Chronic low back pain, response specificity and habitation to painful stimuli

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ABSTRACT The present study examines two related issues. Firstly, is chronic low back pain (CLBP) related to a delayed habituation to an experimental pain stimulus? Secondly, are CLBP patients characterized by response specificity of the paraspinous muscles when confronted with a painful stimulus? CLBP patients (n=22) and healthy controls (n=21) received two series of 10 painful electric shocks. Subjective and physiological (SCR, HR, FPV, frontalis EMG, and paraspinous EMG) measures were obtained. No evidence was found for a global difference between CLBP patients and controls with respect to subjective and physiological habitation. However, CLBP was associated with physiological response specificity of the paraspinous muscles. Paraspinal responses to the pain stimulus were larger in the patient group than in the control group, whereas heart rate acceleration was smaller in patients than in controls. The physiological response pattern of the CLBP patients might be related to a more passive, helpless, and emotional way of processing pain. Whereas subjective experiences habituated, some physiological (including muscular) responses did not. This may play a part in the maintenance of CLBP. Theoretical and clinical implications of the response specificity are discussed.

Introduction

Habituation of human physiological reactions to aversive stimuli, like painful shock, cold pressor, and noise, is a well-documented phenomenon (Wolff, 1978; Tursky, 1974; Strempel, 1976). Habituation of the subjective pain experience of normal volunteers has also been found (Ernst et al., 1986; Brands and Schmidt, 1987; Arntz and van den Hout, 1988). Chronic pain is defined by a persistent (i.e. non-habituating) subjective experience of pain. The present study is concerned with the question how the apparent lack of subjective habituation to pain of chronic low back pain patients is related to habituation processes under laboratory conditions. A small number of studies has addressed the relationship between chronic or recurrent pain and habituation under laboratory conditions\textsuperscript{1} directly.

\textsuperscript{1} Apart from these experiments, a number of studies has found differences between 'high pain behaviour' subjects and 'low pain behaviour' subjects as to physiological habituation to pain stimuli. The 'high pain behaviour group' was found to habituate more slowly (Sternbach and Tursky, 1965; Tursky and Sternbach, 1967). However, these subjects were not chronic pain patients.

Ellertsen and Hammerborg (1982) found that the heart rate responses (HRR) of migraine patients habituated to an aversive auditory stimulus (95 dB) more slowly than the HRR of healthy controls. Using an 80 dB auditory stimulus, Paschier and Ordebeke (1983) reported a similar slower habituation of skin conductance responses (SCR) of migraine patients. However, they could not replicate the slower HRR habituation found by Ellertsen and Hammerborg. Kröner (1984) did not find any significant differences between headache patients of various types (migraine, tension, mixed and unclear) and healthy controls as to habituation of the SCR to a non-aversive auditory signal (70 dB). When auditory signals equal to or greater than 80 dB are classified as aversive and weaker signals as non-aversive, these studies seem to indicate that autonomic habituation of migraine patients to aversive auditory stimuli might be delayed, but that their habituation to non-aversive signals is not. Studying endurance of repeated acute pain (cold pressor test) of CLBP patients and healthy controls, Brands and Schmidt (1987)
found that normals showed clear repetition effects (increased or decreased endurance), whereas CLBP patients did not show any repetition effects at all. Inspired by these findings, Schmidt and Arntz (1987) hypothesized that the habituation process of CLBP patients might be delayed. However, to the best of our knowledge no study has as yet addressed this issue directly.

An important point of interest with respect to habituation and the psychophysiological aspects of CLBP is the notion of response specificity. Lacey and Lacey (1958) suggested a relationship between an exaggerated and rigidly stereotyped response specificity and psychosomatic complaints. Excessive responses of a physiological system are hypothesized to cause a dysregulation in the system, leading to somatic symptoms (Sternbach, 1966; Flor and Turk, 1989). There are some studies addressing the question whether CLBP patients show response specificity, in particular a hypothesized excessive back muscle activity. Contradictory results have been reported. During rest, in different positions and during different motions, paraspinous muscle activity of CLBP patients has been found to be increased (Hoyt et al., 1981; Kravitz et al., 1981), decreased (Wolf and Basmanian, 1977; Collins et al., 1982; Soderberg and Barr, 1983), or similar (Nouwen and Bush, 1984; Cohen et al., 1986) compared to a control group. Thus, the role of tonic paraspinous muscle activity in CLBP remains unclear (see also Flor and Turk, 1989, for a similar conclusion).

