Increased Sympathetic Tone Associated with Transcutaneous Electrical Stimulation

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Transcutaneous electrical stimulation (TES) is being used with increasing frequency in the management of a variety of painful disorders.[1,2] No definite mechanism of action has been agreed upon for its analgesic activity, but modulation of afferent information at the spinal cord level (*i.e.*, gate control) is a popular explanation.[3-5] Little has been published regarding the autonomic effects of transcutaneous electrical stimulation.

We have been using transcutaneous electrical stimulation in our pain clinic for about six months. We have not been using it for patients who have autonomic dysfunction, but we have noticed significant ipsilateral temperature changes during stimulation of extremities for painful disorders of presumed somatic origin. Some patients have increases in temperature, and others have decreases. Temperature change seems to show little correlation with analgesic effect.

Initial beneficial effects of transcutaneous electrical stimulation are frequently short-lived.[1] A placebo effect is a likely mechanism in many such cases, but chronically increased sympathetic tone may play a role in some later failures.

The following case report illustrates the effect of transcutaneous electrical stimulation on peripheral temperature and suggests the possibility of an adverse effect associated with the use of this therapeutic modality.

REPORT OF A CASE

A 75-year-old woman had had a laceration of the proximal portion of the right thumb nine years prior to her admission to the pain clinic. Several weeks after the injury a neuroma had developed, with cicatricial compression of the median nerve to the thumb, and she had undergone nerve decompression with placement of a silicone sleeve.

TABLE 1. Skin Temperatures over the Palms before and during Transcutaneous Electrical Stimulation

	Temperature before Stimulation (C)	Temperature 40 Minutes after Stimulation (C)
September 2		
Right hand (stimulated)	31.8	29.5
Left hand	31.8	32.8
September 5		
Right hand (stimulated)	33.2	29.5
Left hand	33.0	33.1
September 8		
Right hand (stimulated)	30.5	29.0
Left hand	31.0	32.9

Subsequent pain problems in the thumb prompted repeat neurolysis six years later. The patient did well for several weeks after that procedure, but her pain again recurred. The pain persisted unchanged for two years until she was seen in the pain clinic in October 1975. It was diminished only slightly by propoxyphene and aspirin (Darvocet), which she took regularly at bedtime.

The patient complained of severe burning pain and hyperesthesia involving the right thumb, thenar eminence, and web space. She had been unable to touch anything with the thumb for some time. She denied swelling, cold intolerance, increased sweating, or cyanosis of the involved extremity.

Examination revealed a well-healed scar at the base of the thumb, hypotrophic skin changes over the thumb, and severe hyperesthesia of the thumb and web space. No sensory or motor loss was found, and there was no change to suggest vasomotor or sudomotor hyperactivity. Skin temperatures of the two hands were identical.

A diagnostic stellate-ganglion block was proposed to the patient but, since she was quite reluctant to have the procedure done, it was elected to try transcutaneous electrical stimulation first. Stimulation over the radial aspect of the right wrist using a StimTech EPC stimulator† resulted in complete relief of pain within a few minutes. The relief lasted two and a half days, after which the pain returned to its usual level. The patient had noticed that her right hand felt cool for a short while after

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TABLE 2. Skin Temperatures over the Palms Following Stellate-ganglion Block and Subsequent Transcutaneous Electrical Stimulation*

	20 Minutes before Right Stellate Block (C)	1 Minute before Block (C)	20 Minutes after Block (at Onset of Right TES†) (C)	40 Minutes after Block (C)
January 19				
Right hand	25.8	29.0	35.1	35.5
Left hand	30.5	34.0	32.0	30.5

* Note that time was allowed for equilibration with the indoor environment.
† TES = transcutaneous electrical stimulation.

stimulation. Subsequent use of the stimulator in the clinic resulted in complete analgesia, usually lasting 24 to 36 hours, and produced decreases in skin temperature documented by a Yellow Springs temperature monitor with surface thermistors placed over the palsm of the hands (see table 1). The temperature in the treatment room was maintained at a constant 22 ± 0.5 C by a Honeywell thermostat.

The patient acquired a stimulator for use at home and used it once to several times a day for 30 minutes at a time for three months, with good control of her pain. However, she then began to experience pain in her entire hand, arm and shoulder, which had not been present in the past.

On examination in January 1976, two weeks after the onset of pain in the arm and shoulder, she had hyperesthesia of the entire right hand, which was noticeably cooler than the left hand, even though she had not used her stimulator that day. Skin temperature of the right hand was 25.8 C; left hand, 30.5 C. We performed a right paratracheal stellate-ganglion block with 7 ml 1.5 per cent lidocaine, which relieved all pain in the shoulder and arm and reduced the hyperesthetic area to the portion of the hand previously involved. Skin temperature of the right hand rose significantly (see table 2). Transcutaneous electrical stimulation was performed at the usual site 20 minutes after the block, and relieved the residual thumb pain. No further temperature change in the sympathetic-denervated extremity was evident after stimulation.

