The Journal of
Laryngology & Otology

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Correlation between nasal mucosal temperature change and the perception of nasal patency: a literature review

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Review Article

Dr R Tjahjono takes responsibility for the integrity of the content of the paper

Paper presented at the New Zealand Society of Otolaryngology Head and Neck Surgery 72nd Annual Scientific Meeting, 18 October 2019, Dunedin, New Zealand.

Cite this article: Tjahjono R, Singh N. Correlation between nasal mucosal temperature change and the perception of nasal patency: a literature review. *J Laryngol Otol* 2021;**135**:104–109. https://doi.org/10.1017/S0022215121000487

Accepted: 26 September 2020
First published online: 22 February 2021

Key words:
Nasal Obstruction; flow; Thermoreceptors; Temperature; Fluid Dynamics

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Abstract

Background. The mechanism of nasal airflow sensation is poorly understood. This study aimed to examine the role of nasal mucosal temperature change in the subjective perception of nasal patency and the methods by which it can be quantified.
Method. Medline and PubMed database searches were performed to retrieve literature relevant to the topic.
Results. The primary mechanism producing the sensation of nasal patency is thought to be the activation of transient receptor potential melastatin family member 8 ('TRPM8'), a thermoreceptor that is activated by nasal mucosal cooling. Computational fluid dynamics studies have demonstrated that increased airflow and heat flux are correlated with better patient-reported outcome measure scores. Similarly, physical measurements of the nasal cavity using temperature probes have shown a correlation between lower nasal mucosal temperatures and better patient-reported outcome measure scores.
Conclusion. Nasal mucosal temperature change may be correlated with the perception of improved nasal patency. Future research should quantify the impact of mucosal cooling on the perception of nasal airway obstruction.

Introduction

Nasal airway obstruction is one of the most common presenting complaints in otolaryngology practice, and it has a significant impact on quality of life and overall health.[1] Patient-reported symptoms of nasal airway obstruction may be described as nasal congestion, fullness, blockage, stuffiness or discomfort. Patients with nasal airway obstruction report a significantly reduced quality of life compared to the general population, with some studies demonstrating a mean utility value less than that for Parkinson's disease, coronary artery disease, congestive heart failure and moderate chronic obstructive pulmonary disease.[2–7] Evidence also demonstrates that nasal airway obstruction carries a significant health economic expenditure.[1]

Nasal airway obstruction may be assessed both subjectively and objectively. In subjective terms, nasal airway obstruction refers to the perception of reduced nasal airflow, which can be quantified using patient-reported outcome measures, such as the Nasal Obstruction Symptom Evaluation score or visual analogue scale (VAS). Nasal airway obstruction may also be assessed objectively as reduced nasal airflow or increased nasal resistance. Objective diagnostic tests include: rhinomanometry to measure nasal airflow resistance, flow and pressure; acoustic rhinometry to calculate the cross-sectional area at various points along the nasal cavity; and peak nasal inspiratory flow.

Nasal airway obstruction may occur as a result of several conditions where airflow is hindered through the nose. These conditions may be secondary to static or dynamic anatomical restriction, mucosal changes, or a combination of the two. Common anatomical causes include nasal septal deviation (static), inferior turbinate hypertrophy (dynamic) and nasal valve collapse (dynamic), while common mucosal causes include allergic rhinitis and chronic rhinosinusitis.[8]

While some patients can be managed with pharmacological intervention alone, those who do not respond may require surgical intervention. The most common procedures performed for nasal airway obstruction are septoplasty (to correct a nasal septal deviation) and inferior turbinate reduction (to correct inferior turbinate hypertrophy), which may be undertaken individually or simultaneously depending on specific patient's anatomical and disease factors.

However, patients often report persistent nasal airway obstruction post-operatively, despite surgeon satisfaction with the clinical appearance of the post-operative nasal airway and objective testing demonstrating improved and sufficient airflow.[9–11] The rates of surgical intervention failure are reported to range between 23 and 50 per cent.[12–14] As a result, baseline assessment and treatment of symptoms in nasal airway obstruction are highly reliant on subjective opinion and feedback, resulting in inconsistent outcomes.[15]

