Nasal congestion: mechanisms, measurement and medications. Core information for the clinician

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Nasal congestion is a common symptom of nasal disease associated with acute and chronic rhinitis. This review is aimed at the clinician wanting a better understanding of nasal congestion and its treatment. The review aims to provide the clinician with the core information and key references that will aid a better understanding of the aetiology and treatment of nasal congestion.

Nasal congestion

Nasal congestion is caused by swelling of nasal blood vessels that expand to restrict and sometimes completely obstruct the airflow although one or both nasal passages. Nasal obstruction associated with nasal congestion can be distinguished from anatomical obstruction by application of a topical nasal decongestant spray. The topical nasal decongestant constricts nasal blood vessels and opens up the airways. Any restriction in nasal airflow after treatment with a topical nasal decongestant is due to anatomical obstruction such as a deviated nasal septum. The overall feeling of nasal congestion, however, is thought to be due to a combination of factors, including nasal resistance to airflow and more subjective changes including mood, sinus congestion, Eustachian tube function and cool air receptors in the nasal mucosa.

Nasal blood vessels

The blood supply to the nose is via the external and internal carotid arteries and drainage is via the pterygoid plexus and facial vein. Nasal congestion is caused by swelling of specialized capacitance veins in the nasal epithelium. The nasal capacitance vessels are sometimes referred to as venous sinuses, venous sinuses, or venous erectile tissue. The venous erectile tissue is particularly well developed at the anterior end of the inferior turbinate and nasal septum. Swelling in this narrow ‘nasal valve’ region acts to regulate nasal resistance to airflow. The ostia of the paranasal sinuses are also surrounded by a lip of venous erectile tissue, and swelling of these blood vessels in association with a generalized nasal congestion may cause obstruction of the drainage of the paranasal sinuses.

Autonomic innervation of nasal blood vessels

The venous erectile tissue is innervated by a dense network of sympathetic nerves, supplied via the cervical sympathetic nerves, which are distributed to the nose via branches of the maxillary and ophthalmic divisions of the trigeminal nerve and via nasal blood vessels. The sympathetic nerves release noradrenaline and neuropeptide Y, as neurotransmitters that cause an intense vasoconstriction. Interruption of the cervical sympathetic nerve causes Horner’s syndrome with ipsilateral nasal congestion, indicating that there is a continuous sympathetic vasoconstrictor tone to the nasal blood vessels. The venous erectile tissue has little if any...
parasympathetic nerve supply and stimulation of nasal parasympathetic nerves mainly influences glandular secretion and blood flow through the nasal glands.

**Mechanism of nasal congestion**

The control of the filling or swelling of the nasal venous erectile tissue is poorly understood. In 1975, Cauna and Cauna described the presence of cushion or throttle veins. It is thought that through contraction of both circular and longitudinal muscle layers in the wall of these vessels, the throttle veins regulate drainage of the venous erectile tissue, but there is no information on the regulatory mechanism for these throttle veins. Another hypothesis relates the close apposition of the arteries and veins within the periosteal layer and bony canals of the turbinates. Dilatation of the arteries within the bony canals could cause compression of the draining veins, thus restricting the outflow from the venous erectile tissue. This hypothesis also explains the presence of nasal congestion associated with a reduction in sympathetic tone.

**Nasal cycle**

The term nasal cycle has been used to mean ‘spontaneous and often reciprocal changes in unilateral nasal airflow associated with congestion and decongestion of the nasal venous sinuses’. The first documented physiological description was by Kayser in 1895, although he never used the term ‘nasal cycle’. Following a series of direct observations Heetderks stated that ‘the turbinates of one side of the nose were filling while the other side was throwing off secretion. When filling of the turbinates on one side had reached its maximum, the turbinates of the other side were completely empty, thus affording much breathing space’. He determined that this reciprocal change occurred in 70–80% of individuals but, this is disputed by other authors, who concluded that only 21% of volunteers exhibited these airflow patterns.

As yet the functional significance for the nasal cycle in health and disease remains undefined, although an attractive hypothesis is the idea that the nasal cycle may act to share the burden of air conditioning between the two nasal passages. Our understanding of the factors that may influence this nasal cycle are limited. Airflow was thought to be important in the control of the nasal cycle but it has been demonstrated that in a laryngectomized patient, in the absence of airflow, spontaneous congestion and decongestion of the nasal blood vessels still occurs. Oscillation in the activity of the autonomic control centres may exert control over the nasal cycle. In studies on animals, hypothalamic stimulation caused bilateral vasoconstrictor responses and electrical stimulation of alternating sides of the brainstem caused reciprocal changes in sympathetic tone to the nasal blood vessels.

