Heat-Induced Pain Diminishes Vibrotactile Perception: A Touch Gate

A. Vania Apkarian,*,†,[1] Richard A. Stea,* and Stanley J. Bolanowski†

**Department of Neurosurgery, SUNY Health Science Center, Syracuse, New York 13210,*
†Institute for Sensory Research, Syracuse University, Syracuse, New York 13244

Abstract The gate control theory of pain (Melzack and Wall, 1965) suggests that tactile stimuli can decrease the perception of pain. We have found the reverse effect: Heat at levels that induce pain can substantially suppress tactile sensitivity, independently of shifts in attention or arousal. Ten human observers were stimulated by a tonic, pain-producing heat stimulus and vibrotactile stimuli (1, 10, and 100 Hz) coincidentally presented to the right thenar eminence. Vibrotactile thresholds were assessed with the skin at a normative temperature of 31°C and at higher temperatures producing pain. Increases in vibrotactile thresholds (mean change = 7.3 dB) occurred at skin temperatures just below and above those that induced pain. Furthermore, absolute-magnitude estimates of suprathreshold vibrotactile stimuli determined during the same experiments showed decreased sensitivity and psychophysical recruitment. The changes are not attributable to attentional or arousal shifts, since they were not associated with changes in auditory thresholds. Furthermore, the changes occurred just below the subjects' pain thresholds (where nociceptors are presumably activated). This over-twofold diminution of vibrotactile sensitivity suggests that heat stimuli capable of inducing pain can significantly diminish taction, perhaps through a "touch gate" in a manner similar to the gate control theory proposed for pain.

Key words tactile psychophysics, vibrotactile thresholds, magnitude estimation, pain

In 1965, Melzack and Wall proposed the gate control theory of pain. The major tenet of the theory is that large myelinated fibers, conveying innocuous touch information, can inhibit pain perception by closing a spinal cord gate balanced by noxious (small-fiber activity) and innocuous (large-fiber activity) inputs. This theory, consistent with the common behavior of rubbing the skin to relieve pain, generated a large amount of research and was important in designing clinical procedures for pain relief (for references, see Fields, 1987). The opposite interaction—namely, the effect of pain on touch perception—has not been studied, and yet everyday observations clearly indicate that this effect occurs. For example, immersion of the hand in painfully hot water diminishes the touch and wetness perceptions that are commonly associated with palpation of moving warm liquids (as in taking a hot shower). Also, a mild pin prick when first perceived as a "prick" is not accompanied with touch or pressure—perceptions that should be present simply by skin deformation. Similarly, the perception of the movement of a single hair on the dorsum of the hand can be completely abolished by a mild pin prick applied near the stimulated hair (R. B. King, personal communication). Thus, under many conditions where touch, pressure, and pain are coincident, a mild pain seems to inhibit the other cutaneous sensory components. The present study examined the effects of sustained and intense heat stimuli, which produced mild pain, on vibrotactile thresholds and suprathreshold touch perception in humans.

1. To whom all correspondence should be addressed, at Department of Neurosurgery, SUNY Health Science Center, Syracuse, New York 13210.

METHODS

A total of 10 healthy subjects, 5 males and 5 females, participated in the study. All were in good health and signed an informed consent statement after the nature of the study was explained. Five of the subjects had considerable experience in vibrotactile experiments and were tested during six separate sessions in a variety of tasks designed to explore the possibility that painful heat can diminish tactile sensation. These tasks included the measurement of vibratory thresholds, auditory thresholds, and sensation magnitudes, as well as ratings of the level of pain induced by noxious thermal stimuli.

Accepted June 10, 1994

The five naive subjects were tested in two separate sessions in a subset of these tasks. None of the subjects had prior experience in rating experimental pain, but they were informed about its qualitative (i.e., unpleasantness) and quantitative (i.e., intensity) aspects (see Price et al., 1983).

The apparatus used to deliver the vibratory and heat stimuli has been described in considerable detail in Bolanowski and Verrillo (1982), and has been used extensively in studies assessing the capabilities of the tactile system (e.g., Bolanowski and Verrillo, 1982; Verrillo and Bolanowski, 1986; Bolanowski et al., 1988). Vibratory displacements of the skin were produced with a Goodman's 390A vibrator under computer control and applied to the thenar eminence (i.e., the glabrous portion of the hand at the base of the thumb). The vibrator and attached equipment rested on the base of a modified drill-press platen, the height of which could be adjusted. The subject was seated to one side of the stimulator and placed the hand, palm down, onto the stimulator. The vibration assembly, the subject, and the necessary equipment were located in a soundproofed chamber to provide isolation from extraneous mechanical and airborne vibrations.

Stimuli were applied with a 2.9-cm^2 aluminium contactor surrounded by a rigid aluminium annular-shaped surface (33 cm^2), which confined the deformations to the area of stimulation. The contactor and surround were separated by a 1-mm gap. The stimuli were presented around a static indentation of 0.5 mm to ensure continuous contact between the stimulator and the skin. The static indentation was set by first establishing electrical continuity between the contactor and the skin surface, and then advancing the contactor 0.5 mm. The vibratory displacements were monitored with an electromagnetic linear-variable differential transformer (LVDT), which sensed the displacement of the moving element of the vibrator. Displacements were measured in decibels, referenced to 1-μm peak amplitude with the aid of a lock-in amplifier. The vibratory stimuli were presented as sinusoidal bursts having durations calculated at the half-power points of 1000 msec. The rise–fall time at the onset and offset of the stimulus burst was 50 msec and followed a cosine function. The frequencies of stimulation used were 100, 10, and 1 Hz, chosen to selectively activate three of the four psychophysically defined tactile channels of glabrous skin—the Pacinian (P), non-Pacinian I (NP I), and non-Pacinian III (NP III channels), respectively (Bolanowski et al., 1988). It has been shown that variations in skin surface temperatures alone can produce significant changes in tactile thresholds (Green, 1977; Bolanowski and Verrillo, 1982; Verrillo and Bolanowski, 1986). Because of this, the stimuli were chosen to specifically activate the individual channels at frequencies where skin surface temperature changes minimally affect thresholds (Bolanowski et al., 1988).

