Effect of Interferential Current on Perceived Pain and Serum Cortisol Associated With Delayed Onset Muscle Soreness

Randy J. Schmitz, David E. Martin, David H. Perrin, Ali Iranmanesh, and Alan D. Rogol*

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Abstract:
The purpose of this study was to assess the effect of interferential current (IFC) on perceived pain and serum cortisol levels in subjects with delayed onset muscle soreness (DOMS). DOMS was induced in 10 subjects through repeated eccentric contractions of the elbow flexors. Forty-eight hours later subjects were evaluated. Starting at t = 0:00, blood samples were withdrawn from a superficial vein every 5 mitt for 65 min. At t = 0:05, subjects received IFC of 10 bps or IFC of 100 bps. Perceived pain levels were evaluated prior to catheter insertion and at t = 0:35, 0:50, and 0:65. Two mixed-model analyses of variance revealed a significant decrease in perceived pain scores across time for both treatment groups but no significant difference in serum cortisol for the two groups. It was concluded that IFC of high and low beat frequency is effective in controlling the pain of DOMS but does not elicit a generalized stress response as indexed by increasing serum cortisol levels.

Article:
Little information is available on the ability of interferential current (IFC) to relieve pain from musculoskeletal injuries (27). The mechanism of how IFC devices control pain is not truly understood.

IFC has an advantage over traditional transcutaneous electrical nerve stimulation (TENS) because the medium frequencies used in IFC encounter less resistance at the skin and have better current conductance through the skin than the low frequencies used in TENS (15, 16). Therefore, the cutaneous nerves are apparently less irritated (9, 19).

Much of the clinical work done with IFC involves pain control (4, 27). Due to the physical properties of IFC in body tissues, it may be able to produce analgesia via activation of the hypothalamic—pituitary—adrenal (HPA) axis. Pain arises as A-delta and unmyelinated C fibers send afferent signals primarily to laminae I and V in the spinal cord. The contralateral spinothalamic tract is the major nociceptive pathway to the thalamus with the cells of origin concentrated in laminae I and V (16). Under certain pathological conditions, the sensitivity of sensory nerve endings or receptors increases dramatically and is attributed to the release of pain-inducing substances, including bradykinin, serotonin, and prostaglandins (21).

* R.J. Schmitz, D.E. Martin, and D.H. Perrin are with the Athletic Training Research Laboratory, Curry School of Education, University of Virginia, Charlottesville, VA 22903. A. Iranmanesh is with Salem V.A. Hospital, Salem, VA 24153. A.D. Rogol is with the Health Sciences Center, University of Virginia, Charlottesville, VA.

Delayed onset muscle soreness (DOMS) is the sensation of discomfort or pain in the skeletal muscles that occurs following unaccustomed muscular exertion (1). Most soreness increases in intensity in the first 24 hr postexercise, peaks in intensity from 24 to 72 hr, and subsides in 5 to 7 days postexercise (1). DOMS has been attributed to increased tension in contractile elements that physically damages the structural components of the muscle (1). Using a series of eccentric muscular contractions, researchers have shown that DOMS is easily induced in the elbow flexors in subjects (5, 6, 7, 13).

With the identification of the endogenous opioid beta-endorphin, a new theory was proposed that electrical currents of low frequency and high intensity would elicit the release of beta-endorphin from the anterior pituitary and produce an analgesic response (3). Castel (3) proposed that an endogenous opioid, such as beta- endorphin, is released by a variety of factors from the anterior pituitary gland. Many authors have suggested that a current of high intensity and low frequency is ideal for eliciting analgesia via systemically released endogenous opioid peptide (3, 16, 21, 23).

Beta-endorphin is released from the anterior pituitary gland. It shares the precursor hormone proopiomelanocortin (POMC) with adrenocorticotropin (ACTH). ACTH results in the synthesis and release of cortisol from the adrenal cortex (2). Because of the difficulty of conducting and interpreting the beta-endorphin assay, ACTH and cortisol have been used as markers of beta-endorphin release (7, 16).

