only speculative. Further investigation is required to substantiate our findings in the clinical setting.

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TENS Reduces Halothane Requirements during Hand Surgery

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The use of electrical stimulation to treat pain began in ancient times but received little serious attention until Melzak and Wall introduced the gate control theory of pain in 1965.[1] Since then a number of electrical stimulation devices have become available and a considerable amount of clinical research has been devoted to the study of electroanalgesia. With varying success, clinicians have attempted to provide surgical anesthesia with electroacupuncture, transcutaneous cranial electrical stimulation (TCES), and transcutaneous electrical nerve stimulation (TENS).[2–6] None of these attempts have proved sufficient or reliable as the sole anesthetic.

In the treatment of both acute and chronic pain, TENS has been the most extensively studied mode of electroanalgesia. Although some report TENS to be a successful pain treatment,[7,8] there are also equivocal reports.[9,10] Close examination of the methods used reveals that virtually every study of TENS and related modalities of pain treatment has suffered from at least one of the following problems: incomplete elimination of placebo effect, lack of true double blinding, or the difficulties inherent in objective quantification of perceived pain.[11,12] Our study of TENS during general anesthesia is an effort to eliminate the placebo effect, ensure double blindness, and use an objective response variable.

METHODS

With institutional approval we studied 44 informed consenting ASA class I or II patients ranging in age from 18 to 45 yr who were scheduled for elective hand surgery. Patients with any evidence of pulmonary problems were excluded from the study. All subjects fasted for 8 h before receiving anesthesia. Two hours before the anticipated time of anesthesia, subjects received oral premedication or diazepam 0.13 mg/kg (to the nearest 2.5 mg). When the subjects arrived in the operating room holding area, standard monitoring devices were applied: ECG, blood pressure cuff, and precordial stethescope. An intravenous infusion of 5% dextrose in Ringer's lactate solution was begun, and atropine 0.006 mg/kg iv was given. While the patient still was in the holding area, 2 × 2 cm TENS electrodes were placed on the arm designated for surgery. In an effort to locate the TENS stimulation as close to the brachial plexus as possible, one electrode was placed high in the axilla over the axillary artery and the second approximately 6 cm distally. Electrodes then were secured and the surgical tourniquet and padding applied.

After administering curare 3 mg iv, anesthesia was induced with thiopental 2 mg/kg, and muscle relaxation

TABLE 1. Subject Characteristics (mean ± SD)

	Control (n = 22)	TENS (n = 22)
ASA PSI/PSII	16/6	15/7
Age (yr)	31 ± 6	29 ± 7
Sex (male/female)	13/9	12/10
Wt (kg)	77 ± 17	83 ± 16

was obtained with succinylcholine 1 mg/kg iv. When conditions permitted, the trachea was intubated and halothane in oxygen was begun. Initially high concentrations were used and then reduced as indicated by vital signs and finally adjusted to obtain a preselected end-tidal concentration.[13] Ventilation was controlled to maintain a P_aCO_2 between 32 and 38 mmHg. Anesthetic gases were sampled continuously from the endotracheal tube and analyzed for oxygen, CO_2, and halothane by a mass spectrograph (Perkin-Elmer® 1100 Medical Gas Analyzer) and results were continuously recorded (Servogor® 466 Recorder).

The experimenter activated or sham activated, as determined by a random number table, the TENS device as soon as the patient lost consciousness. To obtain maximal safe stimulation while ensuring observer blindness, an output level was used that had been determined previously to be the highest that would not cause motor manifestations.** Before the incision was made, all subjects were checked with a 100-Hz peripheral nerve stimulator at another site to confirm that the effects of succinylcholine had waned sufficiently for movement to occur. Immediately before and after the surgical incision, heart rate and blood pressure were recorded. At the time of incision, an independent observer, who was blind to both TENS status and halothane concentration, determined whether or not gross purposeful movement occurred.[14]

** Codman EPC/Dual: output 30 mA, pulse 0.170 ms, pulse frequency 80 Hz. Codman and Shurleff, Inc., Randolph, Massachusetts 02368.

