EFFECTS OF CHRONIC BACK PAIN ON THE PERCEPTION OF EXPERIMENTAL HEAT PAIN [1, 2]

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Summary.—19 patients between 23 and 65 yr. of age whose chronic back pain was caused by lumbosacral disk disease and 19 healthy volunteers matched for age and sex were studied. Pain thresholds under phasic and tonic heat stimulation and thermal thresholds for warmth and cold were measured on the right hand. The patients rated any current back pain on a visual analog scale. There was a significant negative correlation between current back pain and the threshold for tonic pain, but there was no correlation between current back pain and either the threshold for phasic pain or temperature sensitivity. Hence, current back pain and experimental tonic pain seem to have an additive effect on pain perception because perceptual qualities are similar. Reduced somatosensory perception of chronic back pain patients could be demonstrated for temperature sensitivity and to a lesser degree for phasic pain, but as a consequence of the "opposing" effect of current back pain, not for tonic pain perception.

Changes in the perception of pain in patients with chronic pain and the clinical consequences of these changes have been studied increasingly in recent years. The experimental results have been conflicting, with both increased and decreased pain thresholds having been reported (8, 16, 18, 19, 30). This is, at least in part, because the term chronic pain is used in connection with a great number of different organic and psychophysiological dysfunctions. Consequently, it is unreasonable to expect chronic pain to have a unidirectional effect for all pain patients, and it would therefore appear useful first to evaluate separately the perceptual changes produced by different forms of chronic pain.

From a theoretical point of view, however, the question remains of how an history of chronic pain leads to changes in pain perception. There are already some hypotheses about this process. For example, the decrease in pain sensitivity has been explained by the adaptation level theory (13), whereby current pain is experienced and judged within a subjective framework resulting from an history of severe chronic pain. In contrast, Chapman postulates that patients with chronic pain develop a hypervigilance to aver-

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sive stimuli, which leads to an increase in pain sensitivity (3). Individual differences in the effects of chronic pain have been explained in terms of the concepts of perceptual augmenting and reducing, with an intimate relationship between these concepts and opioid activity being assumed (7, 14).

One shortcoming of all these theoretical arguments is that chronic pain is considered to produce a change in perceptual habits only. However, we must take into account the fact that chronic pain usually is manifest as recurrent pain attacks rather than as a continuous state of pain. For example, Price and coworkers (24) found marked differences between minimum and maximum levels of pain within one week in patients with chronic pain of various origins. In the case of chronic back pain the causes of these variations in current pain intensity are not clear in most of the patients. There may be contributions from nociceptive and neurogenic pain mechanisms as well as situational influences that are the result of learned pain behavior (17, 25). Nevertheless, pain perception is likely to be affected by these variations. We therefore postulated that chronic pain influences pain perception in two ways: as a stable influence, by producing a change in perceptual habits, and as a variable influence, by increasing the likelihood of having current pain.

Unfortunately, there is no solid empirical basis for predictions about the effect of current clinical pain on the perception of experimental pain. Some examples follow of the perceptual effects that have been reported for two types of pain occurring concurrently. In two studies acute intraoral pain had almost no effect on the perception of experimental pain (9, 12). In experiments on "diffuse noxious inhibitory control" with the concurrent use of two pain stimuli, long-lasting pain stimuli reduced the perception of relatively short pain stimuli via a nonsegmental mechanism (21, 28, 29). In contrast, Algom and coworkers postulated that, when two types of pain occur concurrently, the effect on pain perception is additive, and they gave some supporting results from studies with short aversive stimuli (1, 2). These examples indicate that, when two types of pain occur concurrently, pain perception depends largely on the kinds of pain involved. Nevertheless, the study of the perceptual effects of chronic pain should also address the role of the intensity of any current pain instead of disregarding it.

In the present investigation, we studied (a) the perception of experimental pain in a group of patients with chronic back pain of a single origin, lumbosacral disk disease, and (b) the effect of current back pain on pain perception by these patients. With this approach we could avoid misleading results from differing forms of chronic pain and we could investigate the two types of effect of chronic pain postulated, a change in perceptual habits and an increased likelihood of current pain. Past findings for patients with chronic back pain (8, 16, 20, 30) led us to expect that our subjects without current back pain would show an increased pain threshold. As mentioned

earlier, we had no solid basis for predicting the effect of current back pain on pain perception. The same was true for possible interactions between the two types of effect of chronic pain on pain perception.

