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SCIENTIFIC REPORT

Auricular Electrical Stimulation and Dental Pain Threshold

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A modified double-blind evaluation of naloxone reversibility of dental analgesia produced by auricular electrical stimulation (AES) was examined in 40 subjects assigned randomly to one of four groups: AES followed by saline (AS), AES followed by naloxone (AN), placebo AES followed by saline (PS), and placebo AES followed by naloxone (PN). Dental pain threshold was tested using a hand-held dental pulp tester. A second investigator administered the true or placebo AES using an electrical stimulator. A third investigator injected intravenously saline or naloxone. The subjects and investigators 1 and 3 were blind to all treatment conditions. A repeated measures analysis of variance revealed a significant difference among the four groups. The AES groups exhibited a statistically significant 18% elevation of pain threshold, whereas the two placebo stimulation groups (PS and PN) remained essentially unchanged. The mean pain threshold increased to more than 23% for group AS, but fell to less than 12% for the subjects in group AN, who were given naloxone. These findings indicate a small but significant elevation of pain threshold by AES, an effect partially blocked by naloxone, suggesting an endogenous opioid system as one mechanism for AES analgesia.

Electrical stimulation of the acupuncture point Hoku (LI-4), located on the hand, has been found to have similar dental analgesic effects to administration of 33% nitrous oxide.[1] Significantly elevated pain thresholds were obtained when the Hoku point was electrically stimulated either through acupuncture needles inserted into the skin, or through transcutaneous electrical stimulation over the same area; no significant changes in dental pain threshold were reported for subjects receiving placebo acupuncture or subjects in a nontreatment control group.[2] Not only does acupuncture stimulation of Hoku raise the dental pain threshold in human subjects,[3–6] analgesia to dental pain can also be demonstrated following acupuncture stimulation in monkeys. A significant reduction in the jaw-opening reflex to electrical stimulation of the tooth pulp was obtained in monkeys who received an acupuncture needle placed into the same Hoku acupuncture point used in humans.[7]

The selective effect of ear acupuncture upon body pain threshold was first assessed in 1979.[8] Electrical stimulation of needles inserted into six appropriate ear acupuncture points produced a pronounced increase in pain threshold to radiant heat, whereas stimulation of a nonacupuncture point on the ear had no analgesic effect. More recent work[9,10] also demonstrated that subjects given transcutaneous electrical stimulation at four appropriate ear points exhibited significantly greater relief of experimental wrist pain than was shown by either subjects in a placebo auricular stimulation group or subjects in a no-treatment control group. One investigator, however, failed to show significant elevation of dental pain threshold by auricular acupuncture.[11]

Mayer et al[12] were the first investigators to provide scientific evidence that the neurophysiological basis for acupuncture may be partially related to the endogenous opioid system. After demonstrating that acupuncture applied to the Hoku point led to a significant increase in dental pain threshold, they were then able to produce significant reversal of the elevated pain threshold by the intravenous injection of 0.8 mg of naloxone. It was suggested that the natural opioid substance, endorphin, plays an active role in acupuncture analgesia. Subsequently it was observed that naloxone administration reversed the analgesia produced by acupuncture-like transcutaneous

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electrical nerve stimulation (TENS), which consists of low-frequency surface electrical stimulation of acupuncture points, whereas naloxone did not reverse the analgesia produced by high-frequency TENS.[13,14]

This study tested whether auricular electrical stimulation (AES) at auricular acupuncture points altered tooth pain threshold and, if so, was the effect reversible by naloxone.

METHODS

Study Sample

Forty subjects were recruited from the students and staff of the University of California, Los Angeles. The 23 men and 17 women subjects ranged in age from 17 to 45 yr, and were paid for their participation. Each subject was randomly assigned to one of four treatment groups: AES followed by intravenous saline (AS), AES followed by intravenous naloxone (AN), placebo AES followed by intravenous saline (PS), and placebo AES followed by intravenous naloxone (PN). All subjects were screened for the presence of suitable dentition and were eliminated from the study if they were taking any opioid medications, had a cardiac pacemaker, or were pregnant. The informed consent of all subjects who participated in the experimental investigation was obtained after the nature of the procedures and possible discomforts and risks had been fully explained on a form approved by the UCLA Human Subjects Protection Committee.

