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## Snoring: Simple to Obstructive Apnea

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## INTRODUCTION

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Sleep is a basic fundamental process that humans appear to need. Yet sleep is a mechanism about which not all is understood. Average humans spend between six and eight hours per day, or about one-third of their lifetime, sleeping. Sleep is a transient state of altered consciousness with perceptual disengagement from one's environment. Contrary to popular belief, sleep is an active process involving complex interactions between cortical, brain stem diencephalic, and forebrain structures (1). There is still significant metabolism and oxygen consumption during this state of "rest," and any disruption of oxygenation or interruption of this physiological process can lead to both night and day manifestations like snoring, choking sensations, apneic episodes, or daytime somnolence.

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Snoring, the lay term for noisy breathing during sleep, has historically been believed to be just a nocturnal "nuisance" and an obnoxious human habits. In a 30–35-year old population, 20% of men and 5% of women will snore. By age 60, 60% of men and 40% of women will snore habitually (2). Epidemiological studies have shown that for adults over 65 years old, up to 50% have some form of sleep disruption and poor sleep quality (3). Snoring may be a simple nuisance to the patient or sleep partner when not accompanied by other symptoms or complaints. However, it may be part of a symptom complex indicating sleep-disordered breathing (SDB).

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The presence of snoring is a loud "alarm" that alerts one to the possibility of a sleep disorder. Sleep-disordered breathing is a spectrum of diseases related to decreased airflow through the upper airway during sleep, due either to complete or partial upper airway obstruction or increased upper airway resistance. These encompass simple snorers (patients who snore without excessive daytime somnolence and with a normal apnea–hypopnea index), upper airway resistance syndrome (patients with excessive daytime somnolence but who have a normal apnea–hypopnea

1 index), and obstructive sleep apnea (OSA) (patients who snore with both excessive  
2 daytime somnolence and an abnormal apnea-hypopnea index). Overall, these sleep  
3 disorders result in poor sleep quality, fragmented sleep, intermittent nighttime  
4 hypoxemia, reduced percentage of slow wave sleep, and increased sympathetic over-  
5 drive. The results are daytime somnolence, morning headaches, poor concentration,  
6 loss of memory, frustration, depression, and even marital discord.  
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## 8 9 **EPIDEMIOLOGY**

10 Sleep-disordered breathing is more common and pronounced in older adults than  
11 younger adults (4,5). In the largest study of a representative sample of older adults  
12 (65–95 years of age), 62% had an RDI >10, and 24% had an AI >5 (6). In this same  
13 study, SDB was more common in men than in women and in patients with hyperten-  
14 sion than in those without hypertension. In a study of older African Americans  
15 and Caucasians, the prevalence was equivalent, but apnea was more severe in the  
16 African-American group (7). Foley et al. (3) have also shown that up to 50% of  
17 adults over 65 years of age complain of some sort of sleep disruption. Sleep disorders  
18 in the elderly are known to be caused by multiple factors, and many of these sleep  
19 disturbances may be secondary to medical and psychiatric conditions (8,9). In a  
20 study of 1050 individuals with a mean of 74.4 years, 36.7% reported difficulty falling  
21 asleep, 28.7% had sleep continuity disturbance, 19.1% had early morning awakening,  
22 and 18.9% reported uncontrollable daytime somnolence (10).  
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## 24 25 **PATHOPHYSIOLOGY**

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27 The fundamental abnormality in sleep-disordered breathing is in the anatomy and  
28 collapsibility of the upper airway. Snoring is caused by a vibration of the structures  
29 of the oral cavity/oropharynx: the soft palate, uvula, tonsils, base of tongue,  
30 epiglottis, and pharyngeal walls. Partial or complete upper airway obstruction during  
31 sleep can lead to excessive soft tissue or abnormal facial skeletal framework. Patients  
32 with adenotonsillar hypertrophy have a crowded upper airway with very little space  
33 for airflow, while obese patients frequently have soft palate redundancy. It is the  
34 vibration of these soft tissues during sleep that results in snoring, when the bulk of  
35 this soft tissue exceeds a certain amount. It leads to collapse, partial or complete,  
36 of these structures, which then leads to upper airway obstruction during sleep.  
37 Patients with retrognathia will have less space available, therefore increasing the like-  
38 lihood of airway compromise during sleep. There are some authors who believe that  
39 SDB is entirely based on the equilibrium between forces that hold the airway open  
40 and forces that tend to collapse the airway (11). The magnitude of the pressures  
41 collapsing the airway can be measured, and this measurement can be inferred from  
42 the pressure required by continuous positive airway pressure to hold the airway open.  
43 This is known as pharyngeal critical pressure ( $P_{crit}$ ) (12).  
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45 The soft tissues in the upper airway can be divided into the adipose tissues, muscle  
46 groups, and lymphoid tissues. The presence of adipose tissues surrounding the airway  
47 plays a significant role in SDB. There are fat deposits present under the mucosal  
48 membranes as well as surrounding the various muscles in the neck. Adipose tissue is  
49 present in the palate, tonsillar fossa, and even in the pharyngeal walls. Oropharyngeal  
50 fat deposition reduces airway caliber, thereby worsening upper airway obstruction.

