Obstructive sleep apnea (OSA) is a sleep-related breathing disorder that involves a reduces or complete halt in airflow despite an ongoing effort to breathe. It occurs when the muscles relax during sleep, causing soft tissue in the back of the throat to collapse and block the upper airway. This results in partial reductions (hypopneas) and complete block (apneas) in breathing that lasts a minimum of 10 seconds during sleep. Most pauses last between 10 and 30 seconds, but some may persist for one minute or longer. This can cause abrupt reductions in blood oxygen saturation. The brain responds to the lack of oxygen by alerting the body, causing a brief arousal from sleep that restores normal breathing. This pattern can occur many times in one night. The result is a fragmented quality of sleep that often produces an excessive level of daytime sleepiness. Most people with OSA snore loudly and regularly, with periods of silence when airflow is reduced or blocked. They then make choking, snorting or gasping sounds when their airway reopens. A common measurement of sleep apnea is the apnea-hypopnea index (AHI). These include excessive weight, large neck and structural abnormalities reducing the diameter of the upper airway such as nasal obstruction, a low – hanging soft plate, enlarge tonsils or a small jaw with an overbite [6]. There are two sorts of sleep apnea: obstructive and central. Obstructive sleep apnea (OSA) is that the more common of the two. OSA is characterised by repetitive episodes of complete or partial upper airway blockage during sleep [7]. During an apnea episode, the diaphragm and chest muscles work harder as the pressure increases to open the airway. Breathing usually resumes with a loud gasp or body jerk [8]. These episodes can disturb sound sleep, reduce the flow of oxygen to vital organs, and cause heart rhythm irregularities [9]. In CSA, the airway is not blocked but the brain fails to signal the muscles to breath due to instability in the
respiratory control centre [10]. Central apnea is named ass such because it is related to the function of the central nervous system [11].

**Symptoms**
- Snoring
- Daytime sleepiness or fatigue
- Restlessness during sleep
- Sudden awakening with a sensation of gasping or choking
- Dry mouth or sore throat upon awakening
- Intellectual impairment, such as trouble concentrating, forgetfulness or irritability
- Night sweats
- Sexual dysfunction
- Headache
- Insomnia

Symptoms in children might not be as obvious and include:
- Poor school performance
- Sluggishness or sleepiness, often misinterpreted as laziness
- Inward movement of the ribcage when inhaling
- Unusual sleeping positions, such as sleeping on the hands and knees or with the neck hyper-extended
- Excessive sweating at night
- Learning and behavioural disorders
- Bed wetting

**Pathogenesis**

**Central sleep apnea**

There are several forms of central sleep apnea. Period breathing develops in most individuals ascending to high altitude if the altitude is high enough [12]. This is a sort of ventilatory instability produced by ambient hypoxia. Hypoxia at altitude results in high controller gain, leading to hyperventilation and hypocapnia. In this situation the increased control gain is sufficient to overcome the somewhat reduced plant gain (low Pco2) and ventilation becomes unstable as a result, ventilation waxes and wanes between apnea and hyperpnea. For this to occur, there must be in hypocapnia [13]. Thus, the periodic breathing at altitude occurs more commonly in individuals with high controlled gain [12]. However, most such individuals don’t have periodic breathing at sea level. Thus, inherently high loop gain that is augmented further by altitude is required for this respiratory pattern to emerge. Over time at altitude, the periodic breathing resolves due primarily to further decrements n plant gain that cannot be offset by a high controller gain [14]. Idiopathic central sleep apnea is a relatively uncommon disorder seen at sea level in individuals with high controller gain, generally on elevated hypercapnic ventilatory response Pco2: drives ventilation during sleep at sea level as it does at altitude, and patients with idiopathic central sleep apnea tend to have low Pco2 levels, even during wakefulness [15,16] Another form of CSA called chyne-stokes respiration is seen in patients with congestive heart failure and is a product of high controller gain, hypocapnia resulting from lung edema and a long circulation time. This combination of traits is particularly destabilizing to ventilation and yields a chrotmastic crescendo-decrescendo pattern of breathing, with a cycle time approximately 1 minute. As expected in a high loop gain disorder Co2: administration can regularize ventilation [17]. As in idiopathic CSA, Cheyne-stokes respiration occurs primarily during non-REM [Rapid eye movement] sleep, although it can be detected during wakefulness if carefully sought. However, it is uncommon during REM sleep, again likely secondary to decreased controlled gain, because both traits described above are generally required to manifest Cheyne-stokes respiration, it is not seen in all patients with congestive heart failure, even those with severe congestive heart failure [18]. Patients with walking hypercapnia primarily due to ventilatory abnormalities or neuromuscular disease may have central apneas during sleep as well [19,20].

