Dissociation of sensory and affective dimensions of pain using hypnotic modulation

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Received 30 July 1998; received in revised form 29 January 1999; accepted 23 February 1999

Abstract

Understanding the complex nature of pain perception requires the ability to separately analyze its psychological dimensions and their interaction, and relate them to specific variables and responses. The present study, therefore, attempted to selectively modulate the sensory and affective dimensions of pain, using a cognitive intervention, and to assess the possible relationship between these psychological dimensions of pain and changes in physiological responses to the noxious stimuli. In three experiments, normal subjects trained in hypnosis rated pain intensity and pain unpleasantness produced by a tonic heat-pain stimulus (1-min immersion of the hand in 45.0–47.5\textdegree C water).

Two experiments were designed to test hypnotic suggestions to decrease (Experiment one (Section 2.5.1)), or increase and decrease (Experiment two (Section 2.5.2)) pain affect. Suggestions in Experiment three (Section 2.5.3) were directed towards an increase or decrease in pain sensation. In Experiments one and two (Sections 2.5.1 and 2.5.2), the significant modulation in pain unpleasantness ratings was largely independent of variations in perceived pain intensity. Moreover, in Experiment two (Section 2.5.2), there was a significant correlation between the stimulus-evoked heart-rate increase and ratings of pain unpleasantness, but not of pain intensity, suggesting a direct functional interaction between pain affect and autonomic activation. In Experiment three (Section 2.5.3), suggestions to modulate the sensory aspect of pain produced significant modulation of pain intensity ratings, with secondary changes in pain unpleasantness ratings. Hypnotic susceptibility (Stanford Hypnotic Susceptibility Scale form A) was specifically correlated to pain unpleasantness modulation in Experiment two (Section 2.5.2) and to pain intensity modulation in Experiment three (Section 2.5.3), suggesting that this factor relates to the primary process toward which hypnotic suggestions are directed. The specific pain dimension on which hypnotic suggestions act depends on the content of the instructions and is not a characteristic of hypnosis itself. Results are consistent with a successive-stage model of pain perception (e.g. Wade JB, Dougherty LM, Archer CR, Price DD. Assessing the stages of pain processing: a multivariate analytical approach. Pain 1996;68:157–167) which provides a conceptual framework necessary to study the cerebral representation of pain perception. © 1999 International Association for the Study of Pain. Published by Elsevier Science B.V.

Keywords: Pain sensation; Pain affect; Analgesia; Hypnosis; Hypnotic susceptibility; Heart rate

1. Introduction

Pain is widely recognized as a multi-dimensional experience. Sensory processes refer to the quality, intensity, and spatio-temporal characteristics of the sensation while affective-motivational processes refer to its negative valence and aversiveness (Melzack and Wall, 1965; Melzack and Casey, 1968; Melzack and Wall, 1988; Price, 1988). In addition, cognitive activity modulates these processes while autonomic and behavioral responses are conceived as output processes that may, themselves, produce feedback modulation. This study was designed to develop a simple experimental paradigm using hypnotic modulation of sensory and affective pain processes as a tool to further investigate physiological correlates of these separate dimensions.
1.1. Separating sensory and affective dimensions of pain

Several strategies have been used to separate sensory and affective-motivational aspects of pain perception. Studies using multivariate statistical approaches to reveal the underlying structure in pain descriptors and pain responses lead to a number of explanatory factors or dimensions, two or more of which almost invariably reflect sensory and affective aspects (see the review by Fernandez and Turk, 1992). Correlative studies have shown that different experimental stimuli (Rainville et al., 1992), or clinical situations (Price et al., 1987) produce different combinations of sensory and affective responses. Various pharmacological interventions have also been shown to modulate differentially the sensory or affective aspects of pain (Gracely et al., 1978; 1982; Heft et al., 1984), although conflicting results have been reported (Price et al., 1986). The possible confound associated with stimulus characteristics or clinical populations, as well as the various secondary effects of drugs, limit the power of these approaches in dissociating the specific physiological correlates of sensory and affective pain dimensions. Cognitive interventions, on the other hand, allow an investigation of pain dimensions with stimulus and subject variables reliably controlled; moreover, they are easily reversed and are adaptable to a variety of experimental conditions. Price and Fields (1997) hypothesized that the expectation of either sensory or affective relief could be manipulated easily in placebo and hypnotic analgesia through instructions (see also Price et al., 1980). The paradigm of hypnotic modulation of pain perception offers a promising opportunity to study directly the dissociation of pain dimensions through the manipulation of hypnotic suggestions.

1.2. Hypnotic modulation of pain

Hypnosis has been used as a cognitive intervention to produce analgesia in a variety of settings (Crawford et al., 1993; Barber, 1996; Kropotov et al., 1997). However, the relative effect of hypnotic analgesia on pain dimensions is not clear (Price, 1996). Price and Barber (1987) used a combination of suggestions directed toward both the sensory and the affective dimensions of pain, and showed stronger reduction of pain unpleasantness than pain intensity ratings. However, there was an apparent contradiction in the finding that only the smaller reduction in pain intensity correlated significantly with the hypnotic susceptibility of subjects (Kiernan et al., 1995). The use of hypnotic suggestions that incorporated both aspects of pain perception might have led to these results. Suggestions for a decrease in pain affect might require less hypnotic involvement, and be more readily accepted in low susceptibility subjects, compared with suggestions for reduction or elimination of pain sensation (Price, 1996). This explanation would account for the maximum affective analgesia observed in four out of six low susceptibility subjects of Price and Barber (1987). This high level of affective analgesia might have led to a ceiling effect for hypnotic suggestions of reduced pain unpleasantness. This effect can be prevented in experimental settings, by using suggestions designed to modulate pain in both directions, thereby producing hypo- and hyperalgesia (Meier et al., 1993) or decreases and increases in pain threshold (Arendt-Nielsen et al., 1990).

1.3. Pain affect and pain-evoked autonomic activation

A reliable technique for dissociating sensory and affective pain processes opens the possibility of investigating physiological correlates associated with each of these dimensions. For example, activation of the nociceptive system at various levels of the neuraxis induces a number of autonomic responses, some of which are likely to vary along with pain perception (Jänig, 1995). Pain-evoked sympathetic activity has been shown to decrease with hypnotically induced analgesia (Lenox, 1970; Hilgard et al., 1974). The sympathetic response, although admittedly influenced by the physical intensity of the pain stimulation, was found to be highly dependent on perceptual variables. These studies, however, did not distinguish between sensory and affective processes.