The temperature of the skin surface underlying both the contactor and the surround was controlled by circulating water at the appropriate temperature through hollow chambers in both the contactor and the surround. A heating and refrigeration unit and pump controlled both the water temperature and the circulation flow rate. The contactor and surround were placed in series such that the water from the circulating unit first flowed through the contactor and then through the surround, eventually returning to the circulation unit. The skin surface temperature at both the contactor–skin interface and the surround–skin interface, monitored by small thermistors the active surface of which touched the skin, were always within ±0.2°C. The skin surface temperatures used in the present study ranged from 31.0° to 44.6°C, with the higher temperatures capable of inducing the sensation of pain. With this arrangement, the vibratory and heat stimuli were colocalized; the vibratory stimuli were applied to the central 2.9-cm^2 area, and the heat stimuli were applied over an area of 35.9 cm^2. In order to determine the level of pain associated with the heat stimuli, subjects were instructed as to the various qualities of pain, and told to rate only their perception of pain intensity (as described by Price et al., 1983) on a graded analogue scale by manipulating a knob attached to a rheostat with their free hand. A scale on the dial was divided into six categories describing increasing levels of pain, from "no pain" to "intolerable pain," and numbered from 0 to 10 (see Ellenmeier and Westphal, 1991). The total heat duration used during the vibratory testing while the subjects were experiencing pain was a mean of 19.2 min, ranging from 5.5 to 45 min. This range for the duration of the painful heat was large, since sessions where only threshold changes were studied (the sessions with the naive subjects; see above) were shorter than the sessions where threshold and suprathreshold responses were obtained (the sessions with the experienced subjects; see above).

Vibratory thresholds were measured via two different methods. The first method was a two-alternative, forced-choice tracking procedure, which accurately measures detection thresholds uninfluenced by subjective criterion (Zwislocki et al., 1958). This method was used only on the experienced subjects. During the threshold determination, subjects judged which of two sequential intervals of time, demarcated by reference lights, contained the test stimulus. They made their judgments by depressing one of two switches; the responses were recorded by the computer. Feedback as to whether the response was correct or not was provided via a visual (light) cue. The probability that the test stimulus would occur in one interval versus the other was 0.5. The intervals were separated in time by 1 sec. The stimulus intensity was increased by 1 dB for every response error and decreased by 1 dB for every three correct (not necessarily consecutive) responses. In this manner, the criterion used to determine threshold was 75% correct detection over a period of 3 min. The actual period of stimulation was considerably longer than this, since the stimulus inten-

sity was always set at a suprathreshold level at the beginning of each test. Generally, a period of 3–6 min was required before the subject reached the approximate threshold level, at which time the 3-min period of 75% detection was started.

Although forced-choice tracking is the preferred method for determining threshold because it is subject-criterion-free, a considerable amount of time is required to obtain an accurate threshold. Since the experiments reported herein required that volunteers be subjected to painful stimuli, a second method was used to determine vibratory thresholds more quickly. The method was a modified ascending and descending method of limits (Gescheider, 1985), which permitted the evaluation of threshold within 2 min. With this method the vibratory stimuli were first delivered in a decreasing series of intensities, manually adjusted by the experimenter in 2-dB steps, until the subject verbally reported the absence of the vibratory stimulus. At that time the experimenter reversed the procedure, increasing the stimulus in 2-dB steps until the subject reported the presence of the stimulus. Several ascending and descending series were presented until the experimenter was able to determine the approximate threshold. This latter method for determining threshold was used on both the experienced and the naive subjects. The thresholds obtained for the experienced observers with the two methods showed no difference statistically (t test, $p < 0.05$). Because of this, average thresholds were calculated by combining all measurements, regardless of the method by which they were achieved. In most cases, a minimum of three threshold determinations under each pain condition and each vibratory stimulus (i.e., 1, 10, or 100 Hz) were obtained and used to calculate mean threshold values for the individuals. One naive subject without threshold increases at 100 Hz under the painful conditions was insensitive (more than 2 standard deviations away from the group mean thresholds in both hands) to vibrotactile stimuli at this frequency, suggesting abnormality (or absence) of the P channel. This subject's data at 100 Hz were not included in the rest of the analysis. Individual mean values were used to calculate group means.

In addition to the vibratory thresholds, binaural auditory detection thresholds to a continuously presented 1-kHz tone were determined via the modified ascending and descending method of limits already described. Auditory thresholds were determined on only 8 of the 10 subjects. Tones were presented via headphones, which a subject wore throughout an experimental session. When the auditory tests were not being conducted, the subjects were presented with band-filtered white noise through the headphones at a level sufficient to mask any sound produced by the moving element of the vibrator.

Scaling of the sensation magnitude of the vibratory stimuli was performed on the experienced subjects via the modulus-free method of absolute-magnitude estimation (Verrillo et al., 1969; Zwislocki, 1983; Bolanowski and Gescheider, 1989). For this purpose, the subjects first performed an estimation of line length as a simple training task (Stevens, 1975; Zwislocki, 1983). During the experimental session, absolute-magnitude estimation was done by presenting vibrotactile bursts of different amplitudes in a predetermined pseudorandom order. Four to six suprathreshold intensities of equal 8.0-dB increments were presented four times in each trial, with each subject calling out numbers in relation to the perceived magnitude of vibration. The total number of increments was limited by the maximum displacements that could be produced by the vibrator at any particular frequency of stimulation. Data from the last three presentations were used to calculate geometric means of the magnitude estimates for individual subjects. These values were then used to calculate the geometric means of the group.

