TREATMENT OF SEVERE OBSTRUCTIVE SLEEP APNEA SYNDROME WITH A CHINSTRAP


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A chinstrap alone improved severe obstructive sleep apnea as well as or better than the use of CPAP.

Keywords: Obstructive sleep apnea syndrome, chinstrap, polysomnogram

Obstructive sleep apnea syndrome (OSAS) is common and associated with neurocognitive and cardiovascular complications including excessive daytime sleepiness, motor vehicle crashes,1 hypertension, organic heart disease, and stroke.2 Conservative treatment options include weight loss and minimization of alcohol intake. Other treatments include nasal continuous positive airway pressure (CPAP), dental appliances, and surgical interventions.3 We report on successful treatment of severe sleep apnea with the use of only a CPAP chinstrap.

CASE REPORT

A 75-year-old retired physician presented to the Eastern Virginia Medical School/Sentara Norfolk General Hospital Sleep Disorders Center with a chief complaint of morning headaches. He described early morning awakening with a diffuse and dull headache. Head of the bed elevation, as well as aspirin, a combination of aspirin, caffeine, and acetaminophen, and an ice pack frequently alleviated this discomfort. He also described snoring, nocturia, and daytime sleepiness, with an Epworth Sleepiness Scale score of 17/24.

Past medical history included hypertension, benign prostatic hypertrophy, and a transient right homonymous hemianopsia. He was extremely active, including participation in triathlons and hiking in Nepal.

Physical examination revealed an elderly fit gentleman with an irregular pulse of 60 beats per minute. Body mass index (BMI) was 24 kg/m2. The nasal exam revealed leftward septal deflection. He had good dentition with a borderline class 3 malocclusion, normal tongue, Mallampati 1, slightly high-arched hard palate, normal uvula, and no retrognathia.

Diagnostic polysomnography (PSG) was performed utilizing nasal pressure transducer, piezoelectric belts, oximetry, and intercostal EMG for respiratory monitoring. We defined obstructive, mixed, and central apneas in the conventional fashion; we defined hypopneas as demonstrating ≥30% reduction in nasal pressure transducer signal for ≥10 sec, associated with ≥4% oxygen desaturation. At the time of the first study the patient admitted to taking felodipine, aspirin, and a multivitamin. The PSG indicated 260 min of sleep, with 69% of the time spent supine and with 21% REM sleep. He manifested 42 apneas and hypopneas per hour. REM and supine indices were 44 events and 42 events per hour respectively. Of the 184 events, there were 75% obstructive, 6% mixed, and 19% central in origin. He had no arterial oxygen desaturations below 90%. Atrial fibrillation and one 10-beat run of nonsustained ventricular tachycardia occurred. In addition, the patient demonstrated 21 periodic leg movements (PLMs) per hour (1/h associated with arousal). After application of nasal CPAP on the same night, the patient demonstrated 24 apneas/hypopneas per hour (87% of the 68 events were central). The central events were not part of a Cheyne-Stokes breathing pattern. Low oxygen saturation was 96%. PLMs increased to 43/h, with 2/h associated with arousals. PLMs during the diagnostic and therapeutic portions of the PSG did not appear related to respiratory induced arousals.

We recommended nasal CPAP treatment and cardiology consultation. One month later, the patient reported that his headaches, nocturia, and daytime sleepiness all had dissipated on CPAP. After a discussion with a physician friend (also afflicted with OSAS), and given his plans for a trip to Antarctica, the patient decided to treat his OSAS with the CPAP chinstrap and lateral position. Improvement continued in headaches and in sleep quality, and the patient elected to cease use of CPAP and use only the chinstrap.

An echocardiogram performed one and a half months after his sleep study revealed a normal ejection fraction of 73%; an adenosine stress test completed that same day revealed no perfusion abnormalities. Nocturnal oximetry with the chin strap approximately two and a half months after the initial PSG revealed no significant desaturations.

A repeat PSG (BMI 24 kg/m2) with the chinstrap alone was performed approximately 3 months after the first study. At this time the patient was taking felodipine, hydrochlorothiazide, and warfarin. This PSG indicated 404 minutes of recorded sleep (46%
that same study demonstrated that achieving the lateral position, also adopted at home by our patient further improved the effectiveness of these maneuvers. Oral appliances can improve sleep apnea with nonresponders, interestingly, having larger upper airway dimensions. However, oral appliances can be expensive and are not uniformly effective.

In this study, the chinstrap appeared to be effective in improving upper airway soft tissue anatomy. Recently, Eikermann et al. reported increased “pharyngeal collapsibility” with aging. Perhaps the efficacy of the chinstrap in our older gentleman related to just such a “floppy” airway. Future research might consider the utility of a chinstrap for OSAS in different age groups. Whether the chinstrap is a viable alternative intervention for those with complex sleep apnea (and why) might also merit investigation.

The modest reduction in the percent time spent supine could have contributed to the PSG improvement in respiratory instability with chinstrap. However, the diagnostic study did not suggest supine apnea predominance as the overall AHI and the supine AHI were each 42. In addition, our endoscopy results indicated that the chinstrap stabilized the patient’s upper airway.

The use of CPAP might have improved upper airway muscle tone and/or edema, thus leading to an improvement in the second PSG. However, our patient had not used CPAP for 2 months prior to the second study done with the chinstrap. Thus we doubt that residual CPAP effects stabilized the upper airway. It should also be noted that the endoscopic exam was performed in a nonblinded fashion. Thus, any case series evaluating chinstrap efficacy should utilize blinded endoscopic exams.

In sum, this case report indicates that a simple and inexpensive chinstrap deserves further investigation as a modality for the treatment of OSAS.

REFERENCES


Figure 1a—Without chinstrap (mouth open). Notice narrow anterior to posterior airway space as a result of the tongue base and the posteriorly positioned epiglottis.

Figure 1b—With chinstrap (mouth closed). Notice significant improvement of the posterior airway space with the tongue base and epiglottis rotated forward.

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