Research Report

Effect of Transcutaneous Electrical Nerve Stimulation on the Pressor Response to Static Handgrip Exercise

Background and Purpose. A proposed mechanism for the pain-relieving properties of transcutaneous electrical nerve stimulation (TENS) is gating of impulses carried by group III and IV afferent nerve fibers. The purpose of this study was to determine the effects of TENS on the pressor response to static exercise, a response mediated by group III and IV muscle afferents. Subjects. Sixteen subjects (9 men, 7 women) with no known history of cardiovascular, neurologic, or musculoskeletal disease participated. Methods. We measured arterial pressure, heart rate, and sympathetic activity during sustained, 25% maximal handgrip exercise. Each subject performed the handgrip exercise with and without conventional TENS applied to the ipsilateral forearm and, in a separate trial, to the contralateral leg. Results. The sympathetically mediated pressor response to handgrip exercise was blunted when TENS was applied to the ipsilateral forearm, but not when TENS was applied to the contralateral leg. Conclusion and Discussion. These data support the concept that central transmission of neural impulses traveling in group III and IV fibers can be modulated by other afferent inputs converging on the same spinal level. [Hollman JE, Morgan BJ. Effect of transcutaneous electrical nerve stimulation on the pressor response to static handgrip exercise. *Phys Ther.* 1997;77:28–36.]

Key Words: *Blood pressure, Static exercise, Sympathetic nervous system, Transcutaneous electrical nerve stimulation.*

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Transcutaneous electrical nerve stimulation (TENS) has been used in pain management for more than 20 years. One commonly used strategy, termed "conventional" TENS, consists of stimulation of large-diameter, superficial cutaneous nerve fibers using relatively high pulse frequencies (50–100 pulses per second), relatively short pulse durations (2–50 microseconds), and intensities below the motor threshold.[1] The gate control theory of Melzack and Wall[2,3] has been used to explain the effectiveness of this type of sensory-level TENS in reducing pain. According to the gate control theory, the transmission rate of action potentials from peripheral nociceptors to the central nervous system can be modulated by convergence of other afferent inputs at the level of the spinal cord. Specifically, transcutaneous electrical stimulation of group I and II (large-diameter, myelinated) afferent fibers is thought to modulate central transmission of pain impulses carried by group III and IV (small-diameter, lightly myelinated and unmyelinated) fibers via inhibition of second-order neurons located in the dorsal horn.

In addition to carrying information from nociceptors to the central nervous system, some group III and IV afferents mediate the pressor response to sustained isometric muscle contraction.[4–7] This reflex response originates in intramuscular receptors that are chemosensitive to metabolic by-products of contraction.[8,9] In turn, these receptors send afferent impulses via group III and IV fibers and the spinothalamic tract to brain-stem cardiovascular centers[10] where they trigger parallel increases in sympathetic outflow to vascular beds of active and inactive skeletal muscle[11] and substantial increases in arterial pressure.

The goal of our study was to determine the effects of TENS on the pressor response to isometric muscle contraction. We hypothesized that if transcutaneous stimulation of group I and II fibers modifies central transmission of action potentials in group III and IV fibers via a gating mechanism, then application of TENS during a sustained muscle contraction should attenuate the expected increases in arterial pressure and sympathetic neural outflow. Accordingly, we measured arterial pressure, heart rate, and sympathetic outflow to skeletal muscle during static handgrip exercise performed with and without concomitant application of TENS.

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This study was approved by the human subjects committees of the Center for Health Sciences, University of Wisconsin-Madison, and the Middleton Memorial Veterans Administration Hospital.