---

**Fig 01: Central Sleep Apnea**

---

International Journal of Health Care and Biological Sciences
Obstructive sleep apnea
Obstructive apnea have an anatomically small pharyngeal airway like due to either increased soft tissue surrounding the airway or a small bony compartment in which the airway is enclosed [21]. During wakefulness, pharyngeal patency is maintained primarily by reflex-driven, augmented pharyngeal dilator muscle activity, which offsets the positive extraluminal tissue pressure collapsing the airway [22,23]. Normal ventilation is maintained, at sleep onset and/or during REM sleep, reflex muscle activation is reduced as is arousal-modulated excitatory output to the upper airway musculature [24]. Lung volume falls as well [25]. If the airway anatomy is quite deficient, these events alone will likely lead to substantial or complete airflow obstruction, yielding a hypopnea or apnea. As a result, hypoxia and hypercapnia develop, ventilation is stimulated, and often arousal from sleep in response to respiratory activation is required to reestablish airway patency to allow a recovery of ventilation [26,27].

![Image](https://example.com/image.png)

**Fig 02: Obstructive Sleep Apnea**

Diagnosis
If there’s a high suspicion of sleep apnea after evaluating a patient, a sleep study is indicated to determine a diagnosis. Currently, polysomnography, which requires an overnight stay in a sleep laboratory, is the optimum test for diagnosing sleep apnea. It includes evaluation of sleep staging, airflow and ventilatory effort, arterial oxygen saturation, electrocardiogram, body position, and periodic limb movements. Polysomnography, however, may not be readily available. Other options to consider are evaluation using pulse oximetry and portable (home) monitoring of cardiopulmonary channels. Although oximetry is currently being used to diagnose sleep apnea, its sensitivity and specificity are controversial [28]. A variety of home monitors are currently available or being developed that can record both cardiopulmonary parameters (for example, airflow, ventilatory effort, heart rate, and oxygen saturation) and sleep parameters and may be useful in diagnosing sleep apnea [29].

Treatment
The goals of treatment for sleep apnea patients include both physiologic and symptomatic components. Physiologic goals of treatment include eliminating sleep fragmentation, apneas and hypopneas, and oxygen desaturation. Symptomatic goals include eliminating snoring and sleepiness, improving quality of life, and reducing or eliminating comorbidities. Symptomatic improvement, particularly decreased snoring, does not necessarily correlate with physiologic improvement or decreased morbidity [30].

Behavioural approach
Behavioural measures may be the only treatment needed for patients with mild sleep apnea. Behavioral interventions include losing weight, eliminating evening alcohol and sedatives, and proper positioning (avoiding the supine position in bed). Although weight loss (accomplished through a comprehensive program or surgery) may be difficult to achieve, it can be very effective and, in some cases, even curative [31,32]. Patients with mild symptoms may experience improvement using behavioural techniques alone. Appropriate behavioural treatment should be implemented for all patients, even those requiring additional interventions. Patients treated with behavioural techniques should be reevaluated periodically after initiation of treatment [33]. For patients who have improved, continued support and positive reinforcement can sustain their adherence and success. In those patients who continue to experience symptoms, other therapies are warranted [34].

