

Functional electrical stimulation and respiration during sleep

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DECKER, MICHAEL J., JOHN HAAGA, JEAN L. ARNOLD, DONALD ATZBERGER, AND KINGMAN P. STROHL. *Functional electrical stimulation and respiration during sleep*. J. Appl. Physiol. 75(3): 1053–1061, 1993.—During obstructive sleep apnea (OSA), respiratory activation of upper airway muscles, particularly genioglossus, is ineffective during sleep. Functional electrical stimulation (FES) of muscles reportedly reduces the number and length of OSAs. Our goals were to examine the effect of FES on sensation during wakefulness and on OSA events. Studies were performed in 11 subjects: 4 healthy asymptomatic subjects and 7 patients with OSA. Surface electrodes placed on the submental region produced discomfort; however, during sleep, the stimulus intensity producing arousal was significantly greater than that producing barely tolerable discomfort during wakefulness. Additionally, we developed a protocol for placement of fine-wire electrodes into the neurovascular bundle of the hypoglossal nerve, using recognizable radiographic features and computerized axial tomography as guides. In these patients, while awake, optimal wire placement was associated with visible tongue protrusion without discomfort. With both surface stimulation and fine-wire FES, during sleep the stimulus intensity required to produce obvious electroencephalographic arousal was significantly greater than that producing a barely tolerable sensation while awake. During apneic events, the application of surface stimulation had an inconsistent effect, terminating 22% of the apneas, and fine-wire FES also had a limited impact, terminating 23% of the apneic events. We conclude from our studies that subjects tolerate surface and fine-wire FES to higher stimulus parameters during sleep than during wakefulness but that both approaches have an inconsistent effect on apneas during sleep.

sleep apnea; upper airway muscles

DISORDERS OF RESPIRATORY ACTIVATION of muscles include not only those involving chest wall muscles, such as the intercostals or diaphragm, but also those involving upper airway muscles, such as the genioglossus. A reduction in upper airway activation often precedes obstructive sleep apnea (OSA). In this relatively common condition, respiratory activation of pharyngeal muscles, in particular the genioglossus, is ineffective during sleep for the purpose of maintaining upper airway patency. Conceivably, electrical stimulation of these muscles could restore their tone and reestablish upper airway patency.

The concept of electrically stimulating upper airway muscles is not new. Indeed, we and others (4, 6, 8) have demonstrated that electrical stimulation of the genioglossus and/or other pharyngeal muscles will reduce up-

per airway resistance, increase pharyngeal stability, and improve tidal volume in anesthetized animals. In 1989, Miki et al. (5) reported that electrical stimulation applied to the skin in the submental region reduced the number and length of OSAs in humans; however, the mechanism and potential clinical problems of this approach were not reported in any detail. For instance, arousal responses from sleep could have contributed to the observed beneficial effect. Nevertheless, this demonstration suggested a possible role for functional electrical stimulation (FES) in the management of OSA.

The goals of this study were to compare the effect of FES on sensation during wakefulness with its effect on producing arousal or reversal of obstructive apneic events during sleep. To this end, FES was studied in normal subjects and in patients with moderately severe OSA. Studies were performed during wakefulness and sleep.

METHODS

Studies were performed in 11 subjects: 4 healthy subjects and 7 patients with moderately severe to severe OSA based on a desaturation index ($>4\%$ fall in oxygen saturations from baseline values per hour of sleep) of ≥ 35 , as measured during prior studies. The characteristics of those who participated in the surface stimulation protocol are presented in Table 1 and those who participated in the fine-wire stimulation protocol in Table 2. At the time of recruitment, five patients were using nasal continuous positive airway pressure (CPAP), one was using a dental prosthesis, and one was participating in a weight-loss protocol. Studies were approved by the Institutional Review Board at University Hospitals of Cleveland, and all subjects gave written consent.

Submental surface stimulation discomfort thresholds. A protocol of submental surface stimulation was performed on seven subjects. Of the seven, four were control subjects (3 males and 1 female), who reported that they were in a good state of health at the time of study and were not taking any prescribed or over-the-counter medications. The other three were patients previously diagnosed with OSA. Of these three, two were using nasal CPAP and were asked to refrain from using their machines during the night before the trials.

The stimulation device was a Medtronic DYNEX III neurostimulator unit (TENS device). The DYNEX delivers a square-wave stimulus with an adjustable current

TABLE 1. Surface stimulation subject data

Subj No.	Sex	Height, in.	Weight, lb.	Age, yr	Subject Status	Desaturations/Hour of Sleep
1	M	76	182	22	Healthy volunteer	
2	M	68	280	24	Healthy volunteer	
3	M	72	267	27	Healthy volunteer	
4	F	64	130	28	Healthy volunteer	
5	M	64	285	35	Sleep apneic, uses CPAP	100
6	M	74	312	52	Sleep apneic, uses CPAP	75
7	M	72	220	46	Sleep apneic, uses CPAP	66

CPAP, continuous positive airway pressure.

amplitude. The pulse frequency was maintained at 50 Hz and the pulse width at 50 μ s. Connected to the DYNEX were two standard electroencephalographic (EEG) surface electrodes with a 1 cm diameter (Nicolet, Madison, WI). On the basis of the work of Miki et al. (5), we chose to place one electrode over the stylohyoideus and one electrode over the mylohyoideus. Both of these were the working electrodes. The indifferent electrode for each working electrode was another EEG surface electrode placed 50 mm laterally to the working electrode, just below the angle of the mandible. The electrodes were held in place with collodion solution, and each electrode was filled with transduction cream.