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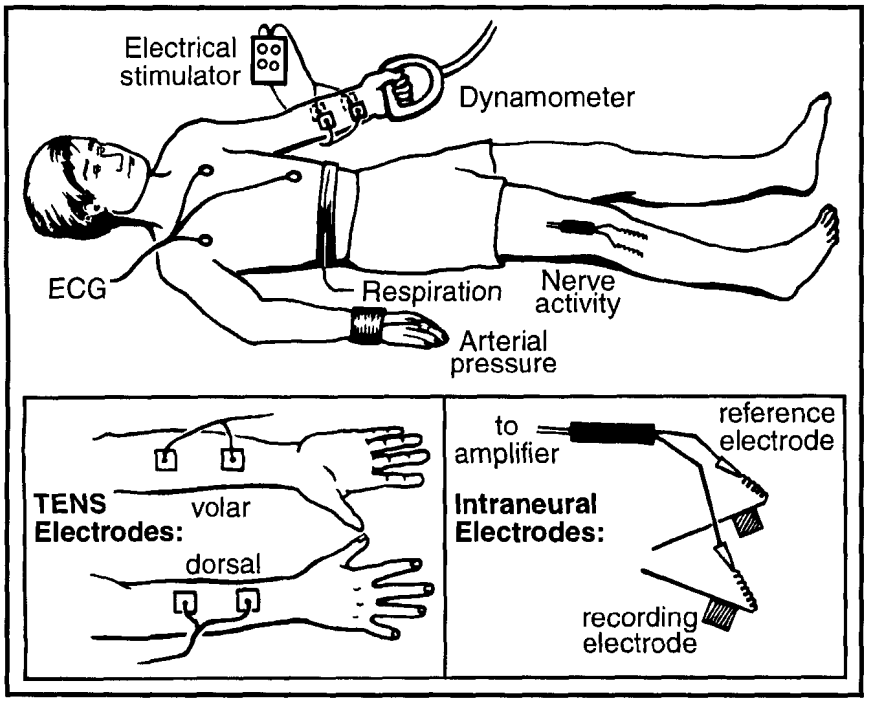

Figure 1.
Diagram showing the experimental setup for protocol 1. An identical setup was used for protocol 2, except that the transcutaneous electrical nerve stimulation (TENS) electrodes were applied to the right lower leg and sympathetic nerve activity was not recorded. (ECG=electrocardiogram.)

Method

Subjects

Sixteen subjects (9 men, 7 women) without known pathology, ranging in age from 19 to 46 years ($\bar{X}$=25, SD=6), were participants in this study. None of the subjects reported a history of cardiovascular, neurologic, or musculoskeletal disease. Informed consent was obtained from all subjects prior to participation.

General Procedure

Subjects were studied while positioned supine, at least 2 hours after a meal, with room temperature maintained at 24°±1°C. Thus, a stable baseline hemodynamic state was ensured by minimizing perturbations due to digestion and thermoregulation. Figure 1 illustrates the experimental setup. Heart rate was measured through a single-lead electrocardiogram. A continuous, beat-by-beat determination of arterial pressure was made by photoelectric plethysmography,* with the probe placed on the middle finger of the right hand. Measurements made with this device correlate well with intra-arterial measurements.[12] Because respiratory variations such as the Valsalva maneuver are known to alter sympathetic outflow and arterial pressure, subjects were instructed to maintain stable breathing patterns throughout the data-collection period. A stable breathing pattern was defined as the absence of sustained changes in rate or depth of breathing. Respiration was monitored using a Lafayette Instrument Model 76607 bellows pneumograph† to ensure compliance with this instruction. Sympathetic outflow to skeletal muscle was recorded via a microelectrode inserted percutaneously into the peroneal nerve.[13] The electrocardiogram, sympathetic neurogram, arterial pressure, respiration, and handgrip force (Lafayette Instrument Model 76618 dynamometer†) tracings were recorded continuously on paper‡ and videotape.§,‖

Recording of muscle sympathetic nerve activity. Recordings of postganglionic sympathetic nerve activity were made by the technique of Vallbo et al.[13] The bony prominence of the fibular head on the lateral aspect of the right leg was identified and marked. Brief electrical impulses (1 Hz, 3–8 mA) were delivered transcutaneously posterior to this mark to identify the location of the peroneal nerve. The skin was then cleansed, and two small epoxy-coated tungsten fine-wire electrodes# were inserted. First, a reference electrode was advanced to an area adjacent to the peroneal nerve. Then, a recording electrode was positioned within a muscle nerve fascicle, which was located by applying brief electrical impulses (1 Hz, 20 μA) to the electrode. Placement of the recording electrode within a muscle nerve fascicle was confirmed by (1) the presence of muscle twitches, not paresthesias, in response to electrical stimulation, (2) the pulse-synchronous nature of the nerve activity, (3) the appearance of afferent activity in response to tapping or stretching of muscle, but not gentle stroking of the skin, in the appropriate receptive field, and (4) the absence of neural activation in response to arousal stimuli. During wakefulness, arousal stimuli evoke increases in sympathetic outflow to skin, but not to muscle.[13] The neural signals were amplified (by 20–50 × 100), filtered (bandwidth of 700–2,000 Hz), rectified, and integrated (time constant of 100 milliseconds) to obtain a mean voltage display of sympathetic activity.

* Finapres model #2300 blood pressure monitor, Ohmeda, 3030 Ohmeda Dr, Madison, WI 53707.

† Lafayette Instrument, PO Box 5729, Lafayette, IN 47903.

