

Effects of Submental Electrical Stimulation during Sleep on Upper Airway Patency in Patients with Obstructive Sleep Apnea^{1,2}

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Introduction

Obstructive sleep apnea syndrome (OSAS) is caused by obstruction of the upper airway during sleep (1-9). It has been postulated that upper airway patency is determined by the anatomic size of the upper airway and the balance of two counteracting forces: one is the negative intraluminal pressure generated by the contraction of inspiratory pump muscles and the other is the upper airway dilating force developed by upper airway muscle contraction (10). Among the upper airway muscles, the genioglossus appears to be of particular importance since it is responsible for pulling the tongue forward and a decrease in genioglossal activity has been reported to narrow or obstruct the upper airway, especially in the supine position, both in animals (1) and in humans (3, 6, 7, 11, 12). We reported in a previous article that electrical stimulation of the genioglossus decreased upper airway resistance in anesthetized dogs (13). Thus, it is expected that genioglossal muscle stimulation also improves upper airway patency in humans and may be applicable to the treatment of OSAS. In this study, we examined the effects of percutaneous electrical stimulation of the genioglossus in patients with OSAS using an apnea demand-type stimulator and investigated the possible applicability of this procedure in the treatment of OSAS.

Methods

Subjects

Six patients (five male, one female) with OSAS previously diagnosed by polysomnography, none of whom had obstructive lung disease or clinical evidence of heart disease or clearly definable upper airway abnormality on clinical examination, were selected for this study. They were all snorers, and four of them had mild daytime sleepiness and slight morning headache. There were two patients with marked obesity, defined as a body weight of at least 150% of ideal. The patients' anthropometric data are summarized in table 1. Skin thick-

SUMMARY We examined the effects of percutaneous electrical stimulation of the genioglossus in six patients with obstructive sleep apnea syndrome (OSAS) during sleep and investigated the possible applicability of this procedure as a treatment of OSAS. Six patients with OSAS were polysomnographically studied in the supine position during all-night sessions with and without electrical stimulation of the genioglossus. Using an apnea demand-type stimulator that we developed, electrical pulses of 0.5 ms (repetition rate, 50 Hz) and 15 to 40 V were delivered through bipolar electrodes (10 mm in diameter) attached to the skin of the submental region when apnea lasted more than 5 s, and was stopped immediately after breathing resumed or after 10 s at the longest. With submental stimulation, the apnea index, apnea time/total sleep time, longest apnea duration, and the number of times per hour that oxygen saturation dropped below 85% decreased significantly compared with those on control nights. The lowest arterial oxygen saturation and the duration of sleep stages III and IV increased significantly. The stimulation employed did not cause arousal, and it did not affect blood pressure or heart rate significantly. These findings show that submental stimulation decreases the incidence of apnea episodes and promotes deeper sleep without accompanying serious side effects, suggesting that the apnea demand-type stimulator may be a noninvasive and effective treatment for OSAS.

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ness measured by calipers in the submental area was approximately 0.8 cm. The apnea index is defined as the number of apneic episodes (> 10 s in duration) per hour over a full night's sleep (2). Each patient gave informed consent to the protocol, which had received prior approval from the Human Research Committee at our institution.

Sleep Studies

Overnight sleep studies were performed using standard polysomnographic techniques, including recording of the electroencephalogram (EEG) (C_4/A_1 ; C_3/A_2), electrooculogram (EOG), submental electromyogram (EMG), and electrocardiogram (ECG) with surface electrodes (14). Studies were performed in the supine position and monitored on television. Respiratory movements of the rib cage and abdomen with tidal volume summation were measured using inductive plethysmography (Respirace®; Ambulatory Monitoring, Ardsley, NY), calibrated using the simultaneous equations method (15). Arterial oxygen saturation was measured continuously using a pulse oximeter (Biox 3700; Ohmeda, Boulder, CO). Airflow at the nose and mouth was recorded with two thermistors. Tracheal breath sounds were also picked up by a microphone attached to the skin overlying the cervical trachea and were band-pass-filtered (200 to 1,000 Hz), rectified, and integrated to display the sound envelopes (16, 17). All variables were recorded continuously on a polygraph (Model

360; NEC San-ei, Tokyo, Japan) and data recorder (A-109; SONY, Tokyo, Japan). Sleep stages were determined according to standard criteria (14).

