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Table of Contents

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Surgical Management of Allergic Rhinitis

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Introduction

The inferior turbinate is composed of a central bony structure surrounded by ciliated pseudostratified columnar epithelium.[1] Underneath the epithelium, there are mucous glands, goblet cells and venous sinusoids in the submucosa,[1] The inferior turbinate is supplied by the inferior turbinate branch of the posterior lateral nasal artery from the sphenopalatine artery.[2] The inferior turbinates humidify the air, trap particles and direct airflow throughout the nasal cavity. Due to these functions, the inferior turbinates are often the first point of contact for allergens in the inspired air. With immune responses prompted by a cascade of inflammatory mediators such as immunoglobulin E, mast cells, histamine, and leukotrienes, mucous glands become stimulated and the vasculature within the inferior turbinate engorges. This results in inferior turbinate hypertrophy, leading to increased nasal discharge and nasal obstruction. For individuals refractory to medical therapy for nasal obstruction, surgical inferior turbinate reduction techniques can be employed to help alleviate the symptoms. For those with rhinorrhea refractory to medical therapy, surgical management aimed at reducing parasympathetic innervation to the nasal cavity to reduce nasal discharge production can be also option.

Nasal septum can contribute to nasal obstruction with deviation and spurs. However, its role in the development of allergic rhinitis is unclear. The septum itself does not undergo the extent of changes that inferior turbinates do in terms of mucosal edema, engorgement, or mucosal gland hyperplasia. Therefore, surgical management of the septum is not the focus for the surgical treatment of allergic rhinitis and will not be discussed in this paper.

The paranasal sinuses can be involved in certain advanced types of allergic rhinitis, such as central compartment atopic disease. If the patient is unresponsive to medical therapy alone, endoscopic sinus surgery can be considered. Certainly, endoscopic sinus surgery can be combined with inferior turbinate surgery in refractory or advanced cases of allergic rhinitis. However, endoscopic sinus surgery techniques are not the focus of this paper. Instead, we will focus on the discussion of various techniques of inferior turbinate reduction, as well as surgical techniques to address rhinorrhea in the treatment of allergic rhinitis.

Turbinate Lateralization

Lateralization of the turbinate is the most surgically conservative technique to reduce the inferior turbinates. Outfracturing the turbinate improves the nasal airflow by repositioning the angle at which the turbinate attaches to the maxillary and palatine bones. A firm instrument, such as a Freer or Cottle elevator, is inserted lateral to the inferior turbinate in the inferior meatus and infractures the turbinate for complete fracture of the turbinate bone. Subsequently, the turbinate is outfractured from the medial aspect. The effect of turbinate lateralization can be sustained for at least 6 months postoperatively.[3] Lateralization of the turbinate may not be sufficient as a stand-alone procedure for the surgical management of severe allergic rhinitis as the bony turbinate tends to return to its original position. It often is combined with other techniques and is performed as a last resort in order to optimize soft tissue dissection without fracture.

Laser-assisted Turbinoplasty

There are various types of lasers available for the treatment of inferior turbinate hypertrophy. These include carbon dioxide (CO_2), potassium-titanyl phosphate (KTP), argonion, neodymium-yttrium aluminum garnet (Nd:YAG), and holmium-yttrium aluminum garnet

(Ho:YAG) lasers.[4] The goal of laser therapy is to induce fibrosis and reduce the turbinate surface area. The laser is applied in a linear or spot fashion, anterioposteriorly along the inferior turbinate or at the anterior aspect of the inferior turbinate. Symptomatic improvement has been reported in the literature with rates from 50–100% effectiveness with heterogeneous follow-up periods. While the advantages of laser-assisted turbinoplasty may include enhanced hemostasis and reduced postoperative pain, the laser equipment can be expensive and is not widely available in many centres, limiting its utility.

Submucosal Soft Tissue Reduction

Electrocautery

Monopolar and bipolar electrocautery can be used to cause thermal injury in the submucosa of the inferior turbinate. This leads to submucosal fibrosis and obliteration of the venous sinusoids. The needle tip(s) is(are) inserted into the submucosa and advanced toward the posterior inferior turbinate in the submucosal pocket. The electrocautery is then activated as the needle is withdrawn. However, studies of electrocautery have not shown promising long-term results.[5]

Radiofrequency Ablation and Coblation

Radiofrequency ablation (RFA) is a thermal ablation technique with the application of radiofrequency (varying from 100 to 4,000kHZ) within the submucosa, thereby leading to coagulative necrosis.[6] Both scar contracture and the formation of scar fibrosis result in reduction of the turbinate bulk with lasting effects, potentially up to 5 years.[7] It is well tolerated with minimal side effects and can be done as an in-office procedure under local anesthesia.[6,7]

Coblation is a technique within the umbrella of RFA and uses the concept of molecular activation dissection in the submucosal layer.[8] With low-temperature tissue disintegration, this technique causes less pain and therefore can be performed more commonly in the pediatric population.[8]

