

# Treatment of obstructive sleep apnea: the role of nasal continuous positive airway pressure

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**SUMMARY** Nasal continuous positive airway pressure (CPAP) has become the treatment of choice for the initial management of obstructive sleep apnea.

**KEY POINTS** Nasal CPAP is usually considered when a patient with obstructive sleep apnea has an apnea-hypopnea index (the mean number of episodes of apnea or hypopnea per hour of sleep) of greater than 20 or if the index is between 5 and 20 and the patient has significant daytime sleepiness or has cardiovascular risk factors such as hypertension, hypercholesterolemia, cigarette smoking, or obesity. ■ The optimum pressure for the individual patient should be determined in a sleep laboratory. ■ Many patients do not actually use CPAP as directed (all night, every night); newer machines, counseling, and measures to reduce side effects may improve compliance. ■ During the first few nights of CPAP therapy, as the patient sleeps more soundly, the risk of apnea and hypoxemia may actually increase; thereafter, the pattern of sleep returns to normal.

**INDEX TERMS:** SLEEP APNEA SYNDROMES; POSITIVE-PRESSURE RESPIRATION  
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**O**BSTRUCTIVE SLEEP apnea (OSA) is a life-threatening condition characterized by repetitive episodes of total or partial closure of the upper airway during sleep resulting in episodic hypoxemia, arousal from sleep, and daytime sleepiness. Some major complications of OSA include traffic and work accidents (due to daytime somnolence), pulmonary hypertension, cor pulmonale, cardiac arrhythmias, hypertension, and increased incidence of strokes and myocardial infarction. Treatment ranges from conservative measures such as weight loss to surgical procedures such as tracheostomy.<sup>1</sup>

Nasal continuous positive airway pressure (CPAP), introduced by Sullivan in 1981,<sup>2</sup> has become the treatment of choice for the initial management of OSA.<sup>1</sup> When used at night at the appropriate pressure, nasal CPAP eliminates episodes of apnea and hypopnea due to upper airway obstruction<sup>3</sup>; the patient therefore enjoys more undisturbed sleep<sup>4</sup> (and less subjective hypersomnolence while awake).<sup>5</sup> In addition, CPAP has been shown to im-

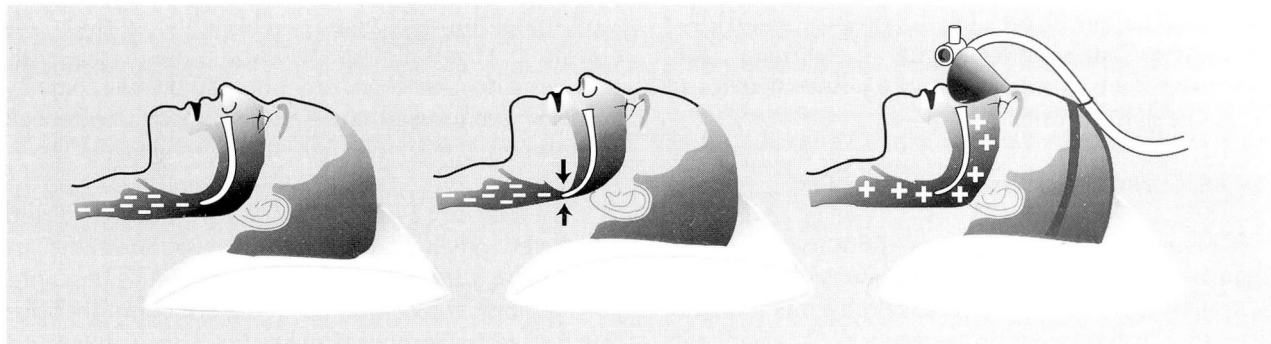


FIGURE. Left, the airway normally remains open during sleep. Middle, in a patient with obstructive sleep apnea (OSA), poor muscle tone allows the tongue and soft palate to be sucked back against the posterior oropharyngeal wall during inspiration, occluding the airway. Patients especially prone to this problem are overweight, middle-aged men who breathe through the mouth and who sleep supine. Right, a nasal continuous positive airway pressure (CPAP) device creates a pneumatic “splint” that keeps the upper airway open, thereby eliminating apnea.

prove prognosis by preventing long-term cardiopulmonary sequelae.<sup>2,6-8</sup>

#### HOW CPAP WORKS

Nasal CPAP creates a positive pressure in the upper airway, thereby providing a “pressure splint” to eliminate apnea (Figure).<sup>1,2</sup> Another proposed mode of action is an increase in functional residual capacity, which in turn reflexively dilates the pharynx.<sup>9</sup> However, the pressure-splint mechanism is generally believed to be the predominant mode of action.