The muscle groups can be divided into those that are vertical and those that are  
horizontal. The vertical group includes the palatopharyngeus, salpingopharyngeus,

1 glossopharyngeus, and the levator veli palatini. The constrictor muscles and the ten-  
2 sor veli palatini comprise the horizontal group. The tongue plays a crucial role in the  
3 upper airway, not only due to its central location in the oral cavity and oropharynx  
4 but also because of its bony attachment. The muscle fibers of the tongue converge  
5 and are primarily attached to the posterior surface of the mandible (genial tubercle)  
6 in the midline. When the mandible is displaced posteriorly (e.g., retrognathia), air-  
7 way compromise may occur at the base of the tongue. This anatomical attachment  
8 of the tongue forms the basis for the genioglossus advancement procedure, which is  
9 done through a mandibulotomy window.

10 The lymphoid tissues are probably the most amenable to treatment. When  
11 there is obvious adenotonsillar hypertrophy, removing the lymphoid tissues may  
12 be curative, especially in children (13).

13 Although mechanically the airway may be visually conceptualized as a simple  
14 conduit, it is very dynamic, and airflow is affected by three main variables. Bernoulli's  
15 principle states that negative pressure develops at the periphery of fluid flow, and as the  
16 flow velocity increases, so does the negative pressure. Therefore, a narrow airway tends  
17 to remain narrow. Fluid velocity increases as a given volume moves through a conduit  
18 of decreasing size (Venturi effect). If the conduit diameter is reduced, then the velocity  
19 and pressure of the fluid or air increases. The final factor is the variable resistor concept,  
20 which states that resistance to flow increases with increasing flow. The net effect is that  
21 upper airway narrowing perpetuates its own narrowing and the airflow velocity and  
22 resistance to flow tend to increase correspondingly.

23 As with most other tissues in the human body, gravity affects the soft tissues in  
24 the upper airway. Lying supine during sleep will cause the soft tissues of the orophar-  
25 ynx to collapse and fall posteriorly, obstructing the airway.

26 Nasal pathologies have been shown to aggravate and contribute to the severity  
27 of SDB, and treatment of the underlying nasal pathology may help, but usually not  
28 cure, the disorder. Controlling and relieving nasal obstruction will improve compliance  
29 with the use of nasal continuous positive airway pressure. Careful nasal examination is  
30 useful to allergic rhinitis, nasal polyposis, or a severely deviated nasal septum.

31 There is a male predominance in OSA, which in most reports is 2:1. This may  
32 be partly a result of hormonal differences since postmenopausal women tend to  
33 develop symptoms on average 5 years after menopause, with a prevalence of OSA  
34 approaching that of males. There are some reports that progesterone will reduce  
35 snoring and sleep apnea in males, while exogenous testosterone can increase upper  
36 airway resistance in females (14,15).

## 37 38 39 **DIFFERENTIAL DIAGNOSIS**

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41 Most patients with SDB complain of snoring, which may be heroic. Frequently, the  
42 sleep partner prompts the patient to see a physician because of concerns over repeated  
43 apneas. Patients may complain of frequent awakenings with a choking sensation,  
44 nocturia, or nightmares. Patients with severe SDB may be unable to sleep supine.  
45 Common patient complaints include early morning tiredness and morning headaches  
46 (attributable to the repetitive nocturnal oxygen desaturations). Morning dry mouth  
47 and throat are caused by mouth breathing and snoring. Other symptoms include  
48 forgetfulness, depression, irritability and, less commonly, impotence.

49 Excessive daytime sleepiness is very common in patients with SDB and is  
50 caused by a combination of frequent arousals, sleep fragmentation, repetitive oxygen  
desaturations, and reductions in delta and rapid eye movement sleep (16,17).