We studied pain perception with two different types of experimental pain: phasic pain produced by heat stimuli of short duration and tonic pain produced by heat stimuli of much longer duration. Because the affective-motivational component of pain perception is greater for tonic than for phasic stimuli (4, 5), we anticipated that perception of the two forms of experimental pain would be influenced differently by clinical pain. To ensure that the observed effects would in fact be pain specific and not due to general alterations of somatosensory perception, we also measured sensitivity to temperature with warm and cold stimuli.

Method

Subjects

The subjects were 19 patients with chronic back pain, whose ages were between 23 and 65 yr. ($M = 43.2$, $SD = 10.4$), and 19 healthy volunteers between 22 and 63 yr. of age ($M = 44.3$, $SD = 10.7$) matched for age and sex (9 women and 10 men each). The experiment was described to them, and they were told that they could stop at any time. All subjects signed informed consent forms. The control subjects were paid for participating.

To be included in the study the patients had to have a definite diagnosis of disk disease in the segment L4-L5, L5-S1, or both and to have suffered from back pain for at least one year. The patients had been having back pain for an average of 10.3 yr. ($SD = 7.4$), and 15 of them had had surgical treatment for their disk disorder at least once. Just prior to the experiment we assessed the current neurological status including lower tendon reflexes, the motor and sensory status of the legs, and lumbosacral motion to reconfirm the diagnosis. We conducted the same tests and an anamnestic interview with the control subjects to exclude any disorders that lead to somatosensory impairment.

A detailed description of the pain status was available for 18 patients: 2 patients described their back pain as persistent, 8 as irregularly intermittent, 3 as regularly intermittent, and 3 as associated with particular activities; 2 could give no specifications. For 13 patients the most recent pain attack had occurred on the day of assessment, for 3 a week earlier and for 2 a month earlier. The duration of the last pain attack ranged from one hour to more than one month.

All patients were inpatients at a rehabilitation center. They were studied as soon as possible after admission. The reason for admission was rehabilitation after either recent back surgery or ineffective conservative outpatient treatment. Patients with acute postsurgical complications were not admitted.

The minimum interval between surgery and our study was one month (5 patients), with a mean of 12.9 mo. ($SD = 23.5$). As treatment outcome with respect to pain symptoms does not appear to differ systematically between surgery and conservative therapy (10), we combined patients with both antecedent treatments if the other criteria were met.

The experiment described in the following was the last part of a series of examinations requiring the subjects to be seated. Two patients developed such severe back pain that they did not complete the session, stopping before the tonic pain threshold had been determined.

Apparatus and Procedure

The stimulator was a temperature-controlled contact thermode that can be both heated and cooled actively and has a stimulation surface of 2×3 cm^2 [modified Marstock thermode, for technical details see (11)]. The apparatus also included a microcomputer to control the procedure and the thermal stimuli, a response panel, and visual and acoustic signalling devices. During all of the procedures the subjects had continuous control over stimulus intensity via the respective response button.

The subjects sat upright at a table on which had been placed the thermode, the signalling devices, and the response panel. For constant pressure against the skin the thermode was mounted on a spring in a half sphere made of PVC. Each subject placed the right hand on the half-sphere with the thenar eminence on the thermode.

Determination of the phasic pain threshold.—Beginning at a base temperature of 40°C, a temperature rise at a rate of 0.8°C/sec. was started 1, 2, or 3 sec. (pseudorandomized intervals) after the visual and acoustical warning stimuli. The subjects were instructed to press a button as soon as they felt pain for the first time. When they pressed the button, the temperature rise stopped immediately and the temperature returned to the base value. This procedure produced a painful stimulation of 1 sec. at most, resulting in only a weak "burning" pain component. There were eight trials. The phasic pain threshold was calculated as the mean of the peak temperatures for the last five trials.