The pattern of breathing during sleep was evaluated as follows. Apnea was defined as the cessation of airflow at the nose and mouth, and of breath sound at the trachea lasting longer than 10 s. On the basis of the Respirace® and airflow signals, apnea was classified as: (1) central apnea when there was cessation of airflow signals with complete absence of thoracoabdominal movements, (2) obstructive apnea when there was cessation of airflow signals despite continuing thoracoabdominal movements, or (3) mixed apnea if there were both central and obstructive components during the apneic event.

Stimulation Studies

Two silver chloride ECG electrodes (10 mm in diameter) were used as stimulation elec-

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TABLE 1
ANTHROPOMETRIC AND SLEEP STUDY DATA
IN PATIENTS WITH OBSTRUCTIVE
SLEEP APNEA*

Male:female	5:1
Age, yr	50.0 ± 10.8
Height, cm	165.3 ± 5.5
Weight, kg	82.9 ± 17.7
Weight/ideal weight, %	127 ± 25
Apnea index, episodes/h	39.2 ± 18.7

* Values are expressed as mean ± SD.

trodes and attached 10 mm apart to the skin under the chin at the submental region. Stimulation consisted of 0.5 ms pulses (repetition rate, 50 Hz) of 15 to 40 volts, delivered by the apnea demand-type stimulator. The stimulator was triggered electronically 5 s after onset of apnea, which was detected by tracheal breath sounds. The sounds were picked up by a microphone attached to the skin overlying the cervical trachea and were band-pass-filtered (200 to 1,000 Hz), rectified, and integrated (16, 17). Apnea was defined as tracheal sound intensity of less than 15% of tracheal sound intensity during tidal breathing (17). Electrical stimulation was stopped after breathing resumed, or after 10 s at the longest, if electrical stimulation failed to alleviate the apnea episode. The intensity of stimulation was previously determined for each patient while awake by choosing an intensity just below the threshold of mild pain. The timing of stimulation was not dependent on the respiratory cycle. For each patient while awake, we observed the forward movement of the tongue while the mouth opened and the subjective feeling was that of the tongue being pulled forward during submental stimulation with an intensity of approximately twice the threshold.

"Control night" was defined as a night studied polysomnographically without submental electrical stimulation. "Stimulation night" was defined as a night studied using the apnea demand-type submental stimulator. All patients had an initial control night. Subsequent control and stimulation nights were randomly ordered, and more than 3 days were allowed between any two test nights. Three patients had one stimulation night, two had two, and one had three. We compared apnea time/total sleep time (%), longest apnea duration, lowest oxygen saturation, the number of times per hour that oxygen saturation dropped below 85%, and sleep stage characteristics between control nights and stimulation nights. Statistical significance was evaluated by paired *t* test, and *p* < 0.05 was considered significant. In the text, data are expressed as means ± SE.

Results

A typical polysomnogram recording obtained during the I/II nonrapid eye movement (NREM) stage of sleep from a patient with obstructive sleep apnea on a



Fig. 1. Recorder tracings on a night with submental stimulation during stage I/II non-rapid eye movement (NREM) sleep. After 1 or 2 s of submental stimulation, indicated by the solid bars, which indicate the stimulation duration, breathing resumed.

night with submental stimulation is shown in figure 1. When tracheal breath sounds (displayed as sound envelopes) ceased for more than 5 s, submental stimulation via the skin was started. Duration of each stimulation is indicated by a solid bar. After 1 or 2 s of submental stimulation, breathing resumed, at which point stimulation was immediately stopped. On this stimulation night, the apnea index decreased from 52.1 to 19.2 episodes/h.