Nasal continuous positive airway pressure
Continuous positive airway pressure (CPAP) is the most effective non-invasive therapy for sleep apnea. To use CPAP the patient must wear a sealed mask over the nose or in some cases, over the nose and mouth during sleep [35]. The mask is connected to a blower forcing air through the nasal passages. CPAP acts as a pneumatic
spline by increasing the pressure in the oropharyngeal airway, thereby maintaining airway patency throughout the ventilatory cycle [36]. This treatment is usually prescribed after polysomnography has first determined the therapeutic level of CPAP pressure required to reduce or eliminate sleep apnea [37]. CPAP is effective in reversing daytime somnolence and eliminating cardiopulmonary sequelae [38]. CPAP used properly, produces rhythmic breathing resulting in the patient feeling dramatically better and being able to function more efficiently. Compared with no treatment or other treatment modalities patients treated with CPAP have a lower mortality rate [39]. Although very effective, CPAP may be difficult for some patients to use [40]. Adherence to CPAP treatment varies greatly but tends to be higher in patients with severe symptoms [41]. The most common reasons for discontinuing CPAP are intolerance of the mask, nasal-related complaints, and the inconvenience of being connected to a machine. Common side effects include nasal stuffiness, rhinitis, facial skin discomfort, and discomfort with the pressure. Humidifiers, nasal steroids or decongestants, intranasal anticholinergic, or different masks may relieve side effects. Variations in pressure application have been developed to offer patients options for improving comfort. Assisting patients to focus on symptom reversal and working with home care companies to ensure proper-fitting and effective equipment will enhance adherence. Followup after the primary month of CPAP treatment should include checking the status kit, assessing patient symptoms and adherence, and assessing the status of coexisting conditions like hypertension. In patients who have achieved significant weight loss, the CPAP pressure may have to be adjusted. If the patient reports continued snoring, the pressure may need to be increased [42].

**Oral/ dental appliances**

Oral or dental appliances could also be an option for patients with mild-to-moderate sleep apnea. However, they are not effective in all patients. Appliances have also been used for patients who snore but do not have sleep apnea. There are various devices that displace the tongue forward or move the mandible to an anterior and forward position to improve patency of the airway [43]. Reported side effects of the devices include excessive salivation and temporomandibular joint discomfort. A dentist or orthodontist experienced in the use of these devices should fit the patient, and a sleep study should be done after the device is fitted to evaluate its effectiveness [44].

**Surgical procedure**

Patients need to understand that no surgical procedure has universal success, and all are invasive and carry risk. Several procedures or a combination of procedures may need to be performed to help sleep apnea patients. It is important that sleep studies be repeated after each surgical procedure to confirm its effectiveness, once there is evidence of adequate healing. When weighing treatment options, it may be useful to let the patient know that CPAP is highly effective when used properly and is safe and reversible [45,46]. Uvulopalatopharyngoplasty (UPPP). During UPPP, an inpatient procedure, the uvula and portions of the soft palate are resected to widen the oropharyngeal airway. Although snoring is temporarily relieved in most cases, apnea may persist. The overall success rate of UPPP is reported to be about 40 percent (when success is defined as achieving an AHI of less than 20) [47]. It is difficult to predict which patients will benefit from this procedure, and long-term side effects and benefits are unknown.

**Nasal Surgery**

Nasal surgery may be used alone or in conjunction with other procedures. However, it is rarely curative alone.

**Tonsillectomy**

In children and adolescents adenotonsillectomy may be useful, even curative [48]. Tonsillectomy alone in adults is not usually helpful [49], but is often done in conjunction with UPPP. Laser-Assisted Uvulopalatoplasty (LAUP): LAUP has received much attention recently as a treatment for snoring. However, its effectiveness in treating sleep apnea is unknown. LAUP differs from traditional UPPP in both surgical technique and setting (office-based). LAUP excises only part of the uvula and associated soft-palate tissues. The resultant shortening of the palate and reduction of the uvula may reduce aleror eliminate snoring. As with UPPP, relief of snoring may occur without improvement in apneic events. Therefore, patients who elect LAUP for snoring may risk delaying the diagnosis of sleep apnea because snoring, a primary symptom, is eliminated [50].

