

ANALGESIC EFFECTS OF TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION AND INTERFERENTIAL CURRENTS ON HEAT PAIN IN HEALTHY SUBJECTS

Gladys L. Y. Cheing and Christina W. Y. Hui-Chan

From the Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Hong Kong

This study examined whether transcutaneous electrical nerve stimulation or interferential current was more effective in reducing experimentally induced heat pain. Forty-eight young healthy subjects were randomly divided into the following groups: (i) transcutaneous electrical nerve stimulation; (ii) interferential current; and (iii) no stimulation. A multi-function electrical stimulator was used to generate the transcutaneous electrical nerve stimulation or interferential current. A thermal sensory analyser was used to record the heat pain threshold. The stimulation lasted for 30 minutes and the heat pain thresholds were measured before, during and after the stimulation. Transcutaneous electrical nerve stimulation ($p = 0.003$) and interferential current ($p = 0.004$) significantly elevated the heat pain threshold, but “no stimulation” did not. The thresholds of the transcutaneous electrical nerve stimulation and interferential current groups were significantly higher than that of the control group 30 minutes into the stimulation ($p = 0.017$). Both transcutaneous electrical nerve stimulation and interferential current increased the heat pain threshold to a similar extent during stimulation. However, the post-stimulation effect of interferential current lasted longer than that of transcutaneous electrical nerve stimulation.

Key words: TENS, IFC, heat pain threshold, pain.

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Correspondence address: Gladys Cheing, Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Hung Hom, Kowloon, Hong Kong. E-mail: rsgladys@polyu.edu.hk

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INTRODUCTION

Various therapeutic currents have been used for modulating clinical pain. Transcutaneous electrical nerve stimulation (TENS) is a low-frequency stimulator that delivers electrical impulses at a frequency of 0–200 Hz. It has been shown to be an effective treatment modality for various types of musculoskeletal pain (1) such as osteoarthritic knee (2, 3) and chronic low back pain (4). Interferential current (IFC) is a medium-frequency (3000–5100 Hz) alternating current with a beat frequency ranging from 0 to 250 Hz (5). Compared with a low

frequency current (about 100 Hz for TENS), IFC produces lower impedance on skin and subcutaneous tissue, therefore the theoretical penetration power should be deeper than that of TENS (5). Studies have demonstrated that IFC is effective in managing pain conditions such as migraine (6) and muscle soreness (7). However, due to the large variability of clinical pain, Taylor et al. (8) did not find any significant difference between the IFC group and the placebo group in managing recurrent jaw pain.

Some research has been carried out into the effect of electrical stimulation on experimental cold-induced pain. Asthon et al. (9) initially did not find that 100 Hz TENS elevated experimentally induced cold pain threshold. However, the same group of researchers (10, 11) confirmed that TENS did elevate cold pain thresholds significantly. Similarly, studies have also shown that IFC delivered at 100 Hz significantly increases ice pain thresholds in healthy subjects, in contrast to no change in the control group (12, 13). Although Stephenson & Johnson (12) postulated that IFC might produce greater antinociceptive effects than TENS when comparing their results with those of previous studies (10, 11, 14), their postulation was disproved by their later research findings (15). Johnson & Tabasam (15) compared the analgesic effects of IFC, TENS and placebo stimulation on cold-induced pain. No significant differences in the pain intensity or unpleasantness ratings were found among the 3 treatment groups. Their findings suggested no differences in the analgesic effects of inferential currents and TENS on cold-induced pain.

Despite the couple of studies done on cold-induced pain, very few studies have been carried out to investigate the influence of electrical stimulation on heat pain. It has been reported that TENS significantly increased experimentally induced heat pain on the cheek in healthy subjects (16). No study has compared the influence of TENS and IFC on heat pain thresholds. TENS and IFC are likely to stimulate similar afferent fibres (i.e. the $A\alpha$ and $A\beta$ fibres). Since the measurement of heat pain threshold in the present study was completed within only a few seconds, it is likely that the measurement mainly involves the fast pain transmission by the $A\delta$ fibres. This study examined whether 30 minutes of TENS or IFC would alter the heat pain threshold in normal healthy subjects. We compared the changes of heat pain threshold *before*, *during* and *after* TENS or IFC; and examined whether or not the heat pain thresholds of these 2 groups would