The patient continued to use the stimulator to control the thumb pain and, four days after the stellate block, pain in the arm and shoulder gradually returned. Infiltration of the scar with 0.25 per cent bupivacaine and triamcinalone diacetate, 25 mg/ml, was performed on three occasions about a week apart, and a repeat stellate-ganglion block was performed two weeks after the first sympathetic block. The pain in the shoulder and arm diminished considerably, though the patient still had intermittent burning pain in the thumb. Use of the stimulator was discontinued after the second sympathetic block.

DISCUSSION

Pain associated with sympathetic dysfunction is atypical in its distribution. Rather than following dermatomal or sclerotomal patterns, it tends to follow "vaso" zones, or vascular areas of distribution.[6] It is poorly localized, often aching in quality, and frequently accompanied by burning dysesthesia.

Within its range of electrical variables the transcutaneous electrical stimulator is capable of stimulating afferent and sympathetic efferent fibers. Our patient manifested little evidence of sympathetic dystrophy prior to the use of transcutaneous electrical stimulation. We postulated that the drop in cutaneous temperature following transcutaneous electrical stimulation was the result of either efferent sympathetic stimulation or afferent stimulation with reflex sympathetic hyperactivity. The absence of a decrease in temperature on stimulation of the sympathetically blocked extremity would point to the latter mechanism. Peripheral sensory nerve stimulation has in fact been shown to cause a pressor response.[7] The generalized pain in the arm and shoulder, burning dysesthesia of the entire hand, and persistent coolness of the skin may represent reflex sympathetic hyperactivity resulting from frequent electrical stimulation, while the long-standing pain in the scar is probably of somatic origin, as it was unaffected by sympathetic blockade.

It would seem prudent to monitor skin temperatures, at least during initial trials of transcutaneous electrical stimulation, and to examine patients periodically for evidence of

autonomic imbalance. Sympathetic blocks may be a useful adjunct to stimulation when such a problem arises. Further study of the effects of transcutaneous electrical stimulation on autonomic function seems warranted.

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Acute Medicine

SODIUM BICARBONATE AND CARDIAC ARREST Administration of alkali during cardiopulmonary resuscitation has become almost axiomatic. The authors question whether there are dangers in such a practice. Among possible problems are hyperosmolality, myocardial depression secondary to increased carbon dioxide tension, and cerebrospinal fluid acidosis. They therefore evaluated the effects of sodium bicarbonate treatment on blood gases and osmolality in mongrel dogs after ventricular fibrillation had been induced electrically. Following cardiac arrest apnea was allowed to supervene for three minutes, after which cardiopulmonary resuscitation was instituted. Nine dogs were treated (1 mEq/kg of 7.5 per cent sodium bicarbonate administered into the superior vena cava over a 15-second period following one minute of resuscitation), and seven served as controls. In untreated animals, *p*H decreased to 7.22 ± 0.04 (SEM) after 30 minutes of resuscitation. The *p*H remained above 7.35 during the first 15 minutes of resuscitation. Pa_{CO_2} was always below control, while osmolality of arterial blood rose from 310 ± 2 mOsm/kg prior to arrest to 334 ± 5 mOsm/kg after 30 minutes. In dogs treated with alkali, *p*H was 7.38 ± 0.05 prior to bicarbonate administration. A peak level of 7.56 ± 0.06 was observed two minutes following injection of bicarbonate. Pa_{CO_2} increased from 27 ± 2.7 torr prior to bicarbonate treatment to 49 ± 10 torr 15 seconds after therapy. Osmolality rose from 309 ± 5 to 349 ± 11 mOsm/kg two minutes following bicarbonate injection. In six human patients treated with bicarbonate during resuscitation similar data were obtained. The authors conclude that the routine use of sodium bicarbonate should be questioned in a number of circumstances: 1) when carbon dioxide elimination is inadequate; 2) in repeated doses when acidosis has not been confirmed objectively; 3) in the face of only a brief period of cardiac arrest when acidosis may be unlikely. (*Bishop RL, Weisfeldt ML: Sodium bicarbonate administration during cardiac arrest. Effect on arterial pH, P_{CO_2} and osmolality. JAMA 235: 506–509, 1976.*) ABSTRACTER'S COMMENT: The authors do well to call attention to possible dangers of any "routine" therapy. However, since cardiac arrest alone produced an increase in arterial osmolality, bicarbonate therapy only contributed partially to this phenomenon. In addition, only laboratory values were furnished. The complete story will not be known until detrimental functional changes resulting from alkalinization have been documented—*PJC*.