Numerous psychophysiological studies of emotions and affective processes have demonstrated reliable modulation of autonomic activity in response to affectively valenced stimuli, compared to neutral stimuli (e.g. Lang et al., 1993). The distinction between sensory and affective dimensions of pain and the hypothesized relationship of the latter with visceral responses is even present in William James’ classical writing (1894, reprinted in 1994) which suggests that ‘a distinction needs to be made between the primary consciousness of the pain’s intrinsic quality, and the consciousness of its degree of intolerability, which is a secondary affair, seemingly connected with reflex organic irradiation’ (p. 208, note 9, James, 1994). A simple application of these long known phenomena to the study of pain would suggest that specific modulation of the affective dimension of pain perception might be associated with changes in pain-evoked sympathetic activation. The evaluation of hypnotic suggestions specifically directed at modulating pain affect afforded the possibility to test this hypothesis and to evaluate the power of the paradigm in revealing physiological aspects of pain responses associated with pain affect.

1.4. Study objectives

The main objective of the present study is to evaluate the possibility of dissociating sensory and affective dimensions of pain, evoked by controlled experimental stimuli, using specifically designed hypnotic suggestions in normal subjects. The hypothesis that hypnotic modulation of pain acts mainly on one or the other of these dimensions is examined by controlling the specific content of the hypnotic
suggestions, and the hypnotic susceptibility of subjects. Finally, the interaction of autonomic activity with the sensory and affective dimensions of pain is assessed within the paradigm of selectively modulated pain unpleasantness.

2. Methods

Three experiments were conducted sequentially in independent groups of normal subjects. Experiment one (Section 2.5.1) evaluated the effect of hypnotic suggestions for reduced pain affect. Experiment two (Section 2.5.2) compared the effect of suggestions for increased pain affect to suggestions for decreased pain affect. Experiment three (Section 2.5.3) evaluated the effect of hypnotic suggestions for increased and decreased pain sensation.

2.1. Subjects

Subjects were recruited from university students and staff. All subjects in Experiments one and three (Sections 2.5.1 and 2.5.3) had no prior experience with hypnosis. Subjects in Experiment two (Section 2.5.2) were partly recruited from an undergraduate class that had been group-tested for hypnotic susceptibility. In Experiment one (Section 2.5.1), ratings of pain intensity and pain unpleasantness were compared between an experimental group (n = five females + six males, Mean ± SD age = 22.4 ± 6.0) receiving hypnotic suggestions for reduced pain affect and a control non-hypnotized group (n = six males, Mean ± SD age = 20.0 ± 0.8) which was presented the same sequence of noxious stimulation. In Experiment two (Section 2.5.2), a single group of subjects was tested in conditions of hypnotic suggestions for increased and decreased pain affect (n = seven females + 13 males, Mean ± SD age = 27.2 ± 9.8). In Experiment three (Section 2.5.3), a single group of subjects was tested in conditions of hypnotic suggestions for increased and decreased pain sensation (n = 10 females + 12 males, Mean ± SD age = 24.3 ± 4.6). All subjects signed a consent form describing the procedure and affirming their right to withdraw from the experiment without prejudice.

2.2. Stimulation

Subjects sat comfortably in a recliner seat in a quiet dimly lit room. Pain was produced by immersion of the hand in 46.5°C (± 0.1°C) circulating water for a maximum of 1 min or until withdrawal. In Experiments two and three (Sections 2.5.2 and 2.5.3), the temperature of the water was adjusted individually in pre-experimental trials to produce pain intensity ratings > 40/100 (see Section 2.4 below) at a level that the subject could tolerate for the complete 1-min stimulation (45.0–47.5°C). Stimulation alternated between right and left hand in Experiment three (Section 2.5.3). The effect of side stimulated and interactions with this variable were non-significant (all P-values > 0.10), so that this factor was dropped from the analysis, and data were averaged across hands. The minimum inter-stimulus interval for a given hand was 5 min in all experiments.

2.3. Hypnotic induction and suggestions

The instructions for hypnotic induction were taken from the Stanford Hypnotic Susceptibility Scale form A (SHSS-A; French version in Bourassa and Leclerc, 1991). Induction instructions were repeated between the pain stimuli to maintain hypnotic relaxation. Indirect suggestions for the modulation of pain affect (decrease: ↓ AFF; increase: ↑ AFF) and pain sensation (decrease: ↓ SENS; increase: ↑ SENS) were adapted from Kiernan et al. (1995). In the first experiment, suggestions were read before and during the pain stimulation. In the second and third experiments, suggestions for pain modulation were read several times only before the onset of the stimulation, and explicit reference to muscle relaxation was removed (see Appendix). These additional controls were implemented to evaluate whether effects observed in Experiment one (Section 2.5.1) were related to differences in muscle activity or auditory verbal distraction during the stimulation.

2.4. Measures

Subjects gave separate numerical ratings (0–100) of the maximum pain intensity and pain unpleasantness experienced during each stimulation. The distinction between pain intensity and pain unpleasantness was explained before each experiment, and separate visual analog scales (VAS) were presented to help subjects understand this distinction (Price et al., 1983; Rainville et al., 1992). In Experiments two and three (Sections 2.5.2 and 2.5.3), the verbal descriptors ‘burning, pricking, stinging sensation’ were added on the pain intensity scale to emphasize the sensory dimension of heat pain (Morin and Bushnell, 1994). In the hypnosis phase of the experiments, subjects were asked to keep their eyes closed and visualize the scales to give their rating. It was emphasized that subjects should report what they actually felt during the stimulation, whatever was suggested before the stimulation.

