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The role of operant conditioning in chronic pain: an experimental investigation

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Abstract

The role of operant conditioning for the development and maintenance of chronic pain was examined in 30 chronic back pain patients (CBP) and 30 matched healthy controls. Half of each group was reinforced for increased, half for decreased pain reports while EEG, EOG, heart rate, skin conductance and muscle tension levels were recorded. Both groups showed similar learning rates, however, the CBP patients displayed slower extinction of both the verbal and the cortical (N150) pain response. In addition, the CBP group displayed prolonged elevated electromyogram levels to the task. These data suggest that CBP patients are more easily influenced by operant conditioning factors than healthy controls and this susceptibility may add to the maintenance of the chronic pain problem. © 2002 International Association for the Study of Pain. Published by Elsevier Science B.V. All rights reserved.

Keywords: Operant factors; Learning; Evoked potentials; Electromyogram

1. Introduction

The operant model of chronic pain (Fordyce, 1976) suggests that positive and negative reinforcement of acute pain behaviors such as moaning, inactivity or medication intake may lead to the development of chronic pain. Although this model has had a significant impact on the treatment of chronic pain, little experimental evidence for its validity is available. Linton and Götestam (1985) used calibrated painful ischemic stimuli and reinforced healthy subjects for increases (up-training) or decreases (down-training) of verbal pain reports. Although the authors reported a within-subject differentiation between increases and decreases of these reports, the study must be interpreted with caution since individual learning curves were very variable and the ratings in the decrease pain condition were higher than the baseline ratings. Furthermore, the subjects could easily predict the physical intensity of the stimuli since their intensity was dependent on the blow-up time of the cuff. Replication studies by Lousberg and collaborators (Lousberg et al., 1996) failed to show clear operant conditioning effects. These authors used electric stimuli instead of the ischemic pain model. For the up-training condition, a significant increase in pain ratings as compared to baseline was observed, whereas the subjects in the control group, that were not reinforced positively or punished for altered pain ratings, showed no change. Pain report increases were, however, higher when punishing responses (requests to better concentrate) were provided. Skin conductance responses also showed a significant increase in the experimental group. However, the replication of this study failed. In an up- and down-conditioning paradigm with an additional unreinforced control group neither the up-conditioning nor the down-conditioning of pain reports could be established. SCR changes were found to be more associated with arousal than with subjective pain intensity. The authors suggested that the modified (punishing statements were now free of reference to attention) verbal feedback prevented the appearance of conditioning effects. Therefore, the previously shown conditioning effects might have been due to enhanced attention rather than operant conditioning.

In addition to verbal pain reports and autonomic measures the amplitude of the somatosensory evoked potential (SEP) related to painful stimuli has also been the target of operant conditioning experiments. The N150–P260 peak to peak amplitude of the SEP has been found to be positively correlated with subjective pain intensity (Bromm, 1984; Chapman and Jacobson, 1984; Chen et al., 1979; Miltner et al.,

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1988a,b). Rosenfeld et al. (1985) reported that half of their subjects showed a strong positive correlation between the reinforced SEP-amplitude changes and pain sensitivity and that the other half showed strong negative correlations between the two variables. Miltner et al. (1988b) reported increased pain reports to painful intracutaneous electric stimulation of the finger when subjects were reinforced for increasing the SEP peak-to-peak amplitude of the N150/P260. A study by Dowman (1996) found no effect of reinforced changes in P200 amplitude on pain sensitivity. The author rewarded the subjects for upward or downward changes of the P200 amplitude during sural nerve stimulation without explicit instructions how they could alter the P200 amplitude. The subjects were asked to find some psychological state that changed the amplitude. After the conditioning procedure was completed, somatosensory sensitivity was tested by intensity ratings of 80 stimuli of eight different intensities varying between just above compound nerve action potential threshold and just below subjective tolerance. Because the pain ratings were recorded after the training, rapid extinction can be responsible for the lack of the conditioning effect on the subjective level. None of the studies tested chronic pain patients in order to determine to what extent they might already have undergone an operant conditioning process and therefore show facilitated learning.

The aim of the present study was to examine the effects of operant conditioning of verbal pain expressions in both chronic back pain patients and healthy controls. It was hypothesized that chronic pain patients might have an extended history of operant conditioning of pain-related responses and might therefore learn the required responses faster and might show greater resistance to extinction. In addition to verbal reports, central and peripheral correlates of pain were assessed to determine to what extent the verbal conditioning effect would generalize.