A typical session during which a subject was tested lasted between 1 and 2 hr, depending upon the particular vibratory tests performed. The number of sessions in which a particular subject participated depended upon whether the subject was in the naive or experienced group. The naive subjects were tested in two sessions, during which tactile thresholds were determined for all three vibratory stimuli. To help ensure that the effects were not confounded by unfamiliarity with the tasks, only the data obtained during the second sessions were used. The experienced subjects were tested in several sessions, during which thresholds were determined and suprathreshold measurements were made. For these experienced subjects, only two vibration frequencies were tested during any one session. Thus at least three sessions per subject were required to obtain results at all three stimulus frequencies. In all, the thresholds of the experienced subjects were tested four times at each stimulus. No differences in the results was found between the naive and the experienced subjects (t test, $p < 0.05$).

At the beginning of a test session, vibrotactile thresholds (naive and experienced subjects) and suprathreshold estimates (experienced subjects only) at 1, 10, and 100 Hz were measured on both the right and left hands at a skin surface temperature of 31°C. These threshold and suprathreshold values provided the normative controls. The procedure took approximately 25 min. The hand was then removed from the stimulator, which was then heated to 40°C. This temperature was selected for use during preliminary experiments, since it was about 1–2°C below most subjects' heat-induced pain thresholds. Following the heating of the stimulator, the right hand was again placed upon it, and the subject was instructed to begin rating the degree of pain produced. At this level of thermal stimulation, which was subthreshold to the sensation of pain, several threshold vibratory tasks were performed on the naive subjects. This period of testing lasted approximately 5–15 min. Subsequently, the skin surface temperature was raised in a stepwise fashion (0.5°C steps) at a very slow rate (2.0–3.0°C/

min or four to six steps per minute, depending upon the subjective responses) until the sensation of pain was signaled by the subject. Typically, the subjects reported the presence of pain at a moderate level (category values of 3–5) within 15 min of placing the hand back onto the stimulator, and it was this level of pain that was maintained by adjusting the skin surface temperature. Thresholds and/or suprathreshold responses were obtained throughout the period (lasting about 20 min) during which the heat stimuli evoked a constant but moderate painful sensation. The particular types and number of tests performed were subject-dependent. After a sufficient number of vibratory tests had been performed, the right hand was removed from the stimulator, and each subject immediately reported the cessation of pain. The stimulator was then returned to the normative control temperature (31°C), this procedure taking approximately 10 min. After the stimulator had returned to the control temperature, the final vibratory tests on the right hand were completed, in the absence of any reported pain. Upon completion of these tests, additional vibratory tests were performed on the left hand. Postpain thresholds were only obtained for the experienced subjects.

RESULTS

A complete trial of vibrotactile threshold measurements before, during, and after painful heating of the right hand is shown in Figure 1 for one observer. Three estimates of vibrotactile thresholds were determined first for the left hand and then for the right hand, with the contactor kept at 30–31°C (baseline temperature). Both left-hand and right-hand vibrotactile thresholds were about −10 dB for 100-Hz stimuli, and about 17 dB for 10-Hz stimuli. Each subject was then instructed to remove the hand, and the contactor was heated to just below 40°C. The right hand was then replaced on the contactor (time = 33 min in Fig. 1), and vibrotactile thresholds were measured again. Within 2 min after placing the right hand back on the contactor, the observer rated the heat stimulus as painful. During this time and for the next 4 min, the vibrotactile thresholds for both 10-Hz and 100-Hz stimuli were continuously increasing. For the next 30 min the contactor temperature was kept relatively constant, although the overall pain ratings continued to increase. The vibrotactile threshold measures, tested at about 10-min intervals, remained elevated for the duration (up to 69 min in Fig. 1). The observer then removed the right hand from the contactor, and the contactor temperature was reduced to the baseline level. The right-hand and left-hand vibrotactile thresholds at 10 Hz and 100 Hz were measured again. As shown in Figure 1, right-hand vibrotactile thresholds at both frequencies, measured 10 min after the cessation of the painful heating, were still higher than those measured before the painful heating. However, the vibrotactile thresholds for the left hand were similar to those