Additional measures included hypnotic susceptibility evaluated using sub-tests of the SHSS-A administered before presentation of painful stimuli in all experiments. In all experiments, subjects were classified in subgroups of low, moderate, or high susceptibility. As hypnotic susceptibility appeared to be a critical factor in the pain modulation observed in Experiment one (Section 2.5.1), precise SHSS-A scores were recorded along the remaining course of the ongoing Experiment two (Section 3.2) (recorded in 10 of the 20 subjects), and throughout Experiment three (Section 3.3) (recorded in all subjects), to allow for non-parametric correlation analyses. Heart rate (HR) was recorded at 0.1 Hz for 1 min before (baseline) and during stimulation in Experiment two (Section 2.5.2) (Datalogue-ACCUSAT pulse oxymeter).
2.5. Experimental designs

2.5.1. Experiment one: pain affect reduction

In the first experiment, the effect of suggestions for \( \uparrow \text{AFF} \) was tested in both a within- and between-subject design. Subjects of the experimental group received five stimuli in each of three experimental conditions: (1) restful awake baseline (pre-hypnosis), (2) hypnotic relaxation with suggestions for \( \uparrow \text{AFF} \), and (3) restful awake after hypnosis (post-hypnosis). Control subjects were presented the same time course of pain stimuli, but received neither the hypnotic induction nor suggestions for pain modulation, and instead, waited quietly between blocks.

2.5.2. Experiment two: pain affect modulation

In the second experiment, the pre-hypnosis stimuli were followed by eight stimuli presented under hypnosis; each preceded by suggestions for \( \downarrow \text{AFF} \) or suggestions for \( \uparrow \text{AFF} \) alternating every two trials. The order of suggestion conditions was counterbalanced across subjects.

2.5.3. Experiment three: pain sensation modulation

In the third experiment, eight stimuli were presented under hypnosis in four experimental conditions, each applied in two consecutive trials. Conditions consisted in (1) pre-suggestion, (2) suggestions for \( \downarrow \text{SENS} \), (3) suggestions for \( \uparrow \text{SENS} \), and (4) post-suggestion. The order of suggestion conditions was counterbalanced across subjects.

2.6. Data analysis

Experimental conditions and groups (Experiment one (Section 2.5.1)) were first compared using analysis of variance (ANOVA) computed separately for ratings of pain intensity, pain unpleasantness, and HR averaged over the 1-min recording periods. Significant effects or interactions were investigated using simple effects and contrast analyses. Analyses of covariance (ANCOVA) with intensity ratings as the main factor and unpleasantness ratings as the co-variate were performed to evaluate the residual effect of the suggestion conditions on pain unpleasantness after accounting for pain-intensity-related variance. The effect of hypnotic susceptibility on pain modulation was tested using two-sample \( t \)-tests comparing subjects showing high and low susceptibility (Experiments one to three (Sections 2.5.1–2.5.3)) and Spearman non-parametric correlation analysis using the SHSS-A scores (Experiments two and three (Sections 2.5.2 and 2.5.3)). In these analyses, pain modulation was calculated by subtracting the subject’s average rating in the \( \downarrow \text{AFF} \) condition from that of the pre-hypnosis condition (Experiment one (Section 2.5.1)), and by subtracting the ratings in the \( \downarrow \text{AFF} \) or \( \downarrow \text{SENS} \) conditions from those of the \( \uparrow \text{AFF} \) or \( \downarrow \text{SENS} \) suggestion conditions (Experiments two and three (Sections 2.5.2 and 2.5.3)), respectively. The relationship between unpleasantness ratings and pain-evoked HR responses was tested in Experiment two (Section 2.5.2) using parametric correlation analyses (Pearson) with and without removing subject- and intensity-related variance (ANCOVA). \( P \)-values were adjusted using Bonferroni correction in all multiple \( t \)-tests and correlation analyses.

3. Results

3.1. Experiment one

In the first experiment, a reduction of pain unpleasantness was produced by the hypnotic suggestions (Fig. 1). In the experimental group, pain unpleasantness ratings decreased during the hypnotic suggestions and post-hypnosis compared to those reported in the pre-hypnosis trials (simple effect of experimental conditions, \( F = 22.11, P < 0.001 \); contrasts, \( P \)-values < 0.01). This decrease was only observed in the experimental group (control group: \( F = 0.52, P = 0.60 \)). Hypnotic modulation of pain unpleasantness was not accompanied by significant changes in pain intensity ratings (all \( P \)-values > 0.05). These results indicate that the hypnotic suggestions for decreased pain affect were effective in reducing pain unpleasantness ratings.

A significant effect of stimulus repetition within experimental blocks was found in both pain unpleasantness ratings for \( \uparrow \text{SENS} \), and (4) post-suggestion. The order of suggestion conditions was counterbalanced across subjects.
(F = 11.9, P < 0.001) and pain intensity ratings (F = 5.16, P = 0.001). From trials one to five, pain unpleasantness and intensity ratings decreased by an average of 12.1 (SD = 18.2) and 4.7 (SD = 12.6), respectively. This small but significant decrease in both pain intensity and unpleasantness ratings could reflect habituation to repetitive presentation of the stimulus. However, this effect did not interact with the group or experimental conditions (all P-values > 0.05), and was, therefore, independent of the hypnotic intervention.

Although pain unpleasantness ratings were highly correlated to pain intensity ratings in the experimental group in the pre-hypnosis and hypnotic suggestions blocks (Pearson r = 0.71, P < 0.001), hypnotism-related changes in unpleasantness could not be accounted for by changes in intensity. Residual unpleasantness was computed by removing the variance shared with intensity ratings in a linear regression model (ANCOVA). Comparison of residual unpleasantness in the pre-hypnosis and hypnotic conditions confirmed a significant decrease in pain unpleasantness independent of changes in pain intensity (paired t-test, t = 2.36, P = 0.04). This result further confirms the selectivity of the effect of hypnotic suggestions in modulating pain unpleasantness.

Baseline HR measured before each pain stimulation decreased with hypnosis in Experiment one (Section 2.5.1) as indicated by the significant interaction between groups and experimental conditions (F = 4.43, P = 0.02; see Table 1A). The decrease in baseline HR was significant only in the experimental group (F = 16.5, P < 0.001; control group, F = 0.96, P = 0.42), in both the Hypnosis and post-hypnosis conditions compared to pre-hypnosis (P-values < 0.001). In contrast to the effect observed in baseline measures, HR during the pain tests decreased with experimental conditions in both the experimental and the control groups (F = 22.0, P < 0.001; main effect of group and interaction, P-values > 0.10). HR during pain decreased significantly from the first to the second block (pre- versus Hypnosis/Test: t = 5.20, P < 0.001) and remained at this level in the third block (Hypnosis/Test versus post-: t = 1.43, P = 0.51; see Table 1A). In view of the hypnotism-related changes in HR measured before but not during the pain tests, further analysis of pain-evoked HR increase was contra-indicated, as differences would most likely reflect changes in baseline, independent of pain (see the law of initial value in Sternbach, 1968).