Submucosal Resection

The goal of submucosal resection of the inferior turbinate is to preserve the mucosa and its ciliary function while reducing the volume of the inferior turbinate. To perform this procedure, the turbinate is infiltrated with a local anesthetic with epinephrine to reduce bleeding and to help with hydrodissection. An incision is made in the anteroinferior aspect of the turbinate. A medial mucoperiosteal flap is raised along the inferior turbinate bone all the way posteriorly using a Freer or Cottle elevator. A lateral mucoperiosteal flap is raised in a similar fashion. Alternatively, some surgeons choose to sacrifice the lateral mucosa while preserving the medial mucoperiosteal flap. The inferior turbinate bone is subsequently resected with a through-cutting instrument. Submucosal resection of the inferior turbinate (including the bone) has shown superior results at the 5-year mark in comparison with electrocautery or submucosal powered turbinate reduction.[9]

Microdebrider-assisted Submucosal Resection

Similar to the traditional submucosal resection, the goal of microdebrider-assisted reduction is to decrease the amount of submucosal erectile tissue while retaining the epithelium. To perform this procedure, a stab incision is made in the anterior aspect of the turbinate. A submucosal tunnel is developed using an incorporated tip elevator or a Freer or Cottle elevator. The microdebrider blade is then rotated toward the submucosal soft tissue and activated. It is crucial to perform this in a controlled fashion to avoid flap perforation. The tip of the microdebrider can also be turned toward the bone for bone resection. However, it may require formal submucosal resection if more extensive bone removal is required. Although microdebrider-assisted turbinoplasty encompasses various heterogeneous techniques, the literature demonstrates the long-term effectiveness of this technique.[10]

Turbinectomy

Total turbinectomy is the most aggressive technique available for the surgical treatment of allergic rhinitis. It can be performed with heavy scissors or endoscopic scissors. However, this practice has fallen out of favour as it can lead to severe long-term complications such as atrophic rhinitis or “empty nose syndrome”.[11] This is likely secondary to the loss of function of the inferior turbinates and leads to excessive mucosal drying, scarring, nasal discharge, and recurrent epistaxis.[12]

Complications of Turbinate Reduction

Most mucosa-sparing turbinoplasty techniques have acceptable safety profiles. Complications of inferior turbinate reduction include bleeding, nasal dryness, and crusting. A meta-analysis focused on long-term outcomes of turbinate surgery showed frequency of 4%, 2%, and 17% for bleeding, nasal dryness, and crusting, respectively, with no reported serious complications.[10] Atrophic rhinitis or "empty nose syndrome" is the most feared complication but it is rare and tends to be attributed to aggressive tissue resection.[11,12]

Surgical Techniques for Rhinorrhea

Vidian Neurectomy

Vidian neurectomy is a well-established procedure with the aim of disrupting parasympathetic supply to the nasal cavity and subsequently reducing the production of nasal secretions. The vidian nerve is formed by the greater superficial petrosal nerve and the deep petrosal nerve. While the deep petrosal nerve is formed by sympathetic fibres from the sympathetic plexus, the superficial petrosal nerve provides preganglionic parasympathetic fibres for the lacrimal, palatine, and nasal glands as well as vasodilator nerves for the nasal mucosa.[13] Vidian neurectomy was initially described by Golding-Wood in the 1960s via a transantral approach.[14] Recently, with the advancement of endoscopic techniques, vidian neurectomy has been reported to be successful in achieving 91% of patient satisfaction.[15] Vidian neurectomy can be performed endoscopically by making a mucosal incision similar to that in sphenopalatine artery ligation. The artery is ligated and a mucosal flap is raised to the face of the sphenoid sinus. The periosteum and the fat of the pterygopalatine fossa are then exposed. The vidian nerve is then identified emerging through the pterygoid canal and ligated.[13] However, vidian neurectomy has fallen out of favour due to complications such as cheek/palate numbness and dry eyes from collateral injury of the nerve fibres innervating the lacrimal glands.[16]

Posterior Nasal Neurectomy

Due to the complications of vidian neurectomy, the posterior nasal nerve has emerged as an attractive target to address rhinorrhea. The posterior nasal nerve carries postsynaptic parasympathetic fibres that innervate the nasal mucosa; it is distal to the pterygopalatine ganglion. Therefore, posterior nasal neurectomy does not carry a risk of dry eyes or palate numbness. The endoscopic technique was first described by Kikawada in 1997.[17] A vertical incision is made in the posterior middle meatus. A mucosal flap is then raised over the palatine bone to identify the sphenopalatine foramen posterior to the crista ethmoidalis. The posterior nasal nerve emerges from the sphenopalatine foramen typically below the sphenopalatine blood vessels. Either the entire neurovascular bundle or the nerve alone can be ligated. A systematic review of posterior nasal neurectomy showed that while it appeared safe and generally efficacious, there is significant heterogeneity in the reporting of outcomes thus limiting any firm conclusions on its effectiveness.[18]

Posterior Nasal Nerve Ablation

With the advent of in-office devices, posterior nerve ablation has become a popular alternative option to posterior nasal neurectomy. Cryotherapy can be used to ablate the posterior nasal nerve in the posterior middle meatus and has been found effective in many single-arm studies.[19] Similarly, radiofrequency neurolysis of the posterior nasal nerve has been found effective and well tolerated.[20] For both of these techniques, clinical benefit in nasal symptoms has been derived and they have shown to improve patient quality of life.