Once the electrodes were in place, stimulation sensitivity thresholds of "first felt," "uncomfortable but tolerable," and "barely tolerable" were established while the subject was awake. Before any stimulation was done, the subjects were told that they would feel a "tingling" sensation. The subjects were instructed to verbally respond as soon as they felt any sensation. The neurostimulator was turned on, and the pulse rate and pulse width were maintained at the previously described levels. The current was increased incrementally until the subjects stated that they felt the tingling sensation. As soon as the tingle sensation was noticed, the stimulator was switched off and the current required to generate the tingle, hereafter called the first felt response, was recorded.

After the first felt response was elicited, the subjects consented to establish an uncomfortable but tolerable threshold. To do this, the neurostimulator was turned on (the previous pulse width and frequency settings were maintained) and the current was increased to the point at which the subject felt that the stimulus was very uncomfortable but not painful. Once again the stimulator was switched off, and the current required to initiate the verbal response of uncomfortable but tolerable was recorded.

Finally, the subjects consented to establish a barely tolerable threshold. While following the previously described steps, the subject was instructed to verbally respond when they felt that the discomfort related to the

stimulation had reached a barely tolerable level. Then the stimulator was turned on and the current increased until the subject indicated that it had reached a barely tolerable discomfort level. The stimulator was switched off, and the current level was recorded. In three of the subjects, this protocol was performed on the day before the surface stimulation protocol and during the day of the protocol.

Protocols during sleep. After thresholds for skin surface stimulation were established, the subject was prepared for polysomnography and allowed to fall asleep. Polysomnographic variables monitored were EEG (C_3-A_2), electrooculogram (EOG; FP_1-FP_2), and electromyogram (P_3-P_4). Airflow was measured by a Fleisch 1.10 pneumotachograph attached to a Validyne pressure transducer (± 2 cmH₂O). The pneumotach was attached to a face mask (Life Design Systems) that covered nose and mouth. Mask pressure was measured by connecting a tube from the pressure port in a nasal face mask to a Validyne pressure transducer (± 50 cmH₂O). Thoracic and abdominal respiratory movements were measured by inductance plethysmography bands positioned around the thorax and abdomen. Oxygen saturation was measured by a Criticare 501+ pulse oximeter. All measurements were recorded on a Grass polygraph at a paper speed of 10 mm/s.

Once the subject was instrumented and in stage II or stage III sleep, submental surface stimulation was initiated. In all seven subjects on whom surface submental stimulation was performed, the initial stimulating parameters were set below those established as first felt. Then, while asleep, the subject was given periodic bursts of submental stimulation while the EEG channel was monitored for any changes indicative of arousal. If low-level stimulating intensities did not alter the airflow or the EEG signal, the current was raised incrementally and the subject was again stimulated.

In the healthy subjects ($n = 4$), to determine the effects of submental stimulation on airflow, pacing bursts were applied during either the inspiration or expiration and current was continually applied for one full respiratory

TABLE 2. Fine-wire stimulation subject data

Subj No.	Sex	Height, in.	Weight, lb.	Age, yr	Subject Status	Desaturations/Hour of Sleep
5	M	64	285	35	Sleep apneic, uses CPAP	100
8	M	75	188	40	Sleep apneic, uses CPAP	52
9	M	74	250	55	Sleep apneic, dental prosthesis	35
7	M	72	220	46	Sleep apneic, uses CPAP	66
10	M	68	192	64	Sleep apneic, weight loss	55
11	M	71	240	52	Sleep apneic, uses CPAP	77

cycle. In the sleep apneic patients ($n = 3$), submental stimulation was applied during apneic events. During some apneas, stimulation was applied immediately on cessation of airflow. At other times, stimulation was not applied until several seconds after the apneic event had begun. In both the healthy and apneic subjects, during the course of the protocol, the stimulating current was increased until it produced polysomnographic evidence of arousal, as defined by observed alpha waves (8–13 cycles/s).

Fine-wire FES. Six patients previously documented with OSA consented to have fine-wire electrodes implanted submentally for the purpose of direct hypoglossal nerve stimulation. Of these six, two had previously completed the submental surface stimulation protocol. All patients were asked to refrain from using their nasal CPAP ($n = 4$) or dental prosthesis ($n = 1$) therapy during the night before the trials. One patient (*patient 10*) had been prescribed nasal CPAP but was unable to tolerate therapy; he was undergoing supervised weight loss.

Stimulation was produced by a Grass single-channel stimulator that outputs a monophasic square wave with a variable voltage, pulse width, and frequency. Attached to the stimulator were fine-wire electrodes that were made from stainless steel wire (Medwire, New York, NY). Each wire electrode had its insulation removed from each end. The wire was inserted into a 25-gauge spinal needle so that the deinsulated end of the wire protruded through the beveled end of the spinal needle. The wire that protruded from the needle was bent back to provide a "barb." The needles were then capped and gas sterilized.

