal obstruction. Aspirin sensitivity is important to exclude, and the patient's response to nasal or oral corticosteroids can be suggestive of an inflammatory cause. Secondary causes of nasal obstruction should be considered in patients who snore, have had life-long nasal obstruction or have a history of asthma or respiratory illnesses. In any patient with hypersomnolence, lethargy, halitosis or cough, or in the face of parental concern about performance, primary and secondary causes of nasal obstruction should be considered.

Examination

When trying to characterise the site of obstruction, the side of the nose affected should be established. Obstruction can be bilateral, unilateral, mainly occurring in one side or variable, changing from side to side. During relaxed breathing with the mouth held closed, a mirror or a piece of cotton wool should be placed under the patient's nostrils and misting (condensation) of the mirror or movement of the cotton wool, which signifies airflow, looked for.

Many patients with nasal obstruction will in fact have some nasal airflow with deep inspiration but there will be limited or no airflow on light nasal breathing. It is, therefore, important to assess the patient during light nasal breathing and forced inspiration. One nostril should be occluded without depressing the side (this may distort the opposite side and interfere with airflow; Figure 2), and again airflow compared on each side.

The site of obstruction is dependant on the section of the nose where the obstruction is maximal. Considering the sections of the nose discussed above, the site of the obstruction may be in the nasal vestibule, at the internal nasal valve, in the nasal vault or in the nasopharynx.

The procedures to follow and the order in which to conduct a nasal examination are shown in Figures 3a to f and discussed in detail below.

Figures 3a to f. Examining the nose. a (top left). With an appropriate light source, position yourself in front of the patient. b (top centre). Inspect the external nose. c (top right). Look closely at the nasal vestibule. d (bottom left). Perform the Cottle manoeuvre. e (bottom centre). Test airflow. f (bottom right). Perform anterior rhinoscopy.

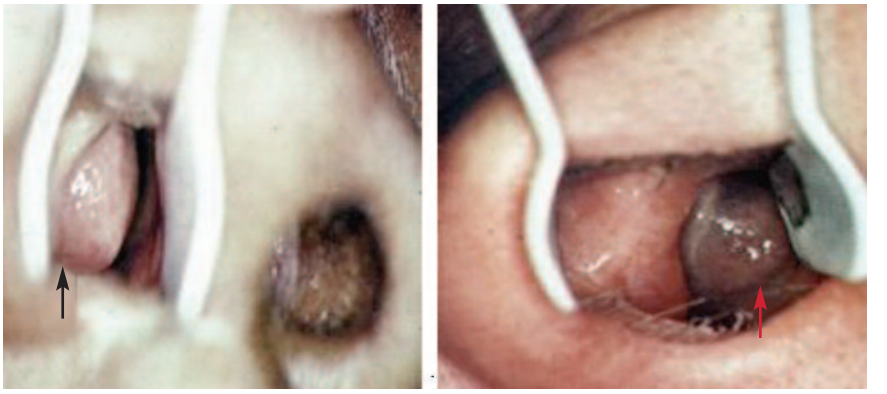
Nasal vestibule

Masses in the nasal vestibule, such as a squamous papilloma, can easily be missed. However, when the mass fills the apex of the vestibule, nasal airflow can be significantly impacted, so it is pertinent to look carefully with adequate light and focus on the apex of the vestibule (Figure 3c).

Internal nasal valve

Although the internal nasal valve is the narrowest part of the nose, it is still physiologically possible for some obstruction to occur at this point. When the internal nasal valve is held open by lifting the nasolabial fold (called the Cottle manoeuvre; Figure 3d) it is normal for a patient to feel improved airflow. If the patient does not feel any improvement it suggests that the obstruction is deep compared with the valve and must be in the nasal vault or nasopharynx. Patients who experience substantial improvement in their airflow from this manoeuvre, and often perform this manoeuvre or similar ones at home, are likely to have an obstruction at the internal nasal valve. This is further demonstrated when the patient can breath easily on light inspiration but any forced inspiration feels obstructed. The observant clinician will be able to see the collapsing internal nasal valve on deep inspiration. Placing a small piece of cotton wool in the apex of the internal nasal valve to hold it open usually improves the patient's airflow, further confirming the diagnosis.