‡ Model TA4000 physiologic recorder, Gould Inc, 3631 Perkins Ave, Cleveland, OH 44114.

§ Model 3000A PCM recording adaptor, AR Vetter Co, Box 143, Rebersburg, PA 16872.

‖ Model HR-D860U videocassette recorder, JVC Company of America, 41 Slater Dr, Elmwood Park, NJ 07407.

MN-10 microneurography electrodes, Iowa Doppler Products, PO Box 2132, Iowa City, IA 52244.

Once an acceptable recording (signal-to-noise ratio >3:1) was obtained, the subject was instructed to maintain the right leg in a relaxed position for the duration of the study. Compliance with this instruction was continuously monitored by inspection of the neurogram for contamination by alpha-motoneuron or mechanoreceptor afferent activities. Both sources of contamination are easily detected by an increase in the density of spikes on the filtered neurogram and by an upward shift in baseline on the mean voltage neurogram. Muscle sympathetic nerve activity is easily identified by its characteristic pulse-synchronous rhythm and its responsiveness to baroreflex stimuli. The postganglionic, sympathetic nature of this activity has been confirmed previously by (1) a conduction velocity of approximately 1 m/s, (2) elimination of activity with injection of local anesthetics proximal, but not distal, to the recording site, and (3) elimination with ganglionic blockade.[14] In the relaxed extremity, when alpha-motoneuron and chemoreceptor or mechanoreceptor afferent activities are absent, the predominant nerve traffic in muscle fascicles of mixed peripheral nerves is sympathetic vasoconstrictor outflow.

Transcutaneous electrical nerve stimulation. A two-channel stimulator[**] with four 6.25-cm^2 polymer-gel electrodes[††] was used. The electrodes were positioned on the left forearm with separate channels on the flexor and extensor surfaces (Fig. 1). The electrodes were placed directly over muscle bellies, which were identified by palpation during resisted wrist flexion and extension.[15] A continuous current output with a balanced, rectangular, biphasic waveform was verified by oscilloscope. Clinically relevant stimulation settings (frequency=60 pulses per second, pulse duration=100 microseconds) were used in all trials. For each subject, the motor threshold was determined by gradually increasing the stimulation intensity until a muscle contraction was palpable. Then, the stimulator output was reduced to a level just under the motor threshold.

Static handgrip exercise. Subjects performed static handgrip exercise of the left forearm for 2 minutes at a work load equal to 25% of a previously determined maximal grip force. Each 2-minute handgrip was preceded by 2 minutes of baseline data collection and followed by 2 minutes of recovery data collection. Dynamometer force output and the target work load were displayed on a dual-trace oscilloscope for continuous subject feedback. At the completion of each 6-minute trial, the subjects were asked to rate perceived effort using the 15-grade Borg scale.[16]

[**] Dynex model 2005 portable stimulator, LaJolla Technology Inc, 11558 Sorrento Valley Rd, San Diego, CA 92121.

[††] ComfortEase, Empi, 1275 Grey Fox Rd, St Paul, MN 55112.

Experimental Design

Subject familiarization and test-retest protocols. Maximal handgrip force of the left hand was determined by taking the highest output obtained in three trials, each 1 second in duration. Subjects were familiarized with the monitoring equipment, and the handgrip exercise protocol was explained. Heart rate and arterial pressure were measured while subjects performed 25% maximal static handgrip exercise for 2 minutes with and without concomitant application of TENS over the contracting muscles. To minimize fatigue, a 10-minute rest period was allotted between trials. This protocol was performed to eliminate the possibility that the novel experience of the test environment, the handgrip exercise, or the TENS application would affect the subjects' neurocirculatory responses to handgrip exercise. The data from this familiarization protocol were not subjected to analysis. On a separate day, subjects returned to the laboratory so that the reproducibility of neurocirculatory responses to handgrip exercise could be evaluated. Heart rate and arterial pressure were measured while subjects performed 25% maximal static handgrip exercise for 2 minutes without concomitant application of TENS. After a rest period of 10 minutes, the handgrip exercise was repeated. Muscle sympathetic nerve activity was not measured as part of the preliminary protocols.

Protocol 1: pressor response to static handgrip exercise with TENS applied to the ipsilateral forearm. On a third day, 25% maximal static handgrip exercise was performed by each subject with and without concomitant application of TENS. The order of the trials was randomized by a coin toss. A 10-minute rest period was allotted between trials. The TENS electrodes were placed within dermatomes that corresponded to the myotomes containing muscles used in the handgrip exercise (C-6 to T-1) (Fig. 1). Heart rate, arterial pressure, and sympathetic outflow to skeletal muscle were measured before, during, and after each handgrip exercise trial.