The purpose of this study was to compare the ability of IFC of high and low beat frequency to control mu sculoskeletal pain as measured by perceived pain scales and serum cortisol levels as an approximation of generalized stress; we used delayed onset muscle soreness as a model of injury.

METHODS

Subjects

Ten healthy subjects (5 male and 5 female) (height 170.9 ± 7.5 cm, weight 64.8 ± 10.4 kg, age 22.8 ± 2.5 years) with no recent history of upper extremity injury, disability, or pain were recruited from the student population at the University of Virginia. Each subject read and signed an informed consent form in compliance with the University of Virginia Human Investigation Committee. All were informed of the purpose of the study, and each was assigned a code number. In no instance was the name of the subject associated with the results.

Procedures

All subjects reported to the athletic training laboratory for the exercise portion of the study between 15:00 and 16:00 hr. To induce DOMS, subjects repeatedly lowered 11.4-kg weights through eccentric contraction from full flexion to complete extension in 3 s with their nondominant limb. This continued until subjects could not maintain the 3-s interval, at which time the weight was decreased 2.3 kg. This continued until subjects reached 2.3 kg, at which time the subjects continued until exhaustion or until 20 repetitions were completed. A technician performed all concentric lifting of the weight to avoid premature fatigue. Subjects were instructed to refrain from the use of any anti-inflammatory and analgesic agents or modalities for the duration of the study.

Forty-eight hours following the exercise session, subjects returned to the Clinical Research Center laboratory at 14:00 hr. Time of treatment was standardized to control for diurnal variations in cortisol. Subjects were then randomly placed into one of two groups. One group (n = 5) received low beat frequency current of 10 bps for 30 min. A second group (n = 5) received high beat frequency current of 100 bps for 30 min.

At the beginning of the evaluation, subjects were assessed for levels of perceived pain using the scale described by Talag (26). Perceived pain levels were also obtained immediately and 15 and 30 min following treatment.

Following skin preparation, a venous catheter was inserted into the unexercised arm in a superficial vein. Four electrodes (carbon rubber, 3 in. round) were placed about the biceps in quadripolar fashion on the exercised arm. Two were placed at the proximal musculotendinous junction (one medial and one lateral) and two were placed at the distal musculotendinous junction (one medial and one lateral). The subjects then sat quietly for 60 min. One 2-m1 blood sample was taken at the end of the 60 min, This first sample served as the zero time indicator for the treatment session (t = 0:00). At t = 0:05, another 2-ml sample was obtained, and the IFC treatment with an Intellect Model 775 MP IFC Unit (Chattanooga Corp., Hixson, TN) commenced. The intensity of current in the low-frequency group was set to maximal subject tolerance. Subject tolerance was defined as the point at which the level of current in the tissue became uncomfortable. This level of current resulted in motor recruitment in all subjects. Intensity of the high-frequency group was set just below motor recruitment (15, 20). We explained this to the subject by stating that if he or she experienced a muscle contraction, the intensity was too high and must be lowered. Two-milliliter blood samples were taken every 5 min throughout the 30-min treatment span. Following the IFC treatment, subjects were monitored for an additional 30 min during which time blood sampling was continued. Following the final sample (t = 0:65), the catheter was removed and direct pressure was administered until bleeding ceased.

Blood samples were centrifuged at 2,500 rpm for 15 min, and the resultant serum was pipetted and stored at —15 °C for analysis. All analyses for cortisol were performed using a standard radioimmunoassay technique (DPC Products, Los Angeles). The minimal detectable amount of serum cortisol is 0.8 jig/d1 for the assay used. The coefficient of variation varied between 6.2 and 10.2% throughout the working range of the assay.

STATISTICAL ANALYSIS

A mixed-model ANOVA (one between, one within) was computed for the perceived pain data. Serum cortisol levels were plotted over time, and the area under the curve for treatment (Samples 1-7, t = 0:00 to t = 0:30) and posttreatment (Samples 8-14, t = 0:35 to t = 0:65) was calculated using the procedure described by Veldhuis and Johnson (29). A mixed-model ANOVA (one between, one within) was also computed for the cortisol values obtained by subtracting treatment from posttreatment. A Pearson Product Moment correlation value was computed for perceived pain and 48-hr cortisol levels.