**Effects of posture**

Postural changes in airflow have been demonstrated in healthy volunteers and this response is often exaggerated in patients with rhinitis, where total unilateral nasal obstruction can occur. On change from sitting to supine posture, the increase in venous pressure of 8 mmHg causes a passive hydrostatic effect, resulting in an increase in pressure within the venous erectile tissue and subsequent nasal congestion. The second mechanism by which nasal airway resistance increases involves a reflex response. On adoption of a lateral recumbent position, alteration of the pressure stimulus to one side of the body causes a reflex change in nasal vasomotor activity. Changes in posture, from erect to supine, predominantly affect the more congested side, probably because of the low sympathetic tone and relative compliance of the venous erectile tissue on that side of the nose.

**Causes of nasal congestion**

Nasal congestion is associated with inflammation of the nasal epithelium and the generation of inflammatory mediators that cause dilation of nasal blood vessels. Nasal congestion is a common presenting complaint of ‘rhinitis’ and as such the causative factors for rhinitis, e.g. allergens, viruses, bacteria and decongestant medication, are all potential causative factors for nasal congestion.

Nasal congestion, associated with nasal allergy or infection, can be explained by the effects of local vasodilator mediators on nasal blood vessels and nerves. These mediators include histamine, prostaglandins, kinins and leukotrienes, which are synthesized locally in the nasal mucosa. Nasal congestion and increase in nasal resistance to airflow, caused by allergen challenge, is due mainly to local effects of mediators, acting directly on the nasal blood vessels causing congestion of the venous erectile tissue. Thus unilateral nasal challenge causes unilateral nasal congestion without contralateral congestion.

Both histamine and prostaglandin E2 have been shown to inhibit the release of noradrenaline from the sympathetic nerve endings. This effect of inflammatory mediators on sympathetic nerve endings may be a further cause of nasal congestion.

**Subjective sensation of nasal congestion**

Nasal symptoms such as itching are quite distinct and easily described by the patient and readily understood by the clinician. This is not the case with symptoms of nasal congestion. In describing nasal symptoms the patient is interested in those aspects that cause discomfort and these symptoms may not correlate to those aspects of the symptoms that are of interest to the clinician such as the patency of the nose as assessed by direct examination with a rhinoscope or
measurements of nasal patency made with rhinomanometry or acoustic rhinometry.

Objective measures of nasal conductance are mainly influenced by the minimum cross-sectional area of the nose, situated at the nasal valve region, whereas the subjective sensation of nasal congestion may be influenced by many other factors, as illustrated in Fig. 1.

As mentioned above, nasal congestion is often unilateral and may alternate from one side of the nose to the other, associated with a ‘nasal cycle’. In cases of unilateral nasal congestion the patient may have a complaint of nasal congestion but the objective measurement of total nasal conductance may be in the normal range.

Sensory thermoreceptors, supplied by the trigeminal nerve are situated in the nasal vestibule and are thought to be involved in the detection of nasal airflow. By acting directly on the calcium channels of the receptors, menthol stimulates the receptors to create a sensation of nasal airflow. Although the ingestion of menthol has been shown to have no effect on nasal airway resistance, as measured objectively, it causes a marked change in nasal sensation of airflow with a subjective sensation of nasal decongestion.

Objective assessment of nasal congestion

The first attempt at objectively measuring nasal airflow was probably performed by Zwaardemaker in 1889, who placed a cold mirror beneath the nose and measured the size of the resultant condensation spots. As indicated by previous authors, in order for an objective test to be universally acceptable a number of criteria must be met. These include:

- a simple non-invasive technique which is quick and easy to perform and interpret;
- a test which is standardized, reproducible and reliable;
- a test which is sensitive and universally applicable.

Most tests available do not fulfil all these criteria, however, and the main advantages and disadvantages of the techniques available are considered briefly below.

Nasal peak flow may be measured as either inspiratory or expiratory measures. The advantage of these methods is that they are quick and easy to learn, and the equipment is cheap, small and transportable. These methods have the disadvantages of alar collapse on forced inspiration and expulsion of secretions on expiration, while both methods are effort dependent and assume normal function of the lower airways. Another disadvantage is that multiple repetitions of this procedure cause a change in the blood content of the venous erectile tissue, which changes the nasal airway resistance with time.

Acoustic rhinometry is a useful method to estimate the nasal anatomy and microvascular volume changes associated with congestion. In this method, sound is presented to the nose via a nosepiece and reflected sound is recorded by means of a microphone. The amplitude and delay in the reflected sound is then calculated, by computer analysis, and the cross-sectional area of the nose, and hence anatomy, can be estimated. This method does not offer any information about the dynamics of nasal airflow and offers the clinician little additional information, in the management of the patient, which will not be gleaned from nasendoscopy and CT scanning.