It has also been suggested that CLBP patients show response specificity in reaction to stress (e.g. Sternbach, 1966). Most studies, however, have not been able to demonstrate a more pronounced EMG-response to several kinds of laboratory stressors in CLBP patients than in normal controls (Wilfling, 1981; Collins et al., 1982; Nouwen and Bush, 1984; Cohen et al., 1986). On the other hand, Flor et al. (1985) did find increased paraspinous EMG in CLBP patients while discussing their pain and while discussing personally relevant stress. Flor et al. claim that the personal relevance of the stressor is the crucial factor. The (tonic) reaction of CLBP patients' paraspinous EMG to an acute pain stimulus (cold pressor test) was measured in two studies (Collins et al., 1982; Cohen et al., 1986). Neither of them found an abnormal paraspinous response. However, the study by Collins et al. (1982) did find increased frontalis EMG and SCR in CLBP patients to the cold pressor test. Whereas tonic EMG levels of CLBP patients do not seem to be abnormally elevated by stress or by pain, it is not clear yet whether CLBP is associated with stronger short term (phasic) paraspinous responses to pain.

The present study addresses physiological and subjective habituation to painful stimuli and response specificity, in relation to CLBP. More specifically, the study aimed at testing the following hypotheses:

(1) compared with healthy controls, CLBP patients show delayed subjective and physiological habituation during repeatedly delivered painful electric shock.

(2) CLBP patients show response specificity, i.e. stronger back muscle reactions to the painful stimulus than controls. In order to test the specificity hypothesis, other physiological responses were also measured to exclude the possibility that CLBP patients show generally stronger responses to painful stimuli.

Method

Subjects

Forty-three subjects participated in the study, 22 CLBP patients and 21 healthy controls matched for age and sex. There were 12 male patients, with a mean age of 43.1 years (range: 33–53 years) and 11 male controls (one control failed to turn up), with a mean age of 42.3 years (range: 34–56 years). The 10 female patients and 10 female controls had a mean age of 45.0 years (range: 28–54 years) and 45.6 years (29–56 years), respectively. The criterion for inclusion in the category of CLBP patients was: having had low backache complaints for at least 6 months without a demonstrable organic pathology (e.g. cancer, ankylosing spondylitis, spinal infections, sciatica, lumbar). Subjects with serious cardiac trouble were excluded from participation. The mean duration of the pain complaints of the CLBP patients was 15 years (median: 15; SD: 8; range: 4–35 years). All patients had sought medical treatment for their complaints. Ten
patients received a financial compensation for being disabled, because of their back pain. Eighteen patients had received no or vague diagnoses of their back pain (like 'weak back', 'stress', 'deteriorated back'), 3 patients had vague diagnoses related to alleged degenerative processes of the spine and one had received 'scoliosis' as diagnosis. One patient had been operated for low back pain (with no success). One patient used a daily 2 mg. diazepam medication and occasionally used paracetamol. The other patients did not use analgesics or other medications related to back pain. All subjects were analgesics-free on the day of testing. Both patients and controls were recruited by means of an advertisement in a local newspaper and received a small financial reward for their participation. There were more patients than controls who received a benefit in accordance with the Dutch Disablement Insurance Act (10 patients vs. 1 control).

Procedure

Before entering the laboratory, the subjects completed several questionnaires (these questionnaires were used in order to describe the samples and test pre-experimental differences). These included: (1) A Dutch version of the Eysenck personality questionnaire, with the subscales neuroticism, psychosomatic complaints, extraversion and defensiveness (ABV; Wilde, 1983); (2) a self-esteem questionnaire; and (3) a depressive complaints checklist, measuring cognitive/emotional, motivational and somatic (sleep) complaints (Bouman, 1987). CLBP patients also rated current back pain level on a 0–100 visual analogue scale (VAS). Subjects were tested individually and were seated in a comfortable armchair placed in a dimly lit, sound-attenuated room. After the physiological measurements had been checked, subjects were asked to relax for 5 min (adaptation period). At the end of this interval, the first baseline measurements were obtained. The shock level was subsequently assessed by increasing the amperage from zero up in steps of 0.5 mA, until the subject indicated that the shock was painful. Having reached the pain threshold level, subjects rated each stimulation on a VAS. The level was further increased until subject rated it more than 50 on a 0–100 VAS (= pre-test rating). This level was kept constant over all trials. Following the assessment of the shock level, subjects were told that they would receive a number of unsignalled shocks of the level that had been determined previously. The number of shocks was not specified in advance. The subjects were requested not to move during the series and were then left alone in the quiet, small room. Shock duration was 1 sec. Intertrial intervals varied between 15 and 45 sec, with a mean of 30 sec. An attempt was made to allow dishabituation to occur by having one of the experimenters enter the room after the 10th shock. He requested the subject to rate the subjective level of pain of the last shock of the series, as well as the subjective level of anxiety during the series on VASs. A rest period which would be followed by the second series was then announced, and the experimenter left the room. Before the start of the second series, there was a 2 min stimulus-free adaptation period followed by a second 15 sec baseline measurement of the physiological parameters. After the second series, the experimenter entered the room and asked the subject to rate the subjective pain level of the last shock of the second series, and the subjective level of anxiety during the second series on VASs. In the presence of the experimenter, a last (announced) shock was given and subjects rated its subjective pain level (post-test rating). Next, the subject was requested to relax for several minutes and the experimenter left the room. After a rest period of 2 min, a third baseline measurement (15 sec) was obtained. Electrodes were then removed and the subject was thanked for participation and paid. All subjective ratings were done in the presence of the same experimenter. Interaction was kept at a minimum level during these ratings.