When the Cottle manoeuvre fails to improve airflow, the nasal vault must be carefully examined. This examination begins with a close inspection of the external nose (Figure 3b). Significant deviation of the nose suggests underlying septal deflection and asymmetrical nasal passages. Similarly, significant tip bulbosity, tip ptosis or alar collapse can all have an impact on airflow. Clues to the underlying disorder may be evident – for example, a nasal crease from rubbing the nose is suggestive of an allergy and widening of the nasal bones is suggestive of massive polyposis or there may be evidence of a previous rhinoplasty, which is a common cause of internal nasal valve collapse.



Figures 4a and b. Anterior rhinoscopy with a nasal speculum and headlight. a (left). Decongested inferior turbinate (arrow). b (right). A nasal polyp medial to the inferior turbinate with a dull grey appearance and mucous streak (arrow).

Examination of the internal nose is best performed with a nasal speculum (Figure 3f). This opens the internal nasal valve and allows an unobstructed view of the nasal passage. Ideally the illumination is along the physician's line of vision, best achieved with a head mirror or a head-mounted light (Figure 3a); however, a hand-held otoscope is also suitable, allowing for anterior rhinoscopy and then careful insertion of the tip to assess the posterior nasal vault. It is always best to assess the nose before any decongestants are given to appreciate the mucosal changes, turbinate congestion and presence of discharge.

Decongesting the nose can be achieved with an alpha agonist, such as oxymetazoline (Dimetapp 12 Hour Nasal Spray, Drixine Nasal, Extra-life Nasex Nasal Decongestant, Logicin Rapid Relief Nasal Spray, Vicks Sinex). The nasal airflow should be retested after decongestion. Improvement suggests that the obstruction is largely mucosal in origin. No visible change to the turbinates after decongestion may indicate alpha agonist abuse (rhinitis medicamentosa). Anaesthetic agents such as lignocaine are frequently added to a decongestant to facilitate the insertion of fibreoptic scopes; however, this can confound the sensation of airflow as mentioned above and incorrectly suggest that the obstruction is not relieved by mucosal decongestion.

Nasal vault

Evaluating the posterior nasal vault can be challenging, and the focus of the examination should be on the septum and any deviation. It should be remembered that septal intrusion on the lower airway is more likely to impact airflow, except at the caudal end of the septum where deviation higher up the airway may narrow the internal nasal valve and significantly impact airflow. The inferior turbinate should then be assessed, noting how prominent it is, if it is touching the mucosa of the septum and if it completely obstructs the view of the posterior passage. Next, the middle turbinate should be assessed, being careful not to mistake it for a polyp, especially if there are polypoid changes (Figures 4a and b). A large middle turbinate may represent a concha bullosa or air-filled turbinate and will impact airflow. Finally, discharge, foreign bodies, blood or masses should be carefully looked for.

Nasopharynx

Examination of the nose is made easier in a specialist practice with rigid or flexible endoscopes available. Failing a direct look at the nasopharynx, there are many clinical clues to the possibility of an obstruction in this region. These include:

- complete nasal obstruction without a preferential side and normal anterior rhinoscopy
- concomitant middle ear effusions given that the Eustachian tube opens at the posterior end of the nasal passage
- snoring
- persistent mouth breathing as seen in the child with large adenoids
- reports of blood being spat out (often mistaken for haemoptysis) prior to an epistaxis
- positional obstruction, especially when lying flat at night.

If suspicion of an obstruction in the nasopharynx is high then a lateral airways x-ray may be helpful in determining if soft tissue masses are present in the nasopharynx.

Objective measures of nasal patency are available in specialist settings. Quantification of nasal congestion is difficult and data on what is normal are unreliable. Most objective measures are used to demonstrate changes in an individual patient such as nasal patency scores pre- and post-treatment or to demonstrate a reaction to a nasal allergen test. The most simple measure of nasal patency is a nasal peak inspiratory flow (NPIF) meter. This is similar to a peak flow meter but the patient is required to inhale rather than exhale. It is reasonably reliable and can be used in a general practice setting.