Conclusion

Allergic rhinitis is a very common condition. Patients refractory to medical therapy can be considered for surgical intervention. Inferior turbinate hypertrophy is the most important contributor to nasal obstruction in patients with allergic rhinitis. A variety of surgical procedures have been developed and described. Most techniques have acceptable safety profiles. There is a lack of consensus on a "gold standard" technique. Meticulous surgical dissection and thoughtful reduction of soft tissue are paramount in achieving a balance between symptom improvement and preserving normal nasal physiology. For the bothersome symptom of rhinorrhea secondary to allergic rhinitis, there are various surgical options available. The posterior nasal neurectomy technique has lower rates of complications compared to vidian neurectomy.

In addition, posterior nasal nerve ablation can be an attractive alternative with the ability to be performed in-office. However, additional studies are needed to characterize the long-term treatment outcomes of these procedures.

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D.L: None declared.
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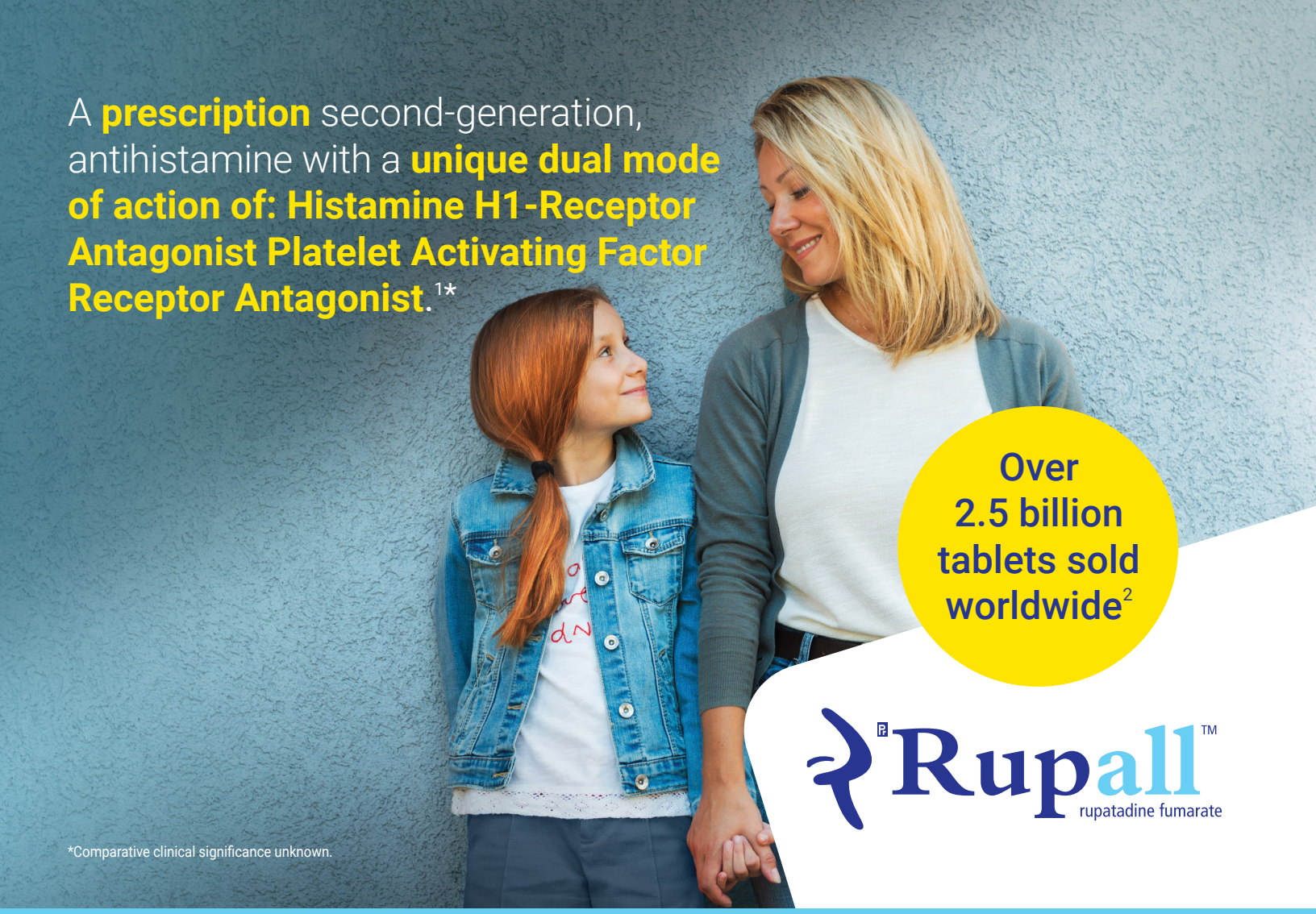

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Adult Obstructive Sleep Apnea: A Practical Guide for the Allergist