Apparatus

Apparatus was placed in an adjacent room. HR was recorded from electrodes placed...
below the sternum and at the left side, at the edge of the chest, and connected to a Beckman Voltage/Pressure/Volume Coupler (type 9853A). Finger Pulse Volume (FPV) was monitored by means of a photocell (Beckman radial transducer 215560) placed on the tip of the first finger of the subject’s right hand. The transducer was connected to a Beckman Voltage/Pressure/Volume Coupler (type 9853A). The time constant was 0.1 sec. SCR and skin conductance level (SCL) were measured by a Beckman Skin Conductance Coupler (type 9844), using the method of constant voltage (0.5 volts). Electrodes were attached to the medial phalanges of the second and third fingers of the right hand with adhesive collars.

SCR, SCL, HRR, and FPV were continuously monitored by a Beckman Polygraph. Bipolar electromyographic surface recordings (EMG) were made from the left lateral frontalis, with a reference electrode placed on the centre of the forehead, by means of a Myotron (type 220). In addition, bipolar EMG surface activity was measured from the part of the paraspinal lower back region that patients localized as their most painful area. Placement of lower back EMG electrodes was matched in control subjects. A Myotron (type 220) was used for recording the paraspinal EMG. For both frontalis and paraspinal EMG low-pass and high-pass filters were set at 1000 and 40 Hz, respectively. The raw EMG was integrated over periods of 1 sec. Integrated voltage was the basis for the data of muscle activity.

For HR, SCR, and frontalis EMG, Beckman Ag-AgCl electrodes (8 mm diameter) were used. Paraspinal back EMG was recorded from Hewlett Packard Surface electrodes (12 mm diameter). SCR recording sites were cleaned with distilled water, whereas HR and EMG recording sites were cleaned with 70% alcohol. An electric stimulator (also placed in the apparatus room) with a maximum capacity of 60 mA administered an electric current (AC) to the subject. Two shock electrodes were attached to the first finger of the subjects left hand. Electric shocks had a duration of 1 sec. The stimulator delivered shocks of constant current.

A microcomputer (PDP Mic-11) controlled the onset of the electric stimuli, inter-trial intervals, and registration. The microcomputer was also used for on-line processing of the cardiac inter-beat (R-R) intervals.

Data definition, reduction and analysis

Baselines of all physiological measures were obtained before the first series, between the two series, and after the second series.

SCLs were directly displayed on the coupler. SCRs were defined as the largest deflection occurring 1–4 sec after stimulus offset. SCRs deviated markedly from normal distribution. After square-root transformation (Levey, 1980), the distributions were approximately normal. Following recommendations by Siddle and Heron (1975), FPV changes (vasoconstriction) were expressed as percentages of the three smallest deflections in a 15 sec post-shock period compared with the smallest deflection in the 10 sec pre-trial period.

HR inter-beat intervals were obtained for six beats immediately following the shock.3 Pilot studies indicated that the HR acceleration response occurs within six beats after the shock. Each interval was expressed as a deviation from the mean pre-trial inter-beat interval, computed over 10 beats. A positive sign indicates a shorter interval, that is, an acceleration. In order to analyse the cardiac waveform, the HRRs were decomposed in orthogonal components (mean, linear, quadratic, cubic, quartic, quintic, etc.) by means of MANOVA trend analysis (Wilson, 1980). Following Wilson’s (1980) instructions, habituation of the HRR was analysed by MANOVA trend analyses of each series on the orthogonal HR response components of each trial.