TABLE 2. Anesthetic Management (mean ± SD)

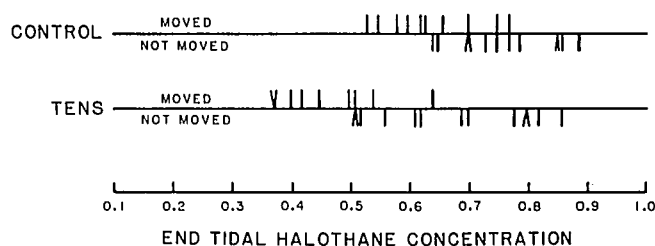|	Control (n = 22)	TENS (n = 22)
Preop diazepam (mg/kg)	0.13 ± 0.02	0.13 ± 0.02
Time: preop to incision (min)	154 ± 54	170 ± 55
Thiopental (mg/kg)	2.09 ± 0.10	2.06 ± 0.11
Time: thiopental to incision (min)	34.1 ± 4.2	30.2 ± 5.4
Duration of TENS stimulation (min)	0	28.4 ± 5.3
Pa_{CO_2} (mmHg) at incision	35 ± 2	36 ± 4

FIG. 1. Individual responses for halothane alone (top) and halothane plus TENS (bottom).

Following the surgical incision, the study was terminated and anesthesia continued as determined by the responsible anesthesiologist who had served as the subject's advocate during the experimental period.

Halothane ED_{50} for both control and treatment groups was determined using the program from Waud.[15] Results were compared using Student's t test for independent means. $P < 0.05$ was considered significant. All results are reported as mean ± SD.

RESULTS

There were no differences in the physical characteristics of the control and TENS groups (table 1). The preanesthetic and anesthetic management of the two groups was likewise similar (table 2). The surgical tourniquet was inflated to 250 mmHg approximately 2 min before the surgical incision. No patient moved in response to tourniquet inflation. Changes in blood pressure and heart rate were similar in both groups and in no instance varied more than 30% from control. The control group had a period of 14.2 ± 2.3 min of stable end-tidal halothane concentration before surgical incision and the TENS group 12.9 ± 2.1 min. The ratio of the difference between inspired and end-tidal to the end-tidal halothane concentration was 0.41 in the control group and 0.54 in the TENS group. The movement/ no movement responses of both groups are shown in figure 1. The calculated halothane ED_{50} for control was 0.69 ± 0.14% and for TENS was 0.53 ± 0.15%. These results were significantly different: $P < 0.001$. No patient developed any complication associated with the use of TENS.

DISCUSSION

The first issue to consider is whether our stated goals were achieved through the experimental design. With regard to subject blindness, all subjects were informed of the purpose of the study and each had a TENS unit applied while awake. The treatment group, however, did not have the TENS unit activated until after they lost consciousness. This is in contrast to other studies in which control subjects received sham TENS, yet the

treatment group clearly received different treatment since they could feel the tingling sensation caused by the electrical stimulation. It is possible that a similar loss of subject blindness occurs during TCES studies.[10,11] The observer who decided whether gross purposeful movement occurred was unaware of the halothane concentrations and whether the patient was in the control or treatment group. Although there is some interpretation on the part of the observer in determining movement/no movement, the assessment is usually accepted as an objective measurement.[14,15] Thus, we believe that our study design eliminated placebo effect, minimized subjective measurement, and was genuinely double blind.

The second issue is whether we showed that TENS lowers halothane fractional MAC for hand surgery. We have shown under experimental conditions that included premedication and thiopental induction and two groups that received virtually identical treatment except for the presence or absence of TENS, that TENS reduced halothane requirements at the time of incision by 23%. Neither of our groups met the technical requirement of 15 min of stable end-tidal halothane concentration prior to incision. While 15 min is considered sufficient, it may not be a necessary condition for accurate MAC determination.[16] Additionally, all subjects were given higher concentration of halothane (3–5%) immediately after intubation, which resulted in end-tidal concentrations greater than the target concentration for periods ranging from 4 to 17 min. Controlled respiration prevented hypoventilation during the period before incision. The ratio of the difference between inspired and end-tidal concentrations to the end-tidal concentration was 0.41 in the control group and 0.54 in the TENS group. This indicates that in the control group, the end-tidal concentration is a better estimate of the arterial concentration and that whatever error occurred in the TENS group tends to overestimate the arterial concentration. Therefore, the effect of TENS in reducing halothane requirements is at least as great as our results indicate. Additionally, the fractional MAC value of 0.69% halothane in our control group is consistent with other reports assessing the effects of diazepam premedication and thiopental induction.[16–19]