2. Methods

2.1. Subjects

Thirty chronic back pain patients and 30 matched healthy volunteers recruited by newspaper advertisements that sought patients with chronic low back pain interested in learning more about their problem participated in the study. The chronic pain patients had been suffering from continuous low back pain for at least 6 months. The patients had mainly descriptive diagnoses such as non-specific back pain, back pain of muscular origin, musculoskeletal pain, etc. pertaining to IASP category XXVI-9. Exclusion criteria for all participants were (a) inflammatory cause of pain (e.g., rheumatoid arthritis), (b) neurological complications (e.g., loss of sensory or motor function, nerve root inflammation), (c) predominance of an unrelated pain syndrome, (d) cardiac pacemaker, (e) major psychiatric illness, (f) drug abuse, (g) allergy to plaster, (h) left-handedness and (i) use of centrally acting analgesics, muscle relaxants or tranquilizers. The experimental groups were matched with respect to gender, age and education (see Table 1). The healthy controls were paid (10 DM/h) for participation and the chronic pain patients could choose between payment ($N = 21$) and psychological counseling ($N = 9$). Half of the subjects in each group were randomly assigned to a down-training group and the other half to an up-training group.

2.2. Experimental design

Upon entering the laboratory, subjects were informed that the purpose of the experiment was to determine the ability of pain patients and healthy controls to estimate various pain intensities. The study was approved by the local ethics committee and adhered to the Declaration of Helsinki in its revised version of Tokyo. The subjects signed informed consent and completed several psychological assessment instruments prior to the laboratory session. After the attachment of the EEG, EOG, EMG and SCL electrodes, a gold electrode was inserted into a small opening in the upper layer of the skin of the middle finger of the left hand as described by Bromm and Meier (1984). An 8-min adaptation period and a 2-min resting baseline period followed. Pain threshold and pain tolerance for the arm and the back muscles were determined in two ascending and descending series of electric stimuli. The duration of a single electric stimulus was 50 ms, the stimulation frequency was 1 Hz. The subjects indicated verbally when pain and tolerance threshold were reached. The mean amperage of the four intensity values obtained for the pain threshold was used as individual pain threshold, the mean amperage of the two tolerance values was used as pain tolerance level. Four individual shock intensities were employed: pain threshold, 25, 50, and 75% of the distance between pain threshold and tolerance. During habituation, 15 electric stimuli at each of the four intensity levels ($N = 60$) were delivered. The training phase consisted of the application of 20 electric stimuli of each intensity ($N = 80$). During extinction, 15 stimuli of each level were presented ($N = 60$). After shock delivery, the subjects rated the intensity of the electric stimulus on a visual analogue scale provided on the computer screen. Additional feedback (computer smiley and monetary gain) was offered to the subjects in the training phase 1 s after they had completed their rating.

In the up-training group, subjects were given positive feedback when their actual pain rating was higher than the average rating of the last ten trials for each intensity level in the habituation period (baseline rating), lower ratings were followed by negative feedback. In the down-training group the positive/negative feedback assignment was defined conversely. Positive as well as negative feedback was arranged in four steps according to the degree of deviance from the baseline ratings. Neutral feedback was provided