#### INITIATING CPAP THERAPY

In OSA, nasal CPAP is usually considered when the apnea-hypopnea index (the mean number of episodes of apnea or hypopnea per hour of sleep) is greater than 20 or if the index is between 5 and 20 and the patient has significant daytime sleepiness or has cardiovascular risk factors such as hypertension, hypercholesterolemia, cigarette smoking, or obesity.<sup>10</sup> Primary snoring (ie, snoring without symptoms suggestive of OSA or without significant apneas or desaturations) does not require CPAP therapy.<sup>10</sup>

The optimum level of CPAP for each patient should be determined in a sleep laboratory. This can be accomplished in one night of testing. At the Cleveland Clinic, we usually have the patient try sleeping with a CPAP unit set to a low level (7.5 cm H<sub>2</sub>O) at home for a few days to get used to it before undergoing sleep testing. The nose mask and the pressure sensation make sleeping a novel experience,

and it is important to explain this therapy in detail. Sufficient pressure must be used to prevent apnea in all sleep stages and, preferably, in the supine and lateral postures.<sup>1,10</sup> The minimum pressure that reduces apneic and hypopneic episodes or hypoxemia to acceptable levels is appropriate. In addition, this pressure should be titrated to eliminate oxygen desaturations, snoring, and arousals. Since higher pressure can be uncomfortable to sleep with, the minimal pressure that reasonably eliminates the majority of the sleep and respiratory disturbances is used. In general, patients need higher pressures when supine and during rapid-eye-movement (REM) sleep. In addition, alcohol intake can depress the upper airway muscles.<sup>11</sup> Therefore, a pressure that is adequate when an alcoholic patient is sober may not be adequate when the patient is intoxicated.

Most patients need a pressure between 7.5 and 12.5 cm H<sub>2</sub>O (overall range 4 to 20 cm H<sub>2</sub>O). The patient should use the same model of machine at home that was used in the laboratory, because different machines may have different characteristics, even at the same pressure settings.<sup>12</sup>

During the first few nights of CPAP use, there is usually a rebound increase in both rapid-eye-movement (REM) sleep and stage 4 non-REM sleep—the deeper level of sleep.<sup>4</sup> Patients sleep more soundly and are harder to arouse.<sup>4</sup> During this REM-rebound period, life-threatening hypoxemia can occur, particularly in patients with severe sleep apnea and hypercarbia.<sup>13</sup> For this reason, some experts recommend that CPAP be initiated in the hospital in patients with congestive heart failure, severe nocturnal hypoxemia, or carbon dioxide retention and

other medical problems such as coronary artery disease.<sup>3</sup> After four to seven nights of treatment, the pattern of sleep normalizes, and daytime sleepiness decreases significantly.<sup>14</sup>

#### PROBLEMS WITH COMPLIANCE

It is very important that the patient use CPAP all night every night, because even one night without treatment has been reported to reverse much of the gain in daytime hypersomnolence in some patients.<sup>1,15</sup>

Even though CPAP is effective, not all patients accept it. Compliance varies from center to center, depending on the definition of compliance used, the staff's skill and experience in initiating therapy, the adequacy of follow-up, and, perhaps, the type of CPAP device and mask used. In studies of unselected OSA patients and in studies based on patients' self-reports, the compliance rate was approximately 75%.<sup>16-18</sup> Similar rates have been obtained in studies that documented CPAP use on the basis of the cumulative time the CPAP unit was turned on.<sup>19,20</sup>

However, in a more recent study, Kribbs et al<sup>21</sup> gave 35 patients CPAP machines that monitored both the duration of use and the actual pressure at the mask. Only 46% of the patients actually used CPAP regularly (defined rather leniently as at least 4 hours on 70% of the nights monitored). In addition, the patients gave inaccurate estimates both of how many nights they used CPAP and of how many hours per night they used it. Hence, these investigators concluded that actual CPAP use in OSA falls short of the therapeutic goal of providing quality sleep all night, every night. This underscores the importance of efforts to enhance CPAP use, especially early in treatment.