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Introduction

Obstructive sleep apnea (OSA) is a common sleep disorder, affecting 15-30% of males and 10-15% of females in North America.[1,2] OSA is characterized by the recurrent obstruction of the upper airway during sleep resulting in apneas and hypopneas. OSA can be diagnosed using a home sleep apnea test (i.e., a level 3 sleep study) or a polysomnogram (PSG) (i.e., a level 1 study or PSG) in a supervised sleep laboratory which quantifies the number of apneas and hypopneas per hour using the apnea-hypopnea index (AHI). In broad terms, the diagnosis is made when an individual with symptoms or cardiac risk factors has an AHI of ≥5, or an asymptomatic individual has an AHI of ≥15.[3] Continuous Positive Airway Pressure (CPAP) is considered the first-line treatment of OSA. Other treatment options include an oral appliance called a mandibular advancement device, or multilevel surgery of the upper airway, especially uvulopalatopharyngoplasty (UPPP).[4] Herein we review OSA in the context of conditions commonly observed in the allergy clinic, namely asthma, chronic rhinosinusitis, rhinitis, and contact dermatitis.

OSA in Asthma

Asthma is a highly prevalent respiratory disease affecting ~8% of Canadian adults.[5] Roughly 5–10% of asthmatics have severe asthma, which is defined by inadequate control, despite intensive treatment with high-dose inhaled corticosteroids (ICS), long-acting beta agonists, or oral corticosteroids (OCS). A broader category, difficult-to-treat asthma, encompasses those whose condition remains unmanaged owing to factors such as treatment non-compliance or the presence of other medical conditions.[6] Among these, OSA is emerging as a significant comorbidity.

Given that the adult prevalence of moderate-to-severe OSA (AHI ≥15) ranges from 6% to 17%,[7] a considerable overlap between asthma and OSA in patients is anticipated. However, emerging evidence underscores a potential bidirectional link between asthma and OSA beyond simple coincidence. A meta-analysis conducted in 2017 found that approximately 50% of asthma patients have OSA and the odds ratio for prevalent OSA in asthmatics was 2.64.[8] Incident OSA is also increased in asthma. The prospective Wisconsin Sleep Cohort study found that the relative risk of developing OSA and OSA with habitual sleepiness in asthmatics was 1.39 and 2.72, respectively, after adjusting for relevant confounders such as body mass index (BMI).[9] A key reason for the increased risk of incident OSA in asthmatics may be the cumulative effects of intermittent bursts of OCS used to treat exacerbations. A recent study from the United Kingdom using primary care databases studied >450,000 asthmatics receiving intermittent OCS and found a strong, dose-dependent relationship between OCS use and OSA, which exceeded the risk between OCS use and commonly cited adverse outcomes such as diabetes and osteoporosis.[10] Conversely, OSA has also been identified as a factor contributing to frequent exacerbations in patients with difficult-to-treat asthma,[11] highlighting the intricate interplay between these conditions.

Despite the clear epidemiologic relationship between OSA and asthma in adults, the mechanisms underlying this association are poorly understood. There does however appear to be an effect of OSA on the inflammatory endotype in severe asthma. A study of more than 300 patients in the Severe Asthma Research Program found that probable OSA (based on questionnaire data) was associated with sputum neutrophilia, but not eosinophilia.[12] Similarly, a prospective cohort of 55 severe asthmatics with OSA, confirmed using home

sleep apnea tests, found that those with OSA had a greater proportion of neutrophils and greater concentrations of the non-type 2 inflammatory cytokine interleukin-18 (IL-18), in induced sputum.[13] Interestingly, in a recent placebo-controlled crossover study of severe asthmatics treated with the biologic treatment benralizumab, patients with a persistently high symptom burden despite benralizumab treatment also had elevated levels of IL-18 in their sputum.[14] Together these data raise the question of whether ongoing respiratory symptoms refractory to biologic treatments could be due to unrecognized OSA.

There is limited evidence available regarding the effects of OSA treatment on asthma. In the only randomized placebo-controlled trial to date (n=37), CPAP therapy for three months did not improve asthma control (as measured by the Asthma Control Test score) but did improve daytime sleepiness and asthma-related quality of life.[15] CPAP also does not impact bronchial responsiveness to methacholine or lung function.[16]

Patients with asthma and OSA frequently also have gastroesophageal reflux disease and obesity and these collinear comorbidities must be considered together.[17] Patients with asthma and fixed airflow obstruction may have sleep-disordered breathing which resembles OSA-Chronic Obstructive Pulmonary Disease (COPD) overlap syndrome, and have significant hypoxemia associated with nocturnal hypoventilation. These patients are probably best referred to a respirologist with experience in sleep-disordered breathing.

In summary, allergists are advised to routinely assess patients with asthma for OSA, especially in cases of long-standing asthma, poorly managed symptoms, frequent bursts of OCS, and high-dose ICS therapy. Home sleep apnea tests are usually sufficient provided the lung function is normal or only mildly impaired. CPAP therapy should be considered, and adherence to this treatment stressed, given its capacity to diminish asthma-related morbidity and enhance life quality.