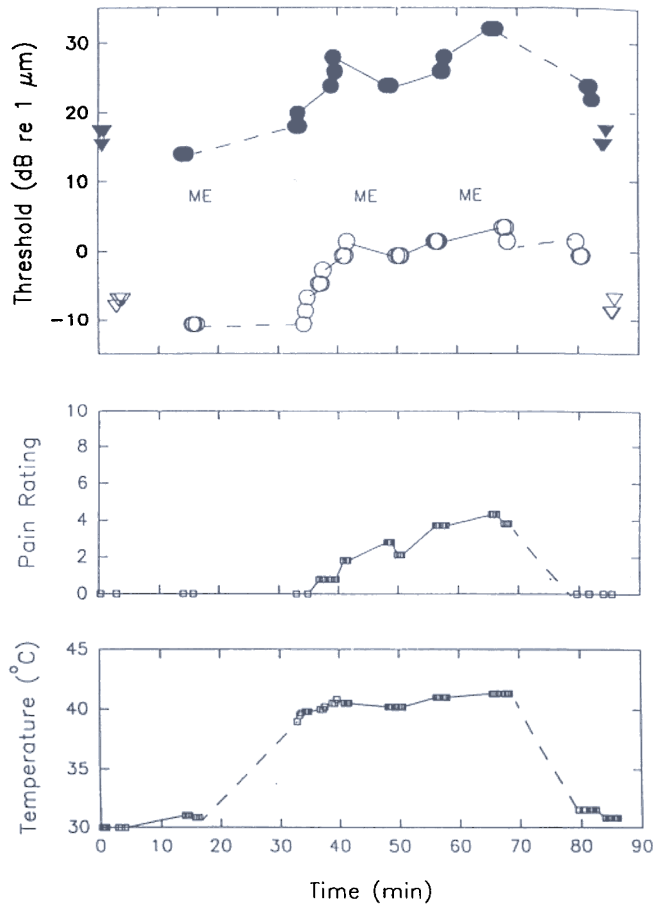

FIGURE 1. Threshold determinations for one observer during a single session. Skin temperature measured on the thenar eminence is shown in the bottom panel. The middle panel shows the observer's pain rating as skin temperature was increased. The top panel shows vibrotactile thresholds determined by the method of limits for the left hand (downward-pointing triangles) and right hand (circles), at 10 Hz (filled symbols) and at 100 Hz (open symbols). Dashed lines indicate times when the right hand was removed from the stimulator. The times when magnitude estimations were done are also indicated (ME).

measured before the application of painful heating for the right hand.

All subjects showed increased vibrotactile thresholds during coincident painful heat at 1 Hz, 10 Hz, and 100 Hz. There were no differences in results between threshold determination methods, between sexes, or between naive and experienced subjects (t tests, $p < 0.05$). Therefore, the data from all subjects were pooled. The group average changes, and the standard errors of the changes, in vibrotactile thresholds during painful heating relative to baseline thresholds are shown in Figure 2 for the right hand (first three bars). Figure 2 also shows the average changes in vibrotactile thresholds between baseline and recovery from the painful heat stimulation (average recovery of 10.4 ±

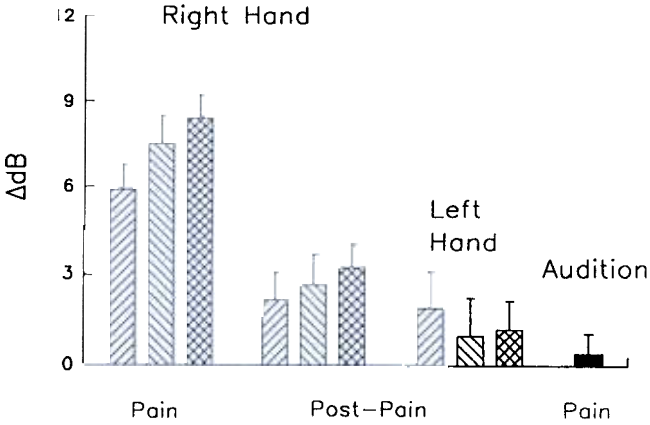

FIGURE 2. Average change in touch thresholds between baseline (no pain) and during thermal stimulation resulting in sustained pain for the right hand (first three bars); between baseline and recovery from pain for the right hand (second three bars); and between left-hand baseline and left-hand postpain thresholds (third three bars). Each condition was tested at three vibrotactile frequencies (1 Hz, left-slanted bars; 10 Hz, right-slanted bars; and 100 Hz, cross-hatched bars). The filled bar shows average auditory threshold change during right-hand pain. Vertical bars represent standard errors. Postpain thresholds for the right hand were elevated; postpain thresholds for the left (contralateral) hand were not changed. Auditory thresholds at 1 kHz were also not changed during painful thermal stimulation of the right hand.

2.25 min [mean ± *SD*]) for the right hand, for all three frequencies tested (postpain, second set of three bars). The right-hand threshold changes during painful heat and during postpain recovery were statistically compared to the right-hand thresholds measured at baseline temperature. Repeated-measures analysis of variance (RM ANOVA) at 100-Hz vibratory stimulation resulted in F (9, 16) = 40.8, $p <$ 0.001. At 100 Hz during painful heat, the threshold increase from baseline was 8.4 ± 0.8 dB (mean ± *SE*), which was statistically significant when tested with Bonferroni's multiple-comparison t test (t = 8.9, $p < 0.05$). At 100 Hz during postpain recovery, the threshold increase from baseline was 3.24 ± 0.8 dB (mean ± *SE*), which was statistically significant (Bonferroni's t test, t = 3.4, $p < 0.05$). With 10-Hz vibratory stimulation, RM ANOVA of baseline thresholds and thresholds during painful heat and after pain resulted in F (10, 18) = 26.4, $p < 0.001$. At 10 Hz during painful heat, the threshold increase from baseline was 7.5 ± 1.0 (*SE*) dB, which was statistically significant (Bonferroni's t test, t = 7.2, $p < 0.05$). At 10 Hz during postpain recovery, the threshold increase from baseline was 2.67 ± 1.0 (*SE*) dB, which was statistically significant (Bonferroni's t test, t = 2.5, $p < 0.05$). With 1-Hz vibratory stimulation, RM ANOVA of baseline thresholds and thresholds during painful heat and after pain resulted in F (10, 18) = 22.0, $p <$ 0.001. At 1 Hz during painful heat, the threshold significantly increased (Bonferroni's t test, t = 6.6, $p < 0.05$) from baseline to 5.9 ± 0.9 (*SE*) dB. At 1 Hz during postpain recovery, the threshold increase from baseline was 2.16 ± 0.9 (*SE*) dB, which was not statistically significant (Bonferroni's t test, t = 2.4, $p > 0.05$). Thus, during the period of strong thermal stimulation where pain was present, there was a mean change of 7.3 dB across all frequencies (over a twofold increase in threshold).