#### SIDE EFFECTS OF CPAP

Patients commonly report the side effects of nasal congestion, nasal dryness, and claustrophobia. They also complain that CPAP is inconvenient; the older devices are heavy, large, and not very portable; and CPAP therapy leads to less intimacy with one's bed partner.<sup>3,21</sup> Many cannot tolerate the pressure sensation. A case of massive epistaxis and pneumocephalus in a patient with a cerebrospinal fluid leak has been reported.<sup>3</sup>

Nasal congestion may necessitate use of intranasal vasoconstrictors, intranasal steroids, and sometimes,

intranasal anticholinergics.<sup>3</sup> Humidification can alleviate both nasal congestion and nasal dryness.<sup>3</sup> Inconvenience and claustrophobia can be overcome by proper psychosocial counselling designed to reduce the patient's fear and make this therapy less intrusive.<sup>21</sup>

The newer CPAP devices are well designed and relatively quiet, and the newer masks are self-sealing, light, and relatively comfortable. These improvements have greatly reduced complications such as noise intolerance, conjunctivitis due to air leaks, and ulceration of the nasal bridge due to mask pressure. In patients who cannot tolerate CPAP, systems that increase the pressure slowly as the patient falls asleep (the "ramp" device) or that reduce the expiratory pressure (the bilevel positive airway pressure—BiPAP—system) may be helpful.<sup>10</sup> A newly developed CPAP system, called "demand" or "smart" CPAP, may eventually replace the currently available CPAP machines. In this device, the machine self-adjusts the pressure for each breath, depending on the airflow and airway resistance. A damaged mask, increasing weight, alcohol use, nasal obstruction, or, very rarely, machine failure can cause CPAP to fail late in the course of therapy.<sup>3</sup> Patients for whom CPAP or its modifications fail may have to resort to surgical therapy.

#### OTHER INDICATIONS

Nasal CPAP has also been useful in central (ie, neurogenic) sleep apnea, sleep apnea with chronic lung disease and heavy snoring, and apnea with nocturnal asthma.<sup>3</sup> The recently described entity called the upper airway resistance syndrome (UARS) is also an indication for CPAP therapy.<sup>22</sup> Patients with UARS complain of hypersomnolence and snoring. Polysomnography reveals repeated arousals but a normal apnea-hypopnea index and oxygen saturation, and esophageal balloon manometry reveals that increasing inspiratory efforts are associated with these arousals, indicating subclinical airway obstruction.

#### LONG-TERM BENEFITS

In addition to alleviating snoring, apnea, and somnolence, long-term CPAP has a number of other benefits. Mood, psychological function, and intellectual function improve,<sup>23</sup> and testosterone and somatomedin levels normalize.<sup>3,24</sup> In addition, sexual

function (as assessed by patients' bed partners) has been reported to improve.<sup>3</sup> Right-sided heart failure, hypertension, and even left ventricular dysfunction have responded to CPAP treatment.<sup>3,25</sup> Long-term follow-up has demonstrated no deleterious effects on lung function.<sup>26</sup>

Although nasal CPAP greatly reduces the severity of OSA, by itself it does not lead to a permanent cure.<sup>27</sup> Lehrhaft et al<sup>27</sup> have shown that sleep apnea declines and respiratory function improves over the first 3 to 12 months of CPAP therapy, and then these stabilize. The swelling, erythema, and edema caused by the mechanical effects of snoring resolve with treatment,<sup>3,27</sup> resulting in increased upper airway dimensions.<sup>28</sup> In addition to thus improving

“sleep architecture,” CPAP leads to improvement in neuromuscular function of the submental, postural, and upper airway muscles.<sup>3</sup> Improvement in respiratory drive can also occur.<sup>29</sup> Finally, CPAP therapy may result in some weight loss, and many obese patients find it easier to lose weight while undergoing CPAP therapy.<sup>3</sup>

**CONCLUSIONS**

Nasal CPAP is a safe, noninvasive form of treatment for OSA and other sleep-related breathing disorders. Properly instituted, CPAP can be very effective and free of significant complications in compliant patients.