1 A thorough sleep history should be obtained to exclude sleep deprivation as a  
2 cause of excessive daytime somnolence (EDS). Daytime somnolence refers to a per-  
3 son's propensity to fall asleep in various situations. The international classification  
4 of sleep disorders defines sleepiness in subjective terms (18):

5 *Mild sleepiness:* Sleep episodes are present only while resting or when little  
6 attention is needed. There is only mild impairment of social function.

7 *Moderate sleepiness:* Sleep episodes are present daily and occur during very  
8 mild physical activities or at times that require a moderate degree of attention. There  
9 is moderate impairment of work or social function.

10 *Severe sleepiness:* Sleep episodes are present daily or during times requiring  
11 mild to moderate attention. There is marked impairment of social or work function.

12 Most clinicians use standardized scales such as the Epworth Sleepiness Scale, in  
13 which the respondent rates the likelihood of sleeping in each of eight situations, with  
14 a maximum score of 24 (19). A score of >10 suggests EDS.

## 16 CLINICAL EXAMINATION

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18 Clinical evaluation can be divided into general medical, systemic (cardiorespira-  
19 tory and neurological), and oral examination with an upper airway assessment. All  
20 patients should have weight and height, body mass index, blood pressure, and neck  
21 circumference recorded (at the level of the hyoid bone).

22 Oral anatomy in the elderly differs from that in younger adults. Brown et al.  
23 (20), using the acoustic reflection technique, reported that the pharyngeal area in  
24 males decreases with age. Other authors have reported that the distance from the  
25 hyoid bone to the mandibular plane (MP-H) is longer in the geriatric age group,  
26 likely due to increased upper airway fat deposition, resulting in increased tendency  
27 for collapse (21). Stanffer et al. (22) showed that older subjects with a significantly  
28 high AHI have smaller upper airway areas and increased resistance compared with  
29 controls. Tonsil size in the geriatric age group is usually not abnormal but is still  
30 graded on a five-point scale (0 = absent, 1+ = small within the tonsillar fossa,  
31 2+ = extends beyond the tonsillar pillar, 3+ = enlarged tonsils but not touching  
32 the midline, 4+ = enlarged tonsils touching the midline). The Mallampati classifica-  
33 tion may also be used to assess upper airway configuration (Fig. 1).