Inspection of the EMG data showed that EMG responses occurred within 7 sec after shock offset. The distributions of the integrated EMG data differed markedly from normal distribution and were positively skewed. Therefore, the EMG data were subjected to a logarithmic transformation. EMG reactions after each shock were expressed as deviations from tonic level (as assessed 10 sec before shock). The 7 integrated EMG values of the 7 sec after shock were summed. Raw (i.e. not corrected for tonic level) summed EMG reactions were also analysed.

Dishabituation of each physiological re-

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3 Because of instrumental failure, HR data of one subject was missing.
spontaneous was tested by contrasting the first response of the second series with the average response of the last three trials of the first series. If there was no indication of dishabitation, the two series were combined in one trend analysis over 20 trials for testing habituation. If there was a dishabitation of the response, the two series were analysed separately. For responses that showed a quick exponential decline to an asymptote (e.g. SCRs, subjective responses), a trend analysis with log trial number was performed to test between group effects in habituation rate. For the other responses (FPV, HRR, EMG), a ‘normal’ trend analysis was performed; between group differences in the linear trend indicate differences in habituation. Between group effects in the mean trend refer to differences in the mean level of responding between patients and normals.

Results

Pre-test differences

The two groups were tested for differences in pre-test variables. CLBP patients differed from controls in several respects. The patients had a lower educational background than the healthy controls (Kruskal-Wallis: $\chi^2 = 11.54$, $p < 0.01$), and indicated that they had more depressive complaints on the depression checklist (cognitive/emotional subscale: $F(1,39) = 5.65$, $p = 0.02$; motivational subscale: $F(1,39) = 12.26$, $p < 0.001$; sleep disturbances: $F(1,39) = 6.90$, $p = 0.01$), as well as more psychosomatic complaints (ABV-NS, $F(1,37)^4 = 26.51$, $p < 0.001$). Differences between CLBP patients and normals did not reach significance with respect to self-esteem ($p = 0.20$), neuroticism ($p = 0.11$), extraversion ($p = 0.24$), and defensiveness ($p = 0.39$). Patients tended to reach the 50 mm criterion on the VAS at a lower amperage than the control group (2.0 mA vs. 3.0 mA, $F(1,41) = 4.12$, $p < 0.05$).

It was decided that the educational level and amperage would be used as covariates in subsequent tests. Depression and psychosomatic complaints were not used as covariates, since they can reasonably be considered as inherent characteristics of CLBP.

4 Because of various missing data the n varies slightly.

Subjective measures

Subjective pain. VAS scores are depicted in Figure 1. Subjective pain decreased in both groups from pretest to the end of the second series (exponential decline, $F(1,39) = 6.72$, $p < 0.02$). The between group effect was n.s. ($F(1,39) = 0.34$, $p = 0.56$).

Subjective anxiety. This was scored after the first, as well as after the second series. There was a significant mean reduction in subjective anxiety from the first to the second series ($t = -3.05$, $p = 0.004$), but patients did not differ from controls ($F(1,39) = 0.33$, $p = 0.57$).

Summarizing, it may be stated that CLBP patients, as well as healthy controls, showed (equal) habituation in their subjective pain levels. No differences were found between patients and controls with respect to subjective pain and anxiety. Thus, no evidence was found to sustain the hypothesis that CLBP patients display delayed habituation.

Physiological measures

SCL/SCR. Patients and controls did not differ with respect to pre-test SCL, between-series SCL and post-test SCL (multivariate $F(3,37) = 1.43$, $p = 0.25$). The mean SCL was 8.86 microSiemens (SD 8.94) for the CLBP patients and 8.25 (SD 5.52) for the controls.

The mean SCRs for each group are depicted in Figure 2.

Habituation was tested by (1) a trend analysis with log trial number (1–10) for the two series separately (exponential decline) and (2) testing the dishabitation of the SCR at the start of the second series. Dishabitation was expressed as the difference between the first response of series 2 and the averaged responses of the last 3 trials of series 1. Pre-test SCL, amperage and educational level served as covariates.

(1) A main exponential decline was found (first series: $F(1,38) = 5.95$, $p < 0.02$; second series: $F(1,38) = 6.19$, $p < 0.02$), but patients and controls did not differ in this respect (first series: $F(1,38) = 0.09$; second series: $F(1,38) = 0.81$). On the whole, patients had marginally higher SCRs than controls in the first series ($t = 1.68$, $p < 0.10$), but not in the second series ($t = 1.13$, $p = 0.26$). However, this effect disappeared when the covariates were left out.

