On the Interaction of Hypnotizability and Negative Affect in Chronic Pain

Implications for the Somatization of Trauma

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The high risk model of threat perception predicts that high hypnotizability is a risk factor for trauma-related somatization. It is hypothesized that high hypnotizability can increase experimentally induced threat or negative affect, as measured by skin conductance level, in a linear or dose-response manner. This hypothesized interaction of hypnotic ability and negative affect was found in a consecutive series of 118 adult patients with chronic pain symptoms. Larger increases in skin conductance levels during cognitive threat were significantly related to higher levels of hypnotizability. In addition, individuals with high hypnotizability retained higher skin conductance levels than individuals with low hypnotizability after stress. The clinical implications of the interaction of hypnotizability and negative affect during threat perception and delayed recovery from threat perception are discussed in terms of cognitive mechanisms in the etiology and therapy of trauma-related dissociative disorders.


The high risk model of threat perception (HRMTP; Wickramasekera, 1979, 1988, 1993, 1995) is a multidimensional model of the mechanisms that underlie the perception of threat that can lead to psychopathology and pathophysiology. The perception of threat or trauma causes fear, which can increase pain perception by increasing myotonia, sympathetic activation (Osterweiss et al., 1987), and electrodermal response (Boucsein, 1992). High hypnotic ability is one of nine risk factors that is predicted by the HRMTP to a) amplify somatic symptoms or b) transduce threat perception into somatic symptoms.

The HRMTP predicts that people who have a high ability to be hypnotized (hypnotizability) will be at risk of stress- or threat-related psychological and somatic symptoms because of their hypersensitivity to the perception of threat (Wickramasekera, 1979, 1988, 1993, 1995). Wickramasekera (1979, 1988) found greater chronic pain intensity related to higher hypnotizability outside of hypnosis. De Benodittis et al. (1989) found that normal highly hypnotizable college students in the nonhypnotized wakening state were more sensitive to both acute sensory pain and distress than a matched control group. Andreychuk and Skrivar (1975) found a significant positive correlation between pretherapy migraine pain severity and high hypnotizability. Stam et al. (1986) and Remler (1990) found that temporomandibular joint and other chronic pain symptoms were associated with higher hypnotizability. Shertzer and Lookingbill (1987) reported a positive relationship between the intensity of chronic urticaria and those with high hypnotizability ("highs"). John et al. (1983) and Kelley (1984) reported a positive relationship between highs and clinical phobias. Highs are also at greater risk of negative moods (Crowson et al., 1991; Velten, 1968). Stutman and Bliss (1985) and Spiegel et al. (1988) found a positive relationship between highs and the severity and frequency of post-traumatic stress disorder. Belicki and Belicki (1986) found a positive relationship between highs and predisposition to nightmares. Wickramasekera (1993) reported a relationship between highs and EEG-defined insomnia (with pathophysiology excluded e.g., apnea, etc.). Pettinati et al. (1990) reported relationships among substance abuse, bulimia (Pettinati et al., 1985), and high hypnotizability. Apfel et al. (1986) reported a relationship between highs and the severity of nausea and vomiting during pregnancy. It is hypothesized that acute and chronic pain, phobias, negative moods, posttraumatic stress disorder symptoms, nightmares, insomnia, substance abuse, bulimia, and nausea may be driven by the interaction between trauma-induced unconscious or conscious a) high negative affect (Watson and Tellegen, 1985) and b) high hypnotizability (Wickramasekera, 1979, 1988, 1993, 1994a, 1994b, 1995). It is known that highs can block (posthypnotic amnesia) true memories from consciousness (Kihlstrom, 1985). Laurence and Perry (1983) and Dywan and Bowers (1983) reported that high hypnotizability was associated with hypnotically

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installed and hypnotically increased false memories in normal college students. People high in hypnotizability may be at risk of a variety of psychological and somatic symptoms during threat perception or trauma. This vulnerability to symptoms in highs may be caused by peculiarities in their perception and memory of trauma. It is hypothesized that sympathetic hyperreactivity as measured by higher skin conductance level (SCL) may provide a physiological index of unconscious or conscious memory and perception of threat or trauma (Bentin and Moscovitch, 1990; Ohman and Soares, 1994; Wickramasekera, 1988, 1993).