Changes in left-hand vibrotactile thresholds between baseline and recovery from painful heating of the right hand (after an average recovery of 13.8 ± 2.46 [*SD*] min) are also shown in Figure 2 for all three frequencies tested (third set of three bars—left hand postpain). Thresholds for the contralateral (left) hand after painful heating of the right hand were not consistently different from baseline (mean threshold increases ± *SE* were 1.17 ± 0.95, 0.97 ± 1.24, and 1.89 ± 1.21 dB; paired t tests, p = 0.18, 0.47, and 0.27, with n = 10, 10, 9 for 1, 10, and 100 Hz, respectively), measured immediately following the right-hand postpain measurements. Auditory thresholds, as shown in Figure 2 (last bar—audition during pain), at the 1-kHz tone were not changed during painful thermal stimulation of the right hand (mean threshold increase ± *SE* = 0.4 ± 0.64 dB; paired t test, p = 0.54, n = 8).

Within a single trial, the baseline thresholds measured for each subject and at each frequency were very consistent, always having standard deviations less than 2 dB and usually close to zero. In contrast, during intense thermal stimulation that elicited pain, the mean threshold change was 7.3 dB (i.e., 4–5 standard deviations above baseline thresholds). The threshold change was abrupt, which enabled us to establish a well-defined temperature at which the threshold to vibratory stimuli changed. The change in vibratory threshold during thermal stimuli occurred at a mean (± *SD*) temperature (41.4° ± 1.1°C) just below the mean (± *SD*) threshold for pain perception (42.0° ± 1.3°C). In 18 trials the thermal stimulus increased slowly, enabling us to separately detect the temperature at which there was a change in vibrotactile threshold and the temperature at which pain was first reported. In all 18 cases pain perception was reported at a temperature above that for a change in vibrotactile thresholds, with a mean (± *SD*) temperature difference of 1.35° ± 0.8°C (Wilcoxon signed-rank test, p = 0.000008). The trial shown in Figure 1 is an example of a case where the change in vibrotactile thresholds and the first report of pain could not be clearly dissociated from each other, although the initial change in vibrotactile thresholds still seemed to occur just before the first report of pain.

Pain ratings were significantly correlated (regression analysis; $p < 0.05$) with temperature, duration of the thermal stimulus, or both, suggesting that pain-mediating mechanisms and not pure temperature effects were involved in mediating the decreases in tactile sensitivity. Indeed, the threshold increase to vibratory stimuli was positively correlated with the pain ratings (r^2 = 0.52 ± 0.32 [*SD*]). Some

subjects had much tighter correlations between vibrotactile threshold changes and pain perception; in the best subject, r^2 was 0.82 ± 0.17 (*SD*) (n = 8 trials). Right-hand postpain vibrotactile threshold elevations at 10 Hz were positively correlated with the maximum pain rating (regression analysis, r^2 = 0.49, p = 0.0006), but not at 1 Hz or 100 Hz. The correlation between recovery interval (time from cessation of painful stimulus to the final right-hand threshold determination) and postpain threshold elevations had a negative slope at all frequencies. This was significant only for 100-Hz trials (regression analysis, slope = $-0.74, r^2 = 0.28, p = 0.03$). The duration of the thermal stimulus did not correlate with postpain right-hand threshold elevations.

Mean magnitude estimates for suprathreshold vibrotactile stimuli before and during painful heating are shown in Figure 3 for all three vibration frequencies. The three panels in the figure illustrate the relationship between the perceived magnitude of the vibrotactile stimuli and stimulus displacement for all three vibration frequencies tested. The results show decreased sensitivity of touch perception during intense thermal stimulation that induced pain at all but the highest levels of skin displacement. Comparing the magnitude estimates, at equivalent displacements, for each individual between baseline and periods of intense thermal stimulation that elicited pain showed that most magnitude estimates were lower during painful heating: At 100 Hz, 13 of 19 estimates were lower during these periods ($p < 0.02$, Wilcoxon signed-rank test); at 10 Hz, 16 of 19 estimates were lower ($p > 0.06$); and at 1 Hz, 19 of 20 estimates were lower ($p < 0.02$). In the 10 instances (of a total of 58) where the magnitude estimates were higher during painful heating, 7 were at the highest tactile intensities tested. Therefore, the magnitude estimates during intense thermal stimulation that produced pain indicate decreased sensitivity to suprathreshold vibrotactile stimuli and suggest psychophysical recruitment; this was first described in audition, where the difference in sensitivities diminish with increasing displacement intensities (Stevens, 1975). When the slopes of the regression lines of the sensation magnitude functions between before and during painful heating were compared, no statistical significance was observed (paired t test, $p > 0.05$). Figure 3 shows that linear regression does not properly fit the magnitude functions; however, it seems that we do not have sufficient data to establish statistically that recruitment was taking place.

DISCUSSION

The results indicate that our subjects demonstrated increased vibratory thresholds and decreased sensitivity to suprathreshold stimuli during intense thermal stimulation that produced sustained coincident pain. A number of factors indicate that this increase in threshold and decrease in sensitivity to touch cannot be attributed to pain-related criteria shifts or to generalized arousal or attentional shifts. Auditory thresholds may change with shifts in attentional states (Sinclair, 1967), but these did not change during the mild to moderate pain used here. Also, the vibrotactile threshold shift occurred just below the pain threshold in each trial.