Equipment

A dental pulp tester (Analytic Technology Corp., Redmond, WA) was used to determine tooth pain thresholds. The digital readout display panel ranges between 0 and 80 units, which according to Kitamura et al[15] corresponds to a rising voltage between 15 and 300 V, with a maximum current of 50 microamps. Each number on the display represents a train of 10 pulses, each pulse with a duration of 0.22 msec and an interval of 9 msec between pulses. The probe tip held against the tooth starts the automatic increase in output, and tooth stimulation ends when the probe is removed from the tooth.

A Stim Flex 400 instrument (Electro Medical Inc., Tulsa, OK) was used to electrically stimulate the surface of the left ear at auricular acupuncture points. It has a hand-held probe with a concentric, bipolar, spring-loaded tip that is pressed against the ear. The specially designed Stim Flex instrument used in this study had a switch on the back that could be placed in either an "A" (active) or "P" (placebo) position. In both the A and P conditions, the instrument produced identical auditory signals; however, it delivered electrical output only in the A position. Even in the A position, the electrical current was below perceptible threshold, and consequently the subject could not consciously distinguish the A from the P stimulation.

Design

This was a modified double-blind, randomized, controlled clinical trial. All subjects and investigators 1 and 3 were blind to all treatment conditions. Investigator 2, who applied the AES, was aware of the A or P position of the Stim Flex instrument, since he applied the instrument to different places on the ear for active and placebo subjects. After obtaining background information, each subject was asked to lay supine on an examination table. Investigator 1, a dentist, selected an unrestored tooth from the upper quadrant of each side of the maxilla, preferably the left and right upper canines. If unsuitable, a premolar or an incisor pair was selected (in that order).

To determine pain thresholds, investigator 1 placed with a gloved hand a sterilized electric pulp tester tip, covered with sodium fluoride conduction gel, onto the gauze-dried mid-facial surface of the tooth being tested. The hand-held wand of the tooth tester was connected electrically to the subject's lip via a sterilized lip clip. These procedures for electric tooth pulp testing have been described previously.[16] The intensity of electrical stimulation of the tooth was allowed to gradually increase until the subject indicated that he or she felt a definite painful sensation. The pulp tester was then removed from the tooth and the digital readout recorded. The subjects were unable to see the digital readout of the pulp tester. The rate of increase of intensity of electrical stimulation was the same for all subjects and was set at 5 on the range of 0 to 10 available on the pulp tester. A test trial on a lower incisor was first given to each subject to allow them to experience the tooth sensations they could anticipate in the subsequent experimental trials. Baseline recordings of dental pain threshold were then obtained by giving three successive stimulations of each tooth, alternately testing the right and then the left upper tooth with a time separation of 30 sec.

After investigator 1 left the room, investigator 2 entered. Investigator 2 cleaned the ear with alcohol and then applied the spring-loaded, constant pressure probe of the Stim Flex 400 to the surface of the subject's left ear. A reference electrode was hand-held by the subject. For subjects in groups AS and AN, ear acupuncture points considered appropriate for the relief of dental pain were stimulated, as shown in Figure 1. Figure 2 shows the placebo auricular points used for subjects in groups PS and PN. The subjects in the placebo AES groups were given sham stimulation in order to avoid any chance of electrical current conducting to the nearby appropriate auricular points. The subjects were advised in both A and P groups that they would be aware of dull pencil-like