(2) Patients did not react more strongly after
Figure 1 Mean subjective pain ratings on a 0–100 VAS of the CLBP and control group (adjusted means). Subjective pain decreased in both groups from pre-test to the end of the first series and continued to do so to the end of the second series and the post-test. There were no between group effects.

Figure 2 Skin conductance responses (SCRs) due to painful shock in microVolts: SCRs were square-root transformed. The figure depicts the mean responses of both groups to the 10 trials in series 1 and the 10 trials in series 2 (adjusted means). There were no differences between groups in SCR habituation.

Figure 3 Heart rate (HR) responses (6 intervals after shock expressed as deviation from the pre-trial inter-beat interval) averaged over all 20 trials for the CLBP patients and the controls. The figure shows that the HR acceleration is more pronounced in the control subjects ($p < 0.02$).

dishabituation than controls ($F(1,38) = 0.65$, $p = 0.43$).

In sum, no evidence was found for hypothesis (1), which states that CLBP patients would show impaired SCR habituation. There was some evidence that CLBP patients show stronger SCRs. The effect, however, was restricted to the first series and reached only borderline significance.

Vasoconstriction. There was no significant main effect of dishabituation of FPV responses (expressed as the difference between the first response of series 2 and the averaged last three responses of series 1) between the series ($F(1,34) = 0.22$), nor was there a between group effect ($F(1,34) = 0.01$). Consequently, the vasoconstriction responses were analysed by means of a trend analysis over all 20 trials. The vasoconstriction responses did not show any habituation at all as reflected by the linear trend ($F(1,34) = 0.07; p = 0.80$). There were no significant differences between CLBP patients and controls as to the mean amount of vasoconstriction ($F(1,34) < 0.001$) and its decrease ($F(1,34) = 0.02$).

HR responses. Pre-trial and baseline HRs were analysed by means of MANOVA. No significant difference between patients and controls emerged. The mean pre-trial inter-beat interval (IBI) in series 1 was 0.84 sec (SD 0.10) for the CLBP patients and 0.82 sec (SD 0.15) for the controls. During series 2, the means were 0.84 (SD 0.10) for the CLBP patients and 0.86 (SD 0.14) for the controls. Figure 3 shows the 6 post-stimulus HR intervals averaged over all 20 trials for patients and controls separately on a beat-by-beat basis. The different shapes of the HR responses for patients and controls appear in a significant patient effect on the quintic component ($F(1,38) = 6.17$, $p < 0.02$). Thus, the controls' response appears to be sharper. There were no other significant between-group effects on the general HR response.

The results of the MANOVA trend analysis of the HRR components, testing habituation of the HRR, are summarized in Table 1.

The first series showed significant general linear trends of all components (multivariate test: $p = 0.001$): HR responses became stronger during the first series. The results of the first series do not support the hypothesis of
Table 1  Linear trends of the components of the heart rate responses.

<table>
<thead>
<tr>
<th>HRR component</th>
<th>Main effect</th>
<th>Patient effect</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>t</td>
<td>p</td>
</tr>
<tr>
<td>series 1</td>
<td></td>
<td></td>
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<tr>
<td>mean</td>
<td>3.48</td>
<td>0.001</td>
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<tr>
<td>x</td>
<td>1.81</td>
<td>0.08</td>
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<tr>
<td>x^2</td>
<td>3.35</td>
<td>0.002</td>
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<tr>
<td>x^3</td>
<td>4.54</td>
<td>&lt;10^-4</td>
</tr>
<tr>
<td>x^4</td>
<td>5.26</td>
<td>&lt;10^-4</td>
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<tr>
<td>x^5</td>
<td>3.21</td>
<td>0.003</td>
</tr>
<tr>
<td>x^6</td>
<td>2.77</td>
<td>0.008</td>
</tr>
<tr>
<td>multivariate</td>
<td>F(7,34)=4.86</td>
<td>0.001</td>
</tr>
<tr>
<td>series 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>-0.98</td>
<td>0.33</td>
</tr>
<tr>
<td>x</td>
<td>-0.36</td>
<td>0.72</td>
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<tr>
<td>x^2</td>
<td>0.13</td>
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<td>x^3</td>
<td>0.15</td>
<td>0.88</td>
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<td>x^4</td>
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<td>x^5</td>
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<td>x^6</td>
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<tr>
<td>multivariate</td>
<td>F(7,34)=0.39</td>
<td>0.90</td>
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</tbody>
</table>

Heart rate responses due to the shock were resolved in orthogonal components (mean, linear, quadratic, etc.), representing the form of the response, and subsequently subjected to a MANOVA linear trend analysis to test whether there were linear trends over trials referring to changes of the HRR form during the series.

delayed habituation in CLBP patients; whereas there are general linear increases in the components of the HR response, patients show smaller linear trends of the first five components (multivariate test: p = 0.025, see Table 1). Thus, series 1 yielded no evidence supporting hypothesis 1 with respect to HR responses.