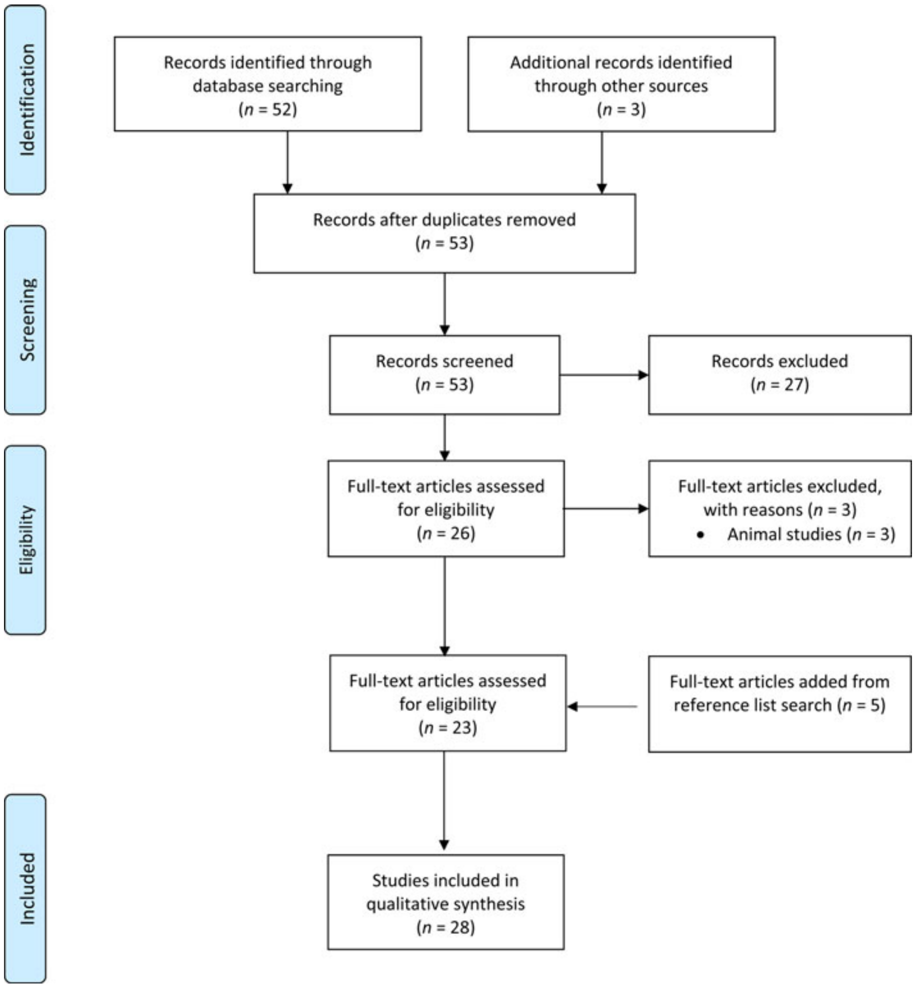

Fig. 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses ('PRISMA') diagram for the literature review.

This discordance between the objective and subjective findings in certain pre- and post-operative patients with nasal airway obstruction suggests that the subjective sensation of nasal patency and airflow may be determined by receptors that do not function primarily by detecting objective nasal airflow. Instead, this observation suggests that the detection of nasal airflow is via an indirect mechanism, which has the potential to be misled in certain scenarios. There is a growing body of evidence to indicate that an important mechanism of nasal airflow sensation may be secondary to mucosal cooling by inspired air and the subsequent change in nasal mucosal temperature across the nasal cavity.[16,17]

Recent evidence has suggested that a thermoreceptor, transient receptor potential melastatin family member 8 ('TRPM8'), is expressed by over 60 per cent of trigeminal afferents in the nasal mucosa.[18,19] This receptor conveys a 'cool' sensation during nasal airflow, which may be interpreted by higher centres as a more patent nasal airway. This may explain why pharmacological modulation of these afferents, such as with the use of menthol or eucalyptol produces a sensation of decongestion, despite no change in the anatomical architecture of the nose.[16]

This literature review aimed to appraise the relevant evidence on the role of nasal mucosal temperature change in the subjective perception of nasal patency. The secondary aim was to determine the methods by which mucosal cooling can be reliably measured.

Materials and methods

A literature review of the topic was conducted through Medline and PubMed database searches. This was initially performed between 30 March and 7 April 2019; however, a repeat search was carried out between 16 and 22 June 2020. The search string used was: (Nasal airway obst* OR nasal obst* OR nasal congest*) AND (Temp* OR nasal temp*) AND (Nasal paten*).

Studies were selected for inclusion if they described the role of thermoreceptors and/or the impact of nasal mucosal temperature change in the perception of nasal patency, either through direct nasal temperature measurement or computational fluid dynamics simulations. This was achieved following a screen of the study title and abstract. Furthermore, the reference lists of the reviewed studies were examined to identify articles not found by the Medline and PubMed searches. Animal and non-English studies were excluded for the purposes of this review.