Hypnotic susceptibility was shown to be associated with the modulation of pain affect. The decrease in pain unpleasantness ratings in response to hypnotic suggestions for ↓AFF was stronger in subjects showing moderate to high susceptibility (n = 6; Mean ± SD decrease: 38.3 ± 15.2) compared to those with low hypnotic susceptibility (n = 5; Mean ± SD decrease: 9.5 ± 6.0). A direct comparison of the hypnotism-related changes in the two subgroups

Table 1
Mean (SD) heart rate before (baseline) and during pain tests in Experiment one (A) and two (B)

<table>
<thead>
<tr>
<th></th>
<th>Pre-hypnosis</th>
<th>Hypnosis with ↓AFF</th>
<th>Post-hypnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Experiment one</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline⁴</td>
<td>72.7 (11.1)</td>
<td>63.0 (9.9)</td>
<td>64.5 (11.0)</td>
</tr>
<tr>
<td>Pain⁴</td>
<td>77.4 (11.2)</td>
<td>69.2 (9.8)</td>
<td>67.8 (11.2)</td>
</tr>
<tr>
<td><strong>Control group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>Pre-test</td>
<td>Test w/o Hypnosis</td>
<td>Post-test</td>
</tr>
<tr>
<td>Pain⁵</td>
<td>70.1 (10.6)</td>
<td>68.1 (9.0)</td>
<td>68.3 (7.1)</td>
</tr>
<tr>
<td><strong>B. Experiment two</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Block 1</td>
<td>↓ AFF</td>
<td>↓ AFF</td>
<td>↓ AFF</td>
</tr>
<tr>
<td>Baseline</td>
<td>63.6 (8.9)</td>
<td>63.3 (7.8)</td>
<td>61.1 (7.4)</td>
</tr>
<tr>
<td>Pain</td>
<td>67.9 (10.3)</td>
<td>70.6 (10.1)</td>
<td>65.4 (8.2)</td>
</tr>
<tr>
<td>Pain-baseline⁵</td>
<td>4.4 (3.6)</td>
<td>7.3 (6.0)</td>
<td>4.3 (3.6)</td>
</tr>
<tr>
<td>Block 2</td>
<td>↓ AFF</td>
<td>↓ AFF</td>
<td>↓ AFF</td>
</tr>
<tr>
<td>Baseline</td>
<td>67.9 (10.3)</td>
<td>70.6 (10.1)</td>
<td>65.4 (8.2)</td>
</tr>
<tr>
<td>Pain</td>
<td>67.9 (10.3)</td>
<td>70.6 (10.1)</td>
<td>65.4 (8.2)</td>
</tr>
<tr>
<td>Pain-baseline⁵</td>
<td>4.4 (3.6)</td>
<td>7.3 (6.0)</td>
<td>4.3 (3.6)</td>
</tr>
</tbody>
</table>

⁴ P < 0.05 in the comparisons of the three experimental conditions in A, and in the comparison of ↓ AFF versus ↑ AFF conditions in B.
confirmed this effect ($t$-test: $t = 3.96, P = 0.003$). Hypnotic susceptibility did not modulate changes in pain intensity ratings or HR ($P$-values > 0.10). These results indicate that higher hypnotic susceptibility was selectively associated with a stronger modulation of pain unpleasantness in response to the hypnotic suggestions.

### 3.2. Experiment two

In Experiment two, hypnotic suggestions for pain affect modulation produced a strong change in pain unpleasantness ($\uparrow$ AFF minus $\downarrow$ AFF: Mean ± SEM = 29.4 ± 22.2) that greatly exceeded the effect on pain intensity ratings (10.2 ± 14.5; see Fig. 2). However, both of these effects were significant (unpleasantness: $F = 26.54, P < 0.001$; intensity: $F = 7.95, P = 0.01$). In addition, the modulatory effect of the suggestion conditions on pain unpleasantness increased with the repetition of the suggestions. This effect is illustrated in Fig. 2 by the larger difference in unpleasantness ratings between the $\uparrow$ AFF and $\downarrow$ AFF condition in the second administration of the suggestions ($suggestion \times block interaction: F = 7.84, P = 0.01$). The modulatory effect of the suggestions on pain unpleasantness also increased with successive trials within each block and suggestion condition (not shown; suggestion $\times$ trial interaction: $F = 15.53, P = 0.001$). These interactions suggest that pain unpleasantness ratings increased with successive blocks and trials in the $\uparrow$ AFF suggestion condition, while they decreased with repetition in the $\downarrow$ AFF suggestion condition. The effects of repetition were not significant on pain intensity ratings (all $P$-values > 0.10). These results indicate that the suggestions for modulating pain affect produced a larger effect on pain unpleasantness than on pain intensity and that this effect increased with repetition of the suggestions.

The observation of a significant effect of the suggestions on both pain unpleasantness and intensity introduced the possibility that pain unpleasantness changes could be secondary to pain intensity modulation. A correlation analysis confirmed the significant relationship between unpleasantness and intensity ratings ($Pearson \ r = 0.70, P < 0.001$); nevertheless, unpleasantness modulation could not be fully explained by intensity modulation. This is demonstrated in Fig. 3, showing residual unpleasantness in each suggestion condition after intensity-related variance was removed in a linear regression model (ANCOVA). Residual unpleasantness was significantly higher in the $\uparrow$ AFF, compared with that of the $\downarrow$ AFF condition (paired $t$-test, $t = 4.95, P < 0.001$). These results confirm that the hypnotic suggestions produced a modulation of pain unpleasantness that exceeded, and was largely independent of, the modulation of pain intensity.

Baseline heart rate was stable across suggestion conditions in Experiment two (Section 2.5.2) (main effect of suggestion: $F = 0.39, P = 0.54$; see Table 1B); however, a general decrease in baseline HR was observed with successive blocks ($F = 8.30, P = 0.01$) and trials ($F = 8.67, P = 0.01$), as additional instructions were given to maintain the hypnotic state. As indicated by the lack of a suggestion main-effect, this decrease in baseline HR was independent of the suggestion conditions ($P$-values > 0.10). Analysis of stimulus-contingent HR also showed a decrease with successive blocks ($F = 5.58, P = 0.03$), and a tendency for a similar decrease with trials ($F = 4.32, P = 0.06$). In this case, there was a tendency for HR during pain to be higher in the $\uparrow$ AFF than in the $\downarrow$ AFF condition ($F = 3.06, P = 0.10$). In view of the comparable baseline HR in the two suggestion conditions, further analysis was performed on the pain-evoked HR increase.