Hypnotizability

Hypnotizability can be operationally defined and quantified in terms of a person’s score on a behavioral test like the Harvard (Shor and Orne, 1962). High hypnotizability can be defined as a mode of information processing in which sequential critical-analytical cognition can be suspended and peripheral attention restricted to the point that profound changes that can have behavioral and biological consequences seem to involuntarily occur in perception, mood, and memory (Wickramasekera, 1979, 1988, 1993). Because hypnotizability is a normally distributed, stable ($r = .71$ over 25 years) individual difference variable (Piccione et al., 1989) that may be partly genetically based (Morgan et al., 1970), it probably existed premorbidly or before the onset of chronic clinical symptoms.

Mechanisms

During verbal information processing, highs seem to perceive events occurring involuntarily or outside of self-control both during hypnosis and outside of hypnosis (Bowers, 1982; Dixon and Laurence, 1992; Dixon et al., 1990). The perception of intrusive, uncontrolable, and unpredictable memories can amplify pain and fear (Mineka and Kihlstrom, 1978). Highs’ perception of sensory or motor events “occurring by themselves” can increase fear, amplify memories of trauma, and drive SCL (Wickramasekera, 1979, 1988, 1993). Vivid diffuse memories in highs can also cause surplus empathy and interpersonal boundary problems that drive up SCL (Wickramasekera, 1988, 1995).

A second hypothesized mechanism of risk in highs is the capacity to inadvertently block from consciousness the perception and memory of pain acquired during a civilian (e.g., rape), auto, military, or industrial trauma (Wickramasekera 1979, 1984, 1988, 1993, 1994a, 1994b). “But out of mind may not mean out of body” (p. 613; Wickramasekera, 1993; see also Wickramasekera, 1979, 1988, 1993, 1994a, 1994b). For example, laboratory studies have shown that some autonomic indicators (blood pressure, SCL) continue to register sensory pain reactivity blocked from consciousness even during verbally reported hypnotic analgesia (Hilgard and Hilgard, 1975). Unconscious or implicit (Kihlstrom, 1987) threatening memories acquired during trauma are transduced into chronic sympathetic activation (SCL) and somatization (Wickramasekera, 1979, 1993, 1988, 1994a, 1994b).

A third mechanism through which highs are at risk of stress-related disorders is their hypothesized propensity to “surplus pattern recognition or to see meaning in a set of data that seems randomly distributed to others” (p. 594; Wickramasekera, 1993; see also 1979, 1988, 1993, 1995). If a person has both high hypnotic ability and high orthogonal trait negative affectivity (Watson and Clark, 1984), the bulk of these meanings are likely to be threatening (Wickramasekera, 1988, 1993, 1994a). High negative affectivity or neuroticism is another risk factor in the HRMTP. Hence, an interaction between hypnotizability and negative affect is predicted to amplify pain perception and drive up SCL.

The perception of threat to physical (tissue damage, etc.) or psychological well-being can trigger negative affect (threat perception) and the hypothalamic-pituitary-adrenal axis (Mason, 1968). Davis (1992) suggests that prefrontal cortex and amygdala are implicated in memory of fear and anxiety. Boucsein’s (1992) review of SCL is consistent with the proposal of Davis (1992). SCL, more than other measures (e.g., frontal EMG, heart rate, etc.) in psychophysiology, normally seems to be involuntary and activated only by excitatory sympathetic nerve impulses (Boucsein, 1992). Hence, SCL is an objective but nonspecific measure of threat perception. Higher levels of SCL have been associated with superior verbal learning and vigilance in signal detection (Boucsein, 1992) and more severe clinical anxiety (Lader and Wing, 1966). Because of SCL’s increase in acute pain (Naifeh et al., 1983) and anatomical proximity to the afferent pain pathways (Boucsein 1992), it may be the best single psychophysiological measure of pain-relevant negative affect.