Results and discussion

Fifty-five studies were identified through the initial search strategy. Twenty-three of these studies were considered relevant to this literature review, with an additional five studies included following a search of the reference lists of studies from the initial search. This is described in a Preferred Reporting Items for Systematic Reviews and Meta-Analyses ('PRISMA') diagram (Figure 1).

Thermoreceptors and nasal airflow sensation

Traditionally, it has been assumed that a patient's perception of nasal patency is dependent on the direct physiological

https://doi.org/10.1017/S0022215121000487 Published online by Cambridge University Press

detection of air flowing through the nose or resistance to flow. Measurements of airflow and resistance can be quantified with objective tests; however, these tools have not been universally adopted for surgical planning given their poor correlation with subjective nasal patency, as well as other intrinsic issues with each test pertaining to operator dependence and poor reliability.[9–11]

There is growing evidence to suggest that the mechanism of nasal airflow sensation may function via indirect means. In particular, there has been significant interest recently in the detection of mucosal cooling by inspired air and subsequent nasal mucosal temperature change.[18]

Recent literature has revealed the existence of transient receptor potential melastatin 8, a thermoreceptor expressed by over 60 per cent of trigeminal afferents distributed in the nasal mucosa.[19] These receptors are located near blood vessels, and activation of these thermoreceptors is linked to local vasoconstriction.[20] Transient receptor potential melastatin 8 has been proposed to be important in the perception of nasal patency as it conveys a 'cool' sensation. The thermoreceptor is classified as a non-selective voltage-dependent cation channel and is activated when inspired air moves through the nasal cavity at high speeds, inducing water evaporation from the epithelial lining fluid. As the temperature of the remaining fluid drops, a reduction in membrane phospholipid fluidity occurs.[20] Transient receptor potential melastatin 8 detects the relative reduction across the nose, leading to neuronal depolarisation, and signalling to the respiratory centre of the brainstem a 'cool' sensation; this is then interpreted as a more patent and open nasal airway, leading to a reduction in accessory and intercostal muscle work in breathing.

Activation of transient receptor potential melastatin 8 receptors occurs along the nasal septum, and the inferior and middle turbinate, in response to humidified air and certain molecules such as menthol and eucalyptol.[21] In contrast, this sensory input is lost when nasal packing or nose clips obstruct the nostrils, or in laryngectomy patients where the upper aerodigestive tract airflow is diverted. Absence of these inputs is thought to cause the sensation of nasal congestion, and consequently increased work of breathing using accessory and intercostal muscles.[22]

Pharmacological modulation of trigeminal afferents has been seen to play a role in the perception of nasal patency.[12,23–25] For instance, the application of topical menthol in the nostrils or hard palate produces a sensation of decongestion, despite causing no actual alteration in nasal morphology, airflow or resistance as determined by objective measurements such as rhinomanometry.[16,17] This may be secondary to direct activation of transient receptor potential melastatin 8 receptors. On the other hand, the injection of local anaesthetic into the nasal vestibule induces a subjective sensation of congestion without objective change in nasal airflow, potentially due to the inhibition of transient receptor potential melastatin 8 receptor activation.[26]

A corroborative study by Zhao *et al.* examined the effect of a number of variables, including air temperature and humidity, nasal cross-sectional area, resistance and mucosal cooling, on the subjective perception of nasal patency in 44 participants.[15] Participants were asked to rate their sensation of nasal congestion by sampling air from three boxes containing untreated room air, dry air and cold air. It was found that participants reported significantly less nasal congestion following inspiration from the dry and cool air boxes compared to untreated room air, in keeping with possible involvement of nasal humidity and temperature in the sensation of nasal airflow. Nasal cross-sectional area and resistance were not significantly correlated to perceived nasal congestion.[15] For these reasons, objective assessments of nasal airflow are often complemented with patient-reported outcome measures to provide a more comprehensive assessment.