A Somatom-Plus computerized axial tomography (CAT) scanner was used for localization of anatomic structures and bilateral placement of the stimulating electrodes. Scans were permitted in a total of six patients to a total of no more than 10 rads of radiation per patient. After radiographic localization of the neurovascular bundle was performed, the skin surface over the mandibular notch two-thirds of the way from the angle of the mandible to the chin was anesthetized with lidocaine (2% solution), and the needle was advanced in 2- to 3-mm increments for anesthetic infiltration. The anesthetizing needle was withdrawn, and a 25-gauge spinal needle with the electrode positioned in its lumen was advanced along the same tract. An image was taken and recorded to localize the tip of the needle and its position relative to the neurovascular bundle at 3-mm increments (Fig. 1, A and B). Once satisfactory placement was observed, the needle was withdrawn, leaving the electrode in place. An additional image was taken and recorded to verify that the tips of the electrodes were in the desired area (Fig. 1C).

Once the fine-wire electrodes were securely positioned and the effects of the lidocaine anesthetic had subsided, a range of stimulating parameters required to elicit subjective responses of first felt and barely tolerable, as well as the objective response of muscle recruitment with tongue protrusion, was obtained. This was done during wakefulness by following the same protocol as that for submental surface stimulation.

Availability of the scanning facility and allowable radiation exposure permitted us to return two patients ~2 h after fine wire insertion to radiographically assess the

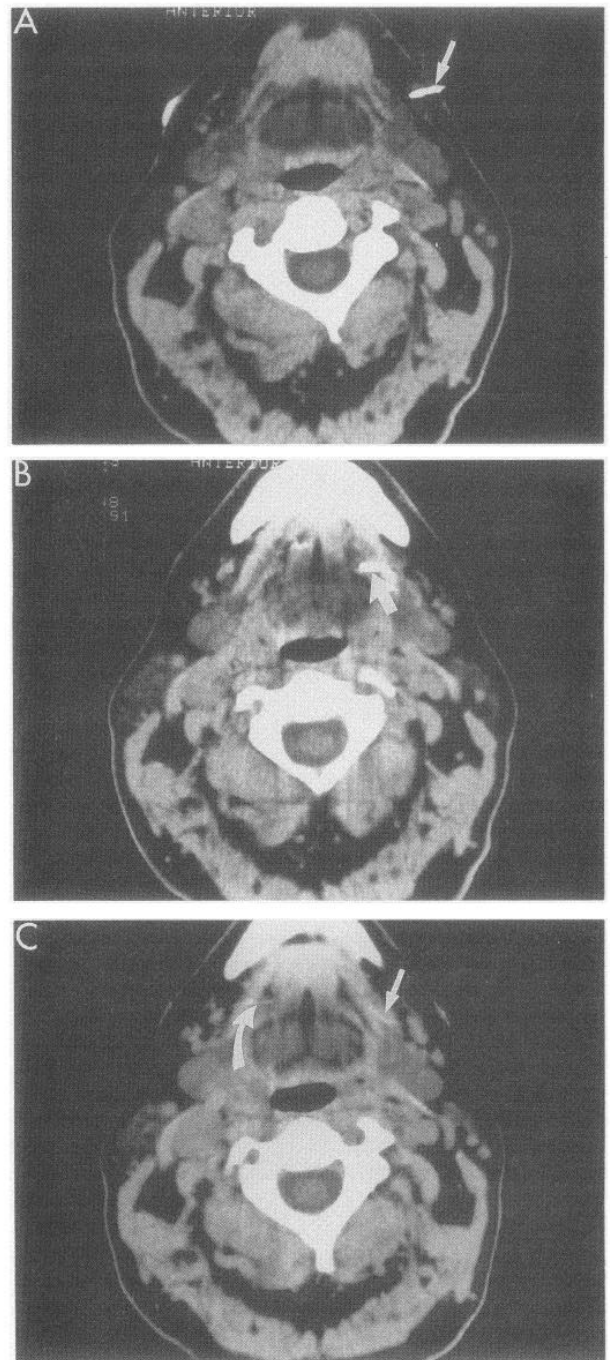


FIG. 1. A: computerized axial tomography (CAT) scan of hypopharynx during electrode placement shows entrance site of needle (arrow) on left side of patient's neck. Needle lies within subcutaneous tissue and is being directed cephalad (see B). B: CAT scan taken at higher level shows tip of needle lying within area of neurovascular bundle behind genioglossus muscle (arrow). Note neurovascular bundle on right side. Margins of artery and vein can be seen, but nerve is not visible. C: final CAT scan of hypopharynx after electrode placement. Note that electrode (curved arrow) on right side of patient is properly positioned through area of neurovascular bundle, with tip slightly in genioglossus muscle. Electrode on left side of patient (arrow) is positioned within mylohyoid muscle and may be too superficial to appropriate structures.