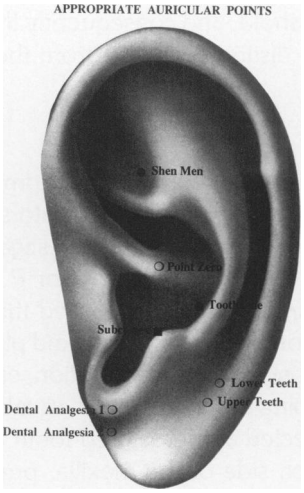

Figure 1. Location of appropriate auricular acupuncture points used for subjects receiving AES, groups AS and AN. The specific ear points related to the relief of dental pain include: Dental Analgesia 1, Dental Analgesia 2, Upper Jaw and Teeth, Lower Jaw and Teeth, and Toothache Point. Master points for AES include: Shen Men, Point Zero, and Subcortex. Open circles indicate raised regions of the ear, filled circles indicate deeper surfaces of the ear, and filled squares indicate hidden regions of the ear.

pressure from the auricular probe as it was pressed against the ear.

Upon completing the 15-min true or placebo AES, investigator 2 left the testing room, and investigator 1 reentered and retested the teeth in the same manner conducted in the baseline condition.

Figure 2. Location of inappropriate auricular acupuncture points used for subjects receiving placebo auricular AES, groups PS and PN. None of these points is used for the treatment of dental pain, such as Apex of Auricle, Apex of Tragus, Helix 1, Helix 2, Helix 3, Helix 6, Fingers, and Elbow points. Open circles indicate raised regions of the ear, filled circles indicate deeper surfaces of the ear, and filled squares indicate hidden regions of the ear.

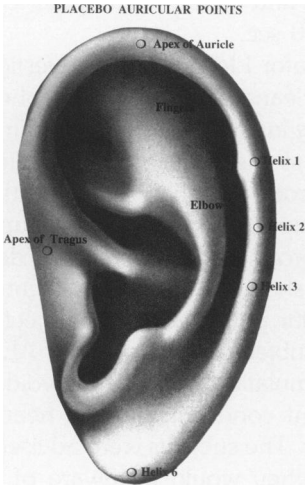

Table 1. Tooth Pain Threshold Average Raw Scores[a]

Group	*Baseline*	*Post-AES*	*Postinjection*
AES, saline	35.5 ± 2.0	42.4 ± 3.1	44.3 ± 3.6
AES, naloxone	38.8 ± 1.5	45.5 ± 1.8	43.1 ± 1.7
Placebo, saline	38.4 ± 2.1	38.0 ± 2.5	39.9 ± 2.3
Placebo, naloxone	37.2 ± 1.5	38.0 ± 1.4	39.6 ± 1.6

[a] Raw scores were obtained from the digital readout of the pulp testing device. Values are the mean ± SE.
AES, auricular electrical stimulation.

After investigator 1 left again, investigator 3, an anesthesiologist blind to the syringe contents, intravenously administered 2 mL of a solution of saline, with or without 0.8 mg naloxone, into the dorsum of the subject's hand or into the antecubital fossa. One minute later, investigator 1 reentered the room and retested the teeth a final time using the same protocol.

Statistics

Research data were based on the digital readout of the dental pulp tester. The mean average of the three separate measures of pain threshold for the left and right upper teeth, and the composite average from both teeth, were computed for the baseline, the post-AES, and the postinjection periods. A mean percent change score was computed by subtracting the mean baseline period from the posttreatment period and dividing the difference by the mean baseline period. A repeated measures analysis of variance (ANOVA) was computed for the pain thresholds obtained for the right and left tooth of each subject, and for the mean average readings from both teeth. A Tukey posthoc test was computed between groups AS, AN, PS, and PN for each statistically significant ANOVA. A Sign test was also computed for the differences in percent change scores between each treatment period.

RESULTS

The repeated measures ANOVA revealed no significant difference in pain threshold for the right and left tooth overall ($F [1, 32] = 0.27$) nor across treatment groups, times, or sex. Therefore, only the composite scores derived by the averaged value of both teeth were used in subsequent analyses. There were also no significant differences related to the sex of the subject (overall $F [1, 32] = 1.58$), so male and female pain thresholds were grouped together.