Protocol 2: pressor response to static handgrip exercise with TENS applied to the contralateral leg. The results of protocol 1 indicated that neurocirculatory responses to handgrip exercise were diminished when sensory-level TENS was applied to skin overlying the contracting muscle groups. To examine the mechanism responsible for this diminution, 4 of the original subjects and 6 new subjects were studied on separate days. Heart rate and arterial pressure were measured while the subjects performed 25% maximal handgrip exercise with TENS and without TENS applied to the contralateral leg. Sympathetic outflow to skeletal muscle was not measured in protocol 2. The TENS electrodes were placed within dermatomes (L-4 to S-1) unrelated by spinal segment to the contracting muscles. To ensure that the number of

Table 1.
Arterial Pressure and Heart Rate Responses to 25% Maximal Isometric Handgrip With and Without Transcutaneous Electrical Nerve Stimulation (TENS) Applied to the Ipsilateral Forearm

	Baseline				Handgrip				Recovery			
	With TENS		Without TENS		With TENS		Without TENS		With TENS		Without TENS	
Variable	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
Systolic pressure (mm Hg)	138	16	136	20	168	23	176	33	139	17	140	10
Diastolic pressure (mm Hg)	77	14	75	17	101	16	107	24	77	12	77	17
Heart rate (beats per minute)	75	12	74	16	91	16	93	21	68	10	69	7
Muscle sympathetic nerve activity												
Frequency (bursts per minute)	29	7	24	8	47	13	60	18	27	6	31	10
Total activity (% baseline)	100		100		203	20	267	13	119	8	117	8

sensory afferents stimulated by TENS was comparable in both protocols, electrodes were placed on an area of the leg with 2-point discrimination[17] similar to that of the forearm. The purpose of this protocol was to determine whether nonspecific stimulation of cutaneous afferents can blunt the pressor response to static muscle contraction.

Data Analysis

Data for each handgrip trial were analyzed by an investigator who was blind to the type of trial (ie, with TENS or without TENS). The average force output during handgrip exercise was calculated by dividing the area under the dynamometer output tracing by the length of the contraction (in seconds). Sympathetic bursts were identified from the mean voltage neurogram using a computer program with a sampling rate of 128 Hz.[18] For purposes of quantification, sympathetic nerve activity was expressed as burst frequency (in bursts per minute) and as total minute activity (in bursts per minute $\times$ mean burst amplitude). Segments of the recording that we believed showed evidence of alpha-motoneuron or mechanoreceptor activity caused by muscle tension were excluded from analysis. Values for arterial pressure, heart rate, and sympathetic nerve activity obtained during the control period and during the final 15 seconds of handgrip exercise were used for analysis. Changes in arterial pressure, heart rate, and sympathetic nerve activity from baseline to the second minute of handgrip exercise during with-TENS and without-TENS trials were compared by paired t tests. Ratings of perceived exertion during the two trials were compared by paired t tests. Values at the $P<.05$ level were considered significant. In the text and figures, data are presented as mean $\pm$ standard deviation.

Results

Reproducibility of Heart Rate and Arterial Pressure Responses to Static Handgrip Exercise

Coefficients of variation, estimates of random variability, obtained in test-retest trials were 0.30 for systolic pressure, 0.36 for diastolic pressure, and 0.35 for heart rate. Paired t tests, estimates of systematic variability, revealed no differences in any of the variables between the first and second trials.

Effects of TENS on Baseline Hemodynamic Variables

Comparable baseline values for systolic pressure, diastolic pressure, heart rate, and sympathetic burst frequency were observed before the initiation of handgrip exercise in the with-TENS and the without-TENS trials (all $P>.10$) (Tab. 1).

Responses to Static Handgrip Exercise Performed With and Without TENS Applied to the Ipsilateral Forearm

Group mean values for handgrip force output were similar in the with-TENS versus the without-TENS trials (8.9 ± 3.2 versus 9.0 ± 3.1 kg, $P>.10$). On average, these outputs represent 25.5% and 25.8% of the maximal grip force, respectively. Ratings of perceived exertion during gripping were not altered when TENS was applied (14.3 ± 1.4 versus 14.6 ± 1.9 Borg units, $P>.10$).