Rhinostereometry is a non-invasive method used to evaluate changes in mucosal oedema of the inferior turbinate. A microscope is mounted on a micrometer table, to allow movement in three angular directions. The subject is fixed exactly to the apparatus by a tooth splint adapted to the teeth. Thus, the nasal cavity is visualized through the eyepiece, and changes of the position of the mucosal surface of the inferior turbinate are registered along a millimetre scale. Changes of 0.18 mm can be detected but this technique remains principally an experimental tool.

The gold standard to date, in objectively assessing nasal airflow resistance, is rhinomanometry, the study of transnasal pressure and nasal airflow. Active anterior and active posterior rhinomanometry are the common methods used in clinical research today.

Nasal resistance to airflow maybe calculated from the following equation:

\[ R = \frac{\Delta P}{\dot{V}}, \]

where \( R \) is resistance to airflow (cmH\(_2\)O/L/s or Pa/cm\(^3\)/s), \( \Delta P \) is transnasal pressure (cmH\(_2\)O or Pa), and \( \dot{V} \) is nasal airflow (L/s or cm\(^3\)/s).

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Figure 1. Factors that influence the patient’s perception of nasal congestion. Nasal resistance to airflow is mainly determined by the cross-sectional area of the nasal valve region at the tip of the inferior turbinate. The patient’s perception of obstruction may be influenced by stimulation of cold receptors in the airway, administration of menthol and mood. Congestion in the ethmoid area, ostia of paranasal sinuses and Eustachian tube cause a perception of congestion and pressure that is unrelated to any change in nasal airway resistance as these areas are distant from the nasal valve.
Transnasal pressure is measured by means of an oral cannula with posterior rhinomanometry and by means of a nasal cannula with anterior rhinomanometry. Total nasal resistance may be determined either directly, using posterior rhinomanometry, or calculated by combining the two separate values of nasal resistance for each nasal passage (obtained with active anterior rhinomanometry), using the following equation:

$$\frac{1}{R(\text{Total})} = \frac{1}{r(\text{Left})} + \frac{1}{r(\text{Right})}.$$ 

Active anterior rhinomanometry, although easy to use, has the disadvantages of only measuring one side of the nose at a time and cannot be used if a septal perforation exists, or in subjects with total obstruction of one nasal passage. Total resistance can be measured directly with posterior rhinomanometry, but this technique requires a period of training. Unilateral measurements of resistance can be made with posterior rhinomanometry by sealing one nasal passage at a time with surgical tape. The equipment used for rhinomanometry is large and cumbersome, however, thus limiting rhinomanometry to laboratory based studies.

Nasal congestion can be quantified in terms of total or unilateral nasal airway resistance. A total nasal airway resistance of 0.3 Pa/cm$^3$/s is generally accepted as the upper limit of normal.3 Because of the spontaneous changes in resistance associated with the nasal cycle, it is not very informative to quote a normal value for unilateral nasal resistance. The range of unilateral nasal airway resistance in a group of healthy volunteers when recorded over a period of 6–8 h has been shown to vary from 0.36 to 1.36, and 0.28–6.3 Pa/cm$^3$/s, indicating almost a fourfold fluctuation in unilateral resistance over this time period.3,21

The partitioning of nasal airflow between the two sides of the nose has been measured by means of nasal spirometry and used to monitor changes in airflow associated with the nasal cycle,22 nasal septal deviation23,24 and rhinitis.25 The nasal spirometer is a portable device, which is easy to use and has shown a good correlation with rhinomanometry for investigating nasal airflow. Nasal spirometry does not give a measurement of nasal airflow resistance but provides a measure of nasal airflow partitioning. In contrast to rhinomanometry, unilateral nasal obstruction is readily measured with the nasal spirometer.

**Treatment**

Treatments for relief of nasal congestion may be considered as environmental control measures, medical therapy and surgical intervention. Environmental control involves avoidance of allergens such as house dust mite, seasonal allergens and animal dander.

Medical therapy can influence nasal congestion by three different mechanisms: (i) by inhibiting the generation of vasodilator mediators associated with rhinitis; (ii) by blocking the effects of vasodilator mediators on nasal blood vessels; and (iii) by mimicking the effects of sympathetic nerve transmitters and causing constriction of nasal blood vessels.