Table II. Recorded heat pain threshold for the transcutaneous electrical nerve stimulation (TENS); interferential current (IFC) and control groups during the study (mean \pm SD)

Time	T ₁ Pre-treatment (-15 min)	T ₂ Pre-treatment (0 min)	T ₃ During treatment (15 min)	T ₄ During treatment (30 min)	T ₅ Post-treatment (45 min)	T ₆ Post-treatment (60 min)	<i>p</i> ^a
TENS	41.6 \pm 3.8	42.0 \pm 3.6	43.6 \pm 4.5	43.9 \pm 3.6	42.4 \pm 3.7	42.0 \pm 3.3	0.003
IFT	41.2 \pm 4.2	41.6 \pm 4.5	43.1 \pm 4.1	43.4 \pm 4.3	42.9 \pm 4.2	42.3 \pm 3.9	0.004
Control	40.6 \pm 4.0	40.4 \pm 3.9	40.6 \pm 3.8	40.3 \pm 3.6	40.5 \pm 3.6	40.6 \pm 3.7	0.994
<i>p</i> -values ^b	0.412	0.412	0.079	0.017	0.067	0.253	

^a *p* values comparing results at different time within each group.

^b *p* values comparing different groups at each time.

experiment lasted for 75 minutes. To reduce the accommodation effect, the intensity of the current in both TENS and IFC groups was increased by 10% at 15 minutes into the stimulation. The control group did not receive any electrical stimulation and no electrodes were placed on their forearms.

Testing was done in a quiet, isolated room. The room temperature was maintained at 21° C. A thermal sensory analyser consisting of a 30 mm \times 30 mm thermode was placed distally to the proximal one-third of the anterior forearm of the dominant hand, which was between the elbow crease and distal crease of the wrist. The location of the thermode on the forearm was marked on the skin. The thermode was attached to the subject's forearm by tightening the Velcro strap by 2 cm, and a mark was made on the strap. A build-in computer program in the thermal analyser controlled the heating process of the thermode. The baseline measurement of the pain threshold was taken at the beginning of the experiment (-15 min). The temperature of the thermode was increased from 32° C at a rate of 1.5° C per second to avoid accommodation of the temperature rise. The highest temperature induced in the thermode was 50° C, to avoid the risk of burning the patient. When the subjects started to feel the heat pain, they were requested to press the mouse immediately with the non-dominant hand. The thermode was removed at the end of each recording period for better heat dissipation.

Data analysis

Repeated measures ANOVA followed by contrast were used to analyse the absolute data. The within-subject factor was "time" and the between-subject factor was "group". Normalized heat pain thresholds with respect to the pre-stimulation baseline observation using the formula were also calculated:

$$\frac{T_n}{(T_1 + T_2) \div 2} \times 100\%$$

where *n* = 1, 2, 3, ... 6, as shown in Figure 2.

T₁, T₂ are the baseline measurements of heat pain threshold.

RESULTS

No significant group difference was found in heat pain threshold

at the baseline, as shown in Table II. The 2 *pre*-treatment values indicate that the baselines were very stable in all 3 groups. As significant interaction was found between "time" and "group" (*p* = 0.008), the analyses were performed separately.

Table III showed the heat pain thresholds that were normalized with the baseline measurement recorded at T₁ and T₂. For the TENS group, the heat pain threshold showed significant changes over time (*p* = 0.003). It increased to 104.3 \pm 6.7% of the normalized value at T₃ (*p* = 0.013) and 105.2 \pm 6.6% at T₄ (*p* = 0.004), both significantly different from the baseline, i.e. (T₁ + T₂)/2 (Fig. 3). It then decreased to 100.6 \pm 3.7% at T₆, i.e. almost back to the baseline level. Similarly, for the IFC group, the heat pain threshold increased significantly over time (*p* = 0.004). The normalized heat pain threshold rose to 104.4 \pm 6.9 % of the control value at T₃ (*p* = 0.026) and further increased to 105.0 \pm 7.2% at T₄ (*p* = 0.020). It then gradually decreased to 102.5 \pm 2.9 % at T₆. However, contrast comparisons showed that the heat pain thresholds of the IFC group at T₅ (103.9 \pm 3.5%; *p* = 0.001) and T₆ (102.5 \pm 2.9%, *p* = 0.004) were still significantly higher than the baseline. In other words, 30 minutes of IFC significantly elevated the heat pain threshold *during* the stimulation, and the effect lasted for at least 30 minutes *after* the stimulation. On the other hand, no significant change in the heat pain threshold was found in the control group throughout the study period (*p* = 0.994). The threshold of the control group remained roughly unchanged from T₁ to T₆. However, there was no significant between-group difference after the intervention, i.e. T₅ and T₆ (all *p* > 0.05).