In the second series, the patient group also showed linear trends that differed significantly from the control group in a direction opposite to what had been hypothesized for mean, linear, and quadratic components (see Table 1). Whereas the controls showed increases in these components during series 2, the CLBP patients showed decreases. Thus, results again contradicted the first hypothesis.

Finally, testing the dishabituation of the HR response indicated a marginally significant main effect (t = 1.39, p = 0.085, one-tailed), but there was no between group effect (t = -0.59, n.s.).

Summarizing, HR responses did not support hypothesis 1: if anything, controls do not seem to habituate. The finding that patients had smaller HR responses than controls indicates that CLBP patients do not show generally stronger autonomic responsiveness to pain.

EMG of the frontalis. Baselines were analysed by means of MANOVA for repeated measures. The patients’ baselines were generally lower than those of controls, as reflected by a multivariate test (F_{1,36} = 6.18, p = 0.002). There were no other significant effects on baselines or changes in baselines. The mean pretrial frontalis EMG during series 1 was 1.51 (SD 0.36) for the CLBP patients and 1.82 (SD 0.45) for the controls. These levels differed significantly between groups (F(1,38) = 10.41, p < 0.005). The means during series 2 were 1.47 (SD 0.39) for the patients and 1.71 (SD 0.40) for the controls. Again, groups differed significantly (F(1,38) = 13.67, p = 0.001).

The frontalis reactions, expressed as deviations from tonic level and summed over 7 sec, are depicted in Figure 4. Since there was neither a dishabituation effect at the first trial of the second series compared to the averaged last 3 trials of series 1 (F(1,38) = 1.87, p = 0.18), nor a between-group effect on dishabituation (F(1,38) < 0.001), the two series were analysed in one MANOVA trend analysis over 20 trials.

Patients generally showed stronger reactions of the frontalis than healthy controls (t = 2.24,
p < 0.03). As to habituation, there was no significant main linear decrease (F(1,38) = 1.72, p = 0.20). There was no patient effect on the linear trend (F(1,38) = 0.17, p = 0.68).

In sum, CLBP patients showed less tonic frontal EMG activity and stronger responses of the frontal EMG after shock. However, the stronger responses may also be attributed to the lower tonic level of the patients (i.e., the law of initial values): when ‘raw’ (not expressed as deviations from tonic level) EMG responses were analysed, CLBP patients showed smaller frontal EMG reactions than controls (t = 2.05, p = 0.05). No evidence supporting the hypothesis that CLBP patients show delayed habituation was found from EMG frontal EMG data.

Paraspinal EMG. Inspection of the data revealed that they were free of movement artefacts. Paraspinal EMG baselines were analysed by means of MANOVA for repeated measures. There were no patient effects on the baseline levels as indicated with a multivariate test (F_{MANOVA}(3,32) = 0.04, p = 0.99). The mean pre-trial tonic paraspinal EMG level during series 1 was 1.22 (SD 0.33) for the CLBP patients and 1.27 (SD 0.36) for the controls (F(1,34) = 0.03). During series 2, these pre-trial levels were 1.19 (SD 0.33) for the patients and 1.20 (SD 0.38) for the controls (F(1,34) = 0.03). Thus, tonic paraspinal levels during the series were not significantly different between groups.

The paraspinal responses are depicted in Figure 5. There was no dishabituation at the first trial of the second series (compared to the averaged last 3 responses of series 1; F(1,34) = 0.08), nor did patients differ from controls with respect to paraspinal response habituation (F(1,34) = 0.86). Therefore, the series were combined in one trend analysis over 20 trials.

There was no significant main effect of habituation (raw responses: F(1,34) = 0.71, p = 0.40; deviation responses: F(1,34) = 0.63, p = 0.43). Overall, patients showed stronger paraspinal reactions than controls (t = 1.79, p = 0.04, one-tailed; as deviation from tonic level: t = 1.96, p < 0.03, one-tailed). The paraspinal responses of CLBP patients did not show less habituation than those of controls (linear trend, raw responses: F(1,34) = 0.004, p = 0.95; deviation responses: F(1,34) = 0.001, p = 0.97).