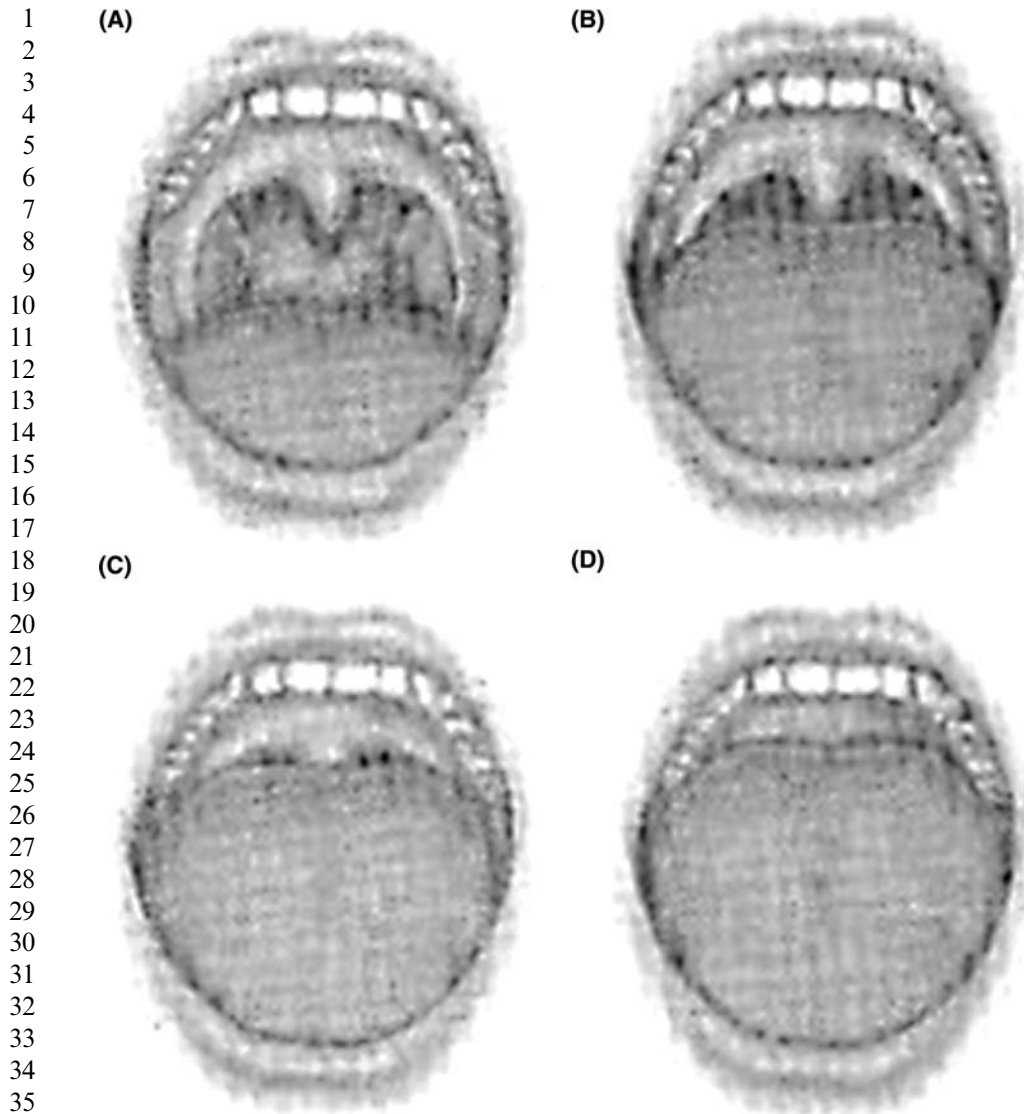
34 No single classification of pharyngeal and upper airway anatomy for patients  
35 with SDB is widely accepted. In 1987, Ikematsu (23) first described the pharyngeal  
36 airway anatomy of heavy habitual snorers. He used six features to classify oropharyn-  
37 geal anatomy: (a) soft palate length ( $\pm 50$  mm), (b) uvula length ( $\pm 11$  mm),  
38 (c) uvula width ( $\pm 10$  mm), (d) pillar arch morphology (parallel, webbed, embedded,  
39 emerging), (e) oropharyngeal narrowing (anterior arch  $\pm 20$  mm, posterior arch  
40  $\pm 15$  mm, shallow oropharynx  $\pm 5$  mm), and (f) enlarged tongue dorsum (oropharynx  
41 not seen with phonation). In 1990, Fujita (24) modified the classification. This  
42 classification is probably the most commonly used by clinicians:

43 *Type I:* soft palate (velopharyngeal) obstruction

44 *Type II:* both soft palate and hypopharyngeal (base of tongue) obstruction

45 *Type III:* hypopharyngeal (base of tongue) obstruction

46 Complementary to Fujita's classification is a dynamic assessment of the upper  
47 airway called the Müller maneuver. It consists of a nasopharyngoendoscopic exami-  
48 nation of the upper airway during which the patient is performing a reverse Valsava.  
49 The site of obstruction is noted, with particular attention to three distinct areas of  
50 potential collapse: retropalatal, lateral pharyngeal walls (lateral to medial), and  
retrolingual. The collapse is quantified on a five-point scale as follows:



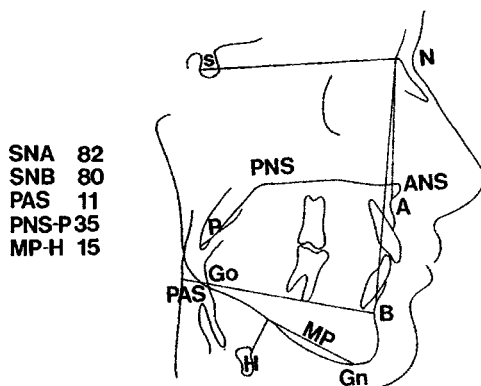
37 **Figure 1** The Friedman classification, which is drawn from the Mallampati classification, is  
38 a general way of describing the upper airway configuration.

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40 0: no collapse  
41 1+: approximately 25% collapse  
42 2+: approximately 50% collapse  
43 3+: approximately 75% collapse  
44 4+: complete collapse, obliterating the airway

45 This dynamic clinical assessment forms the basis for determining the appropri-  
46 ate level for surgical correction.

#### 47 48 **LATERAL CEPHALOMETRY**

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50 Many sleep physicians support the use of lateral cephalograms to estimate the degree of posterior airway space (PAS) narrowing (Fig. 2). Changes in the SNB and SNA angles



**Figure 2** A number of measurements may be identified on cephalometric analysis; the PAS is particularly important in predicting the need for tongue base surgery. *Abbreviation:* PAS, posterior airway space.