Table 1
Demographic and clinical characteristics of the groups

	Healthy Controls		Patients	
	Up M (SD)	Down M (SD)	Up M (SD)	Down M (SD)
Age (in years)	39.33 (11.58)	39.27 (10.31)	43.14 (10.81)	43.27 (11.95)
Sex				
Male (*N*)	5	5	6	5
Female (*N*)	10	10	8	10
Education				
≤10 school years (*N*)	9	6	9	9
>10 school years (*N*)	6	9	5	6
MPI[a] 1 (0–6)				
Pain severity	–	–	3.07 (1.06)	3.38 (1.32)
Interference	–	–	3.39 (1.30)	2.98 (1.39)
Life control	–	–	4.00 (1.20)	3.62 (1.32)
Affective distress	–	–	2.58 (1.28)	2.89 (1.68)
Support	–	–	3.36 (1.50)	3.29 (1.81)
MPI 2 (0–6)				
Punishing responses	–	–	1.07 (1.25)	0.69 (0.65)
Solicitous responses	–	–	2.50 (1.69)	2.95 (1.57)
Distracting response	–	–	2.90 (1.69)	3.03 (1.44)
MPI 3 (0–6)				
General activity	–	–	2.94 (0.74)	2.68 (0.71)
BSQ[b]				
General stress	1.31 (0.91)	1.36 (0.65)	2.22 (0.95)	2.36 (0.79)
PRSS[c]				
Catastrophizing	0.59 (0.67)	0.66 (0.63)	1.50 (0.98)	1.81 (0.94)
Coping	3.52 (1.04)	3.99 (0.72)	3.14 (0.62)	3.17 (0.80)
BDI[d]	5.86 (4.98)	3.87 (3.44)	9.87 (7.05)	12.27 (6.92)
Somatic-Complaints Scale	12.93 (7.82)	12.67 (10.45)	20.07 (8.37)	21.47 (11.36)
Perception Threshold in mA	1.25 (0.66)	1.20 (0.74)	0.72 (0.47)	0.65 (0.65)
Pain threshold in mA	2.27 (0.94)	2.14 (1.29)	1.68 (1.21)	1.56 (1.17)
Pain tolerance in mA	3.06 (1.26)	3.00 (1.71)	2.31 (1.44)	2.41 (1.66)

[a] West-Haven Yale Multidimensional Pain Inventory.
[b] Brief Stress Questionnaire.
[c] Pain-Related Self Statements.
[d] Beck Depression Inventory.

when the actual rating was equal to the baseline rating. Feedback consisted of a computer smiley that moved the corner of the mouth up (positive feedback) or down (negative feedback) depending on the degree of reinforcement. Neutral feedback was represented by a straight line representing the mouth. Additionally, a counter was presented that added (positive feedback) or subtracted (negative feedback) the amount of money earned in addition to the usual payment in four steps. In the case of neutral feedback, the counter remained unchanged.

2.3. Psychological assessment

Patients completed the German version of the West Haven-Yale Multidimensional Pain Inventory (MPI, Flor et al., 1990; Kerns et al., 1985), the Pain-Related Self-Statement Scale (PRSS, Flor et al., 1993; Flor and Turk, 1988), the Beck Depression Inventory (BDI, Beck et al., 1961), the Brief Stress Scale (Flor, 1991), and the Somatic-Complaints Scale (B-L, von Zerssen, 1976). The MPI assesses pain severity, interference, affective distress, social support, life control, and significant others' responses to pain behaviors of the patients as well as the general activity levels. This measure was used to describe the clinical characteristics of the chronic back pain patients. The frequency of coping and catastrophizing pain-related self-statements, as measured by the PRSS, was recorded in order to determine the relationship of deficient coping and operant conditioning of pain ratings. The BDI was used as a measure of depressed mood, the Brief Stress Scale as a measure of general stress. Both, mood and stress, may affect the perception of painful electric stimuli and, therefore, the acquisition rate of the conditioned responses.

2.4. Physiological recordings and instrumentation

Electroencephalographic (EEG) data were recorded from 29 scalp locations using the International 10–20–System (Jasper, 1958) and extending it to a 10–10 system (Fpz, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T3, C3, Cz, C4, T4, CP5, CP1, CP2, CP6, T5, P3, Pz, P4, T6, PO1, PO2, O1, Oz, O2). The EEG channels were recorded based on the

guidelines for EEG recording of Pivik et al. (Pivik et al., 1993). In addition, lateral and vertical EOGs were assessed. Scalp recordings were monopolar referenced to Cz and converted off-line into linked earlobe-referenced recordings. EOG recordings were bipolar. EEG and EOG were recorded by Neuroscan Synamps amplifiers and sampled at a rate of 200 Hz (dc to 70 Hz low pass filter). The first phase of data acquisition began 1000 ms prior to the electric stimulus and was terminated 4 s after the delivery of the electric stimulus. During acquisition, a second measurement phase was used that began 1 s prior to the feedback and continued for 5 s after feedback onset. For the peripheral data, the beginning of the first data acquisition phase was 4 s prior to the electric stimulus. Intertrial intervals varied randomly between 4 and 8 s.