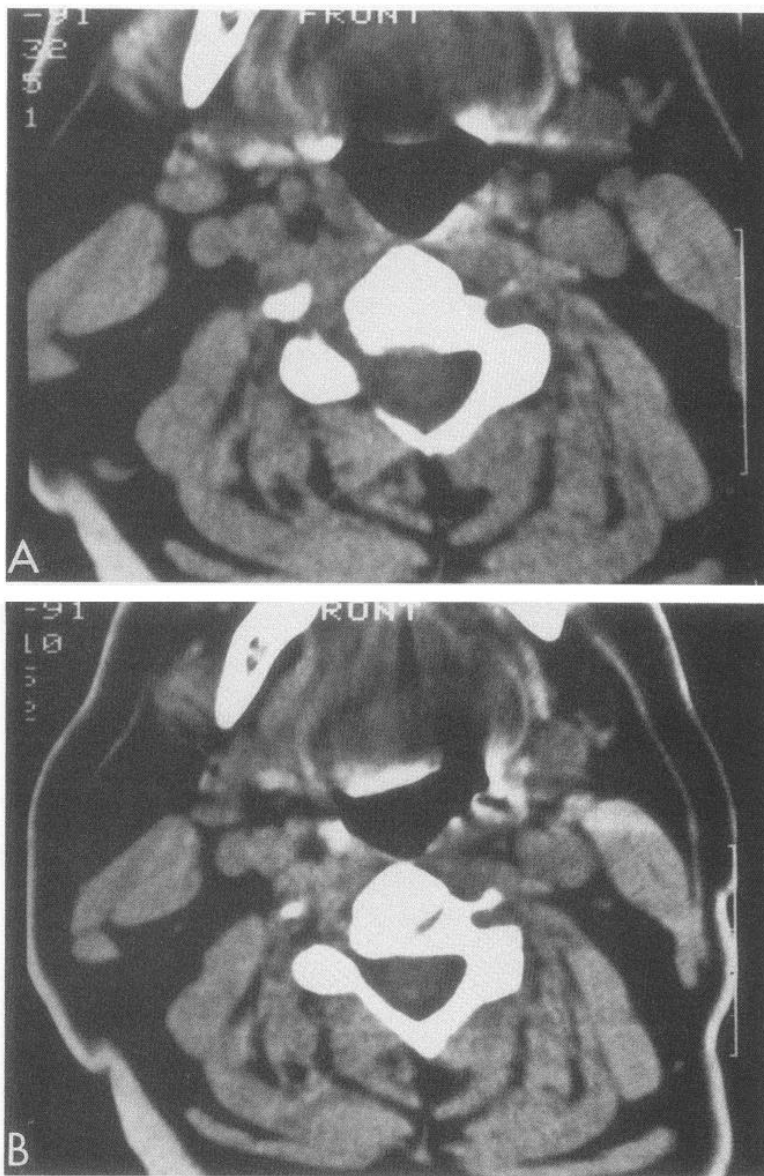


FIG. 2. CAT scan on different patient shows change in configuration of hypopharynx before stimulation (A) and during stimulation (B). Note 24% decrease in upper airway area in B compared with A.

functional response of FES applied to the hypoglossal nerve. Once in the scanner the patients were placed supine and breathed through the nasal route, and at end expiration we electrically stimulated the hypoglossal nerve. A limited number of images were obtained under conditions of no stimulation and during stimulation. These are shown in Figs. 2 and 3. The images were digitized to provide values of upper airway area.

After the stimulation sensitivity thresholds had been established during wakefulness, subjects were prepared for polysomnography and allowed to fall asleep. Once asleep, patients usually began having apneic events within 10–30 min. At this point, the stimulation was initiated, using the same protocol described for the submental surface stimulation. The initial settings were a pulse frequency of 50 Hz with a 0.2-ms pulse width. The 0.2-ms

pulse width was used to minimize stimulation of unmyelinated pain and smaller myelinated sensory fibers. The initial stimulating voltage was set below that required to elicit the first felt response during wakefulness. FES was applied during apneic events. After two to three apneas in which there was no response to FES, the stimulating voltage was increased by 1 V. Throughout the study, the voltage was incrementally adjusted upward until FES caused EEG arousal.

Data analysis. To determine whether a significant increase in submental surface stimulating current was required to move from the thresholds of first felt to uncomfortable but tolerable to barely tolerable, we statistically compared the difference in stimulating currents at each threshold. We then compared the difference between the current that caused arousal from sleep and that causing