Determination of the tonic pain threshold.—Here the stimulus-adjustment procedure used was derived from the "subjective sensitization" technique (15, 27). From a base temperature of 40°C the subjects adjusted the temperature of the pain threshold using a heating and a cooling button. They indicated their final adjustment by pushing a third button. They were then stimulated at this temperature for another 35 sec. After this interval the subjects had to readjust the temperature to the initial level of sensation. [Depending on central temporal summation and peripheral sensitization or fatigue of nociceptive fibers, changes in pain perception strength may take place in an interval of this length, resulting in either an increase (sensitiza-

tion) or a decrease (adaptation). The "pricking" pain component vanishes soon after the onset of stimulation—this usually happens prior to the first adjustment—and a true "burning" pain component remains until stimulation stops (6, 23, 27). The threshold of the "burning" pain component was therefore measured in two runs: before and after a period of prolonged stimulation.] The mean of the two temperatures adjusted was used as the threshold measure for a given trial. There were six trials. The mean of the last five trials was used as the measure of the tonic pain threshold. The beginning and end of the two stimulus adjustment periods and the interval of constant stimulation were signalled visually and acoustically.

Determination of the warm and cold thresholds.—Starting at a temperature of 32°C, a temperature rise (warm threshold) or drop (cold threshold) at a rate of 0.8°C/sec. was started 1, 2, or 3 sec. (pseudorandomized intervals) after the visual and acoustical warning stimuli. The subjects had to push a button as soon as they noticed a change in temperature. Thereupon the temperature returned to the base value. The warm and cold thresholds were assessed in two sets of seven trials beginning with the warm threshold. The mean differences between the base temperature and the peak temperature in the two sets were the measures of the warm and cold thresholds.

The thresholds for warm, cold, phasic pain, and tonic pain were determined in that order.

Assessment of current back pain.—Immediately before and after the threshold measurements the patients assessed the intensity of current back pain on a vertical visual analog scale (VAS) 10 cm in length. The lower end point was labelled "no pain" and the upper end point "unbearable pain." The mean of the two ratings served as the measure of current clinical pain. One patient did not make a rating after the experiment; in this case the initial rating was used in the subsequent analysis.

Results

There were no significant differences between the chronic pain patients and the control subjects with regard to the phasic and tonic pain thresholds (phasic: $p = .14$, tonic: $p = .35$; Mann-Whitney U, one-sided). The phasic pain thresholds were slightly elevated in the chronic pain group (Fig. 1a), but the tonic pain thresholds showed no difference at all (Fig. 1b). The chronic pain patients did have significantly higher warm and cold thresholds, however (both warm and cold: $p < .001$; Mann-Whitney U, one-sided; Fig. 2).

To assess whether the relationships between the various thresholds were similar in the chronic pain patients and the healthy volunteers, we calculated Spearman rank-order correlation coefficient (p values for the one-sided test; Table 1). The correlations between the threshold measures were consistently lower in the pain group. Warm and cold thresholds correlated significantly in both groups, but the thresholds for phasic and tonic pain correlated signifi-

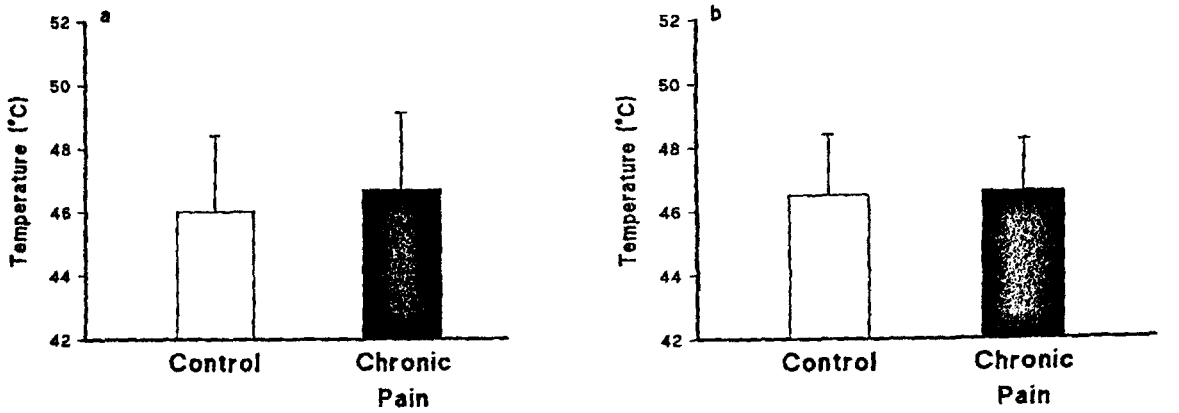

FIG. 1. Pain thresholds: means and standard deviations for phasic (a) and tonic (b) pain thresholds in healthy volunteers ($n = 19$) and chronic pain patients (phasic stimulation: $n = 19$, and tonic stimulation: $n = 17$)

cantly in the control group only. In the pain group the correlation was close to zero, which suggests that here the pain measures for the two types of pain do not measure the same thing.