We compared sleep apnea episodes on control nights with those on submental stimulation nights; figure 2 shows individual apnea index, longest apnea duration, lowest oxygen saturation, and the number of times per hour that oxygen saturation dropped below 85% on control and stimulation nights. Apnea index decreased from 39.2 ± 7.7 to 11.7 ± 4.9 episodes/h (*p* < 0.001). Longest apnea duration decreased from 57.7 ± 7.9 to 30.7 ± 4.2 s (*p* < 0.01). The number of times per hour that oxygen saturation dropped below 85% decreased from 20.1 ± 10.8 to 4.1 ± 2.9 /h (*p* < 0.05). The lowest arterial oxygen saturation increased from $75.5 \pm 3.8\%$ on control nights to $82.7 \pm 2.9\%$ on submental stimulation nights (*p* < 0.005). Apnea time/total sleep time (%) decreased from 29.7 ± 8.1 on control nights to 6.6 ± 3.6 on submental stimulation nights (*p* < 0.005).

The effects of submental stimulation on the frequencies of the three types of apnea are shown in figure 3. Submental stimulation greatly reduced the frequency of obstructive apnea from 29.3 ± 3.8

to 2.0 ± 0.7 episodes/h (*p* < 0.001). Mixed apnea decreased from 7.7 ± 3.9 to 6.9 ± 3.9 episodes/h (*p* < 0.05). In contrast, with submental stimulation, the frequency of central apnea increased 2.2 ± 0.7 to 2.8 ± 0.8 episodes/h, but the difference was not significant.

The effects of submental stimulation on the distribution of sleep stages are shown in figure 4. Stages I and II decreased significantly from 72.3 ± 6.6 to $62.9 \pm 7.9\%$ (*p* < 0.05), whereas stages III and IV increased significantly from 14.3 ± 3.5 to $22.0 \pm 4.8\%$ (*p* < 0.05) on stimulation nights. There was no difference in rapid eye movement (REM) stages from 13.4 ± 3.9 to $15.1 \pm 3.5\%$ with submental stimulation. None of the subjects was awakened by the stimulation, and none complained of skin pain or any other feeling of discomfort the following morning.

We electrically stimulated other parts of the body (arm and leg) as sham experiments under the same stimulating conditions except for the region stimulated. However, in these cases, breathing did not resume. Electrical stimulation did not produce an arousal in EEG and/or a change in the sleep stage. Furthermore, we measured blood pressure by sphygmomanometer and heart rate during wakefulness. Mean blood pressures were 92 ± 2 mm Hg without stimulation and 92 ± 4 mm Hg during stimulation. Heart rates were 67 ± 3 beats/min without stimulation and 66 ± 3 beats/min during stimulation. We could find no significant changes in