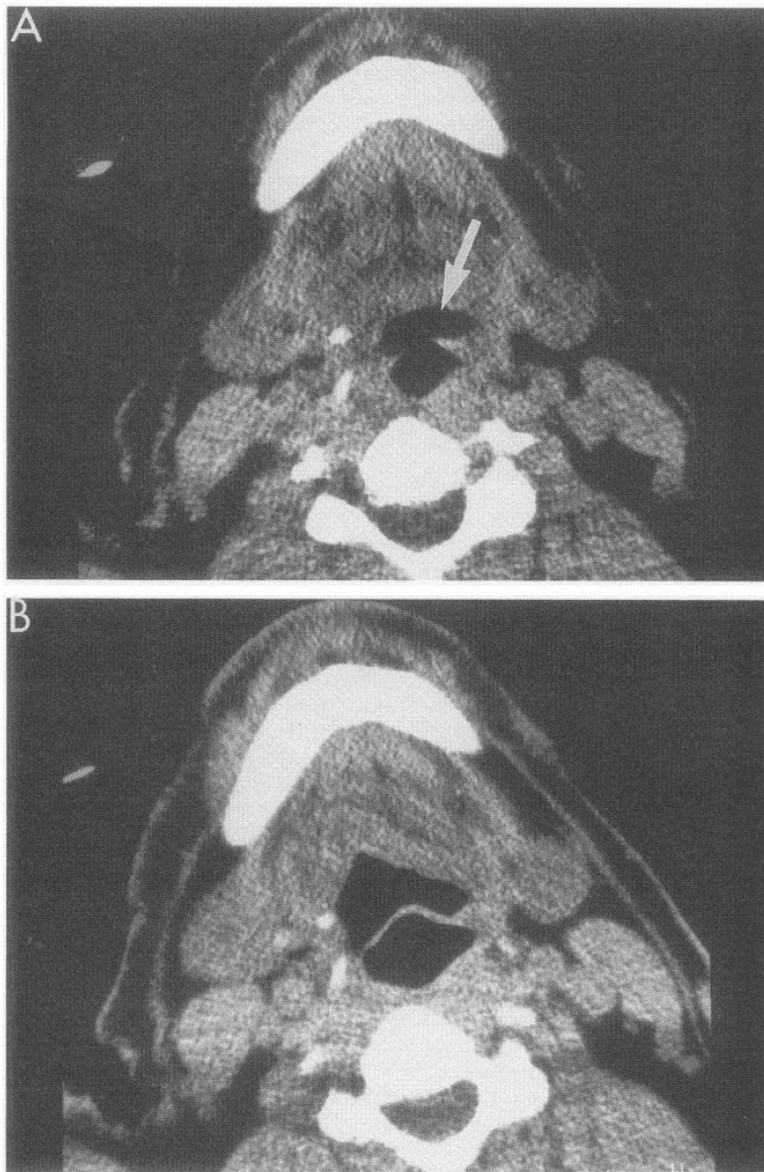


FIG. 3. A: CAT scan taken during prestimulation period shows normally shaped but small hypopharynx (arrow) before stimulation. B: repeat CAT scan taken during stimulation shows significant enlargement of hypopharynx. Note 116% increase in upper airway area in B compared with A.

the barely tolerable sensation while awake. For fine-wire stimulation we compared the voltages required to move from the first felt threshold to that causing tongue protrusion to that of barely tolerable. We also compared the difference in voltage between that causing arousal from sleep and that causing the barely tolerable sensation while awake.

To evaluate the effect of submental surface stimulation on airflow rates in healthy subjects during sleep, in each subject we measured 25 peak inspiratory airflow rates for the breath before, as well as during, submental surface stimulation. The peak airflow rates from the prestimulated breaths were compared with the peak inspiratory airflow rates measured during submental surface stimulation. We evaluated only those breaths in which the stimulating current was equal to or greater

than that causing an uncomfortable but tolerable sensation during wakefulness.

To evaluate the effect of submental surface stimulation on upper airway patency in sleep apneic patients, we evaluated the effects of submental surface stimulation applied during an apneic event. We evaluated only those apneic events in which the stimulating current was equal to or greater than that causing an uncomfortable but tolerable sensation during wakefulness. Comparisons were made between the number of apneas in which surface stimulation was applied and the number of breaths that occurred during FES.

To determine whether fine-wire FES could restore muscle tone and reestablish upper airway patency during apneic events, we evaluated the effects of FES applied during an apnea. We evaluated only those bursts of FES

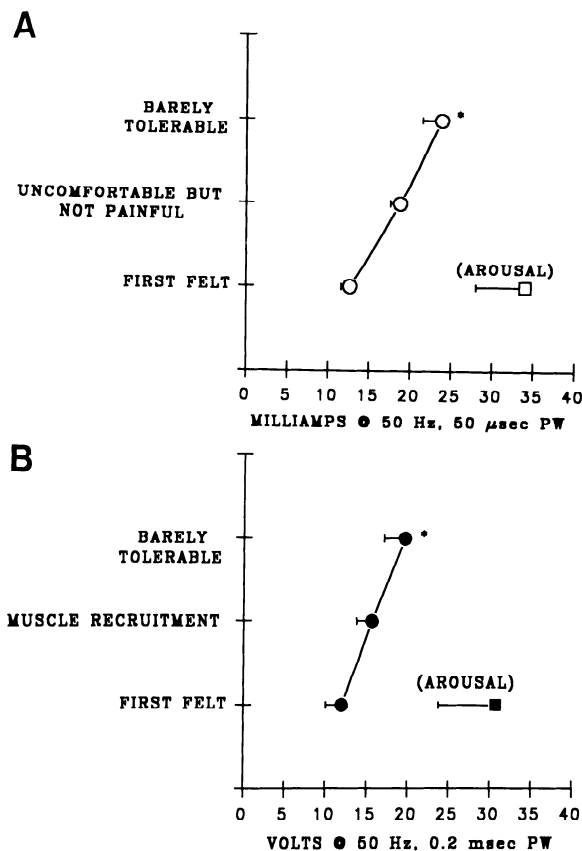


FIG. 4. Difference between stimulating parameters required to elicit first felt sensation during wakefulness to that producing EEG arousals during sleep. Mean $\pm$ SE is displayed at each level. A, surface stimulation data (in amps); B, fine-wire stimulation data (in volts). * Statistically significant difference between barely tolerable threshold and threshold causing EEG arousal from sleep.

in which the stimulating voltage was equal to or greater than that causing tongue protrusion during wakefulness. Comparisons were made between the number of apneas in which FES was applied and the number of breaths that occurred during FES.

A one-way analysis of variance was used to compare stimulus intensities required to produce the responses of first felt through arousal in both the surface stimulation and fine-wire stimulation groups. For the healthy control group, a paired *t* test determined whether statistically significant changes occurred in peak inspiratory airflow rates between the baseline breath and the following breath in which surface stimulation was applied. For both submental and fine-wire FES we used a paired *t* test to determine significance between the number of times FES was applied during apneas and the number of breaths that occurred during FES. The level for statistical significance was $P < 0.05$.