The HR increase in response to the pain stimulus was larger in the $\uparrow$ AFF than in the $\downarrow$ AFF condition (see Table 1B). Moreover, the pain-evoked HR increase showed a positive correlation with pain unpleasantness but not with pain intensity ratings (Table 2). The significant correlation between HR response and unpleasantness ratings was still present after variance related to intensity ratings was accounted for (ANCOVA). In addition, the observed effect did not result from inter-subject differences in the range of pain ratings or HR response. On the contrary, removing subject-related variance (ANCOVA) increased the strength

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**Table 2**

<table>
<thead>
<tr>
<th>Pearson-$r$ ($P^a$)</th>
<th>HR increase</th>
<th>HR increase: within-subject$^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensity</td>
<td>0.19 (0.10)</td>
<td>0.21 (0.053)</td>
</tr>
<tr>
<td>Unpleasantness</td>
<td>0.35 (0.0001)</td>
<td>0.42 (0.00001)</td>
</tr>
<tr>
<td>Res. unpleasantness</td>
<td>0.28 (0.03)</td>
<td>0.38 (0.00001)</td>
</tr>
</tbody>
</table>

$^a$ $P$-values are adjusted using Bonferroni correction for six tests.

$^b$ Residual after inter-subject related variance was removed from both factors (ANCOVA).

$^c$ Residual after intensity-related variance is removed from unpleasantness (ANCOVA).
of the relationship between HR increase and pain unpleasantness, indicating that this was a reliable and highly significant within-subject effect (see Table 2). The significant relationship between HR and unpleasantness ratings confirmed the expected effect of pain-affect modulation on pain-evoked autonomic responses.

The influence of hypnotic susceptibility on the modulation of pain unpleasantness in Experiment two (Section 2.5.2) is illustrated in Fig. 4. Pain unpleasantness modulation (↑ AFF minus ↓ AFF) showed a statistical tendency to be stronger in subjects with high (n = 7; Mean difference ± SD = 44.0 ± 29.6) than low-moderate hypnotic susceptibility (n = 13; 21.4 ± 11.8; t = 2.45, P = 0.07). This effect was confirmed in a subset of subjects (n = 10) by a correlation analysis between the modulation of pain unpleasantness and the SHSS-A score (Spearman, r = 0.74, P = 0.04; Fig. 4A). In contrast, the modulation of pain intensity appeared to be largely independent of hypnotic susceptibility (t = 1.71, P = 0.32; r = 0.69, P = 0.08; Fig. 4A), and there was no demonstrable relationship between the modulation of pain-evoked HR and hypnotic susceptibility (r = 0.15, P = 1.00). These results indicate that only pain unpleasantness modulation was reliably related to hypnotic susceptibility in Experiment two (Section 2.5.2).

3.3. Experiment three

In the third experiment, hypnotic suggestions to increase or decrease pain sensation produced strong changes in pain intensity ratings that were largely paralleled by changes in unpleasantness ratings. The effect of experimental conditions was highly significant on both intensity and unpleasantness, as shown in Fig. 5 (intensity: F = 16.90, P < 0.001; unpleasantness: F = 14.54, P < 0.001). Contrast analyses indicated that intensity ratings in each of the suggestion conditions differed from the pre-suggestion baseline (P-values < 0.01). In contrast, although unpleasantness ratings decreased significantly in the ↑ SENS condition, compared to the pre-suggestion baseline (t = -4.20, P < 0.01), the increase was not significant in the ↑ SENS suggestion condition (t = 2.33, P = 0.12). However, the difference between the two suggestion conditions (↑ SENS versus ↓ SENS) was highly significant for both pain intensity and unpleasantness ratings (P-values < 0.01). Differences between pre- and post-suggestion conditions were not significant (P-values > 0.10). These results indicate that the effect of suggestions for pain sensation modulation was not specific to the pain intensity ratings.

The modulation of pain unpleasantness ratings in response to suggestions for increased or decreased pain sensation could be secondary to changes in pain intensity. A high correlation was again observed between unpleasant-
ness and intensity ratings (Pearson $r = 0.85, P < 0.001$). However, in contrast with the second experiment, no significant difference in residual unpleasantness ratings was found between the ↑SENS and ↓SENS suggestion conditions after removing intensity-related variance (ANCOVA, and paired $t$-test on residual unpleasantness, $t = 1.34, P = 0.20$; Fig. 3). This indicates that modulation of pain unpleasantness by hypnotic suggestions could be explained by variations in pain intensity in Experiment three (Section 2.5.3).

Hypnotic susceptibility appeared to influence the effectiveness of suggestions to increase and decrease pain intensity (Fig. 4B). Pain intensity modulation (↑SENS minus ↓SENS) was stronger in the moderate-high ($n = 13$; Mean ± SD = 39.7 ± 20.9) than low susceptibility subjects ($n = 9$; 15.5 ± 18.0; $t = 2.83, P = 0.02$). The correlation coefficient between pain intensity modulation and susceptibility was similar to the one reported by Price and Barber (1987) and approached significance (Spearman, $r = 0.43, P = 0.09$). In contrast, changes in pain unpleasantness (↑SENS minus ↓SENS) were independent of hypnotic susceptibility (moderate-high: 31.9 ± 25.7; low: 29.8 ± 22.5; $t = -0.20, P = 1.00$; $r = -0.06, P = 1.00$). These results suggest that hypnotic susceptibility affected the hypnotic modulation of pain intensity but not pain unpleasantness in Experiment three (Section 2.5.3).