Hypothesis

It is hypothesized that highs will show a higher level of negative affect (threat) as measured by SCL during experimentally induced cognitive stress (mental arithmetic) than patients with low or moderate hypnotizability. SCL is hypothesized to show a linear relationship to hypnotizability during cognitive threat.
Methods

Subjects

All the subjects were patients \((N = 118)\) suffering from chronic pain symptoms (e.g., muscular and vascular headaches, phantom limb pain, chronic low back pain, temporomandibular joint syndrome, etc.) and were referred by a primary care physician or a specialist (e.g., neurologist, neurosurgeon, urologist, obstetrician/gynecologist, etc.) to the Psychophysiological Stress Disorders Laboratory. These patients were referred because their symptoms were unresponsive to standard medical management (drugs, trigger point injections, physical therapy, anesthetic blocks, surgery, etc.). The mean \((\pm SD)\) age of the men was \(37.8 \pm 2.5\) years. The mean age of the women was \(37.2 \pm 2.5\) years. At entry, all of these patients were taking psychotropic or analgesic medications but none were on anticholinergic medication. A prior study of a more psychiatrically disturbed group of chronic pain patients on psychotropic and analgesic medications showed that only anticholinergic drugs significantly alter SCL (Carr et al., 1985). Careful chart review indicated that medication usage was not systematically related to either sex or hypnotic ability. All measures of hypnotizability and SCL testing were performed during our baseline assessment before any therapy.

In this study, a consecutive series of 72 women and 46 men were tested with the Harvard Group Scale of Hypnotic Susceptibility Form A, (Shor and Orne, 1962) in small groups of two or three patients. Subjects were divided into high (Harvard scores 9 to 12), moderate (Harvard scores 6 to 8), and low (Harvard scores 0 to 5) hypnotizability groups for hypothesis testing. The Harvard test was administered by a research assistant blind to the patient’s SCL status. The Harvard is a standardized behavioral test of hypnotizability on which the scores range from a low of 0 to a high of 12. It is a psychometric measure of high established reliability and validity (Kihlstrom, 1985).

A routine part of the testing on the HRMTP includes a psychophysiological stress profile (Wickramasekera, 1988) in which SCL is monitored under baseline, cognitive stress, and recovery from stress conditions. In the psychophysiological laboratory, patients are oriented, instrumented, and habituated for approximately 10 minutes before testing. A 4-minute eyes open (EO) baseline condition is run with the monitoring of SCL, followed by a 4-minute eyes closed (EC) condition, which is followed by a 4-minute stress (S) period and a recovery period (4 minutes of EO and 4 minutes of EC conditions).

During the stress period, the patients are told they are taking an IQ test and that they should respond to the following standardized mental arithmetic problems as accurately and quickly as possible while their speed of response is conspicuously measured by a large electronic stop watch. Regardless of their performance, they are urged twice to be more accurate and faster. This cognitive social harassment has been found to be adequate stimuli to elicit distress and cognitive threat (Blascovich and Katin, 1993; Wickramasekera, 1988).

SCL was measured with the J & J computer system I-330, which monitors SCL and performs basic data reduction on the SCL signal. SCL was recorded using silver/silver chloride disc electrodes, 12 mm in diameter, attached with electrolyte to the ventral surface of the distal phalanges of the second and third fingers of the subject’s left hand and held in place by Velcro straps following the procedures described by Fowles et al. (1981). The SCL physiological signal is conditioned by J & J Enterprises module and acquired using the J & J I-330 interface with a CompuAdd 333T computer operating under the J & J USE software system. The application is programmed so that each stored data point is an average of 4 seconds of activity, which provides 60 averages for each 4-minute period of the cognitive stress profile. A total of 300 averages are derived for the entire (EO, EC, S, EO, EC) psychophysiological stress profile. Averages of activity for each period or condition are also derived.

Statistical Analysis

The relationship between change in SCL from baseline to stress and hypnotizability was first analyzed by linear regression. Change in SCL from baseline to stress was analyzed by regressing the stress SCL minus baseline SCL difference on hypnotizability, and also by regressing stress SCL on hypnotizability, adjusting for baseline SCL. The analyses were performed with EO as the baseline and again with EC as the baseline.

Hypnotizability was then classified into high (Harvard scores, 9 to 12), moderate (Harvard scores 6 to 8), and low (Harvard scores 0 to 5) groups, and a \(3 \times 2 \times 5\) (hypnotizability \(\times\) sex \(\times\) stress profile condition) repeated measures analysis of variance (ANOVA) was performed to analyze change in SCL over all stress conditions as a function of hypnotizability and gender. Hypnotizability and gender were between-subjects factors, and stress profile condition was the within-subject factor. Means at each stress profile condition were compared with Tukey’s honest significant difference pairwise multiple comparison test.
Results

There was a small but significant positive correlation between change in SCL from baseline to stress and hypnotizability. With EO as the baseline, the slope of the regression line was .3173 ($r = .238$, $p = .0095$), and with EC as the baseline, the slope was .3805 ($r = .274$, $p = .0027$). Figure 1 plots the SCL change with hypnotizability for the EO baseline. The plot with EC as the baseline was similar and is not shown.