When static handgrip exercise was performed with concomitant application of TENS over the ipsilateral forearm, sympathetic activation was attenuated. The amount of sympathetic activation, expressed both as increase in burst frequency and as percentage of increase in total minute activity was smaller in with-TENS trials than in without-TENS trials ($P<.05$) (Tab. 1, Figs. 2–4). The handgrip-induced increase in systolic pressure was smaller during with-TENS trials than during without-TENS trials ($P<.05$) (Tab. 1, Fig. 5). There was a trend toward a smaller diastolic pressure response to handgrip exercise with TENS ($P=.07$) (Fig. 5). In contrast, the

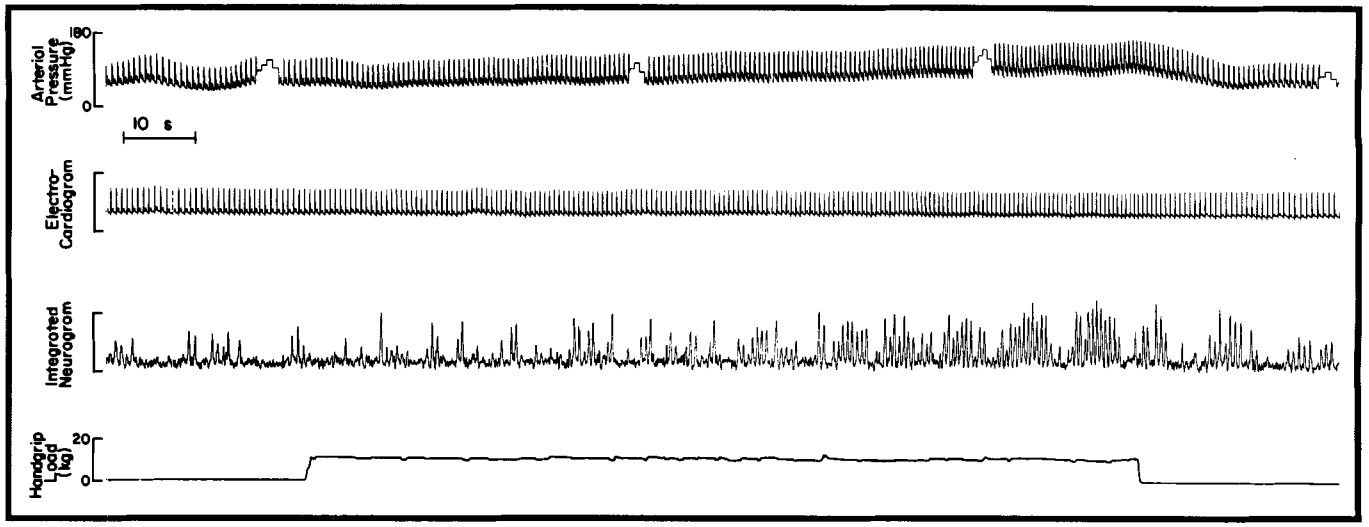

Figure 2.
Original record showing arterial pressure, heart rate, and peroneal neurogram before, during, and after handgrip exercise performed without application of transcutaneous electrical nerve stimulation to the forearm. As expected, static handgrip exercise caused progressive, time-dependent increases in arterial pressure, heart rate, and frequency and amplitude of sympathetic bursts.

