The nose, and obstructive sleep apnoea

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INTRODUCTION

Approximately 10% of Australians have obstructive sleep apnoea (OSA) and the incidence is increasing. OSA is more common in men with an overall prevalence of 25% and 9% in women. In men aged 40-69 years, however, the prevalence is 49%, increasing to 62% in men over 70 years of age. Despite these figures, many of those affected are undiagnosed. OSA is associated with a significant economic burden with lost productivity, increased risk of workplace and motor vehicle accidents, and increased healthcare utilisation.

Symptoms of OSA include daytime somnolence, nocturnal gasping or choking, and nocturia. OSA also negatively impacts mood, and concentration and is associated with a higher incidence of cardiovascular disease, diabetes, impotence, renal disease and chronic obstructive pulmonary disease. In fact, patients with OSA have a 7-fold higher risk of death and heart disease. People with OSA have a poorer quality of life (QOL). OSA is also considered a shared problem as it negatively impacts relationships and the sleep quality of partners. Successful OSA treatment can eliminate or reduce these risks factors and absenteeism and can increase productivity.

OSA is diagnosed via a polysomnogram (PSG). Severity of OSA is graded using scores such as the apnoea hypopnoea index (AHI), respiratory disturbance index (RDI) and the oxygen desaturation index (ODI).

THE IMPORTANCE OF THE NOSE AND AIRWAY RESISTANCE

Nasal airflow is up to 20-30l/min in a normal nose. Nasal airflow simulates breathing and improves upper airway muscle tone via the nasal-ventilatory reflex, particularly during sleep. Interestingly, this effect is abolished when the nose is anaesthetised. The nasal airway accounts for more than 50% of total airway resistance yet, nasal breathing is the preferred air entry route during sleep. In fact, the nasal fraction of breathing in sleep is 95.7%. Further, the AHI and upper airway resistance are lower with nasal breathing than with mouth breathing. Nasal resistance increases in the supine position due to increased venous pooling in the turbinates.

NASAL OBSTRUCTION AND OSA

Nasal obstruction is common in patients with OSA occurring in up to 65% which is much higher than in non-OSA patients. Nasal obstruction increases RDI and snoring duration in those with OSA. This can be explained using a Starling resistor model. Essentially, restricted nasal airflow generates a negative intraluminal pressure downstream in the collapsible segment of the upper airway that lacks rigid support (oropharynx) which causes collapse and an apnoea (Figure 1). In adults,

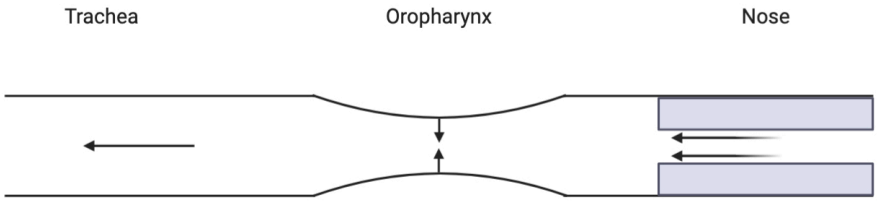

Figure 1: Starling resister model of upper airway collapsibility. Obstruction at the narrow nasal inlet (fixed segment) generates collapsing forces in the downstream collapsible segment of the airway (oropharynx).

relief of nasal obstruction will rarely cure OSA, as other factors such as body mass index and the anatomy of the oropharynx, tongue, hypopharynx and jaw also play a significant role. However, nasal obstruction may contribute to, or exacerbate, OSA and does play a role in snoring, daytime somnolence, AHI, RDI and ODI. Nasal obstruction can also reduce compliance with CPAP masks and oral appliance therapy.

Nasal obstruction can be due to multiple factors. These may be structural, such as a septal deviation, or dynamic, such as nasal valve collapse or turbinate hypertrophy (Figure 2). Oedematous or inflammatory conditions of the nasal mucosa may also contribute to nasal obstruction such as allergic rhinitis or nasal polyposis.

TREATMENT OF NASAL OBSTRUCTION IN PATIENTS WITH OSA

The goal of treatment of OSA is to improve QOL and to reduce risk factors.

Gold standard treatment of OSA is often forms of positive airway pressure including continuous positive airway pressure (CPAP). CPAP has an efficacy rate of approximately 95%, however compliance rates are <40%. Mostly, this is due to discomfort. There are many factors that determine CPAP compliance, some of which are modifiable, such as nasal obstruction.

Treatment of the nasal obstruction will vary however, generally, treatment can be divided into medical and surgical options.

Medical treatment may involve the use of topical intranasal steroid sprays (INSS) or nasal valve dilators. INSS have been shown to reduce the AHI and improve oxygen saturation levels in OSA patients with rhinitis. Nasal dilators have had equivocal results when used in OSA patients.

Surgical treatment for nasal obstruction in OSA may include septal surgery, inferior turbinate reduction, nasal valve surgery and/or endoscopic sinus surgery. Nasal surgery has been shown to improve AHI and Epworth Sleepiness Scores, symptoms of daytime somnolence and sleep quality. Nasal surgery can also reduce required CPAP pressures which can improve tolerance and compliance.

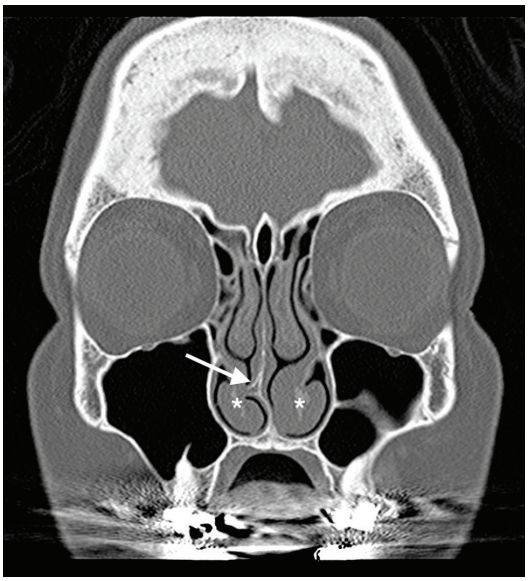

Figure 2: Coronal CT scan of the nasal cavity demonstrating a deviated nasal septum to the right (arrow) and hypertrophied inferior turbinates (asterisks).

CONCLUSION

OSA is increasingly common and negatively impacts the economy and QOL. OSA is associated with significant morbidity, yet effective treatment options exist. OSA is best managed in a multidisciplinary fashion. Nasal obstruction is common in these patients and negatively impacts sleep quality, OSA severity and treatment compliance. Many treatment options exist for nasal obstruction, however, it is imperative that patients understand that relief of nasal obstruction is rarely a cure for OSA but part of a holistic approach to management.

References available on request.