4. Discussion

In each of the three experiments described, hypnotic suggestions directed toward changes in either affective or sensory qualities of pain perception produced a remarkably selective modulation in the subjects’ ratings of these pain dimensions. Suggestions in Experiments one and two (Sections 2.5.1 and 2.5.2), for altering the affective quality of noxious stimuli, resulted in a modulation of pain unpleasantness that exceeded, and was largely independent of, the modulation of pain intensity. Likewise, suggestions in Experiment three (Section 2.5.3), designed to change the sensory aspects of noxious stimuli, produced a significant modulation of pain intensity, but no significant change in residual unpleasantness (after removing the effects of intensity ratings). The influence of hypnotic susceptibility on the modulation of pain perception also appeared to be specific to the suggestions emphasized within the procedure. In Experiments one and two (Sections 2.5.1 and 2.5.2) modulation of pain unpleasantness, but not pain sensation, was statistically related to hypnotic susceptibility, while in Experiment three (Section 2.5.3) the reverse was observed – changes in pain sensation, but not pain affect, were more prominent in subjects with higher hypnotic susceptibility. Finally, pain-evoked HR, measured in Experiment two (Section 2.5.2), was correlated with pain unpleasantness but not with pain intensity ratings. Results are consistent with a successive-stage model of pain processing that distinguishes between sensory and affective dimensions, and suggest an interaction between pain affect and autonomic response. Hypotheses concerning the underlying neurophysiological substrate are suggested.

4.1. Dissociation of pain intensity and pain affect

Results further document the separation of sensory and affective dimensions of pain perception (Price et al., 1987; Fernandez and Turk, 1992; Rainville et al., 1992). Pain modulation was specific to unpleasantness in Experiment one (Section 2.5.1), and was largely independent of changes in pain intensity in Experiment two (Section 2.5.2). These results replicate previous findings indicating a modulation of pain unpleasantness by cognitive variables (Price et al., 1980).

One alternative interpretation for the relative specificity of the reduction in pain unpleasantness ratings observed in Experiments one and two (Sections 2.5.1 and 2.5.2) is that the unpleasantness scale was more sensitive than the intensity scale to changes in pain perception. However, the parallel modulation of pain intensity and unpleasantness in Experiment three (Section 2.5.3) indicates that both scales could adequately reflect changes in pain perception. Moreover, both pain intensity and pain unpleasantness scales revealed habituation to the stimuli in both control and experimental groups in Experiment one (Section 2.5.1). This small habituation effect was independent of the hypnotic intervention and rating scales, and confirms that the experimental paradigm and both scales used in this experiment were sensitive enough to detect small changes in pain perception. Therefore, a difference in the sensitivity of the two scales probably cannot explain the specific and larger reduction in pain unpleasantness produced by the hypnotic suggestions in Experiments one and two (Sections 2.5.1 and 2.5.2), respectively.

4.2. Selectivity of the hypnotic suggestions

The present results differ from those of Kiernan et al. (1995), who failed to demonstrate any differential analgesic effects of hypnotic suggestions directed at either pain sensation or pain affect. However, the inclusion of words with positive affective valence in their sensory suggestions indicates an overlap between the suggestion conditions that might have contributed to the lack of selectivity of the analgesic effect [e.g. ‘and you are just, all mind, floating freely (...) like a cloud in a beautiful sky (...) you might enjoy letting that ankle become more and more numb.’; Kiernan et al., 1995, p. 41]. In the present study, a more strict separation of affective and sensory suggestions was maintained throughout the three experiments (see Appendix), which may account for the higher selectivity of the psychophysical effects observed. In any case, our results confirm the propositions that hypnotic suggestions can act selectively on the processing of pain effect and that modulation...
tion of pain sensation produces a parallel modulation in pain affect.

Other lines of evidence further support our interpretation that suggestions used in the present study acted primarily on either affective or sensory processes. First, the effectiveness of the suggestions in modulating pain unpleasantness increased with successive blocks and trials as suggestions were repeated in Experiment two (Section 2.5.2). The specificity of this repetition effect to unpleasantness ratings is consistent with a predominant action on affective processes. Second, the independent measure of hypnotic susceptibility (SHSS-A) was associated with pain unpleasantness in Experiments one and two (Sections 2.5.1 and 2.5.2) and intensity modulation in Experiment three (Section 2.5.3). A previous study described hypnotic susceptibility as a good predictor of hypnotic sensory analgesia but a poor predictor of affective analgesia (Price and Barber, 1987). Consistent with those findings, we observed in Experiment three (Section 2.5.3) that only pain intensity modulation was related to hypnotic susceptibility. However, stronger modulation of pain unpleasantness was observed in subjects showing higher hypnotic susceptibility in both Experiments one and two (Sections 2.5.1 and 2.5.2) where suggestions targeted pain affect. Considering that hypnotic susceptibility is likely to be more reliably related to changes in the primary process to which suggestions are directed, our results indicate that the cognitive mechanisms involved in hypnotic modulation acted predominantly on the affective dimension of pain perception in Experiments one and two (Sections 2.5.1 and 2.5.2) and on the sensory dimension in Experiment three (Section 2.5.3). The stronger relationship previously observed between hypnotic susceptibility and pain intensity is not a characteristic specific to this pain dimension but probably reflects the specific content of the suggestions for pain modulation.

The central role of instructions in specifically directing expectations of pain modulation toward either the sensory or the affective dimension has been previously emphasized (Price and Fields, 1997). The present results support the importance of suggestion design in the specificity of hypnotic analgesia. However, the issue of demand characteristics and role enactment in hypnosis (e.g. Coe and Sarbin, 1977) offers an alternative explanation for the effects reported in the present study, as well as for their selectivity. At the extreme of this view, hypnotic-related changes assessed by verbal measures might reflect the subjects’ willingness to conform to the perceived task demands and may not represent an actual change in pain perception. Although this position has been seriously criticized (e.g. Hilgard, 1975; Price, 1996), the specific reference to one or the other dimension of pain was, at least, implicit within the hypnotic suggestions, and could have led the subjects to produce what they perceived to be the desired responses. Two observations argue against this interpretation. First, the persistence of the specific pain unpleasantness reduction in the post-hypnotic phase of Experiment one (Section 2.5.1) is contrary to the expected return of the unpleasantness ratings to the pre-hypnosis baseline. The possibility that subjects have spontaneously used their newly learned ability to reduce pain unpleasantness appears more likely. Secondly, the significant modulation of pain-evoked heart rate and its specific correlation with unpleasantness ratings in Experiment two (Section 2.5.2) indicate that pain affect varied consistently with the physiological response to the painful stimuli. These results, along with other neurophysiological correlates of hypnotic analgesia discussed below, support our interpretation that hypnosis-related changes in pain ratings reflect changes in pain processing and not simply demand characteristics of the experimental setting. The relative selectivity of the modulation observed has strong implications on psychological models of pain.