CPAP Rhinitis

CPAP rhinitis occurs primarily due to irritation of the nasal mucosa from the cold, dry air delivered by the CPAP machine. Symptoms typically include nasal congestion, dryness, and irritation, and the nasal mucosa appears dry and erythematous on anterior rhinoscopy. This probably results from damage to the nasal mucosa by cold, dry air under pressure. Nasal symptoms due to CPAP therapy are commonly cited as the reason for treatment non-adherence,[18] and allergists can thus play an important role in symptom control to improve compliance.

Reducing nasal mucosa dryness is the cornerstone of CPAP rhinitis management. Lubricating nasal gel or saline irrigation can relieve nasal discomfort, although petroleum jelly should be avoided due to the theoretical risk of lipoid pneumonia.[19] Adjusting the CPAP humidity settings can alleviate nasal symptoms; however, this does not necessarily improve device compliance. Ensuring a proper mask fit can prevent air leaks, maintain device efficacy, and improve comfort. A common problem is that the water chamber dries out before the end of the night resulting in nasal dryness, and this is usually due to excessive mask leak. Regular mask and filter replacements are recommended in accordance with manufacturers recommendations.[18]

Distinguishing CPAP rhinitis from other forms of rhinitis is vital for proper treatment. Symptoms such as conjunctivitis, frequent sneezing, seasonality, and aeroallergen sensitivity, point toward allergic rhinitis, which may occur alongside CPAP-induced rhinitis. For those with baseline allergic rhinitis, CPAP can exacerbate nasal congestion. Although intranasal corticosteroids usually do not treat CPAP rhinitis,[20] they could help with underlying allergic or vasomotor rhinitis. Patients should be advised to avoid the chronic use of nasal decongestants to prevent rhinitis medicamentosa.

In patients with CPAP rhinitis refractory to medical management, surgical interventions such as nasal septoplasty or turbinoplasty might enhance CPAP adherence. Alternatives to CPAP could also be considered. Oral appliance therapy, using custom-fitted devices, works well for mild-to-moderate OSA.[21] UPPP, suitable for various severities of OSA, involves the surgical modification of the airway to improve airway patency, but is less effective for individuals with a BMI greater than 40 kg/m.[4]

OSA in Chronic Rhinosinusitis

Chronic rhinosinusitis (CRS) is associated with poor sleep quality, snoring, increased AHI,

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and CPAP intolerance.[22] Medical management of CRS with intranasal steroids may improve OSA symptoms and CPAP tolerance by ensuring the comfortable delivery of pressure through the nose. A small case series demonstrated that dupilumab improves OSA symptoms in patients with CRS with polyposis,[23] and further research in this area is warranted given the increasing use of biologic therapies to treat this condition. Flares of CRS due to bacterial infection do not appear to be caused by bacterial colonization of the CPAP reservoir.[24]

When considering referral to otolaryngology for surgical management of CRS, patients should be asked about OSA symptoms, and a home sleep apnea test should be obtained in suspected cases. The presence of significant OSA may tip the balance in favour of surgical intervention for CRS in patients who otherwise would not receive surgery. However, though OSA symptoms may improve with sinus surgery, it is generally not considered curative. In some cases, sinonasal surgery, specifically, nasal septoplasty, turbinoplasty, and polypectomy, may be specifically indicated for OSA treatment.[22]

CPAP mask contact dermatitis

Cases of allergic contact dermatitis caused by CPAP masks have been reported, although it appears to be rare. For example, cases have been reported involving masks containing silicone,[25] neoprene,[26] or dialkyl thioureas,[27] and masks that have been cleaned with benzisothiazolinone.[28] Sensitivity can be confirmed with patch testing. The major commercial manufacturers of CPAP interfaces do not use latex. The presence of an alternative diagnosis should be strongly considered (e.g., periorificial dermatitis in patients receiving ICS). Often, examining the cleanliness and state of repair of the equipment will immediately reveal the cause of the patient's facial irritation.

Conclusion

Atopic diseases are associated with OSA, and allergists may see many patients with symptomatic OSA that interferes with the control of their allergic diseases. Conversely, the allergic conditions themselves, such as asthma, may predispose the patient to OSA. The treatment of OSA may be an important component of the management of asthma, and the presence of OSA may alter the management of CRS. CPAP rhinitis should be considered in patients with rhinitis unresponsive to intranasal steroids. Allergists should be familiar with how patients at risk of OSA are evaluated in their community and initiate appropriate testing or referrals.

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Shared Decision Making in Asthma Treatment: Using Motivational Communication to Elevate Your Consultations

Anda I. Dragomir, PhD
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Introduction

The extent to which patients are adherent to their medication regimen is critical for achieving good asthma control, preventing exacerbations, and optimizing the likelihood that patients can lead full and productive lives.[1] Knowing this, physicians might perceive that their role is to convince their patients to comply with their prescribed medicines using educational or persuasive advice-giving strategies that focus on the health benefits of treatment compliance and the negative health consequences of non-compliance.[2] However, evidence suggests that evoking fear of negative consequences is a poor motivator for both the adoption, and long-term maintenance, of good health behaviours such as medication adherence.[3] In an effort to make the maladaptive behaviour the 'less desirable choice', physicians often inadvertently become associated with the fear messages they share (through classical conditioning) and the negative emotions they elicit (through operant conditioning).[4] This may result in physicians themselves becoming aversive to their patients, leading to patients disengaging from the therapeutic relationship and becoming resistant to treatment recommendations. In fact, treatment success depends not on the imperative to convince and control patient behaviour (which defines compliance), but on the willingness to collaborate with the patient to co-construct a treatment plan that they agree with and want to follow (which defines adherence). The key takeaway is to recognize that non-collaborative communication is ineffective for behaviour change, and may actually be counterproductive, if not harmful.[3]