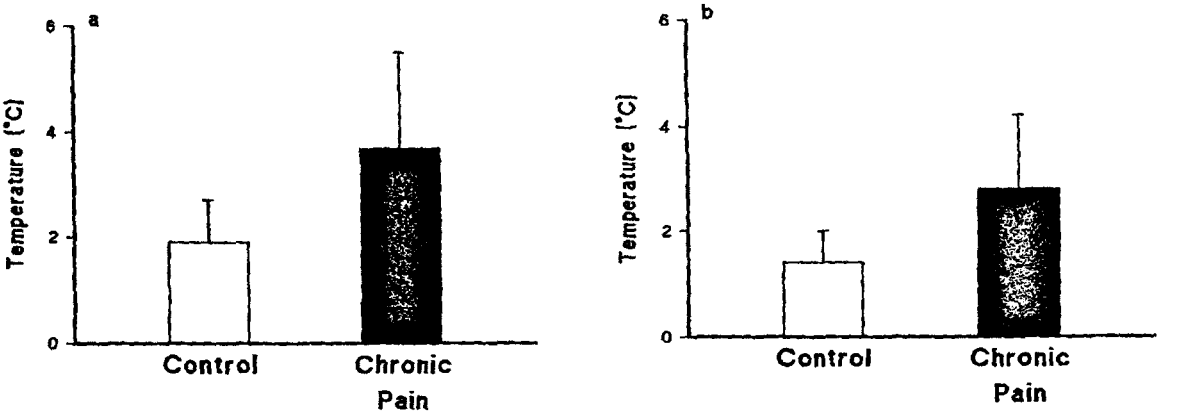

FIG. 2. Thermal thresholds: means and standard deviations for the warm (a) and cold (b) thresholds in healthy volunteers ($n = 19$) and chronic pain patients ($n = 19$)

The correlations between current back pain (visual analog rating) and the four threshold measures were as follows: rating and phasic pain: $r = -0.19$, $p = .22$; rating and tonic pain: $r = -0.51$, $p = .02$; rating and warmth: $r = 0.25$, $p = .15$; rating and cold: $r = 0.31$, $p = .10$. Hence the only significant correlation was between the rating on the visual analog scale and the tonic pain threshold; this was a negative correlation. The relationships between current back pain and the phasic and tonic pain thresholds are shown in Figs. 3a and b, respectively.

The distribution of the rating scores (see Figs. 3a and b) made it seem reasonable to divide the patients into two distinct groups, one with little or no back pain (low scores: rating under 35 mm; $n = 8$) and one with severe

TABLE 1

SPEARMAN CORRELATIONS BETWEEN THRESHOLDS FOR PHASIC PAIN, TONIC PAIN, WARMTH, AND COLD IN CONTROL GROUP ($n = 19$) AND CHRONIC PAIN GROUP ($n = 19$ EXCEPT FOR TONIC PAIN THRESHOLDS, WHERE $n = 17$)

Measure	Control Group			Chronic Pain Group		
	Tonic	Warmth	Cold	Tonic	Warmth	Cold
Phasic Pain	0.53*	0.37	0.39	0.09	0.00	0.25
Tonic Pain		0.34	0.44*		-0.24	0.00
Warmth			0.84‡			0.64†

*$p < .05$. †$p < .01$. ‡$p < .001$.

back pain (high scores: rating above 60 mm; $n = 11$). The correlations between the rating and the four threshold measures were then calculated for these two subgroups separately (see Table 2). Again, there are substantial cor-