RESULTS

The ANOVA revealed a significant decrease of perceived pain levels across time (Figure 1) in both treatment protocols, $F(3, 24) = 25.561$, $p = .0001$, but perceived pain levels did not differ significantly between treatment groups, $F(1, 8) = 4.556$, $p = .0651$.

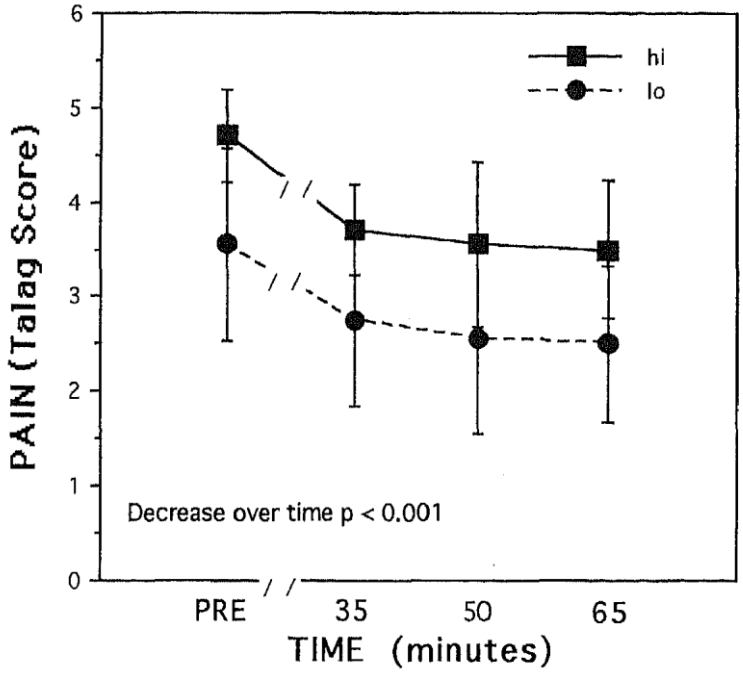

Figure 1— A comparison of the effect of WC of high and low beat frequency on perceived pain levels.

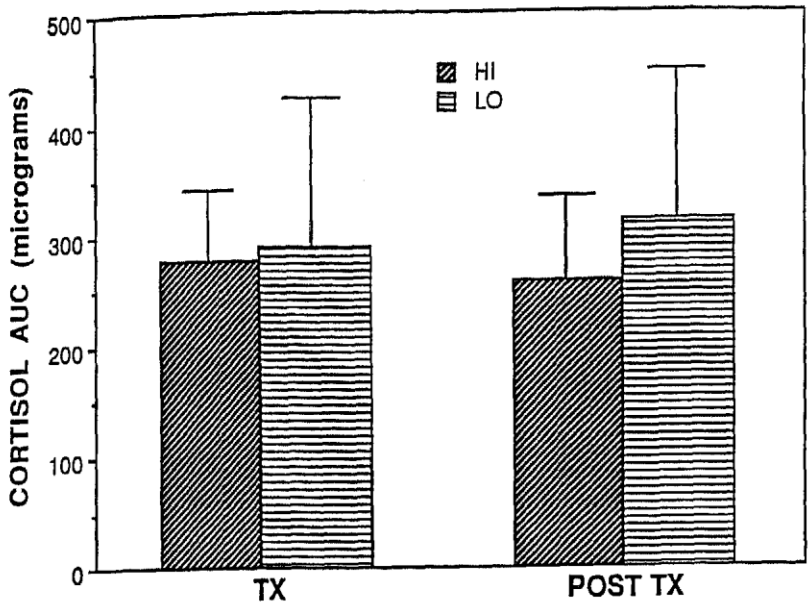

Figure 2 —A comparison of the effect of IFC of high and low beat frequency on serum cortisol levels.