Our preference is to allow patients to take the NIPF meter home and record serial measurements to test the effect of environmental exposure or response to medication. Other objective measures include acoustic rhinometry, anterior and posterior rhinomanometry, and nasal nitric oxide levels.

Imaging

The lateral airway x-ray remains the only worthwhile plain roentgenographic study, and is useful when considering the nasopharynx as the site of obstruction. Obtaining x-rays of the sinuses and nasal bones is of minimal use.

A computed tomography (CT) scan is very useful in providing a view of the nasal structure but should not be used in the initial workup of a patient unless a neoplasm is suspected. The most common causes of nasal obstruction include allergic and viral rhinitis. CT scans performed during an acute episode can be misleading because of significant mucosal oedema.

Indications for a CT scan include concern for sinister pathology (including unilateral obstruction, bleeding or sinonasal cancer), cranial nerve abnormalities or persistent obstruction after a reasonable course of treatment.

A CT scan provides excellent detail of the bony structure of the nasal passages and paranasal sinuses and reasonable soft tissue detail, which is usually enough to determine the presence of a nasopharyngeal mass.

A CT scan does not distinguish fluid from tumour in the sinuses, and any patients with an asymmetrical CT scan or scan showing bony erosion, especially along the skull base, warrant further imaging with magnetic resonance imaging (MRI).

An MRI scan provides a sensitive view of the mucosal lining, distinguishes soft tissue masses from inspissated sinus contents and importantly demonstrates the presence of a meningo-encephalocoele (brain/dural herniation).

Laboratory tests

The evaluation of allergy is an area in which laboratory tests for patients with nasal obstruction are of value. Evaluation can either be performed by skin prick testing or measurement of specific IgE in serum samples using the well-known radioallergosorbent test (RAST) or the more widely used immunoCAP specific IgE blood test. The advantages of blood testing include the safety, standardisation and higher degree of precision, and it is unaffected by medications such as steroids, antihistamines and anticholinergics, which can confound skin prick testing. The disadvantages include higher cost, delayed results and weaker correlation with immunotherapy outcomes. There is little benefit to be gained from testing total IgE or blood eosinophil counts in patients with nasal obstruction.

Another practical laboratory test is to culture purulent discharge from a patient with suspected infectious rhinitis. Care must be taken to obtain the swab from the middle meatus (lateral to the middle turbinate) as contamination from the nasal vestibule can confound results. Nasal smears, looking at eosinophils, neutrophils, fungal cultures and allergen nasal challenges, are best performed in specialist centres.

Management

Strategies for the management of nasal obstruction are based primarily on the patient's history and physical examination findings. Laboratory tests, biopsies and imaging are usually used to confirm the diagnosis and help plan the course of treatment, especially if a surgical approach is required. Alternative diagnoses should be kept as contingencies in the event of treatment failure.[1]

It is important to consider the duration of the nasal obstruction. A stepwise approach is also important, starting with simple measures or observations and progressing to increased medical management, more directed interventions such as immunotherapy and then surgical interventions.

Acute congestion

Acute congestion is characterised by a rapid onset, being self-limited and most commonly being associated with infectious rhinitis. Most patients seek medical help for relief of the nasal obstruction. The key assessment is to exclude purulent discharge and assess the degree of nasal inflammation. More aggressive management is required in patients susceptible to sinus infections.

Humidification and nasal irrigation with hypertonic buffered saline are easy and beneficial adjunctive treatments that are generally underused. A variety of over-the-counter products are available; however, for symptomatic relief the use of hypertonic solutions in a powerful delivery vehicle, such as a squeeze bottle or neti pot, result in more substantial mechanical washing and better distribution.

Topical decongestants are very useful; however, in patients who have recurrent or chronic congestion there is a tendency towards abuse and compounding of the nasal obstruction by an iatrogenic-induced rhinitis medicamentosa. Our approach is to educate patients and restrict the use of topical decongestants to seven days. If nasal congestion persists after five days, a topical corticosteroid spray (e.g. fluticasone [Avamys, Beconase Allergy & Hay fever 24 Hour], beclomethasone [Beconase Allergy & Hayfever 12 Hour], mometasone furoate [Nasonex Aqueous Nasal Spray, Nasonex Aqueous Nasal Spray Alcohol Free], budesonide [Rhinocort Hayfever]) can be introduced. After seven days of use, the topical decongestant can

be changed to an oral decongestant with continuing use of the corticosteroid spray; however, care must be taken in patients with hypertension, arrhythmias, prostatic hypertrophy and anxiety.