Adjusting for baseline SCL produced similar results. With EO as the baseline, the slope of the regression line was .2848 ($r = .218$, $p = .0175$), and with EC as the baseline, the slope was .3500 ($r = .262$, $p = .0041$).

Results of the ANOVA are presented in Table 1. Because the variance-covariance matrix of the stress profile conditions was not compound symmetric (equal variances and equal covariances), a reduced $df$ $p$ value was also computed. The significant hypnotic $\times$ condition interaction indicated that the pattern of change in SCL over the stress profile conditions was not the same for the levels of hypnotizability. Table 2 presents the means and standard deviations for each hypnotizability group at each condition. Figure 2 plots these means and their 95% confidence intervals.

To determine the nature of the interaction, analyses were made for three segments of SCL change: 1) from EO to EC (baseline), 2) from EC (1) to S, and 3) from S to EO (2) to EC (2). For the EO (1) to EC (1) segment, there was no significant difference between any of the hypnotizability groups.

For the EC (1) to S segment, there was a significant hypnotizability $\times$ condition interaction ($p = .0065$), which indicates a significant difference in the amount of increase among the hypnotizability groups. All three groups had significant increases in SCL ($p < .01$ for all), but the low group had a larger increase than either the moderate or high groups. This is reflected in the steeper rise in SCL from EC (1) to S for the high group (Figure 1). Mean SCL at S was significantly greater for the high hypnotizability group than for the low group ($p < .05$).

The analysis for the third segment resulted in significant main effects for hypnotizability ($p = .0349$) and condition ($p < .0001$), but a non-significant interaction ($p = .1682$). All three groups had significant decreases in SCL from S to EO (2) to EC (2),...
and the amount of decrease was similar for the three groups, as indicated by the nonsignificant interaction. The hypnotizability main effect indicates an overall elevation of the high group. Mean SCL was greater for the high group than for the low group at S, EO (2) \( p < .05 \), and at EC (2) \( p < .06 \).

**Discussion**

The significant hypnotizability \( \times \) S condition interaction indicates that the pattern of change in SCL over the S conditions was not the same for low, moderate, and high hypnotizable subjects. Follow-up analyses for the EC (1) to S segment indicated that all three groups had a significant increase in SCL but that the high hypnotizability group had a significantly greater increase in SCL than the other two groups \( p = .0065 \). Thus, it can be concluded that all patients were sensitive to cognitive stress, but high hypnotizable patients were hypersensitive. This supports the hypothesis of an interaction between hypnotizability and negative affect during threat perception. This conclusion is supported by both regression analyses and the ANOVA, in which hypnotizability was grouped into high, moderate, and low categories. The ANOVA also indicated that highs retained higher SCL than low hypnotizable individuals after stress. Put another way, the results indicate that all three groups had a significant increase in SCL but that the highs had a significantly greater increase in SCL than the other two groups \( p = .0065 \). Thus, it can be concluded that all patients were sensitive to cognitive stress, but high hypnotizable patients were hypersensitive. This supports the important theoretically driven hypothesis of an interaction between hypnotizability and negative affect during trauma. This conclusion is supported by both regression analyses and the ANOVA. Put another way, the results suggest a linear or dose-response relationship between hypnotizability and SCL during threat (negative affect) and delay in recovery from threat.

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>( F )</th>
<th>( p )</th>
<th>Adjusted ( p )</th>
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<tbody>
<tr>
<td>Condition</td>
<td>1164.09</td>
<td>4</td>
<td>291.02</td>
<td>61.98</td>
<td>&lt;.0001</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Sex × condition</td>
<td>15.00</td>
<td>4</td>
<td>3.75</td>
<td>.80</td>
<td>.5265</td>
<td>.4539</td>
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<tr>
<td>Hypnotic ability × condition</td>
<td>112.69</td>
<td>8</td>
<td>14.09</td>
<td>3.00</td>
<td>&lt;.0027</td>
<td>.0185</td>
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<tr>
<td>Sex × hypnotic ability × condition</td>
<td>11.63</td>
<td>8</td>
<td>1.45</td>
<td>.31</td>
<td>.9623</td>
<td>.8754</td>
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<tr>
<td>Within cells</td>
<td>2103.51</td>
<td>448</td>
<td>4.70</td>
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</table>