Electromyographic activity was recorded bilaterally from two muscle sites: M. flexor communis digitorum and M. erector spinae. Electrode placements were based on the recommendations of Fridlund and Cacioppo (1986) and Lippold (1967). A Coulbourn S75-01 bioamplifier with a bandpass filter from 90 to 250 Hz was used to collect the EMG data. This signal was rectified and integrated on-line by a Coulbourn S76-01 contour following integrator with a time constant of 20 ms. A sampling rate of 50 Hz was used for the integrated EMG.

The electrocardiogram was assessed using three 8-mm electrodes: left and right lower ribcage and clavicula. A Schmitt-trigger was employed to determine the R-waves for the computation of heart rate. Skin conductance level (SCL) was recorded by two 11 mm-electrodes from the thenar and hypothenar eminence, according to the recommendations of Fowles et al. (1981). A Coulbourn S71-22 skin conductance coupler, which processed the signal with DC coupling and an amplification factor of 50 mV/μs, provided a constant voltage of 0.5 V across electrodes. All peripheral data were assessed via Coulbourn amplifiers and processed by a personal computer. ECG electrode impedances were less than 10 kohms, all other electrode impedances (except for SCL) were maintained below 5 kohms. A ground electrode connected with all peripheral channels and spanning the whole wrist was placed on the left arm. The subjects were seated in a dimly illuminated and sound-attenuated room.

2.5. Data reduction and analysis

EOG artifact was corrected using an algorithm adapted from Gratton et al. (1983). For all experimental phases and intensity levels, the dependent physiological variables (somatosensory evoked potentials (SEP), EMG, SCL, and heart rate (HR) were averaged separately. After baseline correction (referenced to 100 ms preceding the electric stimulus) and averaging within conditions, three peak amplitudes (150, 260 and 300 ms) and the respective latencies were obtained. For the SEP components later than 400 ms no clear peak could be identified. The early SEP components consisted of the negatively deflecting N150 (peaking in the latency range 80–180 ms post stimulus, reference peak latency at Cz), the positively deflecting P260 (peaking in latency range 180–280 ms, reference peak latency at Cz) and the positively deflecting P300 (peaking in the latency range 280–400 ms, reference peak latency at Pz). The peaks were determined by visual inspection. As an additional ERP-parameter, the amplitude difference between the N150- and the P260-component (N150/P260) was analyzed as a potential cortical correlate of subjective pain experience (Bromm, 1984; Chapman and Jacobson, 1984; Miltner et al., 1988b).

For pain ratings and ERP parameters, Greenhouse–Geisser corrected repeated-measures analyses of variance (Jennings and Wood, 1976) were computed using the two between factors ‘group’ (chronic back pain patients versus healthy controls) and ‘direction‘ (up versus down training), and the within factors ‘intensity’ (four intensity levels) and ‘phase’ (habituation, acquisition, extinction). In addition, all experimental phases were segmented into blocks of five trials for all four intensities. For each phase, single ANOVAs for the pain ratings were carried out with the between factors ‘group’ and ‘direction’ and the within factors ‘blocks’ (three blocks in habituation and extinction, four blocks in acquisition) and ‘intensity’. For the pain ratings, additional comparisons were made for the last block of the preceding and the first block of the respective phase. For the ERP parameters, learning curves were analyzed by two ANOVAs with the factors ‘group’, ‘direction’, ‘phase’ (habituation vs. acquisition and acquisition vs. extinction) and ‘blocks’ (first and last block of the analyzed phases). Post-hoc Tukey-tests were performed using Bonferroni-adjusted alpha levels.

3. Results

3.1. Psychological assessment and stimulation data

A summary of the psychological assessment data is shown in Table 1. There were no significant differences in any of the psychological assessment data between the up- and down training groups neither for the chronic back pain patients nor for the healthy controls. Comparisons between the chronic back pain patients and the healthy controls were as follows: As expected, chronic back pain patients showed significantly more catastrophizing ($t(1) = -4.845$, $P < 0.001$) and less coping pain-related self-statements ($t(1) = 2.867$, $P = 0.006$), they experienced significantly more general stress ($t(1) = -4.467$, $P < 0.001$), showed a higher level of depressed mood ($t(1) = -4.122$, $P < 0.001$) and stated significantly more somatic complaints ($t(1) = -3.245$, $P = 0.002$) as compared to the healthy group. There were no significant correlations between stimulation-related pain ratings during habituation, acquisition or extinction and the psychological assessment data.