**REFERENCES**

1. Kryger MH. Management of obstructive sleep apnea: overview. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 2nd ed. Philadelphia: WB Saunders, 1994:736–747.
2. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. *Lancet* 1981;1:862–865.
3. Sullivan CE, Grunstein RR. Continuous positive airway pressure in sleep-disordered breathing. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 2nd ed. Philadelphia: WB Saunders, 1994:694–705.
4. Issa FG, Sullivan CE. The immediate effects of nasal continuous positive airway pressure treatment on sleep pattern in patients with obstructive sleep apnea syndrome. *Electroencephalogr Clin Neurophysiol* 1985; 63:10–17.
5. Rajagopal KR, Bennett LL, Dillard TA, Tellis CJ, Tenholder ME. Overnight nasal CPAP improves hypersomnolence in sleep apnea. *Chest* 1986; 90:172–176.
6. Sanders MH, Moore SE, Eveslage J. CPAP via nasal mask: a treatment for occlusive sleep apnea. *Chest* 1983; 83:144–145.
7. Rapoport DM, Sorkin B, Garay SM, Goldring RM. Reversal of the “Pickwickian syndrome” by the long-term use of nocturnal nasal-airway pressure. *N Engl J Med* 1982; 307:931–933.
8. Sullivan CE, Berthon-Jones M, Issa FG. Remission of severe obesity-hypoventilation syndrome after short-term treatment during sleep with continuous positive airway pressure. *Am Rev Resp Dis* 1983; 128:177–181.
9. Hoffstein V, Zamel N, Phillipson EA. Lung volume dependence of pharyngeal cross-sectional area in patients with obstructive sleep apnea. *Am Rev Resp Dis* 1984; 130:175–178.
10. Kryger MH. Management of obstructive sleep apnea. *Clin Chest Med* 1992; 13:481–492.
11. Krol RC, Knuth SL, Bartlett D. Selective reduction of genioglossus muscle activity by alcohol in normal human subjects. *Am Rev Resp Dis* 1984; 129:247–250.
12. Demirozu MC, Chediak AD, Nay KN, Cohn MA. A comparison of nine nasal continuous positive pressure machines in maintaining mask pressure during simulated inspiration. *Sleep* 1991; 14:259–262.
13. Krieger J, Weitzenblum E, Monassier JP. Dangerous hypoxemia during continuous positive airway pressure treatment of obstructive apnea. *Lancet* 1983; 2:1429–1430.
14. Lamphere J, Roehrs T, Wittig R, et al. Recovery of alertness after CPAP in apnea. *Chest* 1989; 96:1364–1367.
15. Kribbs NB, Pack AI, Kline LR, et al. Effect of one night

- without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1993; 147:1162–1168.
16. Rauscher M, Popp W, Wanke T, Zwick H. Acceptance of CPAP therapy for sleep apnea. *Chest* 1991; 100:1019–1023.
17. Sanders MH, Gruendl CA, Rogers RM. Patient compliance with nasal CPAP therapy for sleep apnea. *Chest* 1986; 90:330–333.
18. Nino-Murcia G, McCann CC, Bhwise DL, Guilleminault C, Dement W. Compliance and side effects in sleep apnea patients treated with nasal continuous positive airway pressure. *West J Med* 1989; 150:165–169.
19. Idatna R. A multicentric survey of longterm compliance with nasal CPAP treatment in patients with obstructive sleep apnea syndrome. *Am Rev Resp Dis* 1990; 141:A863. Abstract.
20. Reeves-Hoche MK, Meck R, Zwillich CW. An objective trial of nasal continuous positive airway pressure treatment compliance. *Am Rev Resp Dis* 1990; 141:A862. Abstract.
21. Kribbs NB, Pack AI, Kline LR, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Resp Dis* 1993; 147:887–895.
22. Guilleminault C, Stoohs R, Clerk A, et al. From obstructive sleep apnea syndrome to upper airway resistance syndrome. *Sleep* 1992; 15:513–516.
23. Bearpark H, Grunstein R, Touyz S, et al. Cognitive and psychological dysfunction in sleep apnea before and after treatment with CPAP. *Sleep Research* 1987; 16:303–309.
24. Grunstein RR, Handelsman DJ, Lawrence S. Neuro-endocrine dysfunction in sleep apnea: Reversal by nasal continuous positive airways pressure. *J Clin Endocrinol Metab* 1989; 68:352–358.
25. Wilcox I, Hedner JA, Grunstein RR. Non-pharmacological reduction of blood pressure in sleep apnea patients by treatment with nasal continuous positive airway pressure. *Circulation* 1991; 84(Suppl II):136. Abstract.
26. Young IH, Mihalyka M, Costus L, Sullivan CE. Long term lung function changes in patients with obstructive sleep apnea during treatment with nasal continuous positive airways pressure. *Thorax* 1987; 42:722. Abstract.
27. Lehrhaft B, Grunstein RR, Sullivan CE. Effects of long term treatment with nasal CPAP on severity of sleep apnea (OSA) and respiratory function. *Eur Respir J* 1991; 4:5855. Abstract.
28. Ryan CF, Lowe AA, Li D, Fleetham JA. Magnetic resonance imaging of the upper airway in obstructive sleep apnea before and after chronic nasal continuous positive airway pressure therapy. *Am Rev Resp Dis* 1991; 144:939–944.
29. Berthon-Jones M, Sullivan CE. Time course of change of ventilatory response to CO<sub>2</sub> with long term CPAP therapy for obstructive sleep apnea. *Am Rev Resp Dis* 1987; 135:144–147.