4.3. Pain unpleasantness and autonomic response

Many psychophysiological observations support a direct relationship between pain perception and the autonomic response. Both cardiovascular response and pain ratings evoked by experimental ischemia have been shown to decrease significantly following hypnotic suggestions of analgesia (Lenox, 1970). Likewise, Hilgard et al. (1974) reported lower pain ratings and smaller HR rise in response to the cold-pressor test during hypnotic analgesia. In the same study, Hilgard et al. also reported a comparable increase in HR in an awake state when the pain test was applied and when the subjects hallucinated pain following hypnotic induction and suggestions. This suggested that HR response is not merely a consequence of physical stress and is closely related to perceived pain. Möttner et al. (1990) further demonstrated that early components of the heart rate response evoked during phasic heat pain correlated specifically with the physical intensity of the stimulus, while the late component correlated with the perceived intensity and not the physical intensity of the stimulus. Taken together these results strongly support a functional interaction between pain perception and autonomic activation that can be separated from the physical characteristics of the noxious stimulus.

The results of Experiment two (Section 2.5.2) further demonstrate a significant correlation between heart-rate response and pain unpleasantness, independent of perceived pain intensity. This finding strongly emphasizes both the distinction between pain dimensions, and the important relationship between pain affect and sympathetic activation. In Experiment two (Section 2.5.2), cognitive processes called upon by the hypnotic suggestions might have acted directly on affective-motivational processes, which in turn could have produced a modulatory effect on the autonomic outflow. Alternatively, the observed modulation of pain-evoked sympathetic activation might be part of, and not simply the result of, the modulation of pain affect (Jänig, 1995). Results of the current and previous studies impose a distinction between the interaction of nociceptive processes
with the sympathetic nervous system, perception of the sensory properties of pain evoked by the noxious stimulus, and the affect associated with pain perception.

4.4. Implications for models of pain processing

The present study provides experimental support for the successive-stage model of pain processing developed from clinical observations by Wade et al. (1996). A similar model is illustrated in Fig. 6 that summarizes the hypothesized processes involved in the present study. Hypnotic suggestions for altering pain sensation resulted in a comparable modulation of both pain intensity and pain unpleasantness (see Fig. 6A), while suggestions for changing pain affect were more selective for pain unpleasantness (see Fig. 6B). Although pain unpleasantness ratings were correlated with pain intensity in all experiments, analyses of covariance in Experiments one and two (Sections 2.5.1 and 2.5.2) indicate that hypnotic modulation of unpleasantness could not be fully explained by variations in pain intensity. In contrast, results from Experiment three (Section 2.5.3) show that changes in pain unpleasantness closely paralleled the modulation of pain intensity, in response to suggestions directed at the sensory qualities of pain sensation. This combination of results is consistent with the model proposed by Wade et al. (1996) in which modulation of pain sensation greatly influences pain affect while the reverse is not necessarily true.

Hypnotic suggestions for modulation of pain affect produced some changes in pain intensity in Experiment two (Section 2.5.2). One might argue that modulation of down-stream affective processes might have exerted modulatory feedback on up-stream sensory processes, leading to the observed secondary changes in pain intensity. However, following this reasoning, changes in pain intensity would also have been observed in Experiment one (Section 2.5.1) in response to similar suggestions directed toward pain affect, and this was not the case. This possibility would also be in contradiction with the model of Wade et al. (1996), in which the pain-affect stage does not exert recursive effects upon the pain-sensation stage of processing. Alternatively, we propose that the modulation of pain intensity in Experiment two (Section 2.5.2) occurred by partial generalization of the suggestions to the sensory dimension of pain perception (see Fig. 6b). In this case, although suggestions were primarily directed toward the affective dimension of pain, they might have acted directly on both dimensions. This interpretation appears more likely, especially in view of procedural differences between Experiments one and two (Sections 2.5.1 and 2.5.2). In Experiment one (Section 2.5.1) suggestions given throughout the stimulation might have acted more specifically on affective processes (see Fig. 6B). In contrast, in Experiment two (Section 2.5.2) suggestions were given only prior to the stimulation. The working memory load, imposed by this latter procedure to maintain the suggestion content active throughout the duration of each tonic stimulation, may have facilitated the partial generalization of the suggestions to both the sensory dimensions of pain (see Fig. 6b). The possible increase in attentional demand associated with the memory load in Experiment two (Section 2.5.2) might also explain this effect, since both pain dimensions are modulated by attentional manipulation (e.g. Miron et al., 1989). The increasing specificity of the suggestions on pain unpleasantness observed with successive blocks and trials is consistent with a gradual decrease in these cognitive loads with repetition.
Limits of the proposed model of pain processing need to be recognized. First, this model is intended to describe the conceptual inter-relationships between pain intensity, pain affect, and cognitive processes, that might apply both to clinical and experimental pain. However, other aspects of pain sensation, such as location, quality, and spatio-temporal characteristics, not modulated in the present experiment, may relate differentially to pain affect (e.g. see Price et al., 1987; Rainville et al., 1992). Second, although the expression ‘successive stage’ implies direction of causation, this relationship may not be reflected in a temporal succession of pain processes. Examination of possible cerebral correlates of these processes (see Section 4.5) illustrates the conceptual puzzle of integrating psychological and neurophysiological models of pain processing.

4.5. Neurophysiological considerations

Numerous lines of evidence support a partial segregation between cerebral regions involved in sensory and affective aspects of pain perception. Human brain imaging studies in normal subjects have shown that the primary and secondary somatosensory cortices (S1 and S2), the insular cortex, and the anterior cingulate cortex (ACC) are reliably activated in response to experimental pain (Jones et al., 1991; Talbot et al., 1991; Casey et al., 1994; 1996; Coghill et al., 1994; Craig et al., 1996; Hsieh et al., 1996; Aziz et al., 1997). In a recent series of brain imaging studies, we showed a significant modulation of ACC activity in response to hypnotic suggestions for pain affect modulation developed in the present Experiment two (Section 2.5.2) (Rainville et al., 1997). The absence of a parallel modulation of S1 activation was taken as evidence that this structure may have less direct involvement in pain affect. In contrast, suggestions directed at the sensory dimension of pain developed in Experiment three (Section 2.5.3) produced the most reliable changes in S1 activation (Hofbauer et al., 1998). These results support the hypothesized involvements of the ACC and S1 in affective and sensory aspects of pain perception, respectively. In the simplest case, basic principles of anatomo-functional localization could suggest a cerebral organization of pain processes with separate cortical regions possibly involved in strictly different aspects of pain perception. In this unlikely case, one might hypothesize that regions involved in affective processes, such as the ACC, are serially driven by regions involved in sensory processes, such as S1 – a relationship that mirrors the psychological model presented in Fig. 6. The observation of a direct spino-thalamo-cortical nociceptive pathway ascending to the ACC (see Craig, 1996) indicates that this simplistic account of the cerebral representation of pain is not adequate. Brain imaging results should not be taken as evidence of a simple one-to-one correspondence between pain processes and cortical structures.