Summarizing, paraspinal EMG data provide evidence for the response specificity hypothesis: CLBP patients showed stronger paraspinal muscle reactions after the shocks. There was no evidence for hypothesis 1: patients did not habituate more slowly than controls. In order to examine the possibility that paraspinal responses of the CLBP patients are related to back pain characteristics, Pearson product-moment correlations were computed between the mean paraspinal response and back pain rated on a VAS at pre-test, resp. duration of
back pain. No relationships emerged (VAS back pain level: $r = 0.004$; duration of back pain problem: $r = 0.03$).

Discussion

The present study indicates that CLBP patients are not characterized by a general impairment in habituation to painful stimuli. Both at the subjective and the physiological level, CLBP patients and healthy controls displayed a comparable rate of habituation to laboratory pain. There is, however, one exception to this: in both series, HR responses of controls increased, whereas the HR responses of CLBP patients decreased. In general, then, CLBP patients' responses do not seem to habituate to pain more slowly. However, some physiological response, notably FPV and EMG, did not show any reliable habituation trends at all.

Under such conditions, potential differences between groups as to habituation rate might be difficult to detect.

Is it possible that the procedure followed in the present study primarily elicited non-habituating reactions, e.g. strong defensive responses of the type described by Sokolov (1963)? The fact that the subjective ratings and SCRs clearly showed signs of habituation renders this unlikely. As far as FPV is concerned, it is known that this response is a slowly habituating variable, even with innocuous stimuli (Furedy and Arabian, 1979). Finally, in contrast to autonomic responses, somatic responses such as EMG activity, have never been widely used in habituation studies, probably because habituation is difficult to demonstrate in these responses. It is worth noting that EMG activity is assumed to be related more to internal, emotional and cognitive processes than to the processing of external stimuli (Cacioppo and Petty, 1981; see also below).

Evidence was found for the hypothesized specificity in physiological reactions of CLBP patients. CLBP patients showed stronger EMG reactions of the paraspinal muscles. With respect to the frontalis reactions, the findings were not unequivocal: the stronger reactions of the CLBP patients may have been caused by lower tonic level of the patients (note that the raw responses, not corrected for tonic level, were smaller for CLBP patients than for the controls). The paraspinal responses appeared to be stronger in the CLBP group, irrespective of considering tonic level. Therefore, the hypothesized specificity of back muscle responses in CLBP patients was supported. As to the SCRs, only a tendency towards stronger reactions was found during the first series, which disappeared during the second series; FPV responses were not stronger and HR responses were weaker than those of the controls. Thus, CLBP patients seem to show a specific physiological response pattern to external pain: the muscular system shows the strongest reactions, and the cardiac response seems to be attenuated. Stronger paraspinal responses could also have been caused by movements, general arousal or stronger HRRs. However, movements after the shock were not detected, general arousal as indicated by the other physiological variables was not higher, and the HRR due to shock was lower in the patients than in the controls. Taken together, these findings imply that it is unlikely that the stronger paraspinal reactions of the CLBP patients are an artefact.

In line with previous research, the tonic paraspinal EMG levels of the CLBP patients, as measured at the baselines and at the pretrial periods during the shock series, were not higher than those of the healthy controls (see Flor and Turk, 1989, for a review). Only the phasic paraspinal responses to the painful stimulus were stronger in the CLBP group than in the control group. This finding seems to contradict the results obtained by Collins et al. (1982). These authors reported that, compared with normals, CLBP patients display larger activity in frontalis EMG and SC during rest, during stressors and during dynamic movement, and do not display larger paraspinal levels. It should be noted, however, that the study by Collins et al. was based on 11 CLBP patients (vs. 22 in the present study), used a rather moderately aversive stimulus (12 sec cold pressor test at 3°C Celsius vs. a more painful stimulus in the present study), and measured tonic EMG level (vs. measurement of phasic EMG reactions in the present study). As pointed out by Flor and Turk (1989), the measurement of tonic EMG—averaging over larger periods of time—might obscure phasic.

3 The HR signal is also picked up by paraspinal EMG measurement.
responses, which might be more directly related to chronic back pain.