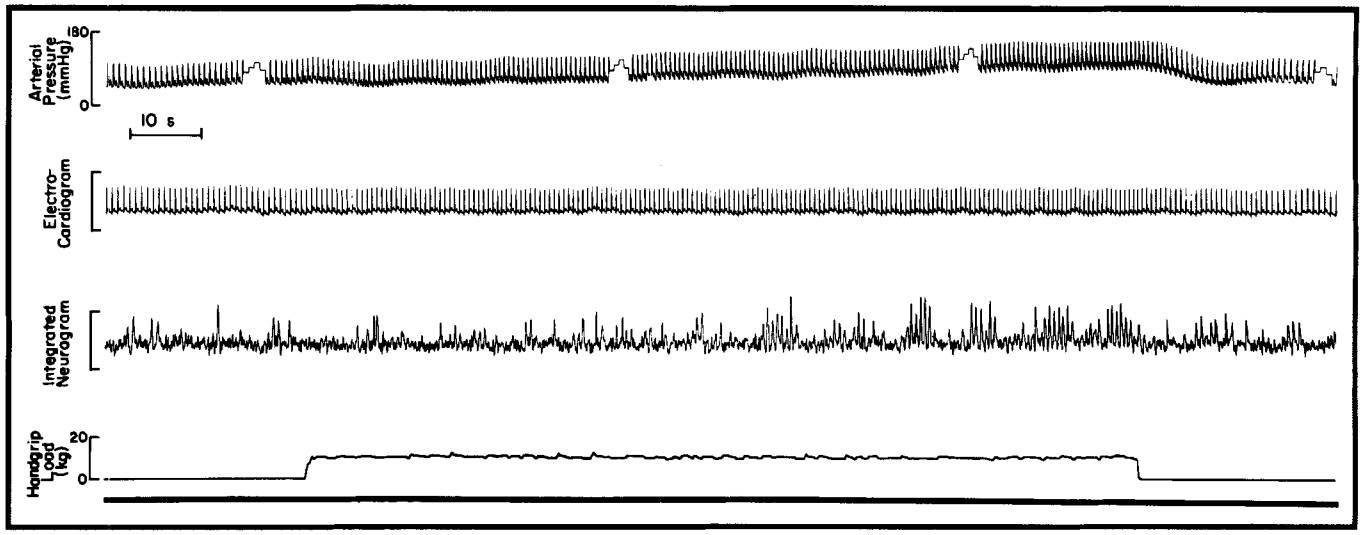

Figure 3.
Original record showing arterial pressure, heart rate, and peroneal neurogram before, during, and after handgrip exercise. Transcutaneous electrical nerve stimulation (TENS) was applied to the forearm throughout the test session. Handgrip-induced increases in arterial pressure and sympathetic nerve activity were smaller in the with-TENS trial than in the without-TENS trial.

heart rate response to handgrip exercise was not modified by TENS ($P>.10$) (Fig. 5).

Responses to Static Handgrip Exercise Performed With TENS Applied to the Contralateral Leg

Transcutaneous electrical nerve stimulation applied to the contralateral leg had no effect on the cardiovascular responses to static handgrip exercise. There was no difference in the handgrip-induced increases in systolic pressure, diastolic pressure, or heart rate between the with-TENS and the without-TENS trials (all $P>.10$) (Tab. 2, Fig. 6).

Discussion

We found that the sympathetically mediated pressor response to static handgrip exercise was attenuated by concomitant application of TENS to skin overlying the contracting muscle groups. In contrast, we found that the pressor response to static handgrip exercise was not affected by application of TENS to segmentally unrelated dermatomes in the contralateral leg. These findings suggest that central transmission of neural impulses traveling in group III and IV afferent fibers can be modulated by other afferent inputs converging on the same spinal level.

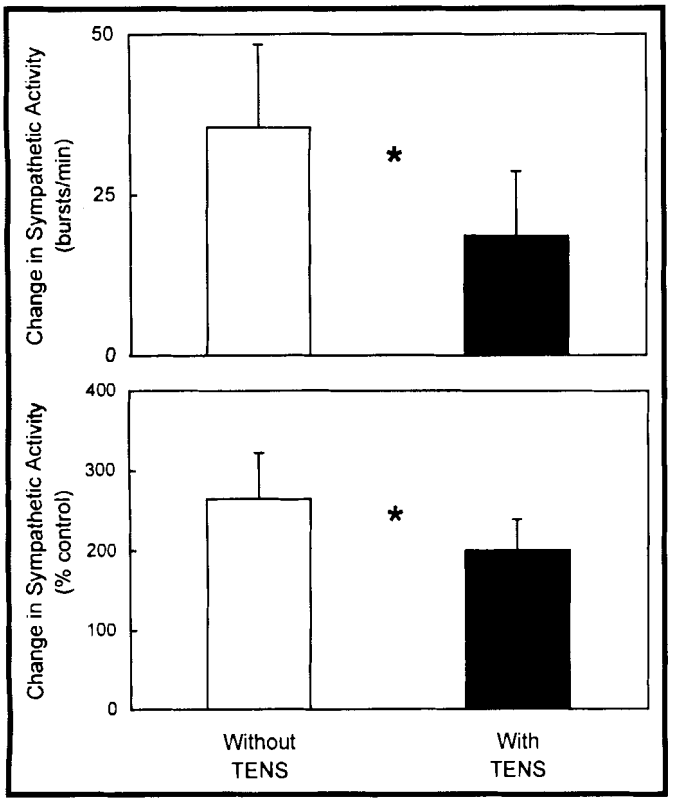

Figure 4.
Bar graphs depict the group mean values for increases in sympathetic burst frequency (upper panel) and sympathetic minute activity (lower panel) during static handgrip exercise performed without transcutaneous electrical nerve stimulation (TENS) (open bars) and with TENS (filled bars) applied to the ipsilateral forearm. Values shown are means ± standard deviation. Asterisk (*) indicates $P<.05$, without TENS versus with TENS.