Involvement of the ACC in affective processes does not preclude its participation in sensory-discriminative processes, as suggested by recent lesion studies (Davis et al., 1994; Talbot et al., 1995). Such a non-serial functional arrangement of cerebral regions involved in pain processing implies multiple representations of intensity-related information (also see Bushnell and Duncan, 1989) and may underlie the usual high correspondence between pain intensity and unpleasantness. Similarly, S1 involvement in sensory aspects of pain processing does not preclude a contribution of this area to affective processes; however, such involvement remains to be demonstrated. Although it is unlikely that all cerebral regions activated during pain play the same role in this complex experience, the functional segregation between regions involved in sensory and affective dimensions of pain is likely to be only partial.

Development of comprehensive psychological models is a prerequisite for better understanding the cerebral representation of pain. In the present study, hypnosis has been used successfully to study basic mechanisms involved in pain perception and their functional inter-relationships. The differential modulation of pain intensity and unpleasantness demonstrates the usefulness and validity of this technique and lends support to a successive-stage model of pain processing. Further research on the anatomical and functional pattern of pain-related cerebral activation is needed to further define the neural mechanisms underlying such interactions between psychological pain processes.

Acknowledgements

We wish to thank Donald D. Price for his insightful comments provided throughout the course of the project. This study was supported by the Medical Research Council (MRC) of Canada. Dr. Rainville has been supported by the MRC of Canada and by the Quebec FCAR.

Appendix A. Suggestions for pain affect

A.1. Suggestions for decreased pain affect ( AFF; Experiment one (Section 2.5.2))

‘During the stimulation, a sensation of well-being will just sweep through all your hand and arm… Although you will continue to experience normal sensation, your experience will seem surprisingly more agreeable… surprisingly more comfortable… surprisingly more restful than you might have expected… When you feel the stimulation, you can be mindful of its intensity… mindful of its burning, pricking, sting quality… but mostly, you will feel particularly comfortable… Almost as if, when you feel the onset of the stimulus, you can feel an onset of well-being quickly spreading into your hand… your arm… into your whole body… and all through your experience’.

[French version: `Au cours de la stimulation, tu auras l'impression que ta main et ton bras s'abandonnent de...']
plus en plus à une sensation de bien-être… Malgré que tu continuerais à avoir des sensations tout à fait normales, ce que tu ressentiras sera étonnamment plus plaisant… étonnamment plus confortable… étonnamment plus reposant que ce que tu envisageais… Lorsque tu ressentiras la stimulation, tu peux être conscient de son intensité, conscient de ses qualités brûlante, picotante, cuisante… mais surtout, tu te sentiras particulièrement confortable… Comme si l’application de la stimulation semblait déclencher une sensation de bien-être croissant qui se propage rapidement dans ton bras, dans tout ton corps, et à travers tout ce que tu ressens’

B.1. Suggestions for increased pain affect (↑ AFF; Experiment two (Section 2.5.2))

‘During the stimulation, you will be fully aware of the sensation of discomfort sweeping through all your hand and arm… Although you will continue to experience normal sensation, your experience will seem surprisingly more unpleasant… surprisingly more uncomfortable… surprisingly more disturbing than you might have expected… When you feel the stimulation, you can be mindful of its intensity… mindful of its burning, pricking, stinging quality… but mostly, you will feel particularly uncomfortable… Almost as if, when you feel the onset of the stimulus, you can feel an onset of discomfort quickly spreading into your hand… your arm… into your whole body… and all through your experience’.

[French version: ‘Au cours de la stimulation, tu seras pleinement conscient du malaise produit par la stimulation dans ta main et ton bras… Malgré que tu continuieras à avoir des sensations tout à fait normales, ce que tu ressentiras sera étonnamment plus déplaçant… étonnamment plus inconfortable… étonnamment plus dérangeant que ce que tu envisageais… Lorsque tu ressentiras la stimulation, tu seras conscient de son intensité, conscient de ses qualités brûlante, picotante, cuisante… mais surtout, tu te sentiras particulièrement inconfortable… Comme si l’application de la stimulation semblait déclencher un malaise croissant qui se propage rapidement dans ton bras, dans tout ton corps, et à travers tout ce que tu ressens’

C.1. Suggestions to decrease pain sensation (↓ SENS; Experiment three (Section 2.5.3); all English-speaking subjects)

‘When you feel your hand in the water bath, you may be surprised to notice how much less intense the sensation is than you might have expected it to be, how it tends to feel only warm… Almost as if, when you feel the stimulation of the water, all you continue to experience is that warmth… As time passes, you can turn down the dial of your sensation, much like turning down the volume dial on a stereo. Perhaps you now feel a tingling… or numbness… which is becoming even more numb or tingling as time passes… and you can feel that feeling of warmth… or tingling… or numbness spread throughout your hand… and it is even possible that you won’t feel anything in your hand at all, almost as if you have forgotten to feel your hand altogether… as time goes on and your hand stays immersed in the water you are becoming aware of how much less intense the sensation is than you expected it to be’.

D.1. Suggestions to increase pain sensation (↑ SENS; Experiment three (Section 2.5.3); all English-speaking subjects)

‘When you feel your hand enter the water bath, you may be surprised to notice how much more intense the sensation is than you might have expected it to be… how the sensation is so much more burning, stinging, or aching than you might have expected it to be… Almost as if, when you feel the stimulation rise in intensity, it continues to rise to levels higher than you expected. As time passes, you can turn up the dial of your own sensation, much like turning up the volume dial on a stereo. The presence of your hand in the water can remind you just how intense the sensation can be, how burning, aching, or stinging it can be,… and you can feel the burning, aching, and stinging sensations spread throughout your hand… as time goes on and your hand stays immersed in the water you are becoming aware of how intense the sensation is. You are becoming aware of the burning, aching and stinging of the water’.

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