**Maxifacial surgery**

(Genioglossal Advancement, Maxillary and Mandibular Advancement). These are specialized procedures that are currently not widely available, although they appear
to be effective in treating sleep apnea [51]. Genioglossal advancement enlarges the airway at the base of the tongue. This procedure may be combined with a UPPP. Maxillary and mandibular advancement enlarges the airway at the level of the soft palate as well as the tongue. Tracheostomy. Tracheostomy is highly successful in eliminating sleep apnea but is very invasive, both physically and psychologically. This procedure is reserved for severe cases where other treatments have failed [52].

**Pharmacological treatment**
Currently, there are no safe and effective medications indicated in the routine treatment of sleep apnea.

**Oxygen**
Administration of supplemental oxygen may improve nocturnal desaturation but is not a satisfactory treatment option by itself because it does not reduce sleep disruption and subsequent daytime sleepiness [53].

**Management considerations**
The efficacy of a chosen treatment modality should be periodically and objectively verified. Sleep apnea patients who undergo surgical interventions need to have sleep studies repeated postoperatively, after healing has occurred. Once effective treatment has been initiated, all patients should be periodically revaluated for recurrence of symptoms such as snoring and excessive daytime sleepiness as well as cardiopulmonary complications. The primary care physician can play a key role in determining if patients are adhering to treatment and in monitoring comorbidities such as hypertension and coronary artery disease. For example, hypertension treatment may need to be adjusted once sleep apnea has improved. Patients who are adherent to treatment for sleep apnea need positive reinforcement, and those who are not adherent may require different treatment options. Patients who are on CPAP need to have their equipment evaluated periodically to ensure that the machine and mask are functioning properly [54].

**Conclusion**
there are a number of phenotypic traits that predispose an individual to the development of sleep apnea. In the case of central apnea, this generally relates to loop gain and circulation time. For obstructive apnea, pharyngeal anatomy, upper airway muscle responsiveness during sleep, arousal threshold, and loop gain may all contribute to apnea presence and severity. The relative contribution of each may vary between patients. It is unclear at this time whether defining these traits in patients with apnea would have therapeutic implications, although this seems possible. Thus, as always, more information is needed.