There are several constraints to the above conclusions. Increased threat elevates SCL, but so many other physical (e.g., total verbal activity) and psychological reactions (e.g., task absorption and cognitive effort). Numerous studies (Glisky et al., 1991) have shown that high hypnotizability is modestly but reliably correlated with high absorption measured by the absorption scale. Greater effort to please the experimenter may account for the higher SCL. Several studies have, however, shown that hypnotizability is unrelated to the need for social approval or social desirability as measured by the Marlowe Crowne Scale (Hilgard, 1966; Riemler, 1990; Wickramasekera, 1995).

This clinical study provided no verbal report measure of distress concurrent with SCL because verbal report data are unreliable measures of distress, particularly in high hypnotizable patients. Previous empirical data (Wickramasekera, 1988, 1994a, 1994b) have shown that verbal report measures (e.g., subjective units of distress) during cognitive experimental stress are incongruent with physiological (e.g., SCL) measures of stress in high hypnotizable patients, perhaps because high hypnotizables can spontaneously block distress from consciousness (Wickramasekera, 1994a, 1994b), as in experimentally induced hypnotic analgesia (Hilgard and

<table>
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<tr>
<th>Low hypnotic ability (N = 26)</th>
<th>Mean (SD)</th>
<th>4.518 (3.411)</th>
<th>4.203 (3.386)</th>
<th>6.944 (4.298)</th>
<th>6.191 (4.173)</th>
<th>5.315 (3.974)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate hypnotic ability (N = 40)</td>
<td>Mean (SD)</td>
<td>5.299 (4.619)</td>
<td>5.288 (4.508)</td>
<td>8.815 (5.610)</td>
<td>7.660 (5.347)</td>
<td>6.999 (5.217)</td>
</tr>
<tr>
<td>High hypnotic ability (N = 52)</td>
<td>Mean (SD)</td>
<td>6.219 (5.000)</td>
<td>5.628 (4.480)</td>
<td>11.003 (7.655)</td>
<td>10.137 (7.680)</td>
<td>8.354 (7.012)</td>
</tr>
</tbody>
</table>

**TABLE 2**

**Skin Conductance (umhos) Means and Standard Deviations**

<table>
<thead>
<tr>
<th></th>
<th>EO1</th>
<th>EC1</th>
<th>S</th>
<th>EO2</th>
<th>EC2</th>
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<tbody>
<tr>
<td>Low</td>
<td>Mean (SD)</td>
<td>4.518 (3.411)</td>
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</table>
Hypnotizability, skin conductance, and trauma

Fig. 2. Hypnotic ability, SCL means, and 95% confidence intervals.

Hilgard, 1994). We have previously shown that in normal college students selected for hypnotizability, not low but only high experimental cognitive stress can produce significant differences in the verbal report of distress (Pomerantz and Wickramasekera, 1988). Numerous prior clinical, and even sub-clinical phobia and anxiety studies have also shown an incongruence or low correlation between verbal report and physiological (e.g., SCL) measures of stress (Lader and Marks, 1971; Lang, 1969; Shedler et al., 1993; Weinberger, 1990) even in people not selected for high hypnotizability. Hence, there is no empirical reason to necessarily expect SCL and verbal report measures of experimentally induced cognitive distress to be congruent, particularly in clinical samples.

These data are constrained by their retrospective nature and the fact that patients served as their own controls. Because hypnotizability is stable across time ($r = .71$ over 25 years) and may be partly genetic, it is likely that it existed at approximately its present levels before the onset of trauma and may have cognitively amplified the pain in highs. It is unlikely that these SCL findings in highs with chronic pain will replicate in normal pain-free college students unless they are matched for risk factors on the HRMTF. These data are also consistent with the hyposensitivity hypothesis of low hypnotic ability (Wickramasekera, 1979, 1988, 1995). It seems that the mechanisms of risk and pathology in low hypnotizable people are not immediately sympathetic (SCL) but behavioral and cognitive, as discussed elsewhere (Wickramasekera, 1979, 1988, 1993).