For between-group comparisons, significant differences among 3 groups were found at T₄ (*p* = 0.017), i.e. 30 minutes into the stimulation. Contrast comparisons indicated that the

Table III. Normalized heat pain threshold in the transcutaneous electrical nerve stimulation (TENS); interferential current (IFC) and control groups over time (mean \pm SD)

Time	T ₁ Pre-treatment (-15 min)	T ₂ Pre-treatment (0 min)	T ₃ During treatment (15 min)	T ₄ During treatment (30 min)	T ₅ Post-treatment (45 min)	T ₆ Post-treatment (60 min)	<i>p</i>
TENS	99.4 \pm 1.6	100.6 \pm 1.6	104.3 \pm 6.7	105.2 \pm 6.6	101.5 \pm 5.1	100.6 \pm 3.7	0.001
IFT	99.5 \pm 2.0	100.5 \pm 2.0	104.4 \pm 6.9	105.0 \pm 7.1	103.9 \pm 3.5	102.5 \pm 2.9	0.006
Control	100.2 \pm 2.0	99.8 \pm 2.0	100.1 \pm 3.8	99.6 \pm 3.8	100.2 \pm 4.8	100.3 \pm 5.2	0.851

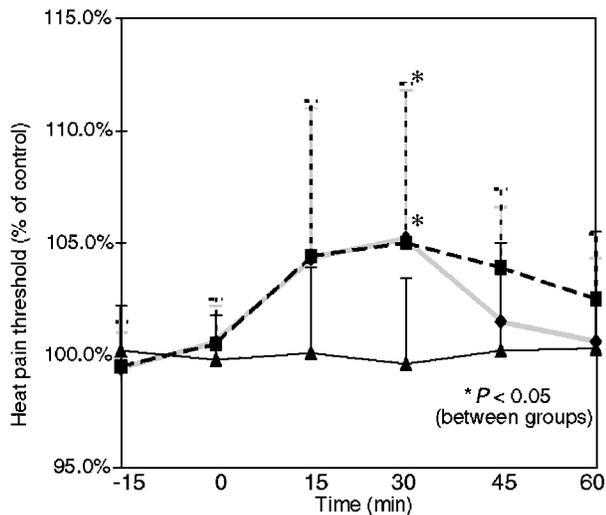


Fig. 3. The thermal pain threshold increased gradually from the baseline value to 105.2% in the transcutaneous electrical nerve stimulation (TENS \blacklozenge) group ($p=0.004$) and 105.0% in the interferential current (IFC \blacksquare) group ($p=0.020$) at T_4 i.e. 30 minutes into the stimulation. In contrast, there was no significant change in the heat pain threshold for the control group (\blacktriangle). The between-group difference reached significance at T_4 ($p=0.017$).

heat pain thresholds of the TENS and IFC groups were significantly higher than the control group at T_4 , but that there was no significant difference between the TENS and IFC groups.

DISCUSSION

To the best of our knowledge, this experiment is the first study comparing the influence of IFC and TENS on heat-induced pain threshold. We demonstrated that 30 minutes of TENS or IFC, but not the control group, significantly elevated heat pain threshold during stimulation in young healthy people. The influence of TENS and IFC on heat pain threshold peaked at 30 minutes into the stimulation (i.e. T_4). After the stimulation (from T_5 to T_6), the heat pain threshold in both groups tended to drop. However, this drop was slower in the IFC group than in the TENS group. In other words, the antinociceptive effects of TENS occurred mainly during stimulation, but the effect of IFC lasted at least up to 30 minutes after stimulation. This could be due to the stronger penetration power of IFC.