The third issue is to determine what our results demonstrate. We have shown that under general anesthesia, devoid of psychologic effects, and with an objective measure, TENS can alter the physiologic response to noxious stimuli in humans. We have not shed any new light on the mechanism of electroanalgesia. The gate control theory suggests that nonpainful stimulation of large-diameter myelinated cutaneous afferent fibers results in closing a gate located in the substantia gelatinosa, thus preventing the transmission of information carried by smaller pain-carrying afferent fibers. Alternatively, there is evidence to invoke a mechanism whereby TENS analgesia is mediated by endogenous analgesics. The discovery of endorphins and other possible endogenous analgesics along with demonstrations that TENS-induced analgesia may be antagonized by narcotic antagonists supports this theory.[20,21] Our results neither confirm nor deny either of these hypotheses.

The final issue is whether TENS has a place in the anesthesiologist's armamentarium as a supplement to general anesthesia. We have not studied TENS during surgery on parts of the body other than the hand or with other agents or techniques, and we have not continued TENS throughout surgery or examined its postoperative effects. Based on our study and the fact that there is virtually no risk associated with the use of TENS, we feel that in patients who are at considerable risk but must be subjected to general anesthesia, the use of TENS may be beneficial.

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Masseter Spasm with Anesthesia: Incidence and Implications

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Masseter spasm after succinylcholine administration is a well-documented event. In many cases it has been the harbinger of an acute malignant hyperthermia (MH) episode.[1-7] Despite this, the incidence of masseter spasm after succinylcholine administration is unknown. In addition, it is not clear whether masseter spasm can develop with induction techniques not utilizing succinylcholine. We therefore examined all cases of masseter spasm that occurred in our operating rooms over a 14-month period and evaluated patients for potential susceptibility to malignant hyperthermia.

METHODS

The medical records were reviewed on all patients from the Children's Hospital in Boston who, from January 1, 1982, through February 28, 1983, were admitted to the recovery room after developing masseter spasm in the operating room. The diagnosis of masseter spasm was established if the attending anesthesiologist found it extremely difficult, if not impossible, to open the patient's mouth after induction of anesthesia. Normal jaw mobility was documented before and after the event.

If succinylcholine was used to facilitate endotracheal intubation, a dose of at least 1 mg/kg given intravenously was considered necessary to assure adequate muscle relaxation.

We reviewed a random sample of 6,500 anesthestic records, or 53% of the 12,169 anesthetics delivered at our institution during that same time period, in order to determine the incidence of masseter spasm overall, and with different anesthetic techniques. The age distribution of patients who experienced masseter spasm was correlated with the age distribution of all patients who were anesthetized during that time; these data were analyzed by the Poisson probability equation.

All patients were seen and evaluated by the anesthesia service preoperatively. All received standard intraoperative care, including monitoring of the pulse, respirations, blood pressure, electrocardiogram, and temperature. By previously established protocol, patients who developed masseter spasm had a blood sample drawn promptly in the operating room for arterial blood gases, serum electrolytes, and creatine phosphokinase (CPK) levels. Postanesthetic care included careful observation, measurement of serial arterial blood gases, and serum CPK levels. Urinalyses were considered positive for myoglobin if they were dipstick positive for hemoglobin/myoglobin in the absence of red blood cells or evidence of intravascular hemolysis.

Whenever possible and after informed consent, muscle tissue was obtained during a subsequent anesthetic that included nitrous oxide, barbiturates, narcotics, nondepolarizing muscle relaxants, droperidol, or diazepam. Dantrolene pretreatment was not given in order to avoid any possible interference with the assay. The specimen immediately was placed in liquid nitrogen and sent with a summary of the clinical history to the nearest