The patients had significantly lower pain thresholds than the healthy controls ($t(56) = -3.27$, $P = 0.002$) and tended to show also lower perception thresholds ($t(56) = -1.96$, $P = 0.055$) and pain tolerances ($t(56) = -1.71$, $P = 0.093$, see Table 1).

3.2. Pain ratings

The overall ANOVA yielded a significant interaction between direction and phase ($F(2, 110) = 41.53$, $P < 0.001$, $\varepsilon = 0.78$) indicating higher pain ratings in the up- as compared to the down-training in the acquisition ($t(1) = -3.51$, $P = 0.001$) and extinction ($t(1) = -3.95$, $P < 0.001$) but not in the habituation phase ($t(1) = -0.46$, ns, See Fig. 1). The main effect phase ($F(2, 110) = 5.87$, $P = 0.008$,$\varepsilon = 0.78$) indicated that the pain ratings were significantly lower in the habituation as compared to the acquisition phase ($t(58) = -2.66$, $P = 0.01$) and significantly higher in the acquisition phase as compared to the extinction phase ($t(58) = 2.41$, $P = 0.02$). Furthermore, there was a significant main effect of intensity level ($F(3, 165) = 94.89$, $P < 0.001$, $\varepsilon = 0.40$) indicating that during all experimental phases the subjects differentiated among the four intensity levels.

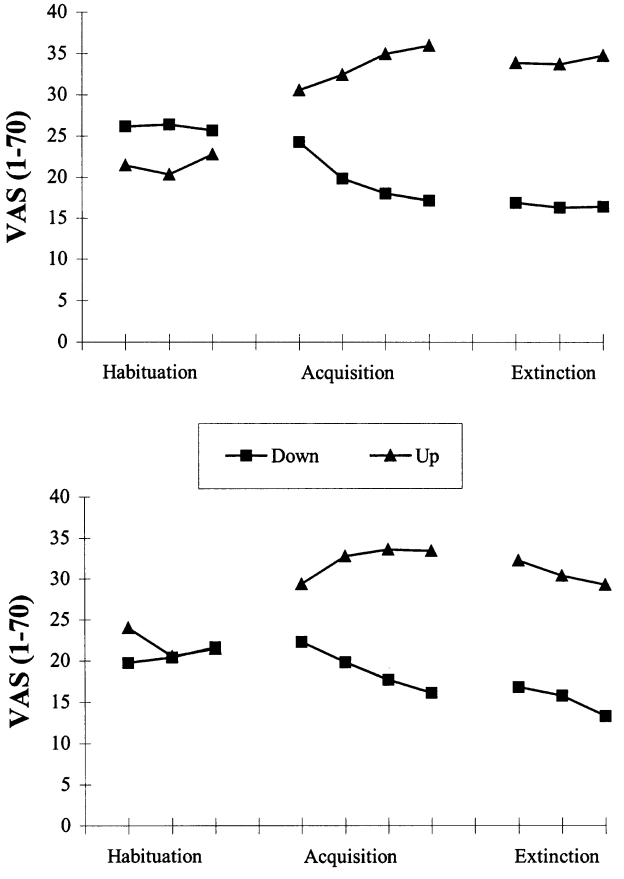

Fig. 1. Pain ratings in the habituation, acquisition and extinction phase of the up- and down-training groups for the chronic back pain patients (top) and the healthy controls (bottom).

The analysis of the blocks for acquisition revealed a significant interaction between direction and block ($F(3, 165) = 16.93$, $P < 0.001$, $\varepsilon = 0.58$) indicating that subjects' pain ratings moved in the expected directions. Post hoc contrasts showed that the pain ratings in the down-training decreased significantly for each subsequent block (all P values <0.05). However, in the up-training, the pain ratings showed a significant increase only between blocks 1 and 3 ($t(29) = -2.12$, $P = 0.04$), and blocks 1 and 4 ($t(29) = -2.53$, $P = 0.02$). The comparison between the last block of the habituation phase and the first block of the acquisition phase also yielded significantly higher pain ratings for the up-training condition ($F(1, 29) = 15.54$, $P < 0.001$).