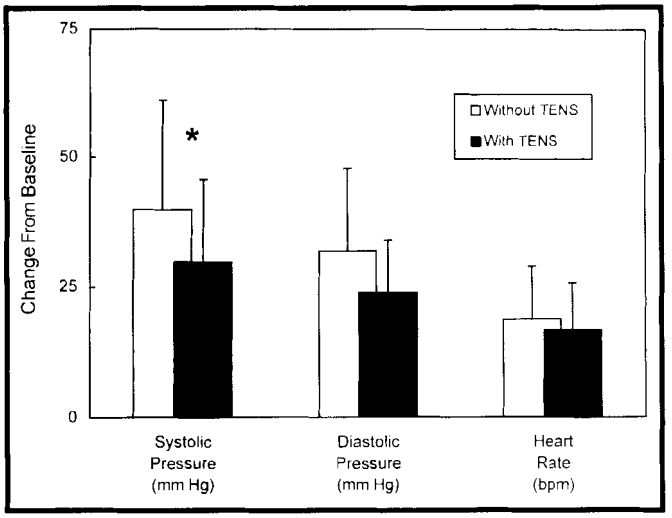

Figure 5.
Bar graphs depict the group mean values for increases in systolic pressure, diastolic pressure, and heart rate during static handgrip exercise performed without transcutaneous electrical nerve stimulation (TENS) (open bars) and with TENS (filled bars) applied to the ipsilateral forearm. Values shown are means ± standard deviation. Asterisk (*) indicates $P<.05$, without TENS versus with TENS.

Our findings provide experimental support for the concept that the pain-relieving properties of conventional TENS can be explained, at least in part, by the gate control theory. This interpretation is predicated on the assumption that static handgrip exercise stimulates the same afferent fibers that are involved in transmitting pain messages. There is a substantial body of evidence that demonstrates that reflex neurocirculatory responses to static muscle contraction are initiated by stimulation of group III and IV afferents[4–7] and that these same afferent fibers mediate pain.[19] Our findings are most analogous to the situation of acute pain, where afferent input from nociceptors can be demonstrated.

The TENS-induced attenuation of the sympathetic nervous system and arterial pressure responses to static handgrip exercise was a consistent and robust finding. This attenuation was observed in 8 of 10 subjects. The differences between with-TENS and without-TENS trials were statistically significant despite the fact that there was a high degree of random variability in the responses (coefficients of variation=0.30–0.35).

We considered the possibility that TENS could have altered the pressor response to static exercise via descending control mechanisms. That is, the level of "central command" necessary to maintain the required force output may have been reduced by TENS. This explanation is unlikely because we used a level of stimulation that was below the motor threshold. In addition, the heart rate response to static handgrip exercise was not affected by TENS. If central command had been reduced by TENS, a smaller increase in heart rate would have been expected.[20] Our findings support the view that peripheral reflex mechanisms are primarily responsible for causing increased sympathetic activity and arterial pressure during static exercise, whereas central mechanisms are primarily responsible for causing increased heart rate.[20,21]

In addition to the gate control theory, another proposed mechanism for the pain-relieving properties of sensory-level TENS is direct peripheral block of neural transmission in afferent fibers.[1] Because this concept has received some experimental support,[22–24] we considered the possibility that physical blockade of afferent impulses in group III and IV fibers was responsible for the observed blunting of the pressor response to static handgrip exercise. Transcutaneous stimulation at the frequency, intensity, and pulse duration used in our study, however, activates only large-diameter, superficial cutaneous afferents.[1] Because muscle afferent fibers are not likely to be stimulated in this manner, we doubt that blocked transmission in muscle afferents was responsible for our findings.

Table 2.
Arterial Pressure and Heart Rate Responses to 25% Maximal Isometric Handgrip With and Without Transcutaneous Electrical Nerve Stimulation (TENS) Applied to the Contralateral Leg

	Baseline				Handgrip				Recovery			
	With TENS		Without TENS		With TENS		Without TENS		With TENS		Without TENS	
Variable	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
Systolic pressure (mm Hg)	112	23	119	25	165	29	166	29	114	19	121	16
Diastolic pressure (mm Hg)	57	12	62	15	90	11	97	19	58	10	66	13
Heart rate (beats per minute)	73	16	75	18	92	15	96	17	70	15	73	20

In anesthetized cats, electrical stimulation of low-threshold skin and muscle afferents has been shown to decrease sympathetic activity and arterial pressure.[4] This mechanism may have been responsible for the attenuated pressor response to static handgrip exercise observed in our subjects. If so, a nonspecific effect of skin afferent stimulation, rather than segmental inhibition of second-order neurons in the dorsal horn (ie, a gating mechanism), could explain our findings. Two lines of evidence argue against this possibility. First, TENS did not affect baseline nerve traffic or arterial pressure. Second, when handgrip was performed with TENS applied to a part of the body unrelated by spinal segment to the contracting muscles, there was no attenuation of the pressor response.