Group mean values for each treatment group are shown in Table 1. During baseline recordings, there were no differences in pain threshold across the four different treatment groups ($F [3, 32] = 1.29$). In contrast, there

was a statistically significant difference among the four groups after active or placebo AES (F [3, 32] = 4.39, $P = 0.01$). Pain threshold values for the subjects given true AES (groups AS and AN) were significantly greater than pain threshold values for the subjects given placebo AES (groups PS and PN). After injection (of saline or naloxone), the groups were once again not statistically different (F [3, 32] = 2.09).

Tukey posthoc comparisons showed that the difference between baseline and post-AES threshold values across the four groups was also highly significant (F [3, 36] = 11.88, $P < 0.0001$). The AS group, however, was not statistically different from the AN group, and the PS group was not significantly different from the PN group.

Following the injection of saline, the mean pain threshold for group AS further increased above the mean baseline value, whereas for subjects in group AN, the mean pain threshold declined. Both groups previously given placebo AES exhibited a slight increase in pain threshold following the injection of saline or naloxone. The difference between baseline and postinjection threshold values across the four treatment groups was statistically significant (F [3, 36] = 5.01, $P < 0.01$). Pain threshold values for the AS and AN groups were not significantly different from each other, nor were the threshold values for the PS and PN groups. However, the AS group was still significantly greater than the PS and the PN group. The AN group was no longer significantly different from the two placebo groups.

A final Tukey comparison was obtained by subtracting the post-AES values for each group from their respective postinjection values. The slight increase in mean values for the AS, PS, and PN groups were not significantly different from each other, but they were all significantly different from the mean decrease in pain threshold exhibited by the AN group. This finding demonstrated that only when naloxone followed an analgesic treatment did it result in a significant decrease in pain threshold.

One-way ANOVA indicated that there were significant differences in percent change scores between the four treatment groups during the post-AES period (F [3, 36] = 8.02, $P < 0.01$) and during the postinjection period (F [3, 36] = 3.52, $P < 0.05$). Figure 3 shows the average percent increase in pain threshold following true AES, which was 18.1%. In contrast, subjects given placebo AES exhibited a decrease of 1.2% for subjects in group PS and only rose by 2.9% in group PN. The average change across both placebo groups was an increase of only 0.85%.

Examination of the individual values for each subject revealed that 18 of the 20 subjects given AES showed at least a 10% increase in pain threshold following auricular stimulation, which is significant by the Sign test ($P < 0.01$). In contrast, only two of the 20 subjects given placebo

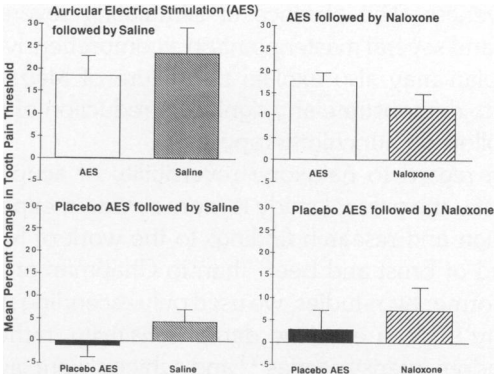

Figure 3. Change (% difference) from baseline of mean tooth pain thresholds. Brackets indicate the standard errors.

AES exhibited at least a 10% elevation of pain threshold. During the postinjection period, nine of the 10 subjects exhibited a decrease in percent change values when AES was followed by naloxone. This difference is significant by the Sign test ($P < 0.05$). For the remaining three groups, a majority of subjects showed an increase in percent change values when the postinjection period was compared to the post-AES period.

DISCUSSION

The primary purpose of this research was to determine if electrical stimulation of auricular acupuncture points could produce statistically significant elevations of tooth pain threshold, and whether this effect could be reversed by the opioid antagonist naloxone. There was statistically positive support for each research question, but the magnitude of each effect was rather small. Nevertheless, our research is in agreement with several previous studies,[17] which found that acute pain thresholds can be elevated by the stimulation of auricular acupuncture points. The present study is the first study to demonstrate the analgesic effect of auricular electrical stimulation on dental pain threshold. Kitade and Hyodo[8] observed pronounced increases in pain threshold to radiant heat following the insertion of needles into appropriate ear acupuncture points. Oliveri et al[10] found the elevated pain thresholds for shocks to the wrist in subjects given transcutaneous electrical stimulation of appropriate auricular points. In both of these studies, stimulation of inappropriate auricular points did not significantly alter pain threshold.