Asthma is a major global health problem affecting as many as 235 million people worldwide.[5] It is characterized by episodic or persistent respiratory symptoms (e.g. cough, wheezing, shortness of breath, chest tightness) and airflow limitation.[5] The treatment of this chronic disease is centred on symptom reduction (i.e. symptom control to maintain normal function) and reducing the occurrence of adverse events such as exacerbations, fixed airflow limitation, and treatment side effects.[6] In all chronic diseases, but particularly in asthma, treatment adherence is paramount to treatment success because asthma management often involves conducting iterative reassessments, leading to frequent treatment adjustments (i.e. pharmacological, non-pharmacological, and treatment of modifiable risk factors) and reviews of the patient's response. Effective asthma management therefore depends on a cooperative partnership between the physician and the person with asthma.[6] Unfortunately, adherence to asthma medications has been shown to be suboptimal across the globe (i.e., as low as 13.8% in China and only as high as 52% in Brazil), with many patients only taking their controller medication when symptomatic and over-using reliever therapies when symptoms worsen, a pattern shown to increase risk of death from asthma.[1]

For physicians, addressing these challenges with patients means learning how to 'communicate for behaviour change'. To achieve this, it may be useful to become familiar with some basic knowledge about human psychology that recognizes all human behaviour—including ambivalence and resistance to change—as being normal and predictable reactions to change, rather than psychopathology.

Ambivalence toward engaging in good health behaviours (both wanting and not wanting

to engage in a behaviour simultaneously) is a natural and predictable reaction to change. This is because many health behaviours are metabolically costly and require effort that may conflict with our established routines and comfort.[7] Our choices are typically guided by pleasure, convenience, and their immediate consequences (we seek good consequences and avoid bad ones). When these preferences are threatened, individuals commonly resort to avoidance as a coping mechanism.[8] Avoidance can also occur when a physician responds to their patient's ambivalence with negative judgement or persistent advice-giving. This can erode trust and result in feelings of shame and self-blame in the patient, which can lead to disengagement and treatment resistance or refusal.[9]

Communicating effectively for behaviour change also requires some basic knowledge of the major determinants of behaviour change, which include motivation (what drives our behaviour) and self-efficacy (which relates to being confident in our ability to succeed at a task). Human motivation exhibits multiple dimensions that can be defined along a continuum from extrinsic (when behaviour occurs in response to external rewards and punishments) to intrinsic/internal (when behaviour occurs to experience pleasure or satisfaction, or because it is consistent with our identities, goals and values).[10] Research has shown that people are more likely to engage in behaviours that are intrinsically/internally motivating (tied to goals and values) and when they feel confident in their ability to succeed. Gaining an understanding of this information can therefore be used to improve medication adherence by eliciting patients' therapeutic goals (e.g., reducing breathlessness and participating in sports) and determining the necessary support to help them succeed in taking their medication as prescribed (e.g., a device that is easy to use or reminders).

When people feel compelled to engage in behaviours that they did not choose for themselves, two outcomes are possible: a) psychological reactance, characterized by defiance against health recommendations, or b) begrudging acceptance leading to learned helplessness.[11] When setbacks occur and people lack intrinsic/internal motivation or confidence, their problem-solving abilities, which enable them to overcome barriers to success, are also limited.[12] Although people might re-attempt the target behaviour for external reasons (e.g., seeking approval, avoiding feelings of shame), over time, prolonged exposure to repetitive failures will result in a deep and persistent state of helplessness (resignation) and eventually, hopelessness (depression). This also tends to result in refusal of the treatment plan or disengagement from disease self-management.[13]

Finally, there are two distinct areas of the brain controlling behavioural choices: the logical executive system (located in the prefrontal cortex), and the emotional system (located in the limbic system). When these systems compete with each other, often the emotional brain prevails.[14] This, coupled with a need for self-determinism—feeling in control of one's life and choices—explains why people do not tend to follow advice and prefer to follow their own beliefs.[10] Physicians attempting to persuade their patients of the benefits of their pharmacological and behavioural prescriptions when a patient is presenting a conflicting opinion or an emotional reason against change can be risky and counterproductive because this approach challenges patients' beliefs, desires, and the comfort and predictability of the status quo.[15] Ultimately, this will also tend to result in treatment disengagement or refusal.

Understanding these basic behavioural concepts allows healthcare providers to recognize how, despite our best intentions and substantial expertise, we may be inadvertently contributing to our patients' resistance and poor adherence to treatment. We may have unintentionally restricted our patients' autonomy by imposing our own motivation for change (e.g., controlling their asthma symptoms) instead of eliciting their own motivation (e.g., being able to keep up with their family on their holidays). For example, we might have unwittingly contributed to our patient's abandoning their exercise routine because they did not receive the necessary support to strengthen their self-efficacy and overcome their setbacks. Our patients might have avoided sharing their obstacles owing to the fear of being judged, because we did not convey acceptance of their ambivalence. We may have unintentionally dismissed a patient's belief, without realizing that we were limiting their sense of agency.