Our results are consistent with those reported by Marchand et al. (16). They investigated the heat pain threshold on the cheek *before*, *during* and *after* 15 minutes of TENS treatment in healthy subjects. They demonstrated that TENS significantly increased the heat pain threshold during stimulation, compared with the baseline value. However, the threshold regressed back to the baseline level *after* stimulation. In the present study, even though we applied TENS for a longer duration (30 minutes), the post-stimulation heat pain threshold was not significantly different from the baseline value ($p > 0.05$). In contrast, our findings demonstrated that the antinociceptive effect of IFC outlasted the stimulation, and thus was longer than that produced

by TENS. The influence of IFC on heat pain threshold was significantly higher than the baseline value even 30 minutes *after* stimulation.

As both TENS and IFC are afferent stimulations that are applied to the skin, it is likely that their analgesic mechanisms are similar, probably involving the gate control theory, the physiological block and the endogenous pain inhibitory system.

The gate control theory was proposed by Melzack & Wall (17) in 1965. They suggested that the substantia gelatinosa in the dorsal horn of the spinal cord acts as a gate control system. Activation of the large diameter myelinated fibers subserving touch, pressure and vibration (i.e. the $A\alpha$ and $A\beta$ fibres) is thought to facilitate the pre-synaptic inhibition of substantia gelatinosa cells on the transmission cells in the dorsal horn, thus reducing pain transmission. TENS is supposed to excite predominantly $A\alpha$ or $A\beta$ fibres, which may reduce the output of the transmission cells, thus reducing the perception of heat pain. This could partly explain why subjects reported an increase in their heat pain thresholds in this study.

The other antinociceptive mechanism is physiological block (18). The C fibres are able to fire when the frequency of an electrical stimulus is below 15 Hz. When the frequency of stimulation increases, the conduction in the C fibres decreases. The application of an electrical stimulus above 50 Hz may result in a physiological block. For $A\delta$ fibres, the physiological block occurs at a higher frequency of 40 Hz. Since both TENS and IFC were applied at 100 Hz in this experiment, a physiological block may have occurred, thus increasing the heat pain threshold.

The endogenous pain inhibitory system is also a well-accepted antinociceptive mechanism. Basbaum & Field (19, 20) proposed that there is a neural network including the midbrain, medulla, and spinal cord levels that monitors and modulates the activity of pain-transmitting neurons. Woolf et al. (21) demonstrated that peripheral electrical stimulation could also excite naloxone-dependent antinociceptive mechanisms, i.e. the endogenous opioid system operating at both spinal and supraspinal levels. If this is the case, it may have led to a reduction in pain perception and an increase in heat pain threshold in the present study.

Our results suggest that the antinociceptive effect produced by IFC is more prolonged than that of TENS. This may be due to the fact that IFC is a medium frequency current that exerts lower resistance to skin than TENS (a low frequency stimulation). Therefore, IFC is likely to be more effective in penetrating through the skin and stimulating the deep nerve tissues underneath. Palmer et al. (22) examined the effects of different IFC and TENS frequencies on sensory, motor and pain thresholds. They found that both IFC and TENS displayed a significant frequency-dependent effect for each threshold. However, IFC was not any better than TENS at increasing the sensory, motor or pain thresholds at different stimulation frequencies. Future studies are needed to examine how the penetrating power of therapeutic currents could affect the antinociceptive effects in humans.

The present study was done on experimental pain because it is a simpler model to test for the effectiveness of pain treatment.

Experimental pain is usually induced in a standardized way in healthy subjects. As they are relatively homogeneous within a group, the different responses of different groups could be explained by group allocation, rather than individual variations. In contrast, patients suffering from clinical pain tend to have variations in terms of the history, severity or cause of pain. It is difficult to form a homogeneous group at the baseline. As a result, patients within a group may respond differently to the same intervention. However, further studies need to be conducted to compare the relative effectiveness of TENS and IFC on clinical pain, because experimental pain may differ from clinical pain in some aspects. The heat-induced pain applied in our study is a localized, well-defined and sharp sensation, which is similar in nature to acute pain. However, clinical pain could involve chronic pain, which often involves a diffuse and dull sensation (23). These 2 types of pain are also different in the affective aspect; one may be more anxious about experimental pain but more depressed about clinical pain. Therefore, the relative effectiveness of the therapeutic currents may vary with these 2 types of pain. Further studies are needed to compare the effectiveness of IFC and TENS in managing clinical pain.