The ANOVA for the extinction phase revealed a significant interaction between group and block ($F(2, 110) = 3.328$, $P = 0.043$, $\varepsilon = 0.63$). During extinction, the ratings of the healthy controls decreased continuously independent of the training condition (decrease from block 1 to 2: $t(29) = 2.55$, $P = 0.016$); block 2–3: $t(29) = 2.68$, $P = 0.012$); block 1–3: $t(29) = 3.37$, $P = 0.002$), whereas the ratings of the chronic back pain patients did not show extinction (all P values ns).

3.3. Somatosensory-evoked potentials

N150. The overall ANOVA revealed that the N150-component of the somatosensory evoked potential (SEP) was in general significantly enhanced in the chronic back pain patients as compared to the healthy controls (effect group: $F(1, 55) = 5.34$, $P = 0.025$, see Fig. 2). There was no significant intensity effect ($F(3, 165) = 0.459$, ns).

Furthermore, the overall ANOVA showed significant phase differences ($F(2, 110) = 8.100$, $P = 0.001$, $\varepsilon = 0.78$) indicating a general habituation of the N150 component of the SEP over the course of the experiment (see Fig. 3).

Closer analysis of the phases of the experiment revealed a significant interaction between group and direction during extinction ($F(1, 55) = 3.94$, $P = 0.05$), indicating signifi-

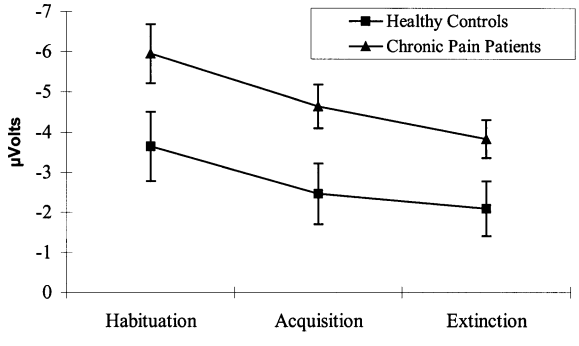

Fig. 2. N150 amplitude (SEM as error bar) for the chronic back pain patients and the healthy controls in the habituation, acquisition and extinction phases (averaged across up- and down training groups and across intensity levels).

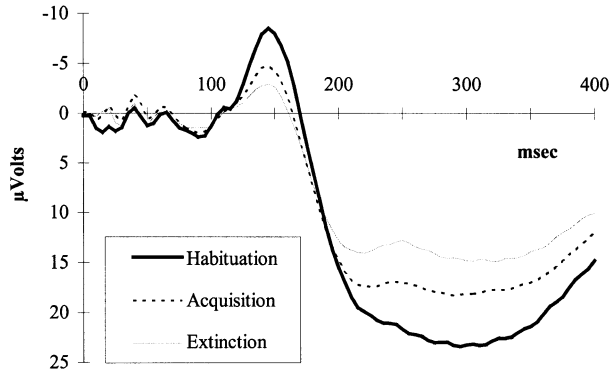

Fig. 3. ERP waveforms for the three experimental phases averaged across all subjects and four intensity levels.

cant extinction of the N150-amplitude in the up-training condition in the healthy controls ($t(14) = -4.40$, $P = 0.001$ see Fig. 4) but not the chronic back pain patients ($t(14) = -0.80$, ns).

The ANOVA for the first and last blocks of the habituation and acquisition phases revealed a significant group main effect ($F(1, 55) = 4.43$, $P = 0.040$). Furthermore, the three-way interaction between direction, blocks and phase was significant $F(1, 55) = 5.483$, $P = 0.023$). For the down-training, the N150 amplitude during the last training block was significantly lower than in the habituation blocks (first block of the habituation vs. last down-training: $t(29) = -3.155$, $P = 0.004$; last habituation block vs. last down-training block: $t(29) = -4.20$, $P < 0.001$). For the up-training, the N150 amplitude during the first block of the habituation was enhanced as compared to the first block of the acquisition ($t(28) = -2.75$, $P = 0.01$) but no significant amplitude differences between the habituation phases and the last block of the up-training could be found. The ANOVA for the blocks of the extinction phase showed no significant effects.