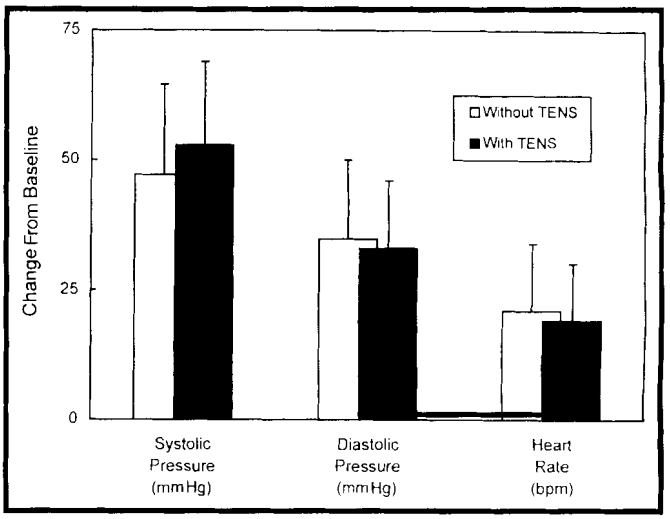

Figure 6.
Bar graphs depict the group mean values for increases in systolic pressure, diastolic pressure, and heart rate during static handgrip exercise performed without transcutaneous electrical nerve stimulation (TENS) (open bars) and with TENS (filled bars) applied to the contralateral leg. Values shown are means ± standard deviation.

Our findings are consistent with those of previous studies with experimental animals that provided both direct and indirect evidence that TENS can modify central transmission of noxious stimuli via a gating mechanism. In anesthetized monkeys, TENS inhibited C-fiber-induced activity in spinothalamic-tract neurons of the lumbosacral spinal cord.[25] This response was not altered by injection of naloxone, indicating that the mechanism of inhibition did not involve endogenous opioid substances. In anesthetized cats, both spontaneous and noxiously evoked activity of lumbar dorsal horn cells were reduced by application of TENS.[26] Furthermore, in lightly anesthetized rats, electrical stimulation of dissected skin nerves at clinically relevant frequencies profoundly inhibited the flexion response to noxious stimuli.[27]

An initial report based on eight case studies suggested the potential usefulness of high-frequency, low-intensity electrical nerve stimulation in treating humans with chronic pain.[28] Since then, prospective, randomized, placebo-controlled studies of the clinical efficacy of sensory-level TENS have yielded inconsistent results. The most convincing positive evidence comes from studies of high-frequency, low-intensity TENS in the setting of acute pain.[29,30] In contrast, studies of the effects of high-frequency, low-intensity TENS on chronic pain have yielded mainly negative results.[31–33] As the authors of the most recent of these reports point out, the perception of chronic pain may be dependent, at least in part, on learned pain behavior rather than peripheral nociceptor stimulation.[32]

The findings of our study are not directly generalizable to the clinical setting because we did not study the effects of TENS on pain. Our research design, however, has the advantage of being based on measurements of neurocirculatory function instead of perceptions of pain. Although clinically relevant stimulation settings were used in our study, our experiments with asymptomatic individuals do not provide evidence for the clinical effectiveness of TENS in pain management. The perception of pain is a complex psychophysiologic phenomenon that involves descending as well as ascending neural pathways subserved by multiple neurotransmitters and receptors. In addition, distinct mechanisms may be responsible for chronic and acute pain perception.

Conclusion

Our data demonstrate that application of TENS can attenuate the reflex pressor response to static exercise in humans. This effect was seen when TENS was applied to dermatomes related by spinal segment to the contracting muscles but not when TENS was applied to unrelated dermatomes. These findings support the view that central transmission of neural impulses traveling in group III and IV afferent fibers can be modulated at the spinal cord level by concomitant input from other fiber types. The clinical implication is that these findings provide a physiologic substrate for use of conventional TENS in management of acute pain. Future studies are planned to determine whether TENS modifies the neurocirculatory and respiratory responses to dynamic exercise, a form of muscular work that is commonly encountered during activities of daily living.

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