15 are correlated with postoperative changes in AHI. Other studies have also shown that  
16 increasing PAS values by a minimum of 10 mm have significantly better surgical outcomes.

## 18 DIAGNOSIS OF SDB

20 The gold standard for differentiating simple snoring from OSA is an attended  
21 polysomnogram. It is appropriate to obtain a sleep study for any snoring patient  
22 with a history of excessive daytime somnolence, with or without physical findings  
23 suggestive of SDB, especially in patients with cardiovascular comorbidities.

24 Severity of OSA can be classified according to the AHI and lowest oxygen  
25 saturation level. The severity is usually graded as the worst of the two (Table 1).

26 Powell and Riley (25) suggest that any patient with an AHI  $\geq 20$  warrants some  
27 form of intervention and treatment. Patients with an AHI  $< 20$  with EDS, associated  
28 desaturation  $< 90\%$ , arrhythmias associated with obstructive events, or complica-  
29 tions of SDB also require treatment.

## 32 SNORING TREATMENT

33 Snoring is considered a social disease with no medical significance. Consequently, it is  
34 not covered by insurance, and treatment is available but must be paid for out of pocket.  
35 This has led to a number of treatment modalities, the efficacies of which are pretty  
36 similar but differ mainly in pain outcomes and costs. The majority of benign snoring  
37 is secondary to palatal flutter. Thus, treatment is directed at the soft palate. Scarring  
38 of the palate has been the objective of all treatments. The theory is that the more collagen  
39 deposited into the soft tissues, the stiffer they are and the less likely to flutter. In patients  
40 with excessive soft tissues, resection of redundant tissue has also been advocated.

**Table 1** Different severity grades of OSA

	AHI	LSAT (%)
Mild	5–19	86–90
Moderate	20–39	70–85
Severe	$> 40$	$< 70$

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*Abbreviations:* AHI, apnea–hypopnea index; LSAT, lowest oxygen saturation level; OSA, obstructive sleep apnea.

### Laser-Assisted Uvulopalatopharyngoplasty

This is perhaps the first treatment that was advocated for treatment of benign snoring. In this procedure, a CO<sub>2</sub> laser is used to vaporize the attachment of the uvula to the soft palate. Furrows are made in the soft palate itself. The resultant healing causes a decrease in tissue due to the uvulectomy and stiffening of the soft palate due to the laser-induced thermal effect. Success rates with this procedure have been reported between 80% and 90% in the short term and 60–70% in the long term. The morbidity is minimal, and the drawbacks have to do with the pain of the procedure. Narcotics are usually required for 7–10 days. Occasionally, bleeding and nasopharyngeal incompetence have been reported but are exceedingly rare.

### Somnoplasty

The high cost of the laser, along with the pain from the procedure, prompted the development of a microwave-energy delivery system to the soft palate. The Somnus Corporation pioneered this, and the procedure called somnoplasty was developed. In this procedure, energy is delivered to the submucosal surface through a proprietary specialized device. The resultant soft tissue protein coagulation causes a stiffening of the tissue with a decrease in snoring and palatal flutter. Results are similar to all other snoring procedures with successes of 80% and long-term results of 60–70%. The major advantage with this procedure is that pain is minimal with it. Usually, non-narcotics are sufficient for pain control. Multiple treatments are usually required, and the proprietary nature of the device makes its cost high.

### Snoroplasty

First pioneered by a number of otolaryngologists at the Walter Reed Medical Center, this procedure involved injection of a sclerotherapy agent into the soft palate. The resultant scarring and soft tissue cauterization effect decreased palatal flutter and led to stiffening of the soft palate with retraction and decrease in volume of noise. Results were reported to be extremely good with single injections, and they seemed to maintain themselves over the long term, as with other procedures. Pain was between that of the laser-assisted procedure and the somnoplasty procedure, with most patients requiring some form of narcotic control. The major drawback is that the drug pioneered in this procedure is no longer manufactured in the United States and must be purchased through a foreign country.

### Other Procedures

People have described using electrocautery and coblation techniques for the same desired effects. Results from single-institutional studies and by single authors are similar to those reported above. A relatively new procedure based on implanting poly-Teflon implants to stiffen the palate has recently been described with a high extrusion rate but good success.

### SUMMARY

Overall, all of the procedures for snoring have been described as being 70–80% successful in the short term, while decreasing to 60% or so in the long term. No randomized control studies comparing different techniques have been reported.

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