Several explanations can be offered for the observed paraspinous response specificity of CLBP patients. Firstly, this response specificity might be a characteristic of CLBP patients already present before the development of chronic pain, and possibly predisposes to CLBP. Secondly, the response specificity might be related to a disturbance of the physiology in the paraspinous muscles, correlated with the back pain problem. Since there is converging evidence that the tonic paraspinous EMG levels of CLBP patients are not elevated, the present finding of stronger phasic paraspinous responses might indicate that the tonic state of the paraspinous muscles of CLBP patients is characterized by increased activation (not measurable by means of tonic EMG). Such an action readiness might be related to, or caused by, a disturbed biochemical balance in the muscles that eventually causes pain through triggering pain receptors in the muscle. This hypothesis implies that CLBP patients also show larger phasic paraspinous responses to non-pain stimuli (e.g., loud noise). Thirdly, several psychological explanations can be offered. Whereas SCRs are related to arousal, muscular responses are assumed to be related to the emotionality of the subject's response (Cacioppo and Petty, 1981; Frijda, 1986). Thus, the CLBP patients' stronger paraspinous responses (and their frontal-sponses—if we assume that they were not an artefact caused by the baseline differences) might indicate that CLBP patients react more emotionally to pain than normals. The finding that normals showed a stronger cardiac response than CLBP patients might be related to this. According to Lacey (1959) cardiac acceleration augments the cortical inhibition of afferent information transmission.

It has, indeed, been suggested that cardiac acceleration is especially pronounced in low-emotional subjects (Lykken, 1967). Thus, the greater HR acceleration in the control group might reflect a stronger and more efficient inhibition of afferent pain information. It must be admitted that this post-hoc interpretation is complicated by the identical subjective pain intensity ratings of CLBP patients and controls. On the other hand, the greater sensitivity of the CLBP patients to the painful stimulus (as inferred from their lower objective shock levels) is in line with the interpretation that CLBP patients process pain more emotionally. The interpretation of the HR data is, however, complicated by several factors. Firstly, the smaller HR reactions of the CLBP patients were not hypothesized, which, in combination with the relatively large number of physiological parameters, increases the likelihood that it was a chance finding. Secondly, the interpretation is complicated by the lower objective shock level in the CLBP group which—despite employing this level as a covariate—might have reduced the HR acceleration. However that may be, it might be worthwhile to further investigate the speculation that CLBP patients process painful stimuli more emotionally than normals.

Another psychological interpretation of the larger muscular responses and smaller HR acceleration in the CLBP group might be that this response pattern is a manifestation of a helpless attitude towards pain. Since cardiac activity is controlled by both the sympathetic (increasing HR) and the parasympathetic (decreasing HR) systems, and the sympathetic activation appears to be equal in the two groups (which is indicated by comparable SCRs and vasoconstriction), it seems that the CLBP patients reacted to pain with more parasympathetic activation than the normals. According to Obrist (1981), parasympathetic activation is related to passive coping (e.g., undergoing aversive experiences helplessly; the combination of smaller HR acceleration and increased muscular reactions might be considered as a kind of freezing response). It should be noted that several authors have suggested that the emotional disturbances of CLBP patients can be understood as part of learned helplessness resulting from their failure to control their pain (e.g., Turk and Salovey, 1984; Turk and Holzman, 1986; Rudy, Kerns and Turk, 1988; Arntz and Schmidt, 1989). Admittedly, the problems with
interpreting the HR data mentioned above makes this interpretation also rather speculative.

Returning to the issue of paraspinal muscle specificity, its importance becomes even more clear when two issues are considered. First, although it is well-established that subjects react to stress with specific and relatively stable psychophysiological response patterns, these response patterns are rarely dominated by muscle activity in normal subjects (Robinson et al., 1987). However, the present data suggest that paraspinal muscles are the most reactive system in CLBP patients. Second, the lack of habituation of the EMG response appears to be highly relevant. Assuming that CLBP patients generally react more strongly to various aversive experiences as well as to theirown pain producing sensations with paraspinal muscle activity, and assuming that muscle tension produces pain (as is hypothesized by muscle tension/spasm-pain theories of CLBP, cf. Dolce and Raczyński, 1985; Flor and Turk, 1989), the lack of habituation of this response might be an important factor in the maintenance of the problem. If muscle responses do not diminish spontaneously, CLBP patients may be helped by being trained to relax their muscles (which is done in relaxation training and in biofeedback), or by being taught to perceive various experiences as less aversive (which is done in various psychotherapies by increase of perceived control or by reinterpretation of aversive experiences).

Finally, the lack of difference in habituation between CLBP patients and healthy controls should not be generalized too easily. It is possible that differences will be found when other pain stimuli are considered. In the present context, stimuli that are at perception or pain threshold level, stimuli that are prolonged, and stimuli that are more problem-related (e.g. experimentally induced back pain) are highly relevant. It may well be, that differences in habituation can be found with these stimuli, but, as the present data suggest, such differences cannot be attributed to a fundamental habituation deficit of CLBP patients.

References