Better results are obtained from any topical nasal medication when delivery is performed with the patient in a dependent position such as the Mygind position (lying supine on a bed with the head hanging off the end and eyes looking at the floor). Immediately after the drops are applied the patient is instructed not to sniff. Sniffing is a natural response to the insertion of topical medication; however, it draws the drops into the nasopharynx rather than leaving them to be adequately exposed to the mucosa and allowing the mucociliary flow to distribute the medication widely across key areas.

With any presentation of acute congestion, septal haematomas, septal abscesses and foreign bodies should be considered. Although rare, if these conditions are missed they can result in significant and unnecessary morbidity. A history of trauma almost always precedes a septal haematoma and a septal abscess is most commonly the result of a septal haematoma left uncontrolled. Both result in a devastating loss of septal cartilage causing a saddle-nose deformity. A septal abscess can progress to an intracranial thrombosis in the cavernous sinus.

Both septal haematomas and septal abscesses almost always present with bilateral nasal obstruction. Septal haematomas are usually preceded by epistaxis. On examination, there is a cherry red swelling in the midline obstructing both nostrils. By manipulating the swelling with a cotton bud, a fluctuant mass is revealed. Occasionally after trauma to the nose there can be bruising of the septum and in a patient with a deviated septum this can mimic a unilateral haematoma. However, unilateral haematomas are rare and simple manipulation of the mass will reveal firm cartilage and not the characteristic soft fluctuant feel of a septal haematoma.

Both septal haematomas and septal abscesses require urgent incision and drainage. An abscess is best managed with intravenous antibiotics and close observation.

Until proven otherwise, the presence of a foreign body should be considered with any acute onset of unilateral nasal obstruction with purulent discharge. Foreign bodies are especially prevalent in children and developmentally delayed patients. They can be bilateral and more than one may be present, so close examination with adequate light should be performed after decongesting the nose. A purulent discharge will obscure the foreign body and needs to be suctioned before examination. The foreign bodies that cause most concern are the small metallic batteries found in many children's toys. These result in significant collateral damage, especially to the septum, and patients with these foreign bodies should be referred urgently to a specialist. If there is a history of battery insertion, parental concern, rust-coloured discharge or suspicion on anterior rhinoscopy, prompt referral is recommended.

Recurrent congestion

Congestion recurring beyond the duration of viral symptoms (more than five days) merits physician evaluation. A diagnosis of acute bacterial sinusitis can be made if the congestion lasts more than 10 days and is associated with one or more of the following:

- facial congestion
- pain
- purulent anterior or posterior discharge
- hyposmia
- fever.

or at least two of the following:

- headache
- cough
- lethargy
- halitosis
- ear pressure/pain
- dental pain.

This is a clinical diagnosis and a CT scan should not be ordered unless a complication is present or an appropriate course of treatment has been implemented. Treatment involves a 10-day course of an antibiotic such as amoxycillin, cefuroxime (Zinnat) or clarithromycin (Clarac, Clarihexal, Clarithro, Kalixocin, Klacid) with at least twice daily nasal saline irrigation, followed by the application of an appropriate topical corticosteroid. Both the saline and corticosteroid should be continued for a minimum of two weeks, but ideally for a full course of six weeks to minimise rebound symptoms. There is no clear role for antihistamines, anticholinergics or oral decongestants in the treatment of recurrent congestion and the use of topical decongestants risks aggravating problems as mentioned before.