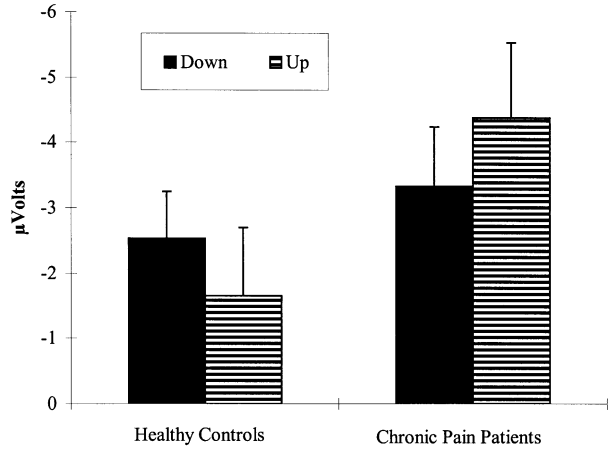

Fig. 4. N150 amplitude (SEM as error bar) for the chronic back pain patients and the healthy controls for the up-and down-training conditions in the extinction phase.

P260.The overall ANOVA for P260 revealed that amplitudes decreased significantly over the three phases ($F(2, 110) = 81.960$, $P \leq 0.001$, $\varepsilon = 0.72$) indicating a general habituation effect (see Fig. 3). Furthermore, there was a significant intensity effect ($F(3, 165) = 4.22$, $P < 0.009$). Post hoc tests revealed that P260 amplitudes were significantly higher at intensity level 4 than at levels 1 ($t(58) = -2.82$, $P = 0.007$) and 2 ($t(58) = -2.58$, $P = 0.012$). Level 3 elicited a significantly higher P260-amplitude than level 1 ($t(58) = -2.33$, $P = 0.024$). The ANOVA for the blocks of the habituation and acquisition phase revealed no significant effects.

Peak-to-peak amplitude N150/P260.For the N150/P260 amplitude, the overall ANOVA showed a significant phase effect ($F(2, 110) = 90.69$, $P < 0.001$, $\varepsilon = 0.70$) indicating habituation over the time course of the experiment (see Fig. 3). Post hoc tests revealed decreasing amplitudes between habituation and acquisition ($t(58) = 9.56$, $P < 0.001$) and between acquisition and extinction ($t(58) = 6.70$, $P < 0.001$). Furthermore, higher intensity levels elicited larger N150/P260 amplitudes ($F(3, 165) = 4.96$, $P = 0.005$, $\varepsilon = 0.82$). Post hoc tests showed that N150/P260 amplitudes differentiated significantly between intensity levels 1 and 4 ($t(58) = 10.96$, $P = 0.002$), levels 2 and 4 ($t(58) = 4.63$, $P = 0.036$) as well as between levels 1 and 3 ($t(58) = 7.73$, $P = 0.007$). The analysis for the segmented extinction phase revealed no significant effects.

P300. No significant group or interaction effects could be observed. Again, there was a significant general habituation effect ($F(2, 110) = 129.011$, $P < 0.001$) (see Fig. 3) and a significant amplitude difference among the four intensity levels ($F(3, 165) = 30.276$, $P < 0.001$).

None of the latency analyses became significant suggesting that stimulus latencies were not affected by the conditioning procedure. There were no significant group or condition differences in the phase before the feedback. These data are therefore not presented.

3.4. Peripheral measures

There were no baseline differences in the bilateral m. communis flexor digitorum and the bilateral m. erector spinae EMGs. Furthermore, no significant group or direction differences could be detected for the electromyographic activity of the right forearm. The ANOVA with the between factor 'group' and the within factor 'time' course (baseline, 0–500 ms, 500–1000 ms) for the left flexor communis digitorum revealed a significant two-way interaction between group and time course ($F(2, 114) = 18.460$, $P < 0.001$, $\varepsilon = 0.9160$) and a significant main effect of time course ($F(2, 114) = 24.884$, $P < 0.001$). Post hoc tests showed that the chronic back pain patients had significantly higher EMG-levels than the healthy controls in the late period after stimulus onset (500–1000 ms) ($t(1) = -4.467$, $P < 0.001$). Within group tests showed that there were significantly enhanced EMG-levels in the first 500 ms as compared to

the baseline in both groups ($P < 0.05$), however, only the chronic back pain group showed a prolonged enhanced EMG ($P < 0.001$). The ANOVA for the left m. erector spinae revealed a significant interaction between group and time course ($F(1, 55) = 24.402$, $P < 0.001$). Closer analysis of this effect showed that EMG activity was reduced in the period 500–1000 ms after electric stimulus onset compared to the early period (1–500 ms) ($t(29) = 3.558$, $P = 0.001$) in the healthy controls. In contrast, the chronic back pain patients showed enhanced EMG activity in the period 500–1000 ms as compared to 1–500 ms ($t(28) = -3.739$, $P = 0.001$).