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REFERENCES

1. Robinson AJ. Transcutaneous electrical nerve stimulation for the control of pain in musculoskeletal disorders. *J Orthop Sports Phys Ther* 1996; 24: 208–226.
2. Taylor P, Hallet M, Flaherty L. Treatment of osteoarthritic pain. *Physiotherapy* 1981; 69: 266–268.
3. Lewis D, Lewis B, Sturrock RD. Transcutaneous electrical nerve stimulation in osteoarthrosis: a therapeutic alternative? *Ann Rheum Dis* 1984; 43: 47–49.
4. Marchand S, Charest J, Li J, Chenard JR, Lavignolle B, Laurencelle L. Is TENS purely a placebo effect: a controlled study on chronic low back pain. *Pain* 1993; 54: 99–106.
5. Low J, Reed A. *Electrotherapy explained*. 2nd edn, Oxford: Butterworth Heinemann; 1994, pp. 39–116.
6. Truscott B. Interferential therapy as a treatment for classical migraine: case reports. *Aust J Physiother* 1984; 30: 33–35.
7. Schmitz RJ, Martin DE, Perrin DH, Iranmanesh A, Rogol AD. Effect of interferential current on perceived pain and serum cortisol associated with delayed onset muscle soreness. *J Sport Rehab* 1997; 6: 30–37.
8. Taylor K, Newton RA, Personius WJ, Bush FM. Effects of interferential current stimulation for treatment of subjects with recurrent jaw pain. *Phys Ther* 1987; 67: 346–350.
9. Asthon H, Ebenezer I, Golding J, Thompson JW. Effects of acupuncture and transcutaneous electrical nerve stimulation on cold-induced pain in normal subjects. *J Psychosom Res* 1984; 28: 301–308.
10. Johnson MI, Ashton CH, Bousfield DR, Thompson JW. Analgesic effects of different frequencies of transcutaneous electrical nerve stimulation on cold-induced pain in normal subjects. *Pain* 1989; 39: 231–236.
11. Johnson MI, Ashton CH, Bousfield DR, Thompson JW. Analgesic effects of different pulse patterns of transcutaneous electrical nerve stimulation on cold-induced pain in normal subjects. *J Psychosom Res* 1991; 35: 313–321.
12. Stephenson R, Johnson M. The analgesic effects of interferential therapy on cold-induced pain in healthy subjects: A preliminary report. *Physiother Theory Pract* 1995; 11: 89–95.
13. Johnson MI, Wilson H. The analgesic effects of different swing patterns of interferential currents on cold-induced pain. *Physiotherapy* 1997; 83: 461–467.
14. Johnson MI, Ashton CH, Thompson JW. Analgesic effects of acupuncture-like transcutaneous electrical nerve stimulation (TENS) on cold-induced pain (cold-pressor pain) in normal subjects. *Eur J Pain* 1992; 13: 101–108.
15. Johnson MI, Tabasam G. A double-blind placebo-controlled investigation into the analgesic effects of interferential currents and transcutaneous electrical nerve stimulation on cold induced pain in healthy subjects. *Physiother Theory Pract* 1999; 15: 217–233.
16. Marchand S, Bushnell MC, Duncan GH. Modulation of heat pain perception by high frequency transcutaneous electrical nerve stimulation (TENS). *Clin J Pain* 1991; 7: 122–129.
17. Melzack R, Wall PD. Pain mechanisms: A new theory. *Science* 1965; 150: 971–979.
18. De Domenico G. Pain relief with interferential therapy. *Aust J Physiother* 1982; 28: 14–18.
19. Basbaum AI, Fields HL. Endogenous pain control mechanisms: review and hypothesis. *Ann Neurol* 1978; 4: 451–462.
20. Basbaum AI, Fields HL. Endogenous pain control systems: brainstem spinal pathways and endorphin circuitry. *Annu Rev Neurosci* 1984; 7: 309–338.
21. Woolf CJ, Mitchell D, Barrett GD. Antinociceptive effect of peripheral segmental electrical stimulation in the rat. *Pain* 1980; 8: 237–252.
22. Palmer ST, Martin DJ, Steedman WM, Ravey J. Alteration of interferential current and transcutaneous electrical nerve stimulation frequency: effects on nerve excitation. *Arch Phys Med Rehabil* 1999; 80: 1065–1071.
23. Jette DU. Effect of different forms of transcutaneous electrical nerve stimulation on experimental pain. *Phys Ther* 1986; 66: 187–193.