In order to address these important clinical behavioural challenges, we co-developed, along with behaviour change experts and chronic disease physicians, a training program in motivational communication. This program was designed to better support physicians in their efforts to motivate and guide patients in their self-management efforts.[16] Motivational Communication (MC) is defined as an evidence-based, time-efficient

Figure 1. The 11 core competencies of Motivational Communication; *courtesy of Anda I. Dragomir, PhD and Kim L. Lavoie, PhD*

communication style used by health care providers to promote sustained patient engagement and support self-management of chronic conditions.[17] MC is a communication style that was designed to be seamlessly integrated into a typical clinical consultation, without requiring additional time.[18] MC is time saving because it focuses on addressing the actual barriers to change as stated by the patient. Persuasive, education-driven interventions tend to assume patients are not changing because they do not know a behaviour is important. This leads to physicians providing potentially time-consuming and unnecessary information at the expense of spending that time strengthening motivation or self-efficacy. The goal is to be more strategic about what information is provided while emphasizing patient-physician collaboration in the spirit of non-judgemental acceptance, tolerance, and respect that avoids argumentation. MC represents a subtle departure from other motivational approaches (e.g., motivational interviewing), which tend to discourage using other evidence-based behaviour change techniques such as goal setting and positive reinforcement. It also incorporates elements from cognitive-behavioural theories such as identifying value-based determinants for change.[19]

How does MC work? MC presents 11 core communication competencies that can be easily integrated into any clinical consultation (**Figure 1**). While mastering all 11 competencies can be a long-term goal, adopting one or two competencies may be more attainable in the short term. Research has shown that doing so is associated with significant increases in patient motivation and confidence, health behaviour change, and improvements in the therapeutic relationship.[18] For example, adopting this approach can include choosing to use more open questions to explore ambivalence and elicit change-talk, or employing reflections to communicate acceptance and avoid argumentation and impatience. It can also involve simply working more collaboratively with patients to understand their treatment goals and preferences.[20] See **Table 1** for an example which compares MC to a more traditional persuasive consultation style. If you are considering using MC in your practice, or if you wish to obtain additional training, contact the Montreal Behavioural Medicine Centre (**https://mbmc-cmcm.ca/**).

Conclusion

In conclusion, the imperative for effective asthma management extends beyond mere prescription adherence; it necessitates a profound shift in the patient-physician interaction. By prioritizing shared decision-making, the principles of MC can enable physicians to bridge the gap between treatment recommendations and patient engagement. This approach not only acknowledges the complexities of human behaviour but also respects the autonomy and intrinsic motivations of individuals living with asthma.

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Notes	Traditional Approach	Motivational Communication Approach	Notes
Agenda set independently Closed question explores adherence	**Physician:** Let's have a chat about your medication use. Have you been using your daily inhaler as prescribed?	**Physician:** Would it be ok to talk about your medication? Tell me, what medications are you taking and how often do you take them?	Asking permission to set the agenda, open question to explore adherence
[Patient provides 'correct' answer to closed question, but it may not be valid]	***Patient:*** *Yeah.*	***Patient:*** *Sure. I use my controller inhaler twice a day, and the rescue inhaler as needed. But I noticed I have been using the as-needed inhaler more often than usual, which worries me.*	[Patient provides a more elaborate answer; expresses a concern]
Argumentation, impatience	**Physician:** Ok, then I don't understand why you are having so many symptoms. If you are using your controller medication as prescribed, your asthma should be well controlled on this dose of medication.	**Physician:** It's great that you seem to be using your inhalers as prescribed, but your rescue inhaler use concerns you, which is understandable. What do you think might be causing you to use your rescue inhaler more?	Reflects on good behaviour and validating concerns Open question to explore rescue inhaler use
[Patient gets defensive and expresses some resistance]	***Patient:*** *Well, I don't understand it either, or why I came to see you. I am also using my rescue inhaler several times a week, and it's not really helping. I am not sure what's going on.*	***Patient:*** *Well, I have to admit that I don't like the idea of taking steroids every day because of the long-term side effects, so sometimes I skip doses of my controller medication, especially when I feel well. Maybe it's finally catching up with me...*	[Patient admits they are not always adherent due to concerns about side effects]
Argumentation, impatience, and unsolicited advice-giving	**Physician:** As you know, taking too much rescue medication is not good for you, and can lead to a phenomenon called "rebound effect," where your symptoms worsen over time and your asthma becomes harder to control. But if you use your daily controller as prescribed, you should not need to use the rescue inhaler so much.	**Physician:** Many of my patients have expressed the same concerns. One of the challenges associated with treating asthma is that sometimes, the medications are so effective at treating the underlying inflammation, some people think the medication is no longer needed. What do you think about that?	Validates and normalizes feelings; Gives information neutrally without giving advice; Asks open question to elicit feedback
[Patient expresses some ambivalence – she takes the medication, but has concerns]	***Patient:*** *I know, and I am taking it, but I really don't like the idea of taking steroids because of all the side effects, so sometimes I skip doses when I feel well.*	***Patient:*** *I understand that using my controller medication regularly is what helps reduce my attacks, which is important. However, I was feeling so much better for once, so I thought I could ease back a bit thinking I would be ok.*	
Lack of acceptance, negatively judging, fear-based messaging	**Physician:** Feeling better doesn't mean your asthma has disappeared. You are probably feeling better because you are using your controller medication as prescribed. Skipping doses when you feel well can lead to attacks, which can require more intensive treatment and even hospitalization. This is probably why you are feeling so breathless.	**Physician:** I understand. It's natural to want to avoid taking more medication than we need.	Validation, acceptance, and empathy through reflective listening