Allergic rhinitis

Allergy remains the most common cause of recurrent nasal obstruction, even in the absence of a clear allergy history. Allergic rhinitis is classified as either intermittent or persistent and either moderate or severe according to the ARIA (Allergic Rhinitis and its Impact on Asthma) guidelines. In addition to this, it is generally accepted that these patients broadly fall into a further two groups – 'sneezers and runners' or 'blockers' (Table 3).[5] The blockers group present with recurrent nasal congestion and no clear history of allergy. This group can easily be screened using either skin prick testing or immunoCAP serum testing for common inhalant allergens. It is important to note that 'other' allergies, especially food allergies, do not present with isolated nasal symptoms and a thorough history will always reveal other systemic problems.

Management of the patient with an allergy starts with identification of the allergen and other triggers. Allergen avoidance is often difficult but warrants a trial. Regular saline irrigation especially during peak allergy season, helps reduce the allergen load in the nose and is a

useful adjunctive treatment. As mentioned before, hypertonic solutions do not decongest the nose but there is evidence that improved sensation of nasal breathing may be the result of increased mucous clearance and sensory feedback.

Topical corticosteroids

The early introduction of topical corticosteroids is especially important in nasal 'blockers' and patient education is vital because patients need to be informed that steroids are a long-term treatment. They do not give acute relief and need to be continued for at least two weeks to see maximal effect. Topical steroids need to be appropriately applied, and evidence is emerging that steroids in droplet form are probably more effective than those in metered dose bottles.

Not all corticosteroids are the same and some patients will respond better to one type than another. Mometasone (Elocon, Nasonex Aqueous Nasal Spray, Nasonex Aqueous Nasal Spray Alcohol Free, Novasone) and fluticasone (Avamys, Beconase Allergy & Hayfever 24 Hour, Flixonase Nasule Drops, Flixotide) are the only two corticosteroids with negligible systemic absorption. Other steroids such as budesonide (Budamax, Entocort, Pulmicort, Rhinocort), beclomethasone (Beconase Allergy & Hayfever 12 Hour, Qvar), triamcinolone (Aristocort, Telnase, Tricortone) and even dexamethasone (Dexmethsone) may be equally efficacious or better but increased amounts of systemic absorption warrant caution when prescribing them to children.

Antihistamines

Oral antihistamines are effective treatments in allergic patients but have minimal impact on nasal obstruction. Topical antihistamines are very useful for rapid symptom reduction during acute episodes and have a role in preventing attacks. They have some effect on nasal obstruction and a trial in allergic patients with nasal obstruction is worthwhile. Two topical antihistamines are currently available over the counter, levocabastine (Livostin Nasal Spray) and azelastine (Azep Nasal Spray). Azelastine can leave a bitter taste in about one-third of patients and therefore has a lower compliance.

Table 3. Distinguishing 'sneezers and runners' from 'blockers' with allergic rhinitis[5]

Symptom	Sneezers and runners	Blockers
Sneezing	Especially paroxysmal in bouts	Little or none
Rhinorrhoea	Always present, watery, anterior and sometimes posterior	Variable, can be thick mucous and generally more posterior
Nasal itching	Yes, often	No
Nasal blockage	Variable	Often severe
Diurnal rhythm	Worse on awakening, improves during the day and usually worsens again in the evening	Constant day and night, may be worse at night and is often severe
Conjunctivitis	Often present	Not present

The goal of managing the 'blockers' group is to:

- establish the diagnosis
- isolate the allergen(s) and attempt allergen avoidance
- start topical steroids, regular saline irrigation and topical antihistamines early on with appropriate counselling
- avoid topical decongestants and use oral decongestants sparingly
- refer to the appropriate specialist if this regimen fails.