RESULTS

Figure 4A graphically displays the submental surface stimulation currents required to elicit responses of first

felt, uncomfortable but tolerable, barely tolerable, and EEG arousal from sleep. The data are displayed as means $\pm$ SE. We found a statistically significant difference in the stimulating current needed to move from one threshold to the next. Also, the current required to cause EEG arousal from sleep was significantly greater than that producing the barely tolerable sensation during wakefulness. In Fig. 4A, only the arousal stimulus for five of the seven subjects is displayed. One control subject never fell asleep, and one sleep apneic patient spontaneously awoke before the stimulating current exceeded the uncomfortable but tolerable threshold.

Figure 4B graphically displays the fine-wire voltage required to produce a first felt response, muscle recruitment with tongue protrusion, a barely tolerable response, and EEG arousal from sleep. We found a statistically significant difference in the stimulating voltage needed to move from one threshold to the next. The voltage required to cause EEG arousal from sleep was significantly greater than that causing the barely tolerable sensation during wakefulness. In Fig. 4B, data from only four of the six subjects are displayed for EEG arousal from sleep. The other two subjects had spontaneously awakened during the protocol.

While performing the submental surface stimulation protocol we were able to measure stimulating currents on two separate occasions in three of the subjects. We found no significant variation between stimulating parameters used during the first and second occasions to elicit the subjective responses during wakefulness. We found that in sleep apneic patients it was not possible to systematically assess arousal responses to FES in each stage of sleep. This was due to their frequent apneas and arousal responses that caused a fragmentation of their sleep state.

Submental surface stimulation: control subjects, sleep. In three of the four healthy subjects we were able to compare peak airflow rates measured during sleep before and during submental surface stimulation. As mentioned above, the fourth subject was not able to fall asleep. Group comparisons ($n = 75$ unstimulated to stimulated breaths) indicated that there was a small but statistically significant increase of 0.56 ± 2.31 (SD) l/min ($P < 0.05$) in inspiratory peak airflow rates between the preceding breath and the stimulated breath and a small but statistically significant decrease of 1.3 ± 4.3 l/min in expiratory peak airflow rates between the preceding breath and the stimulated breath ($P < 0.05$).

Submental surface stimulation: sleep apneic patients, sleep. We applied submental surface stimulation to three sleep apneic patients during apneic events. In two of the three patients we were able to apply current equal to or greater than that causing an uncomfortable but tolerable sensation during wakefulness. The third patient had a spontaneous arousal while the stimulating currents were still below this level. Once awake this patient did not fall back to sleep.

In two sleep apneic patients, we evaluated the effect of submental surface stimulation applied during 44 ± 24 apneic events per patient. We found that ventilation resumed during or immediately after cessation of submental surface stimulation 22% of the time.

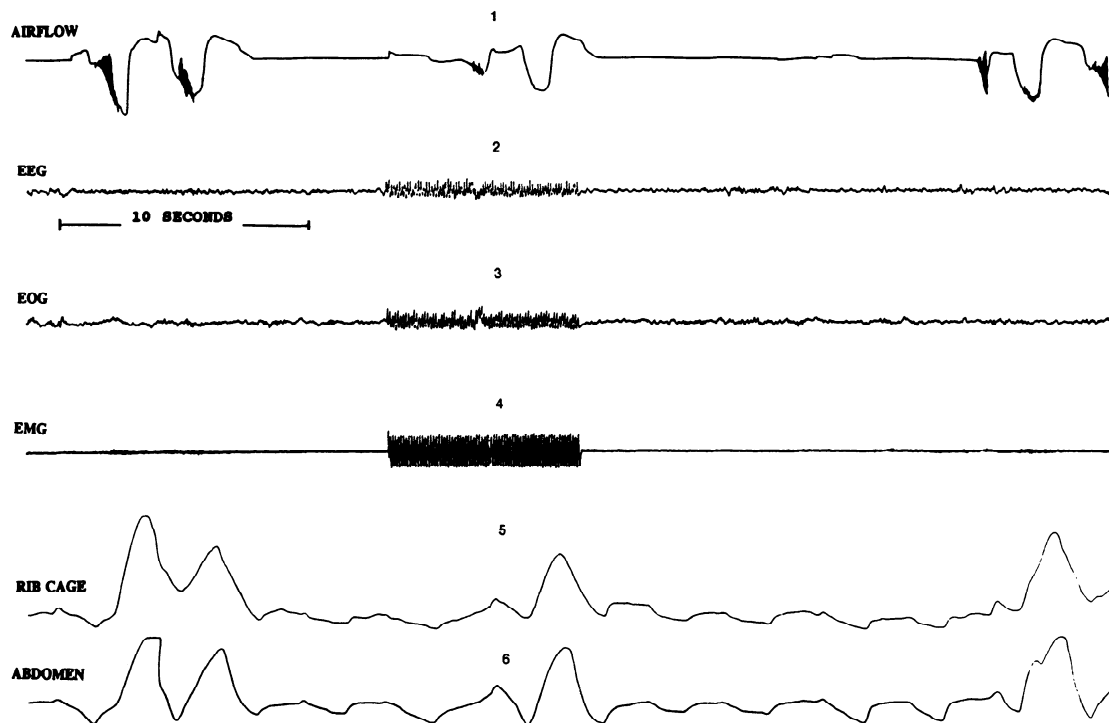


FIG. 5. Polysomnograph recording illustrating effect of fine-wire functional electrical stimulation of hypoglossal nerve. Stimulation parameters were 14 V, 50 Hz, and 0.2-ms pulse width. When stimulation was applied during apneic event, airflow resumed. Airflow then ceased with termination of stimulation. EEG and EOG channels do not show any sign of arousal associated with stimulation. EMG, electromyogram.