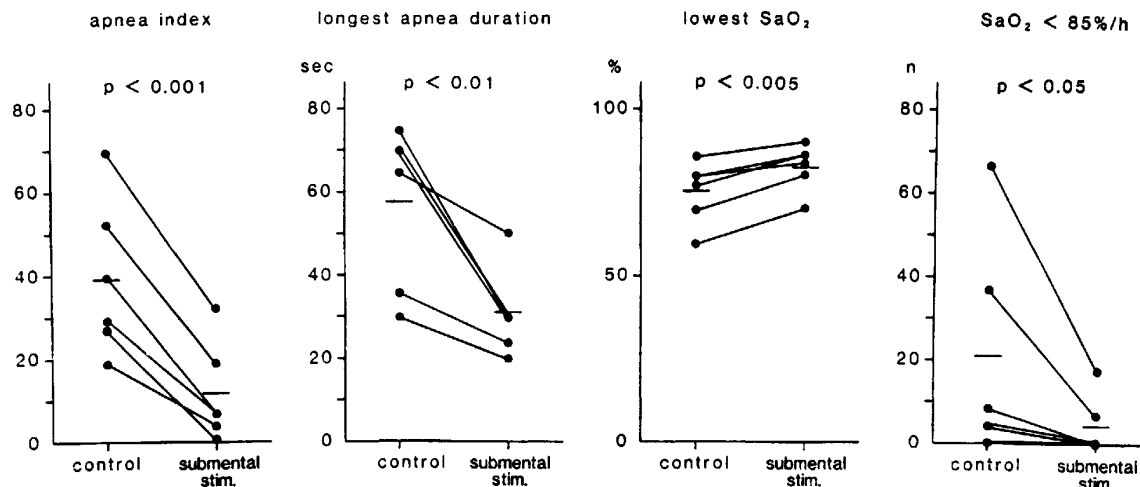


Fig. 2. Effect of submental stimulation (submental stim.) on apnea index, longest apnea duration, lowest oxygen saturation (SaO_2), and the number of times per hour that oxygen saturation dropped below 85% ($\text{SaO}_2 < 85\%/h$). Short horizontal lines represent mean values for six patients. In all patients, submental stimulation decreased apnea index, longest apnea duration, and $\text{SaO}_2 < 85\%/h$. Stimulation increased lowest SaO_2 .

blood pressure or heart rate produced by possible indirect stimulation of the carotid bodies on both sides.

The results from patients who experienced two or three stimulation nights were reproducible.

Discussion

We studied the effects of electrical stimulation of the submental region on sleep apnea episodes in patients with OSAS. Submental stimulation decreased the fre-

quency of apnea episodes, apnea time/total sleep time, and the longest apnea duration. This resulted in a decrease in the number of times per hour that oxygen saturation dropped below 85% and an increase in arterial oxygen saturation. Moreover, submental stimulation decreased sleep stages I and II, and increased stages III and IV. These findings suggest that submental stimulation using the apnea demand-type stimulator may be an effective treatment for OSAS.

In the present study, to detect apnea episodes for triggering the stimulator, tracheal breath sounds were recorded with a microphone attached to the skin overlying the cervical trachea (16, 17). This is more convenient than using thermistor type flow indicators since we would have had to monitor both nasal and oral air flow, because mouth breathing frequently occurs in the absence of nasal breathing and vice versa. Although with inductive plethysmography it is possible

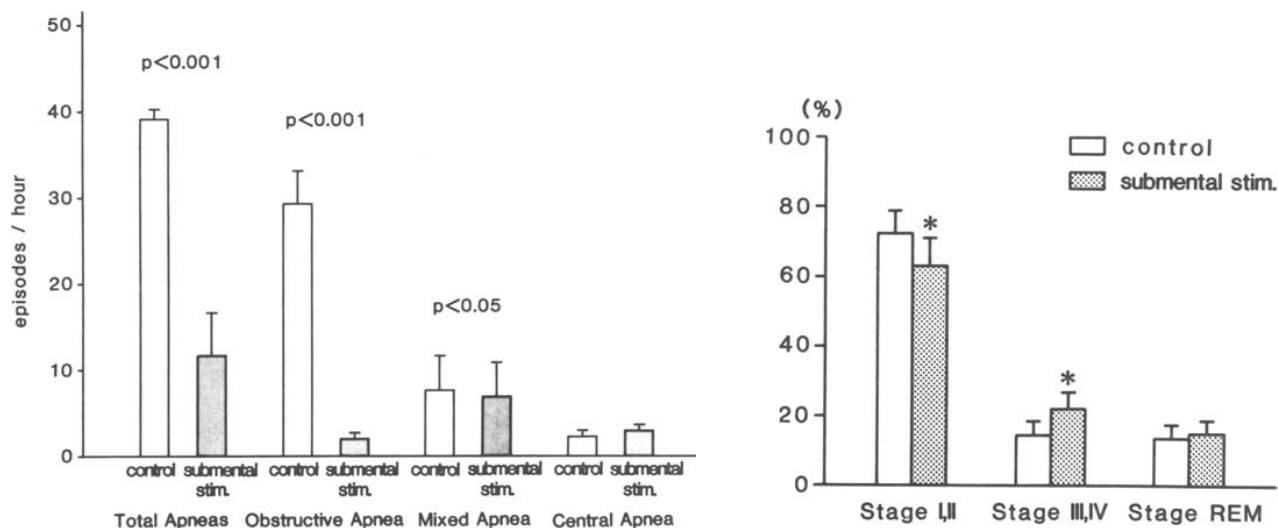


Fig. 3 (left). Differential effects of submental stimulation on the frequency of central, mixed, and obstructive apnea types in six patients. Data are expressed as the mean $\pm$ SE. Submental stimulation greatly reduced the frequency of obstructive apnea, but did not decrease central apnea.