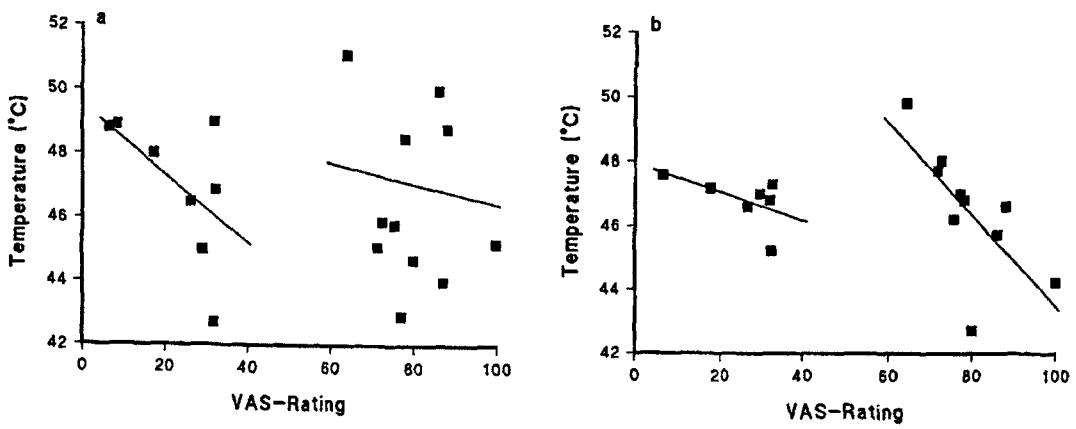

FIG. 3. Scatter diagram for the relationship between VAS rating of current back pain and phasic (a) and tonic (b) pain thresholds. The linear regression of "pain threshold" on "back pain" was calculated separately for patients with low VAS ratings (<35 mm, $n = 8$) and those with high VAS ratings (>60 mm, $n = 11$); for phasic stimulation $n = 19$ and for tonic stimulation $n = 17$

relations between current back pain and the tonic pain threshold only. However, the negative correlation is significant only in the subgroup with severe back pain. From Fig. 3b it is clear that in this subgroup, with one exception, there was a strong linear relationship with a negative slope.

TABLE 2

SPEARMAN CORRELATIONS BETWEEN RATINGS OF CURRENT BACK PAIN ON VISUAL ANALOG SCALE AND THRESHOLDS FOR PHASIC PAIN, TONIC PAIN, WARMTH AND COLD IN CHRONIC PAIN GROUP ($n = 19$ EXCEPT FOR TONIC PAIN THRESHOLDS, WHERE $n = 17$) WITH SUBJECTS SUBDIVIDED INTO LITTLE AND SEVERE CURRENT PAIN GROUPS

	Phasic	Tonic	Warmth	Cold
Little Pain ($n = 8$)	-0.22	-0.45	-0.06	-0.11
Severe Pain ($n = 11$)	-0.13	-0.87*	0.17	0.04

*$p < .001$.

Discussion

In the present study we postulated two types of effect of chronic back pain, a change in perceptual habits and an increase in the likelihood of current back pain. When we considered the two types of effect simultaneously, which we thought necessary (see Introduction), the perception of experimental pain appeared to be influenced in a complex manner. An unexpected finding—if the effect of chronic pain is seen solely as a change in perceptual habits—was that under tonic stimulation, there was no difference at all between the patient and control groups. This finding can be explained, however, if the influence of current back pain is considered. Above a certain minimum level of severity current back pain seemed to increase pain sensitivity and therefore counteracted any threshold elevation based on a change in perceptual habits. As a result, the effect of chronic back pain observed in other studies (8, 16, 20, 30), namely, an increase in the pain threshold, may have been masked. But the current back pain influenced pain perception under tonic stimulation only, not under phasic stimulation. Consequently, the phasic pain thresholds in the patient group showed a slight elevation. Hence, as we expected, these findings indicate that in studies of pain perception in chronic pain patients the current pain state can be of importance. This has been corroborated recently in a study by Peters and coworkers (22), in which, in addition to the usual criterion of chronicity (duration of back pain more than six months), daily pain attacks were required. As in our study, no differences in pain threshold between the patients and the control subjects were found. We therefore conclude that the current pain state is as important as the chronicity of pain in investigations of pain perception and that the two influences may offset each other.