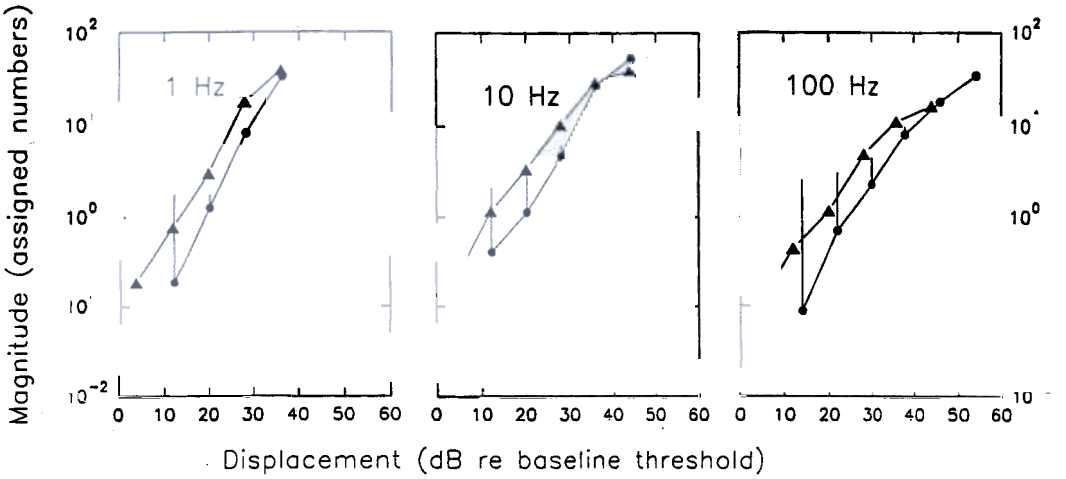

FIGURE 3. Average sensation magnitude functions obtained for vibrotactile stimuli before (baseline) and during intense thermal stimulation that produced coincident pain in the right hand in five subjects. Suprathreshold vibrotactile stimuli were presented relative to the average baseline threshold for all three frequencies (1, 10, and 100 Hz). The open circles represent the average vibrotactile thresholds determined during painful heating. The baseline thresholds for the observers relative to peak displacement (mean ± *SD*) were 33 ± 3.2, 27 ± 2.7, and −2.5 ± 3.8 dB re: 1 μm, at 1, 10, and 100 Hz, respectively. Filled triangles represent the average baseline magnitude estimates; filled circles indicate the average magnitude estimates during coincident pain. The standard errors are shown as upward vertical bars for the painful heating sessions only. The standard errors for the baseline curves were similar.

are unlikely. We do not mean to imply that attentional shifts during pain have no effect on touch perception, particularly in the case of unexpected, intense painful stimuli, but rather that these made at most a minimal contribution in this study.

The pain thresholds determined in our subjects (42.0° ± 1.3°C) are lower than those usually reported for the volar hand in previous psychophysical and physiological studies, these values being about 45°C (see Hardy et al., 1967; Price, 1988). This apparent discrepancy in pain thresholds is primarily attributable to the large size of the contact thermal stimulator used in our study (35.9 cm^2). Increasing the size of a contact thermode decreases pain thresholds (Kojo and Pertovaara, 1987) and increases pain perception (Price et al., 1989). Kojo and Pertovaara (1987) showed that increasing the size of a Peltier thermode from 1.3 cm^2 to 11.8 cm^2 decreased pain thresholds from over 46°C to 43°C for the volar forearm. The large size of our stimulator was dictated by the need for coincident presentation of thermal and vibratory stimuli, and could not be varied. However, in two of our subjects, thresholds were also determined by a 1.5-cm^2 contact thermode and were about 46°C. Obviously, the slow rise rate and long duration of the thermal stimulus also contributed to the pain thresholds and pain perceptions determined.

The three tactile channels (P, NP I, and NP III) that were evaluated in this study are sensitive to skin temperature changes. For example, warming the P channel in the innocuous range (32–40°C) *decreases* threshold and shifts the best response frequency (Green, 1977; Bolanowski and Verrillo, 1982; Verrillo and Bolanowski, 1986). The threshold decreases that occur with skin warming have been attributed to local physical changes in the skin and/or the receptor. In the present study, we have shown that when skin heating reaches a level where pain is reported and nociceptors can presumably be activated, threshold *increases* and sensitivity *decreases*. Although earlier psychophysical studies of effects of skin warming on touch showed this trend with temperatures above 40°C (Weitz, 1941; Green, 1977; Stevens, 1979; Bolanowski and Verrillo, 1982; Verrillo and Bolanowski, 1986), the effect was unrecognized or not commented upon in relation to possible interactions between touch and pain sensory modalities. However, Nathan (1960) studied the alterations in perceptions of touch and pin pricks during clinical pain and reported that pain relief resulted in "sharpening" tactile perception, which may be a chronic manifestation of the phenomenon we describe here. It should be stressed that the present study used intense thermal stimuli to induce pain. On the basis of the previous studies, we can conclude that merely increasing thermal energy will not, at the vibrotactile stimulus frequencies used, increase vibrotactile thresholds. Thus it is likely that the activation

crucial for the effects observed. However, given perimental paradigm used, we cannot dissociate b intense heating and pain for the diminution in perception.

This study has not directly addressed the natu site of suppression of touch by the heat-induced pain ever, the presented data strongly suggest that the site be located centrally, not peripherally. The changes in perception during the periods of heat-induced pain w the opposite direction to those observed when the ski face was heated to innocuous temperatures (see al Also, since the vibrotactile thresholds increased at a te ature unique for each subject, at a seemingly fixed pro to each subject's pain threshold, it is likely that the was not a result of physical changes in the skin or th organ, but a result of the activation of nociceptors positive correlations between vibrotactile threshol creases and pain ratings during pain, and the negative c lations during recovery from pain, are also consistent the notion that the effect is mediated through nocicep Because noxious and innocuous primary afferents ar known to interact peripherally, the effect must be n ated centrally.