Airflow pattern changes and effects on nasal airflow sensation

Static air temperature and environmental humidity are important in the dynamic heat loss and cooling of nasal mucosa. However, it is also important to consider the interaction between the individual's nasal airway structures, baseline thermosensory sensitivities and inspired airflow in thermoregulation. Differences in nasal structure and physical conditions may result in varying degrees of nasal mucosal cooling, leading to varying changes in the perception of nasal patency amongst different people.[15]

A study on the air-conditioning capacity of the nasal cavity using three-dimensional (3D) nasal cavity reconstructions by Naftali *et al.* demonstrated that the inferior and middle turbinates and the septal and lateral nasal walls (60–70 per cent) have the highest contribution in overall heating of inspired air.[27] Other structures contributing to overall heating of inspired air include the anterior and posterior nasal walls, and the floor and roof of the nasal cavity. Repeat simulations in this study demonstrated a decrease in the heating of inspired air by 12 per cent without the middle turbinate, and by 16 per cent without the inferior turbinate. These findings are attributed to the alterations in airflow patterns and the loss of air-conditioning capacity following removal of the inferior and middle turbinates.[27]

In addition, turbulence is a known determinant of nasal mucosal cooling, as temperature changes and particle filtering are more pronounced within areas of turbulent airflow in comparison to areas with laminar airflow, particularly around the turbinate mucosa.[28,29]

The effects of alterations of nasal airflow in relation to nasal patency can be illustrated in those with nasal septal deviation, where significant abnormalities cause an alteration in airflow and mucociliary clearance. Septal deviations tend to shift airflow inferiorly, leading to reduced middle turbinate airflow and reduced nasal mucosal cooling.[30] Furthermore, turbulence is created when inspired air contacts the convex side of the deviated septum, causing drying of the nasal mucosa – this is the current accepted mechanism, outside of digital trauma, to explain why there is increased risk of epistaxis in this group of patients.[30,31]

Another example is 'empty nose syndrome', a rare and controversial condition in which patients with anatomically patent nasal cavities (usually following a sinonasal procedure for nasal airway obstruction) report severe, often debilitating nasal obstruction, crusting and dryness. It is hypothesised that reduced airflow turbulence from a lack of contact of the inspired airstream with the nasal mucosa leads to an abnormal airflow pattern, producing minimal mucosal cooling, in a similar manner to a narrow nasal cavity with inadequate airstream.[32] Therefore, the development of future treatments for nasal airway obstruction may be directed towards improving the patient's nasal mucosal cooling function and thermosensory ability to achieve better outcomes.

https://doi.org/10.1017/S0022215121000487 Published online by Cambridge University Press

Computational fluid dynamics

This section concerns computational fluid dynamics and its role in modelling nasal physiology. Computational fluid dynamics is a branch of fluid mechanics used to analyse the flow of incompressible substances (including fluid and air) across rigid structures. High-powered computers are used to perform the calculations required to simulate the interactions of gases and liquids within surface boundaries under a set of known conditions. In otolaryngology, computational fluid dynamics models are derived from high-resolution computed tomography (CT) or magnetic resonance imaging scans of the paranasal sinuses. Following segmentation of the nasal geometry, nasal physiology is simulated, allowing airflow, heat changes, water vapour and transport of inhaled particles to be analysed.[33–35] Calculations of airflow through the reconstructed nasal cavity are typically performed based on the Navier–Stokes equation (laminar model).[36]

Computational fluid dynamics simulations have gained popularity recently, following increased insight into the intricacies of nasal airflow and sinonasal function. For instance, it was found that the peak nasal mucosal heat loss (and therefore nasal mucosal temperature change), which mainly occurs in the mucosa posterior to the nasal vestibule and, to a lesser extent, in the middle meatus, significantly correlates with better perception of nasal patency.[37] Furthermore, computational fluid dynamics simulations revealed that airflow in the middle meatus accounted for over 30 per cent of total nasal airflow. In addition, there was very little air exchange between the nasal cavity and paranasal sinuses during quiet breathing; however, it was predictably increased following sinus surgery.[38,39]

Studies have compared computational fluid dynamics variables with the subjective perception of nasal patency, with the aim of objectively diagnosing the cause of reduced nasal airway patency and predicting and evaluating treatment outcomes. Casey *et al.* compared intranasal airflow distribution in nasal airway obstruction patients and healthy individuals.[31] The nasal airway obstruction patients were found to have significantly reduced airflow in the middle region of the nasal cavity. The reduced airflow correlated with the sensation of reduced nasal patency, which was quantified using the VAS and Nasal Obstruction Symptom Evaluation scores.[31] In addition, computational fluid dynamics simulations have been conducted in patients with nasal airway obstruction before and after surgery. This has revealed positive correlations between computational fluid dynamics variables, such as airflow and heat flux, with VAS and Nasal Obstruction Symptom Evaluation scores.[23,33] These studies provide some evidence that mucosal cooling has significant clinical relevance to perceived nasal patency. Furthermore, computational fluid dynamics models have the potential for future applications in virtual surgical planning and the evaluation of patients with nasal airway obstruction.