One explanation for the increase in pain sensitivity produced by the current back pain under tonic stimulation is that suggested by Algom and coworkers (1, 2). According to their theory, after separate primary processing of the stimuli pain perceptions from different somatosensory channels may be integrated additively, which would then determine the over-all level of pain. One prerequisite for such an integrative process is a certain degree of similarity among the perceptions. This prerequisite appears to be met in the case of experimental tonic pain and clinical pain. Perception of both of these types of pain, in contrast to perception of pain under phasic stimulation, has both a sensory-discriminative and an affective-motivational component (4, 5). Our tonic procedure, which produces a prolonged "burning" pain and thereby activates the affective-motivational pain component (6), seems also to have met this criterion. This could mean that current back pain and tonic pain contribute additively to the threshold level, and hence if back pain is severe the pain threshold is reached even with mild tonic pain stimuli. This could explain the strong negative correlation between the severity of back

pain (above a certain intensity) and the pain threshold under tonic pain stimuli. Inasmuch as such an additive effect did not exist for the pain threshold under phasic stimulation, it is understandable that the correlation between the two types of pain thresholds was close to zero for the pain patients. Similar predictions can be derived from the generalization hypothesis, which claims that for chronic pain patients, the greater the similarity between the experimental and clinical pain, the greater the similarity between the patients' responses to the experimental and clinical pain (25).

The finding that current back pain had no effect on thermal sensitivity is compatible with findings for tooth pain, where thermal sensitivity was also unaffected by the painful condition (9, 12). This makes it unlikely that the effects of current back pain on the tonic pain threshold seen in our study were unspecific changes in perceptual skills, such as deterioration of attention. Thermal sensitivity was reduced in the chronic pain group, however. Similar changes in thermal sensitivity and in somatosensory perception in general in back pain patients have been reported by other authors (20, 26, 30).

Based on the findings in the present study we put forward the following hypothesis about somatosensory perception in patients with chronic back pain: chronic back pain tends to change perceptual habits, resulting in a reduction in somatosensory perception. In addition, chronic back pain predisposes patients to have episodes of current back pain, which, above a certain minimum level of severity, affect pain perception, but not somatosensory perception in general. The effect is one of a subjective sensitization. Whether or not this happens, however, also depends on the similarity of the clinical and experimental pain. One prerequisite for similarity is that the experimental pain have an affective-motivational component, as clinical pain usually does. It remains to be seen whether these arguments will have to be restricted to back pain of organic origin or can also be applied to other forms of clinical pain.

REFERENCES

1. ALGOM, D., RAPHAELI, N., & COHEN-RAZ, L. Integration of noxious stimulation across separate somatosensory communications systems: a functional theory of pain. *J. exp. Psychol.: Hum. Percept.*, 1986, 12, 92-102.
2. ALGOM, D., RAPHAELI, N., & COHEN-RAZ, L. Pain combines additively across different sensory systems: a further support for the functional theory of pain. *Percept. mot. Skills*, 1987, 65, 619-625.
3. CHAPMAN, C. R. Pain: the perception of noxious events. In R. A. Sternbach (Ed.), *The psychology of pain*. New York: Raven, 1978. Pp. 169-202.
4. CHEN, A. C. N., DWORKIN, S. F., HAUG, J., & GEHRIG, J. Human pain responsivity in a tonic pain model: psychological determinants. *Pain*, 1989, 37, 143-160.
5. CHEN, A. C. N., & TREEDE, R-T. The McGill Pain Questionnaire in the assessment of phasic and tonic experimental pain: behavioral evaluation of the "pain inhibiting pain" effect. *Pain*, 1985, 22, 67-79.
6. CHÉRY-CROZE, S. Painful sensation induced by a thermal cutaneous stimulus. *Pain*, 1983, 17, 109-137.

