TRANSCUTANEOUS ELECTRICAL STIMULATION

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Abstract

Transcutaneous electrical stimulation as one of several ways to provide relief for patients with chronic neuropathic or neuromuscular pain. It will be sufficient as the only therapy for some and a useful adjunct for pain relief in many others. The key to successfuluse is an appropriate diagnosis. Pain is a common complaint that is frequently modified by ecological. Behavioral and socioeconomic factors. It is unlikely that trans-ptaneous stimulation or any other form of pain relief will affect these aspects of the pain sindrome. Pain is a common complaint that is frequently modified by ecological. Behavioral and socioeconomic factors. It is unlikely that trans-ptaneous stimulation or any other form of pain relief will affect these aspects of the pain sindrome.

INTRODUCTION

There have been several studies which demonstrate increase in pain threshold to experimental induced pain following transcutaneous electrical stimulation. Why these changes in pain threshold occur is less clear. There is additional information that determines that changes in parameters of stimulation can modify the effects. Much more has been done with parameter change with internal stimulators than with transcutaneous stimulation. The original stimulators provided a range of 60-100 Hz. Short bursts appear to be as effective as prolonged stimulation and square waves are usually employed. However, many different wave forms have been tested and seem to be effective. Effects are not parameter specific.

There is also controversy about whether the effects of TENS are reversible by naloxone. In our own work, we were unable to determine any effect of naloxone. Other investigators have shown that low-frequency stimulation pain relief is reversible by naloxone

MAIN TEXT

Transcutaneous electrical stimulation was originally based upon the general concept enunciated by Melzak and Wall in their gate-control theory. The hypothesis was that the stimulation of peripheral receptors would block or modify pain transmission from nociceptors probably at the spinal cord level and thus reduce pain. Several variations of this theme have been presented. It has been suggested that the block may be local at the receptor level or involve peripheral nerves. The stimulation may activate an inhibitory system at the level of the dorsal horn, or it may activate a descending inhibitory tract originating principally from thalamus and brainstem.

A placebo response have also been invoked. There are enough controlled studies that compare TENS with sham simulation, subliminal stimulation, and other kinds of sham therapies to demonstrate that the effect of stimulation is beyond what can be explained by placebo.

While very little direct research has been done on the possible effects of TENS, there is much material from pain in literature that provides additional information about possible bodes of action. There are accepted causes of pain, which include acute tissue injury, peripheral nerve injury, peripheral nerve compression, or spinal instability. All activate nociceptors. There are theoretical causes of pain for which there is strong experimental evidence. Nociceptors may be activated by chemical agents, such as might be derived from a degenerated disk. Nociceptors sensitization after injury is a well-accepted event, and associated mechanosensi-

tivity, such as occurs in types 2 and 3 neurons, has also been demonstrated. There is definite evidence that sensitization of a c-fiber nociceptors, which is usually confined at the site of the injury, is a first event in pain perception. Central sensitization of A nociceptors, which occurs in the site of injury and the surround, is also known to occur and sensitization of AB non-nociceptors, which produces allodynia, has also been demonstrated. The possibility that electrical stimulation modifies the sensitization

Process either through its local effects or at the level of the dorsal horn is a reasonable supposition. An experimental model that is used to investigate peripheral nerve injury also indicates that much of the perceived pain is mediated through adjacent uninjured nerves. A blocking effect preventing this spread of the pain signal is certainly possible.

Central facilitation of the reduction of pain threshold is known to occur. This central facilitation is reduced by pain-relieving procedures, and this is another possible explanation of the effects of electrical stimulation of the nervous system. The central inhibition may also be an important phenomenon. The central inhibition is impaired when pain is continuous and reappears when pain is controlled. Increasing the effectiveness of central inhibition is another possible explanation.

CONCLUSION

The use of electrical stimulation for pain control has a long history over the last 40 years or more. In the early 1970s, well-engineered controllable devices were developed and since that time, many well-controlled studies have indicated the value of the technique. There are many applications for which stimulation is a reasonable alternative to other symptomatic methods of pain control. Many other poorly designed and critical reports are in the literature describing transcutaneous electrical stimulation which fast been inexpertly applied to patients. When an appropriate diagnosis has been made and fe patient educated in the use of the technique, it remains a valuable adjunct in pain management that will provide satisfactory relief to many.

References

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