**Authors contribution**
all authors contributed equally to this work.

**References**
1. Mozaffarian D, Benjamin EJ, Go As, et al., for the American heart association statistics committee and stroke statistics subcommittee heart disease and stroke statistics = 2016 update: a report from the American heart association circulation 2016; 133; e38=360.
2. peppard PE, Young T, Barnet JH, et al. increased prevalence of sleep-disordered breathing in adults. Am J epidemiol 2013; 177:1006=14.
3. Javaheiri S, dragger LF, Lorenzi-filho G. sleep and cardiovascular disease: present and future. In: kryger MH, roth T, dement WC, editors. Principles and practices of sleep medicine 6th edition. Philadelphia, PA; Elsevier, 2017;1222-8.
4. Javaheiri S, Dempsey JA. Central sleep apnea. Compr physiol 2013; 3:141=63.
5. Lyons OD, bradely TD. Heart failure and sleep apnea. Can J cardiol 2015; 31:988-908.
6. May AM, Blackwell T, Stone PH, et al, for the Mr OS sleep (outcomes of sleep disorders in older men) study group. Central sleep-disordered breathing predicts incident atrial fibrillation in older men. Am J respir crit care med 2016; 193:783-91.
7. Javaheiri S, plackwell T, ancoli- Israel S, et al, for the osteoporotic fractures in Men study research group. Sleep – discorded breathing and incident heart failure in older men. Am J respir crit care Med 2016;193;561-8.
8. Javaheiri S. Cardiovascular diseases. In: Kryger MH, Avidan RB, Berry RB, editors. Atlas of clinical sleep medicine. 2nd edition. Philadelphia, PA; saunders, 2014;316–28.
9. Dempsey AS, Mchharry DG, Malhotra A. pathophysiology of sleep apnea. Physiol REV 2010; 90:47-112.
10. Jordan JA, Veasey SC, morgan BJ, et al. obstructive sleep apnea. Lancer 2014; 383:736-47.
11. Mendelson lyons OD, yadollahi A, et al. effects of exercise training on sleep apnea in patients with coronary artery disease: a randomised trial. Eur respir J 2016; 48:142-50.
12. White DP, Gleson K, pichett CK, rannels AM, cymerman A, weil JV. Altitude acclimatization: influence on periodic breathing and chemo responsiveness during sleep. J appl physiol 1987; 63:401-412.
13. Zhou XS, shahe buddin S, zahn BR, babaek MA, development of hypocapnic apnea/hypopnea during NREM sleep. J appl physiol 2000; 89:192-199.
14. Somers V, javaheri S. cardiovascular effects of sleep-related breathing disorders. In: Kryger MH, roth T, dement WC, editors. Principles and practices of sleep medicine, 6th edition. Philadelphia PA: elaevier, 2017;1243-52.
15. Xie A, wong B, Phillipson EA, Slutsky AS, bradley TD. Interaction of hyperventilation and arousal in the pathogenesis of idiopathic central sleep apnea. Am J reserpir crit care Med 1994; 150:489-495.
16. Xie A, Rutherford R, rankin F, wong B, Bradley TD. Hypocapnia and increased ventilatory responsiveness in patients with idiopathic central sleep apnea. Am J respir crit care Med 1995;152:489-495.
17. Lorenzi-filho G, rankin F, Bradley. T. effects of inhaled carbon dioxide and oxygen on Cheyne stokes respiration in patients with heart failure Am J respir crit care Med 1999; 159:1490-1498.
18. Sin D, fitzgerald F, parker J. risk factors for central and obstructive sleep apnea in 450 men and women with congestive heart failure. Am J respir crit care Med 1999;160:1101-1106.
19. Mellins R, balfour H, turion G, winters RW. Failure of automatic control of ventilation (ondines curse). Medicine (Baltimore) 1970; 49:487-504.
20. Tassinari C, dalla Bernardina B, cirignotta F, Ambrosetto G. apnoeic periods and the respiratory related arousal patterns during sleep in the pickwikian syndrome. Bull physiopathol respir (Nancy) 1972; 8:1087-1102.
21. Schwab RJ, gupta KB, getter WB, Metzger LJ, Hoffman EA, pack Al. upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing: significance of the lateral pharyngeal walls. Am J respir crit care Med 1995:152:1673-1689.
22. Fogel R, Malhotra A, Pillar G, Edwards J, shea S. whigte D. genioglossal activation in patients with obstructive sleep apnea versus control subjects: mechanisms of muscle control. Am J respir crit care Med 2001; 164:2025-2030.
23. Nan lunteren E. muscle of the pharynx: strucual and contractile properties. Ear nose throat J 1993; 72:27-29,33.
24. Wheatley J, white D, mezzanotte W, white D. influence of sleep on response to negative airway pressure of tensor palatini muscle and retropalatal airway. J appl physiol 1993; 75:2117-2124.
25. Ballard R, Irvin CG, Martin RJ, Pak J, Pandey R, White DP. Influence of sleep on lung volume in asthmatic patients and normal subjects. J Appl Physiol 1990; 68:2034-2041.
26. Gleson K, Zwillich CW, White DP. The influence of increasing ventilatory effort on arousal from sleep. Am Rev Respir Dis 1990; 142:295-300.
27. Berry RB, McNellis MI, Kouchi K, Light RW. Upper airway anaesthesia reduces phasic genioglossus activity during sleep apnea. Am J Respir Crit Care Med 1997; 156:127-132.
28. Gyulay S, Olson LG, Hensley MJ, King MT, Allen KM, Saunders NA. A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. Am Rev Respir Dis 1993; 147:50-3.
29. Rosen CL, D’Andrea L, Haddad GG. Adult criteria for obstructive sleep apnea do not identify children with serious obstruction. Am Rev Respir Dis 1992; 146:1231-4.
30. Young T, Palla M, Dempsey J, Skatrud J, Weber S, Baddr S. The occurrence of sleepdisordered breathing among middle-aged adults. N Engl J Med 1993;328:1230-5.
31. Smith PL, Gold AR, Meyers DA, Haponik EF, Bleecker ER. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. Ann Intern Med 1985; 103:850-5.
32. Sugerman HJ, Fairman RP, Baron PL, Kventus JA. Gastric surgery for respiratory insufficiency of obesity. Chest 1986;90:81-6.
33. National Heart, Lung, and Blood Institute. Fact Book: Fiscal Year 1993. U.S. Department of Health and Human Services, U.S. Public Health Service, National Institutes of Health, February 1994.