Fine-wire stimulation: awake CAT scan images. In all patients in whom fine-wire electrodes were inserted, once the lidocaine anesthetic subsided (~ 2 h) we were able to apply voltage to the electrodes and observe genioglossus muscle recruitment with subsequent tongue protrusion. The images presented in Figs. 2 and 3 illustrate that in the two patients who were returned to the CAT scanner for FES, upper airway configuration changed when stimulation was applied. In *patient 11* (Fig. 2), during fine-wire FES, the area of the airway decreased by 24% from its baseline value. In *patient 10* (Fig. 3), during fine-wire stimulation, the area of the airway increased by 116% from its baseline value.

Fine-wire stimulation: sleep. In six sleep apneic patients we applied FES during apneic events. In five of the six patients we were able to apply voltages equal to or greater than that causing tongue protrusion during wakefulness. The sixth patient had a spontaneous arousal while the stimulating voltages were below that voltage causing tongue protrusion. Once awake this patient could not fall back to sleep.

In the five patients in whom the voltage was equal to or greater than that causing tongue protrusion during wakefulness, we evaluated the effect of FES applied during 41 ± 26 apneic events per patient. With respect to the number of times that FES was applied, we found that ventilation resumed during or immediately after cessation of FES in 23% of the cases.

Figure 5 is one example from the polysomnographic recording from one subject (*subj 8*) whose weight was

proportional to his height. In this tracing, *channel 1* indicates a complete cessation of airflow. *Channels 5 and 6* reveal abdominal and thoracic motion, respectively, indicating this to be an obstructive apneic event. The electromyogram is displayed on *channel 4*; however, its resolution has been diminished by the photocopy reduction. The rapid pen deflections seen on *channel 4* are the stimulating bursts with electric stimulation parameters of 14 V, 50 Hz, and a 0.2-ms pulse duration.

In this example, there was an immediate resumption of airflow (as illustrated by *channel 1*) with initiation of fine-wire stimulation. As soon as the stimulation ceased, the airflow signal also ceased. Therefore, in this one example, fine-wire stimulation appeared to restore airway patency. Also, the EEG and EOG channels (*channels 2 and 3*, respectively) do not show any obvious changes or signs of arousal after FES that could have accounted for the changes in the airflow signal. Signal artifact obscures the EEG and EOG channels during FES so the presence of a microarousal cannot be determined. Although fine-wire stimulation of the hypoglossal neurovascular bundle appeared to elicit changes in the airflow signal 23% of the time, these changes were not reproducible in a systematic fashion.

Figure 6 is another example of fine-wire stimulation from the same subject. Once again fine-wire stimulation was initiated shortly after the airflow ceased. In this example, as in the previous, the airflow was abruptly restored in association with stimulation. However, when FES was terminated and its accompanying electrical in-

FUNCTIONAL ELECTRICAL STIMULATION FOR APNEA

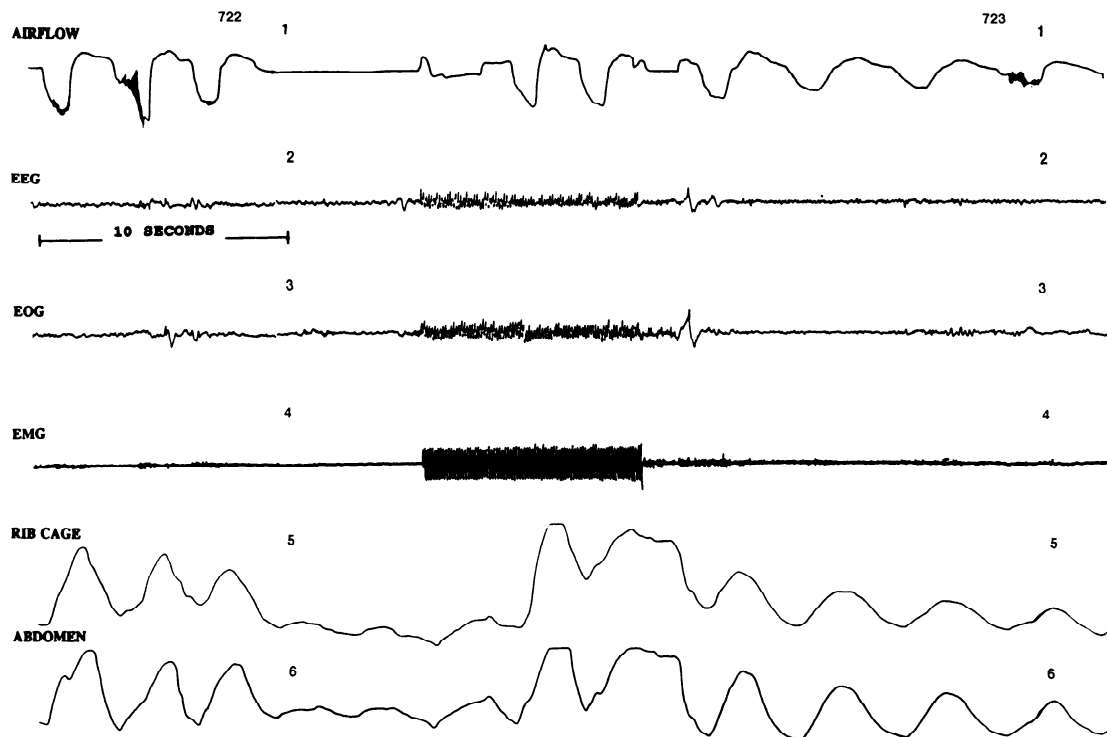


FIG. 6. Polysomnograph recording from same subject as in Fig. 5. Same stimulation parameters were used. However, EEG and EOG channels exhibit small spike that may indicate that arousal may have occurred to terminate apnea.