Fig. 4 (right). Difference in sleep stage characteristics between control and submental stimulation nights. Data are expressed as the mean $\pm$ SE. Submental stimulation decreased stages I and II and increased stages III and IV.

to detect apnea episodes from zero sum signals, the method is still somewhat burdensome. A microphone can easily detect the presence or absence of tracheal breath sounds, and thus indicate apnea.

Because it has been reported that patients with OSAS tend to have more apneas in the supine position than in the lateral position during sleep, postural change could affect the results. However, from television monitoring we know that all the sleep studies were done basically in the supine position. Therefore, we believe that postural effects on apnea episodes were negligible in this study.

Harper and Sauerland (3) mentioned the possibility that upper airway obstruction is aborted by artificial activation of the genioglossus. We electrically stimulated via the skin the submental region of patients with OSAS. The subjective feeling during submental stimulation was that of the tongue being pulled forward, and the forward movement of the tongue with the mouth open was observed during submental stimulation with an intensity twice the threshold while awake. Therefore, it is likely that submental stimulation via the skin stimulates mainly the genioglossus. However, some other muscles, principally the geniohyoid and mylohyoid muscles, may have also been recruited during submental stimulation. Geniohyoid muscle activity as well as genioglossal muscle activity is thought to play an important role in maintaining upper airway patency (18).

There are several points in determining the effectiveness of submental stimulation on apnea episodes. First is the stimulation frequency (pulse repetition rate). The electrical stimulation technique has been applied to various skeletal muscles such as the adductor pollicis, the quadriceps, the sternomastoid, and the diaphragm (19–22). Muscle function, expressed by force frequency curves, is nearly identical in these muscles. We therefore assume that force frequency curves of the upper airway muscles are similar to those of other skeletal muscles. From these force frequency curves, the absolute force reaches a maximum at stimulation frequencies near or above 50 Hz (22). In our recent work we also found that electrical stimulation of genioglossi in anesthetized dogs decreased with increase in stimulation frequency, and approached a plateau at a minimum value above 50 Hz (13). However, in a pilot study, stimulation with frequencies higher than 50 Hz was found to be painful.

We therefore chose 50 Hz as a standard stimulation rate.

A second point is pulse duration. It has been reported that there is a marked increase in the tension evoked when the pulse duration is increased to the range of 0.2 to 1.0 ms and a plateau is reached at a duration of 1.0 ms (23). Longer durations do not affect the force appreciably but do increase discomfort. We chose a pulse duration of 0.5 ms to allow a low enough stimulating voltage to be used to minimize the potential skin irritation produced by electrical stimulation.

A third point is timing of the onset of stimulation. In three of the patients with OSAS, we started submental stimulation when apnea lasted more than 10 s. Arterial oxygen saturation often dropped below 90%. Stimulation after 5 s from the onset of apnea prevented oxygen saturation from dropping significantly, and so was more effective than stimulation beginning after 10 s of apnea.

A fourth point is the duration of stimulation. Submental stimulation was stopped immediately after breathing resumed or after 10 s at the longest. If the central portion of mixed apnea lasted more than 15 s, the submental region was stimulated only during periods of no respiratory movement. In such cases, even if the upper airway was open, breathing would not be resumed because of a lack of respiratory movement. In this study, submental stimulation greatly reduced the frequency of obstructive apnea, but did not decrease central apnea episodes. This may be the reason why the apnea index did not decrease to the normal range (apnea index < 4 episodes/h). In two patients, we tried continuous stimulation, but it was less effective than intermittent stimulation. Further experiments are necessary to find the optimal duration of stimulation.