Weinberger (1990) and associates have found that "repressive coping" is associated with higher sympathetic activation and higher SCL. Hypnotizability, however, is an independent pathway into somatization empirically unrelated to repressive coping (Wickramasekera, 1995). Pennebaker et al. (1987) found that the conscious inhibition of trauma was associated with higher SCL and that the verbalization and/or writing of traumatic material was associated with a reduction in both the verbal report of distress and SCL. DeGoode (1991) found that higher SCL in chronic pain patients was related to better therapy outcomes. Wickramasekera found a decline in SCL related to treatment success in somatizers in psychophysiological psychotherapy (Wickramasekera, 1988, 1993, 1994b). Psychophysiological psychotherapy (PPP) matches self-soothing low arousal induction procedures (Wickramasekera, 1977) like self-hypnosis or biofeedback to patient features (high or low
hypnotizability) to disinhibit and desensitize unconscious perceptions, memories, and moods. PPP uses autonomic monitoring concurrent with psychotherapeutic self-exploration to identify and track unconscious perceptions and memories. Pre- to post-therapy declines in SCL may be an objective measure of reduced threat and trauma.

Higher SCL in highs may not be necessarily associated with higher rates of conscious verbal reports of specific traumas or distress. Highs can block the perception (verbal report) of threat from consciousness in the clinic (Wickramasekera, 1988, 1994a, 1994b) and also in the laboratory (Hilgard and Hilgard, 1994). "But out of mind, is not out of body" (p. 162, Wickramasekera, 1994b; see also Wickramasekera, 1988, 1993, 1994a, 1994b, 1995), and trauma or loss abolished from consciousness may drive somatization. Accurate unconscious memories can be marked by enhanced skin conductance response (Bentin and Mascovitch, 1990) that is dependent on the amygdala (Bechara et al., 1995). Higher SCL in high hypnotizable people may be caused by their cognitive propensity to surplus empathy, surplus pattern recognition, and their unconscious retention of unprocessed (critical-analytical) traumatic memories (Wickramasekera, 1988, 1993, 1994a, 1994b). It is well documented both in the laboratory and in the clinic that highs can reduce the verbal report of pain perception without abolishing its autonomic nervous system correlates (Hilgard and Hilgard, 1975). It is hypothesized that they may also learn inadvertently to amplify pain perception, particularly if they have high statistically unrelated (orthogonal) trait neuroticism (Wickramasekera, 1979, 1994a, 1995). Wickramasekera (1988) has suggested that disorders such as chronic pain and post-traumatic stress disorder may be treated, at least adjunctively, with PPP (Wickramasekera, 1988, 1993, 1994b, 1995). PPP uses self-hypnosis to teach patients cognitive self-soothing skills that seem to reverse the direction of activity of one of the very risk factors (high hypnotizability) that can increase threat. PPP can also be very cautiously (Dywan and Bowers, 1983; Laurence and Perry, 1983) used with highs to increase access to any unconscious traumatic memories (Brown, 1995) that may, based on current stimulus or response generalization (Wickramasekera, 1976, 1988), trigger intrusive memories, sympathetic activation, and chronic pain. Low hypnotizable patients require some form of systematic low arousal induction (e.g., biofeedback) or sensory restriction therapy (Barabasz and Barabasz, 1993) prior to PPP to increase their response to self-exploration and psychotherapy (Wickramasekera, 1977, 1988).

It is hypothesized that what is today called dissociation in the psychiatric literature (Spiegel et al., 1993) is empirically the coincidence of a) high or low hypnotic ability and b) high negative affect and is, in fact, the basis of several amplified psychological and somatic symptoms, including chronic pain (Wickramasekera, 1979, 1988, 1993, 1994a, 1995). A hundred years ago, Charcot proposed and Bernheim opposed the theory that high hypnotizability generated psychopathology and pathophysiology. Wickramasekera (1979, 1988, 1993, 1994a) proposed and has now shown experimentally that it is not high hypnotizability per se, but its interaction with negative affect (Watson and Tellegen, 1985), that can increase sympathetic activation (SCL). Increased chronic sympathetic activation can drive both psychopathology and pathophysiology through neuroendocrine and immune mechanisms (Herbert and Cohen, 1993; O'Leary, 1990).

References


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