Pathophysiology of OSA

The human upper airway has evolved as a multipurpose structure and allows for speech, swallowing and breathing. The upper airway is made up of muscles and soft tissue but does not have any hard bony tissue that would prevent muscle or soft tissue from collapsing or compressing.

During wakefulness, the upper airway has the ability to close momentarily while changing shape, to allow for speech and swallowing. The collapsible part of the upper airway spans the hard palate to the larynx.

It has been shown in many studies that the airway lumen is smaller in patients with OSA than normal adults. There are several pathophysiological factors that make a person susceptible to developing a sleep-disordered breathing condition, such as OSA.

The collapse of the upper airway during sleep occurs as a result of the loss of compensatory tonic input to the motor neurones of the upper airway dilator muscles, predominantly the genioglossus and the tensor palati muscles.8

The ability of a person to compensate for these collapses will determine how often the cycle repeats itself, hence the severity of the disease. Several neurotransmitters and neuromodulators have now been identified as contributing to the regulation of the upper airway patency, but not much progress has been made in pharmacological terms to counteract this collapse.

It is important to differentiate between the various apnoea types: obstructive and central apnoea. While the former is a result of the mechanical collapse of the upper airway, the latter is a result of either reduction or cessation of brainstem activity regulating the respiratory muscles and the failure to send a message to the respiratory muscles to breathe. Each type of sleep apnoea is managed differently.1

As it stands, those at risk are categorised.2,3,4,5 The underlying mechanism that affects the categories is listed in Table 1 below.

Factors contributing to the pathophysiology of obstructive sleep apnoea (OSA)

- neck circumference
- high blood pressure (hypertension) and diabetes
- airway size and shape
- chronic nasal congestion and anatomical obstruction
- gender: males twice at risk as females
- ethnicity: higher risk in Asian and Afro-Caribbeans
- age: increased risk as one gets older
- genetics
- smoking
- alcohol


Table 1: Risk categories
Anatomical factors predisposing to airway collapse

Site of collapse of the airway
The pharyngeal airway comprises hard tissues, such as the hard palate, the maxilla and mandible, the nasal turbinates and the hyoid bone anteriorly and the cervical vertebrae posteriorly. The pharynx is divided into four sections:

- nasopharynx – turbinates to the superior part of the soft palate
- retropalatal pharynx – hard palate to the inferior margin of the soft palate
- oropharynx is subdivided into retroglossal pharynx, which extends from the soft palate to the epiglottis and the hypopharynx from the epiglottis to the larynx
- area between the retropalatal and retroglossal pharynx is also called the velum

See Diagram 1.

Bony structures that predispose to OSA
There is a bony cage made up of hard tissues such as the mandible, maxilla, hard palate and hyoid bone and any structural abnormalities of these displaces the soft structures, such as the tongue, to precipitate airway obstruction. Extreme examples are those born with Treacher Collins syndrome or Pierre Robin or any such congenital craniofacial malformation described earlier is typical of an OSA subject. Any inflammation of the airway soft tissues may contribute to airway obstruction. Some faculties believe that increased fat pads predispose to medial airway compression but there are others who believe there to be no significant effect. Rostral fluid can lead to increased pressure in the blood vessels in the neck and can lead to collapse such as seen in patients with cor pulmonale, renal or heart failure.

Table 2 (overleaf) demonstrates the several risk factors that predispose a subject to developing OSA, but I will concentrate on those that are relevant to the dental professional. The reader is directed to relevant literature in the reference section for further reading.

Soft tissues
A disproportionate volume of soft tissue compared to the hard tissue cage, but it does not take much to contribute to such a collapse.

Bony protuberances along the cervical vertebrae can lead to airway obstruction from the back of the airway (see Diagram 2).

The hyoid bone is a suspended bone detached from the rest of the cervical spine. It has been shown that the more inferior the hyoid bone, the more likely the tongue is to be displaced inferiorly in the hypopharyngeal area, hence an increased risk of developing OSA.

The most important and common skeletal abnormality that predisposes a subject to develop OSA is a short mandibular length. Cephalometry has shown that short airway length may be a significant cause of developing OSA.

General factors
Gender and race
It is well documented that OSA is present in twice as many men as women, with statistics showing 4% of men and 2% of women having OSA (the figures...
representing just 20% of those who have the disease but remain undiagnosed).

It is not certain why men are twice as likely to develop OSA than women but it is thought that men are more susceptible to airway collapse due to a higher degree of pharyngeal resistance. It may also be the effect of testosterone and the higher incidence of obesity in men than women, although this fact may be subject to discussion as female obesity rises. The statistics alter as women reach ageing leads to reduced muscle tone, hence a higher incidence of airway collapse.

**Obesity**
Weight is significant in the development of OSA. There is a defined association between obesity and pharyngeal resistance leading to OSA. Management of OSA in obese patients with weight loss programmes is beneficial and a proven method. The association between obesity and OSA may be the increase in fat deposits in the neck with increased extraluminal pressure and airway narrowing. MRI studies have shown increased deposits of fat in the collapsible part of the airway as well as the tongue base that may explain airway obstruction in these patients in the oropharyngeal area. As the fat pads influence the airway muscles directly, it is the deposition around the neck that is more critical than all over obesity, hence the relevance of neck circumference.

**Pharyngeal muscles**
Since OSA occurs during sleep only, neuromuscular factors must play a part in the development of the disease, in addition to anatomic factors mentioned above. Such factors include UA muscle tone, sensation and function.

**Other potential mechanisms for UA collapse**
Surface tension: the stability of the UA may be influenced by surface adhesive forces and fluid elasticity. Sensory changes
The pharyngeal muscles are sensitive to local stimuli and, in particular, negative intrapharyngeal pressure, which causes activation of these muscles. An inability to detect mechanical stimuli, such as increased UAR, may reduce dilator muscle activity, leading to UA collapse. Chronic trauma to UA structures by low-frequency vibration due to snoring may also play a role.

**Conclusion**
Obstructive sleep apnoea and snoring is a multifactorial disease and the underlying mechanism is complicated. A patient may have more than one coexisting factor precipitating the apnoea episodes and this should be borne in mind when managing their care.

[References]