A fifth point regards the intensity of stimulation. We first set the intensity of stimulation for each patient below the pain threshold while awake. Subjects did not wake up because of the stimulation, and did not complain of skin pain or any other feeling of discomfort on the following morning. Some patients said that they slept well. Indeed, stages I and II decreased significantly and stages III and IV increased significantly on submental stimulation nights. This suggests that submental stimulation via the skin with the intensity determined by the method described here promoted deeper sleep rather than inducing an arousal effect.

In this study, during some apnea epi-

sodes (less than 10% of total episodes) submental stimulation did not open the upper airway, even during obstructive phases. A possible explanation for this is that the intensity of genioglossal muscle stimulation was insufficient to improve the balance between activation of the upper airway and chest wall inspiratory muscles and opening of the upper airway. Furthermore, failure of stimulation to terminate obstructive apneas may be dependent on the sleep stage. The genioglossal muscle activity during NREM apneas decreases but does not cease. In contrast, this muscle activity during REM apneas almost ceases. Therefore, we speculate that the stimulating intensity required to open the upper airways for REM apneas is greater than for NREM apneas. However, in the present study, we found no significant differences in termination of obstructive apneas between those occurring during NREM and REM sleep. Possibly the pre-set intensity of stimulation was above the threshold necessary to open the obstructed upper airway during both NREM and REM sleep.

Skin thickness in the submental area may limit the effectiveness of stimulation. Although submental skin thickness in two grossly obese patients (0.9 to 1.2 cm) tended to be thicker than in other patients (0.6 to 0.8 cm), the obese patients did not significantly differ in their response to electrical stimulation compared with the other patients. The intensity of stimulation set while awake, however, tended to be greater in the former than in the latter. There are possibly other factors that were not addressed by the present study, e.g., mucosal secretion and surface adhesion. Mucosal secretion increases upper airway resistance. If tight surface adhesion in the pharynx occurs during apnea, it becomes hard to open the upper airway and a higher intensity of stimulation may be required to open the obstructed upper airway. If the stimulation intensity were increased, the upper airway might be opened, but at the same time might arouse or awaken the patient.

Sham electrical stimulation performed in other parts of the body did not initiate breathing. We also examined, in one patient with central sleep apnea syndrome, the effect of submental electrical stimulation using this apnea demand-type stimulator during central sleep apnea episodes. However, stimulation did not decrease the frequency or duration of apnea episodes. These findings suggest that the improvement of upper air-

way patency by electrical stimulation is not due to arousal.

Recent studies suggest that airway obstruction occurs when airway collapsing forces caused by inspiratory suction overcome pharyngeal airway dilating forces associated with inspiratory muscle activity (10). It has been proposed that an imbalance in the activity of upper airway dilating muscles (like the genioglossus) and the diaphragm might lead to airway obstruction in humans, particularly during sleep (1, 7, 24). Studies in animals and human subjects have shown that inspiratory activity, particularly tonic activity, of various upper airway muscles decreases from wakefulness to sleep (3, 25–27). In addition, studies in animals and human subjects have established that upper airway resistance increases during sleep (28, 29). Moreover, Hudgel and coworkers (30) found a nonlinear relationship between activation of the upper airway and chest wall inspiratory muscles over a wide range of central respiratory neural output in human subjects, and a close relationship between the ratio of upper airway to chest wall inspiratory muscle activity and upper airway resistance. These findings are consistent with the hypothesis that patients with OSAS have attenuated upper airway muscle tone during sleep. On the other hand, Anch and associates (31) have reported that pharyngeal dilator EMG activity is increased during occluded breathing in sleeping obese patients. However, in both cases, it appears that the dilating force of the upper airway muscles is not sufficient to keep the upper airway patent during obstructive sleep apneas. Therefore, electrical activation of upper airway muscles is expected to improve upper airway patency in various pathophysiologic conditions that cause obstructive sleep apneas (3).

In three patients, we examined the effects of submental stimulation using this apnea demand-type stimulator on two or three separate nights and found reproducible results. However, long-term effects remain under investigation. Our results suggest the possibility that this apnea demand-type stimulator may be used to treat OSAS as an alternative to surgery in an emergency or in the sleep clinic.

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