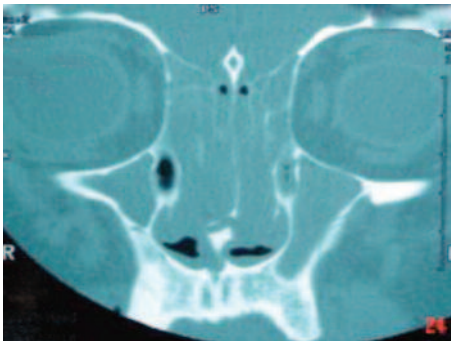
Surgical management

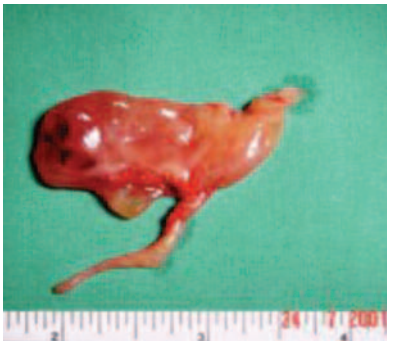
One of the causes of medical management failing and a common cause of recurrent nasal obstruction is the presence of an anatomical defect. It is common for a deviated septum or hypertrophic turbinate to incompletely obstruct airflow and remain undetectable; however, in the presence of concurrent pathology, such as allergic or nonallergic rhinitis, it can result in significant obstruction. The most common defects causing obstruction include septal deviation, turbinate hypertrophy, septal perforation and nasal valve collapse. An anatomical defect should always be considered in recalcitrant cases of obstruction. If that is suspected, referral of the patient to an ear, nose and throat specialist for further assessment and possible surgical repair is warranted.

There are numerous surgical approaches to these anatomical defects. Care should be taken because repair of one defect can result in other problems, such as a narrowed internal nasal valve after a septal reconstruction. There are various ways of addressing turbinate hypertrophy including outfracture, cautery, radiofrequency, laser, submucosal resection, turbinoplasty and turbinectomy.

An approach that is too aggressive can result in reduced nasal sensation and a perceived nasal obstruction. Scarring from turbinate surgery can also result in nasal obstruction and the result of many of the techniques used are only temporary, with turbinate obstruction commonly recurring three to five years later. It is also important to counsel patients that anatomical defects are often only part of the problem and addressing them will not have an effect on the underlying mucosal pathology.

Other causes of recurrent obstruction include erosion and crusting of the





Figures 5a and b. a (left). Coronal CT scan demonstrating complete sinus opacification. b (right). A nasal polyp following surgical removal.

septum. This usually results from local trauma, either digital or from topical medications, but inflammatory disease, such as relapsing polychondritis, or granulomatous conditions, such as Wegener's granulomatosis, always need to be considered.

Chronic obstruction

Patients who experience no relief from nasal congestion usually have a physical obstruction, which is either anatomical or pathological. In addition to the anatomical defects mentioned above, close examination may reveal a nasal or nasopharyngeal mass such as polyps (Figures 5a and b), benign and malignant neoplasia, granulomatous masses or adenoid hypertrophy. Perennial allergy, vasomotor rhinitis or atrophic rhinitis may account for a chronically obstructed nose when no physical obstruction is apparent.

Vasomotor rhinitis

Patients with vasomotor rhinitis experience almost constant, clear rhinorrhoea and have considerable nasal obstruction. Vasomotor rhinitis results from an imbalance of autonomic innervation to the nose due to an array of complex and multifactorial conditions. It is generally a diagnosis of exclusion in chronic nasal obstruction associated with rhinorrhoea – a physical obstruction will be excluded and the patient will fail to respond to conventional therapy.[2] One management plan is to start patients on topical anticholinergic medication, such as ipratropium (Atrovent Nasal, Atrovent Nasal Forte), a topical steroid and a topical antihistamine. If this effectively improves symptoms, then one of the three medications should be stopped every two weeks in order to isolate the most effective treatment. If there is only minimal improvement with the anticholinergic medication, a trial of increasing the frequency and dose is often effective.

Nasal mass

The presence of a nasal mass always warrants referral. Features that cause particular concern include a unilateral mass, bleeding, associated cranial nerve loss, visual changes and pain. Office biopsy of nasal masses is strongly discouraged given the risk of excessive bleeding, leakage of cerebrospinal fluid and false-negative results when a herald inflammatory polyp is biopsied missing the malignancy behind it. Although rare, these are diagnoses that warrant consideration to facilitate prompt detection.

Conclusion

Focusing on a patient's nasal obstruction allows the physician to significantly improve a patient's quality of life, while at the same time avoiding potential diagnostic pitfalls. The GP is well equipped to manage most conditions causing nasal obstruction alone or in conjunction with specialist referral. MT

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COMPETING INTERESTS: Dr Kalish is an invited speaker at a conference being hosted by Care Pharmaceutical in March. Professor da Cruz: None.