There was no difference in cortisol levels between treatment groups, $F(1, 8) = 0.250$, $p = .631$, and no mean effect of pre- to posttreatment was observed, $F(1, 8) = 0.031$, $p = .864$. Figure 2 presents the cortisol data. The Pearson Product Moment correlation revealed no significant correlation between perceived pain and 48-hr cortisol levels ($r = .257$, $R2 = .066$, $p > .05$).

DISCUSSION

We found that IFC of high and low beat frequency significantly decreased perceived pain levels across time in both treatment groups. However, neither high or low beat frequency treatment protocols had a significant acute effect to raise serum cortisol levels.

Cortisol values ranged from 4.2 to 19.5 μg/dl (normal clinical range = 2-10 μg/d1). These data resulted in very large standard deviations, as demonstrated by Figure 2. The pretreatment levels were within normal range for the time of day. This demonstrated that the stress of DOMS at the 48-hr mark is much different than the stress brought about by an acute injury that would acutely raise cortisol levels (11).

A reason for failure of cortisol level elevation may be the intrinsic factors of the IFC used in this study. The unit that was used to deliver IFC in this study operated with a carrier frequency of 5000 Hz. The resulting phase duration of the current study was 100 μs. A current with this phase duration may not be able stimu-current study was 100 μs. A current with this phase duration may not be able stimulate the A-delta and C fibers that travel to the HPA axis. This value is at or below suggested parameters (21).

The reduction in pain levels for both groups suggests that pain control was achieved regardless of the IFC treatment protocol. One explanation may be the gate control theory of pain suggested by Melzack and Wall (18). According to this theory, activity in afferent large diameter fibers (A-beta and A-alpha) projects to the substantia gelatinosa (SG) of the dorsal horn of the spinal cord. The large diameter fibers then inhibit the input of nociception fibers (A-delta and C) to the first transmission cell. Kloth (16) suggested that if a beat frequency of 2 bps is selected and adjusted to produce a comfortable tingling paresthesia without muscle contraction, then A-beta and A-alpha fibers would be recruited.

Another limitation of this study was that no placebo group was used. Therefore, the reduction of pain in both treatment groups may be due to a placebo effect. Another possible explanation for the reduction of pain may be peripheral mechanisms acting upon primary afferent nerves that may be potential targets for circulating beta-endorphin produced by the anterior pituitary (14, 22, 24).

That there was no significant correlation between perceived pain and cortisol levels at 48 hr postexercise suggests that our method of DOMS induction has no effect over cortisol release after the acute stage.

The failure to elevate cortisol levels acutely via IFC treatment in our subjects suggests that POMC was not released from the anterior pituitary gland, although present data do not rule out such a mechanism. This is in agreement with others who have reported that TENS-induced pain control was not reversed by the opiate antagonist naloxone hydrochloride (8, 20), although neither of these studies used IFC as the stimulating current.

Within the central nervous system, the major physiological role of the endogenous opioid peptides, including beta-endorphin, is thought to be pain modulation by binding to specific opiate receptors and blocking pain transmission in afferent neurons at the spine and supraspinal

levels (2). However, the role of opioid peptide synthesized in the anterior pituitary gland and released into the systemic circulation is much less clear (2, 12, 25, 28). It has been suggested that ACTH and beta-endorphins are released concomitantly into the peripheral circulation (2, 17). Malizia et al. (17) studied the effect of electroacupuncture on peripheral ACTH and beta-endorphin levels and found that both rose in response to electroacupuncture. Guillemin et al, (10) studied the effect of peripheral ACTH and beta-endorphin levels in the responses of male rats to acute stress and found that ACTH and beta-endorphin levels did rise concomitantly.

CONCLUSION

Both high and low beat frequency IFC reduced perceived pain levels across time. Once again, this may be due to a placebo effect; no placebo treatment was used in the investigation. No significant effect existed with low beat frequency or high beat frequency IFC with respect to eliciting the central release of cortisol into the circulation. These data are not consistent with (but do not rule out) the theory that TENS of a low frequency and high intensity will elicit the release of POMC. Continued use of this therapeutic modality for pain modulation necessitates a more complete understanding of how this modality modulates pain, which requires properly controlled studies in the future.

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