By reducing cytokine and chemokine release, corticosteroids have a strong anti-inflammatory effect, thereby reducing actual cellular infiltration.26 Topical preparations are usually prescribed as the reported risk of developing hypothalamic-pituitary-adrenal axis suppression is apparently low when compared with systemic steroids. The maximum benefit of corticosteroid treatment is not immediate, however, but may take several days to be apparent (Table 1).27

Another group of drugs thought to inhibit the release of vasodilator mediators is the cromones, the most commonly prescribed being disodium cromoglycate. The specific mechanism of action is still unknown but their action is linked to the cell wall of the mast cell and/or to the intracellular events that follow the allergen binding to IgE.27 However, the frequency of dosing schedule, at 4–6 times per day, often leads to poor compliance with this group of drugs.

Histamine is the major mediator involved in the development of nasal congestion associated with allergy. The vasodilator action of histamine is predominantly on H$_1$ receptors but some stimulation via H$_2$ receptors is also thought to occur.28 Antihistamines prescribed for relief of nasal symptoms are H$_1$ receptor antagonists and have no H$_2$ antagonist action. Thus, if used alone in the treatment of nasal congestion, anti-histamines rarely offer the patient full symptomatic relief.

Immediate relief of nasal congestion is offered by a group of drugs called decongestants. Preparations available for clinical use include $\alpha_1$-adrenergic agonists (e.g. phenylephrine), $\alpha_2$-adrenergic agonists [e.g. amines (xylometazoline), imidazoles (oxymetazoline)], noradrenaline releasers (ephedrine and pseudoephedrine) and drugs that prevent the re-uptake of noradrenaline (cocaine, tricyclic anti-depressants).27 $\alpha_2$-adrenergic agonists are contained in many of the ‘over the counter’ preparations. Oxymetazoline has a longer duration of action than xylometazoline and appears more likely to cause rebound nasal congestion.29 The adverse effects of this class of drugs was first high lighted in 193230 and in 1945 Lake coined the term ‘rhinitis medicamentosa’ for the condition defined as nasal hyper-reactivity, mucosal swelling and tolerance, induced or aggravated, by the overuse of topical vasoconstrictors.27 A key factor in the development of rhinitis medicamentosa is rebound nasal congestion that occurs after the effect of the decongestant has declined. Oral decongestants generally have a weaker effect on the relief of nasal congestion than topical preparations but they carry a much lower risk of causing rebound congestion.27

Surgical intervention is well recognized as a modality of treatment for nasal congestion and is performed primarily to restore the respiratory function of the nose, by improving nasal airflow. Many authors recommend surgery to the inferior turbinates as an effective treatment. Many techniques for reduction of the turbinates have been described including...
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<th>Drug class</th>
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<th>Application</th>
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<th>Pharmacological receptors</th>
<th>Onset/duration of action</th>
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<tr>
<td>Sympathomimetics</td>
<td>Significant decrease in subjective symptoms and nasal airway resistance, overall oral decongestants have a weaker effect on the relief of nasal congestion than topical preparations</td>
<td>Ephedrine, 0.25–0.5%, oral: usual doses up to 60 mg TDS</td>
<td>Topical/oral</td>
<td>Tachycardia, anxiety, restlessness, insomnia, tremor, dry mouth</td>
<td>Patients with hyperthyroidism, diabetes mellitus, IHD, hypertension, renal impairment, glaucoma, prostatic enlargement</td>
<td>Interaction with MAOIs, Phenelzine, Moclobemide may lead to hypertensive crisis</td>
<td>( \alpha ) and ( \beta ) adrenergic agonists</td>
<td>Readily and completely absorbed, more potent but less prolonged action than adrenaline</td>
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<td></td>
<td></td>
<td>Pseudoephedrine, 60 mg, 3–4 per day</td>
<td>Oral</td>
<td>As for ephedrine, also skin rashes. Rarely hallucinations in children</td>
<td>As above</td>
<td>As above</td>
<td>Non-selective ( \alpha ) adrenergic agonist</td>
<td>Readily absorbed, half-life 5–8 h</td>
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<td></td>
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<td>Phenylephrine, oral: 5–20 mg, 3–4 doses per day, topical: 0.25–0.5%, four doses per day</td>
<td>Oral/topical</td>
<td>As for ephedrine, may cause a prolonged rise in blood pressure, reflex tachycardia and bradycardia</td>
<td>As above, extreme caution in infants/elderly, may cause powerful systemic side effects</td>
<td>As for ephedrine, Angle – closure glaucoma, severe hyperthyroidism. Cross-sensitivity reported in a patient hypersensitive to pseudoephedrine</td>
<td>Predominantly ( \alpha_1 ) adrenergic agonist</td>
<td>Low oral bioavailability due to first pass metabolism, systemic absorption follows topical administration</td>
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<td>Oxymetazoline, Topical: 0.05% BD</td>
<td>Topical</td>
<td>As for ephedrine, also nausea, headaches, dizziness, transient irritation</td>
<td>As for ephedrine, great caution, if at all, in infants and young children</td>
<td>As for ephedrine, may induce an acute attack of porphyria, therefore unsafe in porphyric patients</td>
<td>( \alpha_2 ) agonist</td>
<td>Systemic absorption occurs following topical administration, acts in a few minutes, lasts up to 12 h</td>
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<tr>
<td></td>
<td></td>
<td>Xylometazoline, topical: 0.05–0.1%, 2–3 doses per day</td>
<td>Topical</td>
<td>As for oxymetazoline</td>
<td>As for ephedrine</td>
<td>As for ephedrine, infants &lt;3 months</td>
<td>( \alpha_2 ) agonist</td>
<td>Absorbed through mucous membranes following topical application, acts in 5–10 min, lasts up to 10 h</td>
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</tbody>
</table>