Our results, therefore, can be interpreted as indic the existence of a neural mechanism or "touch gate" can actively inhibit touch perception during pain, as m fested by increased tactile thresholds and decreased su threshold sensitivity. The most parsimonious interpreta of these results is that activation of peripheral nocicept even at magnitudes just below pain perception (but wl nociceptors are already active), decreases the encoding vibrotaction in the vicinity of the noxious stimulus. T implies that touch sensation confounds pain, and that clos the touch gate enhances the perceptual discrimination pain relative to touch. This suppression of touch by p runs contrary to the old notion, inspired by Head and Holr (1911) and restated in recent textbooks (Carpenter and Sut 1983), that discrimination of painful stimuli (intensity a location) is achieved by the medial lemniscal system rat than pain pathways.

Independent evidence exists for this effect of heat- duced pain on touch that also suggests the central site suppression. Cerebral blood flow studies of sustained pa perception in humans, employing single-photon emissi computed tomography (SPECT), have indicated that neu activity in the contralateral somatosensory region is dimi ished (Apkarian et al., 1992). These results were in fact tl original impetus for the present investigation, and they ha recently been corroborated by an experimental pain activa tion study using a positron emission tomography (PE slow-bolus blood flow technique (with a single-subject com parison between six controls and pain states; A. K. P. Jones personal communication). Assuming that inhibition of th

somatosensory cortex implies disruption of touch perception, the results of the present psychophysical study may be regarded as confirming the predictions of the SPECT study. Similar to our SPECT study, blood flow studies of patients with chronic pain states show decreased activity in the somatosensory thalamus and cortex (DiPiero et al., 1991; Canavero et al., 1993). On the other hand, activations of multiple cortical areas, including the somatosensory cortex, have been observed in PET studies when the experimental pain was phasic (Jones et al., 1991; Talbot et al., 1991; Casey et al., 1992). Electroencephalographic studies also indicate decreased cerebral activity during sustained pain (Backonja et al., 1991). Furthermore, blocking the activity of C fibers (fibers that transmit noxious and thermal information; see Fields, 1987) results in the immediate expansion of the receptive fields of somatosensory cortical cells, implying again that nociceptive input inhibits cortical neurons (Calford and Tweedale, 1991). Nathan's (1960) study is important as well, since he showed that in patients with chronic pain, relief by a variety of independent interventions (nerve block, nerve section, intrathecal anesthesia) resulted in the sharpening of touch perception for the duration of pain relief. These studies, therefore, indicate that multiple central regions thought to be important in encoding touch become inhibited during activation of nociceptors or during painful states.

The inhibition of touch by pain observed at the cortical level may be a reflection of a touch gate operating at a lower level. The gate of Melzack and Wall (1965) resides in the spinal cord; consistent with this idea is the extensive evidence showing that most descending modulatory inputs to the spinal cord dorsal horn preferentially inhibit nociceptive responses (see Gebhart, 1986). However, it is unlikely that the touch gate is located at the spinal segmental level, since touch is conveyed primarily through the dorsal column nuclei, which receive direct peripheral input from dorsal column tracts that bypass spinal cord processing. On the other hand, the second-order dorsal and dorsolateral postsynaptic pathways may be the source for the segmental nociceptive input controlling the hypothesized touch gate, which is perhaps located in the dorsal column nuclei (see Willis and Coggeshall, 1991). Another possibility is that the putative touch gate resides in the lateral thalamus, where there is considerable convergence between innocuous and nociceptive inputs (Gingold et al., 1991).

The suppressive effect of touch (large-fiber stimulation) on experimental pain has recently been demonstrated during skin stimulation (Zoppi et al., 1991) and during dorsal column stimulation (Marchand et al., 1991). The opposite effect of heat-induced pain on touch perception has not been recognized until now. While touch can suppress pain, pain, too, can diminish touch. Whether this latter effect results in significant deficits in everyday performance remains to be examined.

This study was partially funded by NIH Grant Nos. NIDCD DC00098 and DC00380 to Stanley J. Bolanowski and by the Department of Neurosurgery and the Perkins Foundation to A. Vania Apkarian.

REFERENCES

Apkarian, A. V., R. A. Stea, S. H. Manglos, N. M. Szeverenyi, R. B. King, and F. D. Thomas (1992) Persistent pain inhibits contralateral somatosensory cortical activity in humans. Neurosci. Lett. *140*: 141–147.

Backonja, M., E. W. Howland, J. Wang, J. Smith, M. Salinsky, and C. S. Cleeland (1991) Tonic changes in alpha power during immersion of the hand in cold water. Electroenceph. Clin. Neurophysiol. *79*: 192–203.

Bolanowski, S. J., Jr., and G. A. Gescheider (1989) *Ratio Scaling of Psychological Magnitudes*, Erlbaum, Hillsdale, NJ.

Bolanowski, S. J., Jr, G. A. Gescheider, R. T. Verrillo, and C. M. Chekosy (1988) Four channels mediate the mechanical aspects of touch. J. Acoust. Soc. Am. *84*: 1680–1694.

Bolanowski, S. J., Jr., and Verrillo, R. T. (1982) Temperature and criterion effects in a somatosensory subsystem: A neurophysiological and psychophysical study. J. Neurophysiol. *48*: 836–855.

Calford, M. B., and R. Tweedale (1991) C-fibres provide a source of masking inhibition to primary somatosensory cortex. Proc. Roy. Soc. Lond. *243*: 269–275.

Canavero, S., C. A. Pagni, G. Castellano, V. Bonicalzi, B. Marilena, S. Duca, and V. Podio (1993) The role of cortex in central pain syndromes: Preliminary results in long-term Technitium-99 hexamethylpropyleneamineoxime single photon emission computed tomography study. Neurosurgery *32*: 185–191.