7. COGER, R. W., KENTON, B., PINSKY, J. J., CRUE, B. L., CARMON, A., & FRIEDMAN, Y. Somatosensory evoked potentials and noxious stimulation in patients with intractable, non-cancer pain syndromes. *Psychiatry Res.*, 1980, 2, 279-294.
8. COHEN, M. J., NALIBOFF, B. D., SCHANDLER, S. L., & HEINRICH, R. L. Signal detection and threshold measures to loud tones and radiant heat in chronic low back pain patients and cohort controls. *Pain*, 1983, 16, 245-252.
9. EKBLOM, A., & HANSSON, P. Thermal sensitivity is not changed by acute pain or afferent stimulation. *J. Neurol. Neurosurg. Psychiatry*, 1987, 50, 1216-1220.
10. FLOR, H., & TURK, D. C. Etiological theories and treatments for chronic back pain: I. Somatic models and interventions. *Pain*, 1984, 19, 105-121.
11. FRUHSTORFER, H., LINDBLOM, U., & SCHMIDT, W. G. Method for quantitative estimation of thermal thresholds in patients. *J. Neurol. Neurosurg. Psychiatry*, 1976, 39, 1071-1075.
12. HANSSON, P., EKBLOM, A., LINDBLOM, U., & MARCHETTINI, P. Does acute intraoral pain alter cutaneous sensibility. *J. Neurol. Neurosurg. Psychiatry*, 1988, 51, 1032-1036.
13. HELSON, H. *Adaptation-level theory: an experimental and systematic approach to behavior.* New York: Harper & Row, 1964.
14. KNORRING, L. VON, ALMAY, B. G. L., JOHANSSON, F., & TERENIUS, L. Endorphins in CSF of chronic pain patients, in relation to augmenting-reducing response in visual averaged evoked response. *Neuropsychobiology*, 1979, 5, 322-326.
15. LAUTENBACHER, S., MÖLTNER, A., LEHMANN, W. P., GALFE, G., HÖLZL, R., & STRIAN, F. SDT analysis of experimental thermal pain with "signal" and "no-signal" being determined psychophysically. *Percept. mot. Skills*, 1989, 68, 1019-1030.
16. LIPMAN, J. J., BLUMENKOPF, B., & PARRIS, W. C. V. Chronic pain assessment using heat beam dolorimetry. *Pain*, 1987, 30, 59-67.
17. LOESER, J. D. Pain due to nerve injury. *Spine*, 1985, 10, 232-235.
18. MALOW, R. M., GRIMM, L., & OLSON, R. E. Differences in pain perception between myofascial pain dysfunction patients and normal subjects: a signal detection analysis. *J. Psychosom. Res.*, 1980, 24, 303-309.
19. MALOW, R. M., & OLSON, R. E. Changes in pain perception after treatment for chronic pain. *Pain*, 1981, 11, 65-72.
20. NALIBOFF, B. D., COHEN, M. J., SCHANDLER, S. L., & HEINRICH, R. L. Signal detection and threshold measures for chronic back pain patients, chronic illness patients, and cohort controls to radiant heat stimuli. *J. abnorm. Psychol.*, 1981, 90, 271-274.
21. PERTOVAARA, A., KEMPPAINEN, P., JOHANSSON, G., & KARONEN, S-L. Ischemic pain non-segmentally produces a predominant reduction of pain and thermal sensitivity in man: a selective role for endogenous opioids. *Brain Res.*, 1982, 251, 83-92.
22. PETERS, M. L., SCHMIDT, A. J. M., & VAN DEN HOUT, M. A. Chronic low back pain and the reaction to repeated acute pain stimulation. *Pain*, 1989, 39, 69-76.
23. PRICE, D. D. *Psychological and neural mechanisms of pain.* New York: Raven, 1988.
24. PRICE, D. D., HARKINS, S. W., & BAKER, C. Sensory-affective relationships among different types of clinical and experimental pain. *Pain*, 1987, 28, 297-307.
25. SCHMIDT, A. J. M., & ARNTZ, A. Psychological research and chronic low back pain: a stand-still or breakthrough. *Soc. Sci. Med.*, 1987, 25, 1095-1104.
26. SELTZER, S. F., & SELTZER, J. L. Tactual sensitivity of chronic pain patients to non-painful stimuli. *Pain*, 1986, 27, 291-295.
27. SEVERIN, F., LEHMANN, W. P., & STRIAN, F. Subjective sensitization to tonic heat as an indicator of thermal pain. *Pain*, 1985, 21, 369-378.
28. TALBOT, J. D., DUNCAN, G. H., BUSHNELL, M. C., & BOYER, M. Diffuse noxious inhibitory controls (DNICs): psychophysical evidence in man for intersegmental suppression of noxious heat perception by cold pressor pain. *Pain*, 1987, 30, 221-232.
29. WILLER, J. C., ROBY, A., & LE BARS, D. Psychophysical and electrophysiological approaches to the pain-relieving effects of heterotopic nociceptive stimuli. *Brain*, 107, 1095-1112.
30. YANG, J. C., RICHLIN, D., BRAND, L., WAGNER, J., & CLARK, W. C. Thermal sensory decision theory indices and pain threshold in chronic pain patients and healthy volunteers. *Psychosom. Med.*, 1985, 47, 461-468.

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