However, computational fluid dynamics modelling and analysis have been complicated by the nasal cycle. Computer models are typically derived from radiological images, which are taken from a single snapshot in time, and will often show the nose part-way through a cycle. This will result in one side being congested while the other will appear decongested, potentially distorting the computer model and subsequent computational fluid dynamics analysis. In order to avoid this problem, decongestants may be used prior to scanning, which will result in bilateral mucosal decongestion. However, while this will result in mucosal symmetry in the model, it may not accurately represent true physiology.[40]

Recently, Gaberino *et al.* attempted to circumvent this problem by creating virtual mid-nasal cycle models of 12 patients who underwent sinonasal surgery.[41] This was done by comparing the extremes of mucosal congestion and decongestion of the middle and inferior turbinates from pre- and post-operative CT scans for each patient. Following correction of the nasal cycle, the study found an increased correlation between subjective and objective measures of nasal patency. Results from this study further emphasised the confounding impact of the nasal cycle in computational fluid dynamics analysis, and the importance of nasal cycle correction in virtual surgery planning in the future.[41]

Physical measurement of nasal mucosa temperature

While computational fluid dynamics simulations demonstrate that it is possible to quantify inspiratory mucosal heat loss through 3D modelling of a patient's nasal anatomy, limitations of this modality exist. These include radiation exposure from CT scanning, the cost of scanning, and the time required to obtain the medical images, construct the nasal anatomy model and conduct the simulation. In addition, computational fluid dynamics models are computer simulations with resultant assumptions and limitations, and they may not represent the actual physiology. In order to increase the applicability of nasal mucosal temperature in clinical practice, several studies have been conducted that aimed to measure temperature through physical modalities.

Lindemann *et al.* measured the nasal mucosal temperature at various intranasal sites during respiration without interruption of nasal breathing.[42] This was achieved by placing a miniaturised thermocoupler in the nasal vestibule, nasal valve area, anterior turbinate area and nasopharynx. The mean nasal mucosal temperature ranged between 30.2 ± 1.7°C and 34.4 ± 1.1°C, with the highest temperature detected in the nasopharynx and at the end of expiration.[42] A subsequent study by Lindemann *et al.* recorded nasal mucosal temperature using the same methodology, but temperature values were then compared with rhinomanometrical data.[28] That study found an inverse correlation between nasal mucosal temperature and nasal airflow, further indicating that mucosal cooling may be a significant mechanism in the perception of nasal patency.

Willatt and Jones examined the correlation between subjective nasal patency and nasal mucosal temperature.[43] Specifically, they compared the VAS score with nasal mucosal temperature recorded using a non-contact infrared thermometer to the anterior nasal septum at the level of the piriform aperture in 62 individuals. Participants were asked to perform quiet breathing during the temperature recording. The study found that the lower the nasal mucosal temperature, the higher the VAS score, with better subjective sensation of nasal patency.[43]

Similarly, Bailey *et al.* conducted a study comparing VAS and Nasal Obstruction Symptom Evaluation survey scores with nasal mucosal temperature recordings, using miniaturised thermocouples inserted against the nasal septum at the level of the nasal vestibule and head of the inferior turbinate, of 22 healthy individuals.[25] Participants were asked to perform 60 seconds of quiet breathing followed by three deep breathing cycles. Higher mucosal temperature oscillations with lower inspiratory mucosal temperatures were seen in deep breathing; in addition, lower temperatures measured on the right vestibule had significant correlations with better VAS and Nasal Obstruction Symptom Evaluation scores.[25]

Conclusion

Nasal airway obstruction is a common, yet complex condition that is not yet fully understood. Several modalities exist to objectively assess the character and severity of nasal airway obstruction, such as acoustic rhinometry and rhinomanometry; however, these modalities have not been universally adopted for surgical planning because of poor correlation with subjective nasal patency, among other limitations.

Recent studies have raised the intriguing possibility that mucosal temperature change may be the primary determinant in patients' perceptions of nasal patency. These investigations have shown correlations between lower intranasal temperatures and better subjective perception of nasal patency, by either physical temperature measurements or computational fluid dynamics airflow simulations, mostly in healthy subjects.

Thus, future research in nasal airway obstruction should be directed towards the quantification of mucosal cooling and the development of an objective test for surgical planning. Such a test could be based on computational fluid dynamics analysis of nasal heat flux and physical measurements of nasal temperature.

Competing interests. None declared

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