terference subsided, an alteration in the EEG and EOG channels was evident. This indicates that the restoration of airflow may have been the result of an arousal.

DISCUSSION

Our results indicate that 1) FES produces arousal from sleep but at higher stimulation levels than those producing barely tolerable discomfort during wakefulness; 2) percutaneously inserted stimulating wires, appropriately placed, afford the opportunity for tongue protrusion at minimal discomfort with FES; and 3) in our hands, neither surface stimulation nor fine-wire stimulation has a consistent effect in terminating obstructive apneas during sleep.

Submental surface stimulation. During wakefulness, we suspect that sensation and discomfort associated with submental surface stimulation resulted from activation of skin afferents rather than from muscle contraction (7). When the subject was asleep, submental surface stimulation produced arousal at a significantly higher intensity than that producing barely tolerable discomfort during wakefulness. This phenomenon may result from the general property of diminished responses to stimuli during sleep and may explain, in part, the tolerance of patients for submental FES, as reported by Miki et al. (5). Submental surface stimulation, as it was done in our study, had an inconsistent effect on upper airway patency. There are substantial differences in the techniques and devices employed by Miki et al. and our study;

these or other factors, such as patient selection, increased pharyngeal stability, or training, could explain the previously observed clinical utility of submental surface stimulation. However, although we could not detect any consistent effect of submental stimulation in apnea termination, our small sample size limits our speculation of its role in treating OSA.

Fine-wire stimulation. Because of frustration with submental surface stimulation, we developed the protocol for percutaneous placement of fine-wire electrodes near the neurovascular bundle of the hypoglossal nerve. There are recognizable radiographic features to identify the course of the hypoglossal nerve, and the technique of CAT scan-guided needle placement was employed to implant fine-wire electrodes at those specific anatomic sites. The wires are radiographically visible once the needle is withdrawn.

In two patients circumstances allowed us to observe changes in pharyngeal size with fine-wire FES during wakefulness. Even though both patients displayed tongue protrusion with stimulation, by CAT imaging there were differences in the size and shape of the oropharynx with FES. We believe that these changes resulted from placement of fine wires in relation to the arborization of hypoglossal bundles to different extrinsic muscles or to asymmetric excitation of the main trunk of the hypoglossal nerve that shows geographic localization of fibers to specific distal muscle groups. With regard to the latter, studies in animals have demonstrated that different nerve segments of the hypoglossal nerve cause ei-

ther tongue protrusion or retraction when stimulated (1, 2). Furthermore, unlike spontaneous nerve activation where large fibers are recruited last, with FES large fibers are recruited first (7) and are an additional source of variability with regard to wire placement. Consequently, FES could produce tongue protrusion with or without increases in size of the oropharynx; we presume this depends on the specificity and coordination of the muscles stimulated.

The effects of fine-wire FES on sleep apnea and/or obstructive apneas were not dramatic. Surface stimulation and fine-wire stimulation terminated apneic events 22 and 23% of the time, respectively. This occurred without gross evidence of arousal as defined by body movement or change in EEG state; however, an effect on arousal cannot be excluded definitively by these measures. We feel that the alleviation of approximately only one in five apneic events indicates a generally poor patient response to FES.

Our experience indicates that surface stimulation for tongue protrusion is associated with an unfavorable balance between discomfort and/or arousal from sleep and tongue protrusion and/or mechanical effect during wakefulness or sleep. These observations support those of Edmonds et al. (3), who found that submental surface stimulation had no effect on apneic events other than to terminate them by causing arousal. On the other hand, the balance is more favorable for fine-wire FES applied to the hypoglossal neurovascular bundle, since visible tongue protrusion occurs during wakefulness without discomfort. This latter approach is feasible and should be explored further. Neurophysiological localization of stimulating wires should be explored as an alternative or adjunct to radiographic placement.

We believe that our study is not definitive for the following reasons. It may be that the muscles innervated by the hypoglossal nerve may not be the key muscles involved or that FES of the hypoglossal nerve results in mechanical action with opposing effects on pharyngeal size. Also, we did not mechanically quantitate the degree of tongue protrusion or changes in oropharyngeal size during fine-wire FES; consequently, we cannot predict whether the force of muscle contraction resulting from FES would open or reopen the airway. Indeed, FES could make apneas worse if tongue retrusion occurs.

Our criteria for successful fine-wire stimulation was resumption of airflow during an apneic event and that the EEG signal maintained the same observed amplitude and frequency before and after bursts of FES. However, we cannot definitively exclude the effect of microarousals, since the gross electrical interference and signal artifact caused by FES would obscure the EEG signals during application of FES. Therefore, we cannot be confident that microarousals did not occur even during the pacing bursts when apneas were broken.

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