Lin[11] failed to demonstrate any elevation of dental pain threshold when needles were inserted into a single auricular acupuncture point. Treatments described in several ear acupuncture references[18–20] suggest that several auricular points should be stimulated in order to achieve clinical

effectiveness. The absence of stimulating several local points and several master points in a comprehensive treatment plan may also explain the failure of Melzack and Katz[21] to demonstrate any significant reduction of chronic pain following auriculotherapy.

With regard to naloxone reversibility of acupuncture analgesia, the present study is more similar in experimental design and research findings to the work of Mayer et al[12] and of Ernst and Lee[22] than to Chapman et al.[23] As in the former two studies, we used only ascending stimulus intensity trials to examine dental sensitivity, rather than the random intensity series,[23] and subjects were given 0.8 mg of naloxone rather than 1.2 mg. Descending stimulation is not available for the Analytic Technology pulp tester and was deemed too aversive for human volunteers. While a traditional dose-response curve would predict that 1.2 mg of naloxone would be a more effective analgesic antagonist than 0.8 mg, the lower dose of naloxone was still sufficient to demonstrate partial reversibility.

The naloxone challenge does not provide conclusive proof that acupuncture analgesia is due to an endogenous opioid system.[23] More direct evidence of the endorphinergic basis of auriculotherapy is provided by Abbate and associates.[24] Assaying β-endorphin concentrations in subjects undergoing surgery, they observed a significant increase in plasma β-endorphin after AES combined with nitrous oxide inhalation, whereas control subjects given nitrous oxide without acupuncture showed no such elevation. An alternative theory[23] is that acupuncture analgesia may be attributable to the phenomenon of stress-induced analgesia. This view, however, would not account for the experience of many of our subjects who received active AES and found the treatment to be very calming and relaxing. The only stressful procedure that several subjects commented upon was the IV injection. Stress-induced analgesia may account for the slight elevation of dental pain threshold shown in the postinjection period by all groups other than group AN. In the latter case any analgesia afforded by the aversive injection would arguably have been overbalanced by the naloxone reversal of the AES analgesia.

Bossy[25] delineated several neurological mechanisms by which acupuncture may work. Besides initiating the release of endorphins, Bossy also suggests that body and ear acupuncture may turn on the spinal gate control pathway by activating supraspinal mechanisms in the thalamus and in the midbrain periaqueductal gray. That both of these brain areas are organized somatotopically[26] may account for the somatotopic organization of the auricle first observed by Nogier[27] and later confirmed by Oleson et al.[28] AES may be a peripheral procedure for eliciting the same stimulation-produced analgesia that is activated by deep brain stimulation.[29] Electrical stimulation of the brain has been shown to raise tooth shock thresholds in cats[30] and monkeys[31] and may provide a possible central mechanism by which AES produces an elevation of dental pain threshold in man.

AES is more commonly used in a clinical setting for the relief of chronic rather than acute pain. In their review of the acupuncture literature, Richardson and Vincent[32] documented the use of auricular and body acupuncture for the relief of headaches, temporomandibular disorder pain, and cervical pain. Multiple treatments of AES may be both additive and cumulative, especially in a chronic pain treatment setting. The use of acupuncture may also be valuable in chronic conditions where conventional therapies have failed. For acute dental procedures, a combination of AES therapy, body acupuncture, and nitrous oxide might yield higher levels of analgesia than any of these procedures used alone. While the magnitude of the analgesia demonstrated in this study was small, and the reversibility by naloxone even more limited, the effects were nonetheless reliable and statistically significant.

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