34. Fletcher EC, DeBuhnke RD, Lovoi MS, Gorin AB. Undiagnosed sleep apnea in patients with essential hypertension. Ann Intern Med 1985;103:190-5.

35. Sanders MH, Kern NB, Stiller RA, Strollo PJ, Martin TJ, Atwood CW. CPAP therapy via oronasal mask for obstructive sleep apnea. Chest 1994;106:774-9.

36. American Thoracic Society. Indications and standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. Am J Respir Crit Care Med 1994;150:1738-45.

37. Waldhorne RE, Wood K. Attended home titration of nasal continuous positive airway pressure therapy for obstructive sleep apnea. Chest 1993;104:1707-10.

38. Riley RW, Powell NB, Guilleminault C. Maxillofacial surgery and nasal CPAP. A comparison of treatment for obstructive sleep apnea syndrome. Chest 1990;98:1421-5.

39. Keenan SP, Burt H, Ryan CF, Fleetham JA. Long-term survival of patients with obstructive sleep apnea treated by uvulopalatopharyngoplasty or nasal CPAP. Chest 1994;105:155-9.

40. Polo O, Berthon-Jones M, Douglas NJ, Sullivan CE. Management of obstructive sleep apnoea/hypopnoea syndrome. Lancet 1994;344:656-60. 28. Kribbs NB, Pack AI, Kline L.

41. Kribbs NB, Pack AI, Kline LR, et al. Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. Am Rev Respir Dis 1993;147(5):1162-8.

42. Fletcher EC. The relationship between systemic hypertension and obstructive sleep apnea: facts and theory. Am J Med 1995;98:118-28.

43. Guilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. Am J Cardiol 1983;52:490-4.

44. Shepard JW Jr., Garrison MW, Grithers DA, Dolan GF. Relationship of ventricular ectopy to oxyhemoglobin desaturation in patients with obstructive sleep apnea. Chest 1985;88:335-40

45. Hung J, Whitford EG, Parsons RW, Hillman DR. Association of sleep apnoea with myocardial infarction in men. Lancet 1990;336:261-4.

46. Rapoport DM, Garay SM, Epstein H, Goldring RM. Hypercapnia in the obstructive sleep apnea syndrome. A reevaluation of the “Pickwickian syndrome.” Chest 1986;89:627-34.

47. Millman RP, Carlisle CC, McGarvey ST, Eveloff SE, Levinson PD. Body fat distribution and sleep apnea severity in women. Chest 1995;107:362-6

48. American Sleep Disorders Association. Practice parameters for the use of laserassisted uvulopalatoplasty. Sleep 1994;17:744-8.

49. Fletcher EC, Munafo DA. Role of nocturnal oxygen therapy in obstructive sleep apnea. When should it be used? Chest 1990; 98:1497-504.

50. Phillips BA, Schmitt FA, Berry DTR, Lamb DG, Amin M, Cook YR. Treatment of obstructive sleep apnea: a preliminary report comparing nasal CPAP to nasal oxygen in patients with mild OSA. Chest 1990.

51. Friberg D, Gazelius B, Hokfelt T, Nordlander B. Abnormal afferent nerve endings in the soft palatal mucosa of sleep apnoics and habitual snorers. Regul Pept 1997;71:29-36.

52. Boyd J, Petroff B, Hamid Q, Fraser R, Kimoff R. Upper airway muscle inflammation and denervation changes in obstructive sleep apnea. Am J Respir Crit Care Med 2004;170:541-546.