Notes	Traditional Approach	Motivational Communication Approach	Notes
	Patient: *I thought that I could ease back a bit if I was doing well. But now my symptoms seem really out of control, and my rescue medication is no longer working.*	***Patient:*** *Exactly! I was feeling normal for once, and I thought it would be good to take less steroids.*	[Patient agrees with physician, does not express any resistance]
Hostility and impatience, fear-based messaging	**Physician:** That's probably because there is more underlying inflammation. Therefore, we need to get you back on track with taking your daily controller as prescribed, and I will have to increase the dose in order to get your symptoms under control, which unfortunately may cause some additional side effects.	**Physician:** Tell me, what would it mean for you if your symptoms were well controlled again, and you could feel good everyday?	Open question to explore motivation
	Patient: *I'm really not happy about the idea of taking a higher dose, but I am really not doing well, so I guess I have no choice.*	***Patient:*** *It would mean a lot! I felt really good for about a month. I slept well and could keep up with my friends during workouts. I wasn't out of breath walking up the hill to the office everyday. It was great.*	[Patient describes advantages of good control]
Hostility and impatience, fear-based messaging	**Physician:** I really think this is best. If you want to avoid serious complications, you need to take your medication consistently. Here is your prescription, and we can follow up in a few months.	**Physician:** Sounds like taking your daily controller medication really helped manage your symptoms and allowed you to feel more in control of your life.	Validation through reflective listening
	Patient: *Ok, thanks.*	***Patient:*** *Yeah, it did. I guess I was so excited about feeling good that I forgot how I got there. It's a good reminder to go back to using my controller every day as prescribed.*	[Patient agrees that using their daily controller helped them achieve good control]

Table 1. Comparing Motivational Communication to traditional advice-giving consultation style; *courtesy of Anda I. Dragomir, PhD and Kim L. Lavoie, PhD*

Case: A 45-year-old female patient with moderate asthma, usually well controlled, reports experiencing increased breathlessness, increased use of her rescue inhaler, and greater difficulties performing daily activities.

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Most serious warnings and precautions

Serious infections: patients may be at increased risk for developing serious bacterial, fungal, viral and opportunistic infections that may lead to hospitalization or death; more frequently reported serious infections were predominately viral. If a serious infection develops, interrupt treatment until the infection is controlled. Risks and benefits of treatment should be carefully considered prior to initiating therapy in patients with chronic or recurrent infection. Monitor for signs and symptoms of infection during and after treatment, including the possible development of tuberculosis in patients who tested negative for latent tuberculosis infection prior to initiating therapy.

Malignancies: lymphoma and other malignancies were observed in patients taking JAK inhibitors to treat inflammatory conditions and were more frequently observed in patients with rheumatoid arthritis (RA) during a clinical trial with another JAK inhibitor versus TNF inhibitors.

Thrombosis: including deep venous thrombosis, pulmonary embolism, and arterial thrombosis have occurred in patients taking JAK inhibitors to treat inflammatory conditions. Many of these events were serious; some resulted in death. Consider risks and benefits prior to treating patients who may be at increased risk. In a clinical trial in patients ≥50 years of age with RA, a higher rate of all-cause mortality and thrombosis occurred in patients treated with another JAK inhibitor versus TNF inhibitors. Patients with symptoms of thrombosis should be promptly evaluated and treated appropriately.

Major adverse cardiovascular events (MACE): including non-fatal myocardial infarction, were observed more frequently in patients ≥50 years of age with RA during a clinical trial comparing another JAK inhibitor versus TNF inhibitors.

Other relevant warnings and precautions

- Driving or operating machinery
- Dose-dependent increase in blood lipid parameters, lipid monitoring and management
- Hematological abnormalities
- Use with potent immunosuppressants
- Vaccination
- Monitoring and laboratory tests
- Fertility
- Women of childbearing potential
- Pregnancy and breastfeeding
- Geriatrics

For more information

Consult the Product Monograph at http://pfizer.ca/pm/en/CIBINQO.pdf for important information regarding adverse reactions, drug interactions and dosing information, which have not been discussed in this piece. The Product Monograph is also available by calling 1-800-463-6001.

AD=atopic dermatitis; JAK1=Janus kinase 1.
* Clinical significance unknown.

Reference: CIBINQO Product Monograph, Pfizer Canada ULC.

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