The ANOVA for SCL did not reveal any significant effects. The analysis of heart rate showed a significant main effect of phase $F(2, 110) = 6.193$, $P = 0.003$) indicating significantly enhanced HR levels in the extinction as compared to the habituation phase ($t(58) = -3.332$, $P = 0.002$).

4. Discussion

It can be concluded from the data that pain reports in both healthy controls and chronic back pain patients can be brought under operant control. Whereas the healthy controls showed fast extinction, the chronic back pain patients maintained their elevated pain ratings throughout the extinction phase and thus counteracted the general habituation of the pain ratings that was present in the data. The early SEP-component of the chronic pain patients was generally elevated and the pain threshold was reduced. This may reflect an enhanced reactivity to painful stimuli in chronic back pain patients as shown in previous MEG and EEG studies of our group (e.g., Flor and Birbaumer, 1994; Flor et al., 1994; Flor et al., 1997a,b) indicating cortical hyperreactivity to painful electric stimulation in chronic pain patients. If operant conditioning of overt pain responses leads to the formation of new cortical and subcortical cell assemblies (e.g., Flor and Birbaumer, 1994; Flor et al., 1994), then pain-related EEG responses should reflect this conditioning process as well.

The up-down differentiation of the verbal reports was, however, not mirrored in the N150 component during acquisition although this effect was overlaid by a general habituation of the N150 component. The significant decrease of N150 during down training and the lack of habituation of the N150 in the up training may be viewed as indicative of a conditioning process. However, compared to subjective pain ratings, the cortical pain response showed delayed conditioning with the up/down differentiation occurring only in the fourth as compared to the first acquisition block. During extinction, N150 remained elevated in the CBP patients indicating that the conditioning effect of the chronic back pain patients remained present on the cortical level. N150 has been related to activation of the anterior cingulate (Dowman, 1994a,b; Dowman and Darcey, 1994) as well as primary and secondary somatosensory cortex (Desmedt and Tomberg, 1989; Raichle, 1994) and the modifiability of this component by the reinforcement of verbal pain reports has important implications for the processing of painful stimuli in chronic pain patients. These data suggest that external reinforcement of pain behavior enhances the subsequent processing of painful stimuli. To what extent the affective or the sensory pain components (Rainville et al., 1997) are modified by operant conditioning cannot be determined from this study because source analyses were precluded by reduced signal to noise ratios in the patient group.

P200, which reflects late evaluative processes in the pain experience, was not influenced by operant conditioning nor was the N150/P260 peak to peak amplitude as a general indicator of pain processing. These data suggest that it may be the early processing of pain reflecting sensory rather than later cognitive-evaluative processes that are influenced by operant learning. Operant conditioning of components earlier than 100 ms might give additional information on the stages of processing of pain responses.

The analysis of the peripheral measures suggests that the altered verbal pain reports do not generalize to general indicators of pain and arousal such as heart rate, skin conductance or muscle tension. However, the muscle tension data confirm previous reports of stress-related muscle tension increases in chronic back pain patients (Flor et al., 1992; Moulton and Spence, 1992). Only the patients responded with prolonged increased muscle tension levels to the operant conditioning task. The stronger response of the left side of the body reflects (a) that the painful stimulus was applied to the left finger and (b) confirms previous reports of more pronounced pain responses of the right cerebral hemisphere (Brennum et al., 1989; Jensen et al., 1992). The increase of heart rate in the extinction phase for both the patients and the healthy controls may reflect the uncertainty induced by the removal of reinforcement (Kelsey, 1991).

Taken together, the results of this study show that the operant conditioning of verbal pain reports leads to delayed extinction in chronic pain patients and generalizes, moreover, to the cortical level. The lack of significant correlation between resistance to extinction and chronicity suggests that the prolonged operant conditioning effects might precede, not follow, the onset of chronic pain and thus constitute a predisposing factor.

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