Carpenter, M. B., and J. Sutin (1983) *Human Neuroanatomy*, Williams & Wilkins, Baltimore.

Casey, K. L., S. Minoshima, K. L. Berger, R. A. Koeppe, T. J. Morrow and K. A. Frey (1992) PET analysis of brain structures differentially activated by noxious thermal stimuli. Soc. Neurosci. Abstr. *18*: 883.

DiPiero, V., A. K. P. Jones, F. Iannotti, M. Powell, D. Perani, G. L. Lenzi, and R. S. J. Fracowiak (1991) Chronic pain: A PET study of the central effects of percutaneous high cervical cordotomy. Pain *46*: 9–12.

Ellenmeier, W., and W. Westphal (1991) On the 5absoluteness" of category and magnitude scales of pain. Percept. Psychophys. *49*: 159–166.

Fields, H. L. (1987) *Pain*, McGraw-Hill, New York.

Gebhart, G. F. (1986) Modulatory effects of descending systems on spinal dorsal horn neurons. In *Spinal Afferent Processing*, T. L. Yaksh, ed., pp. 391–416, Plenum Press, New York.

Gescheider, G. A. (1985) *Psychophysics: Method, Theory and Application*, Erlbaum, Hillsdale, NJ.

Gingold, S. I., J. D. Greenspan, and A. V. Apkarian (1991) Anatomic evidence of nociceptive inputs to primary somatosensory cortex: Relationship between spinothalamic terminals and thalamocortical cells in squirrel monkeys. J. Comp. Neurol. *308*: 467–490.

Green, B. G. (1977) The effect of skin temperature on vibrotactile sensitivity. Percept. Psychophys. *21*: 243–248.

Hardy, J. D., H. G. Wolff, and H. Goodell (1967) *Pain Sensations and Reactions*, Hafner, New York.

Head, H., and G. Holmes (1911) Sensory disturbances from cerebral lesions. Brain *34*: 102–254.

Jones, A. K. P., W. D. Brown, K. J. Friston, L. Y. Qi, and R. S. J. Frackowiak (1991) Cortical and subcortical localization of responses to pain in man using positron emission tomography. Proc. Roy. Soc. Lond. *244*: 39–44.

Kojo, I., and A. Pertovaara (1987) The effects of stimulus area and adaptation temperature on warm and heat pain thresholds in man. Int. J. Neurosci. *32*: 875–880.

MARCHAND, S., M. C. BUSHNELL, P. MOLINA-NEGRO, S. N. MARTINEZ, and G. H. DUNCAN (1991) The effects of dorsal column stimulation on measures of clinical and experimental pain in man. Pain *45*: 249–257.

MELZACK, R., and P. D. WALL (1965) Pain mechanisms: A new theory. Science *150*: 971–978.

NATHAN, P. W. (1960) Improvement in cutaneous sensibility associated with relief of pain. J. Neurol. Neurosurg. Psychiat. *23*: 202–206.

PRICE, D. D. (1988) *Psychophysical and Neural Mechanisms of Pain*. Raven Press, New York.

PRICE, D. D., P. A. MCGRATH, A. RAFII, and B. BUCKINGHAM (1983) The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. Pain *17*: 45–56.

PRICE, D. D., J. G. MCHAFFIE, and M. A. LARSON (1989) Spatial sumation of heat-induced pain: Influence of stimulus area and spatial separation of stimuli on perceived pain sensation intensity and unpleasantness. J. Neurophysiol. *62*: 1270–1279.

SINCLAIR, D. (1967) *Cutaneous Sensation*, Oxford University Press, London.

STEVENS, J. C. (1979) Thermo-tactile interactions: Some influences of temperature on touch. In *Sensory Functions of the Skin of Humans*, D. R. Kenshalo, ed., pp. 207–218, Plenum Press, New York.

STEVENS, S. S. (1975) *Psychophysics*, Wiley, New York.

TALBOT, J. D., S. MARRETT, A. C. EVANS, E. MEYER, M. C. BUSHNELL, and G. H. DUNCAN (1991) Multiple representations of pain in human cerebral cortex. Science *251*: 1355–1358.

VERRILLO, R. T., and S. J. BOLANOWSKI, JR. (1986) The effects of skin temperature on the psychophysical responses to vibration on glabrous and hairy skin. J. Acoust. Soc. Am. *80*: 528–532.

VERRILLO, R. T., A. J. FRAIOLI, and R. L. SMITH (1969) Sensation magnitude of vibrotactile stimuli. Percept. Psychophys. *6*: 366–372.

WEITZ, J., (1941) Vibratory sensitivity as a function of skin temperature. J. Exp. Psychol. *28*: 21–36.

WILLIS, W. D., JR., and R. E. COGGESHALL (1991) Sensory pathways in the dorsal funiculus. In *Sensory Mechanisms of the Spinal Cord*, Vol. 2, pp. 245–306, Plenum Press, New York.

ZOPPI, M., M. R. VOEGELIN, M. SIGNORINI, and A. ZAMPONI (1991) Pain threshold changes by skin vibratory stimulation in healthy subjects. Acta Physiol. Scand. *143*: 439–443.

ZWISLOCKI, J. J. (1983) Group and individual relations between sensation magnitudes and their numerical estimates. Percept. Psychophys. *33*: 460–468.

ZWISLOCKI, J. J., F. MAIRE, A. S. FELDMAN, and H. J. RUBIN (1958) On the effect of practice and motivation on the threshold of audibility. J. Acoust. Soc. Am. *30*: 254–262.