Table 1. Classifies the various medications used to treat nasal congestion and lists references on efficacy, side effects, precautions, contra indications and the pharmacological receptors mediating decongestion.
<table>
<thead>
<tr>
<th>Drug class</th>
<th>Evidence of efficacy of drug class</th>
<th>Frequently used drugs within each class[^38]</th>
<th>Application</th>
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<tr>
<td>Corticosteroids</td>
<td>Recent meta-analysis confirmed the superiority of topical corticosteroids to anti-histamines in the treatment of allergic rhinitis for all nasal symptoms[^38]</td>
<td>Beclomethasone, Betamethasone, Budesonide, Dexamethasone, Fluticasone, Flunisolide, Momethasone, Triamcinolone</td>
<td>Topical</td>
<td>Local: dryness, irritation of nose and throat, epistaxis (usually mild). Rarely ulceration, nasal septal perforation, raised intraocular pressure[^37], glaucoma. Dexamethasone spray and betamethasone drops can rarely provoke systemic effects[^27]</td>
<td>Presence of intranasal infection, immediately postsurgery (prior to healing), pulmonary TB, prolonged use in children may cause growth retardation (height should be monitored)[^37,40]. Adjuvant steroid treatment for asthma and eczema may lead to the possibility of an additive effect and steroid loading[^41]</td>
<td>Hypersensitivity reaction[^37]</td>
<td>Reduction in cytokine and chemokine release. Decrease in cellular infiltration of antigen presenting cells[^25]</td>
<td>Slow onset of action, usually after 12 h, maximum efficacy develops over days and weeks when used regularly[^27,28]</td>
</tr>
<tr>
<td>Cromones</td>
<td>Generally less effective than corticosteroids and anti-histamines in allergic rhinitis[^27]</td>
<td>Disodium cromoglycate, solution: 2.5–5 mg as a 2–4% solution, six doses per day. Powder: 10 mg QDS[^37]</td>
<td>Topical</td>
<td>Rarely local irritation[^7]</td>
<td>Generally well tolerated[^37]</td>
<td>Hypersensitivity reaction[^37]</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The table is meant only as a guide of commonly used medications, doses are quoted for the adult, and the manufacturers instructions should always be referred to for precautions and contraindications.
partial resection, which was popularized by Mabry in 1984.\textsuperscript{31} By causing submucosal fibrosis, Quine et al. described how the use of submucosal diathermy to the inferior turbinates acts to ‘splint’ the airway in a state of relative decongestion, thus increasing nasal airflow by decreasing the amplitude of spontaneous changes in unilateral nasal airflow.\textsuperscript{32} This state of relative decongestion may then allow improved delivery of medical treatment such as a topical steroid spray.\textsuperscript{33}

As discussed previously, in many patients it is difficult to determine if there is any link between septal deviation and symptoms of nasal congestion. The use of septal surgery for relief of nasal symptoms thus remains a topic of debate,\textsuperscript{34} although some studies report postoperative satisfaction levels of up to 85%.\textsuperscript{35}

**Conclusion**

Nasal congestion is one of the most common complaints associated with acute and chronic conditions of rhinitis. The changes in nasal congestion, associated with the nasal cycle and changes in posture, often complicate the presentation of the symptom for both the clinician and patient. In the majority of acute conditions of rhinitis associated with infection and allergy, over the counter treatments can provide symptom relief. In cases of chronic nasal congestion, where medical therapy has not provided symptom relief, nasal surgery may be necessary to correct any anatomical problem such as a deviation of the nasal septum.

**References**

30. **Fox** N. (1931) The chronic effect of epinephrine and ephedrine on the nasal mucosa. *Arch. Otolaryngol.* 13, 73–79