Observation of a Paradoxical Temperature Increase During Cognitive Stress in Some Chronic Pain Patients

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A total of 224 chronic pain somatoform disorder patients without obvious pathophysiology or psychopathology were found to have colder hands than nonpatients. A paradoxical temperature increase (PTI) in response to a cognitive stressor (mental arithmetic) was noted in a subset of these chronic pain patients. Patients were defined as “PTI” responders if, during cognitive stress, an increase in digital temperature occurred over a prior eyes closed resting condition. It was found that 49.4% of males and 42.6% of females in a total sample of 224 patients demonstrated PTI. The PTI patients had significantly colder hands than non-PTI patients prior to stress. A concurrent SCL measure of sympathetic activation found no difference between the PTI and non-PTI groups either at baseline or during cognitive stress. It appears from this data that PTI is specific to the peripheral vascular system of these patients and may be a marker of psychophysiological dissociation or trauma blocked from consciousness.

KEY WORDS: stress; paradoxical vasodilatation; hand temperature.

INTRODUCTION

The critical role of explicit and implicit (unconscious) memory in the cognitive appraisal of threat to a person’s well-being have been emphasized in human studies of physiological responses to threatening psychological and physical stimuli (Lazarus, 1991; Wickramasekera et al., 1996; Wickramasekera, 1998). The High Risk Model of Threat Perception (HRMTP) predicts that threat perception and activation of the hypothalamic-pituitary-adrenal axis (HPAA) occurs when a challenge to a persons psychological well-being or physical integrity by major life change or minor hassles exceed their support systems and coping capability (Wickramasekera, 1979, 1983, 1986, 1988, 1993, 1998). It is suspected that the perception of “threat” can have neuroendocrine and immune consequences through both sympathetic activation and stress hormone receptors on immune cells (Dantzer, 1991; Naliboff et al., 1991). The HRMTP predicts that chronic pain patients will

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have colder hands than people without chronic pain and will show greater ANS dysregulation than people of moderate hypnotic ability (Wickramasekera, 1988, 1998).

For example, during mental stress, heart rate, blood pressure, and skin conductance (EDR) levels increase but peripheral skin temperature in the digits normally drop (Mittleman & Wolff, 1939). This paper will focus on the peripheral skin temperature and EDR changes that occur in the digits of people who are subjected to a timed mental arithmetic stressor. Standardized mental arithmetic stress according to the reactivity model (Blascovich & Katkin, 1993; Frederickson & Matthews, 1990; Wickramasekera, 1976, 1988, 1993, 1998) appears to be a temporally stable (Blascovich & Katkin 1993; Arena & Hobbs, 1993) and clinically significant method of activating the autonomic nervous system (ANS).

In the last 25 years, one of us has clinically observed that a subset of patients with chronic somatic symptoms during the cognitive stress of psychotherapy or mental arithmetic actually show an increase in digital temperature (Wickramasekera, 1976, 1979, 1988, 1993, 1994). This phenomena has been labelled PTI or paradoxical temperature increase. Halperin, Cohen, and Coffman (1983) and Cooke, Creager, Osmundson, and Shepherd (1990) have also noted paradoxical digital vasodilation during cognitive stress (mental arithmetic) in both male and female Raynaud’s patients and even some normal women. Halperin et al. (1983) found PTI even after sympathetic blockade. Barlow and Cerny (1988) observed PTI or “idiopathic flushing” (Aldrich, Moattari, and Vnuk, 1988) in some patients during panic attacks. It is hypothesized that the PTI described here is a peripheral vascular specific ANS dysregulation driven by implicit or unconscious (Kihlstrom, 1987) chronic threat perception reflecting an increase in sympathetic tone that may have psychophysiological implications for stress related diseases (Wickramasekera, 1979, 1983, 1988, 1993, 1998).

The electrodermal response (SCL) is a nonspecific peripheral measure of sympathetic nervous system reactivity to threat (Boucsein, 1992). Unlike, for example, heart rate, SCL is exclusively innervated by the sympathetic nervous system (Boucsein, 1992), which may be implicated in “stress” related disorders (Mason, 1972). The SCL response is reported in this study to provide a different but concurrent physiological measure of the efficacy of the experimental stressor (mental arithmetic) and as a manipulation check.

For this study a PTI patient was defined as one who showed an increase of .1 degree F or more during cognitive (mental arithmetic) stress or threat. This definition was empirically based on the variability of the 60 temperature measurements taken at each 4-minute period. On average, a temperature difference of slightly less than .1 was about 2 standard errors from the mean of the 60 temperature measurements regardless of profile condition. Therefore, PTI was defined as an increase in temperature of .1 degree F or more.

**METHODS**

**Participants**

There were 224 patients, with a mean age of 37.79 years, \((SD = +12.92\) years) who presented chronic vascular or muscular headache (51%) and other chronic pain symptoms (49%). There were 83 males, mean age = 38.05 years, \((SD = +11.92\) years), and a range of 15–66 years. Of the 141 females, the average age was 37.61, \((SD = +13.61\) years), and a range of 12–80 years. All of these patients received a *Diagnostic and Statistical Manual of*

The first “cognitive stress response profiles” (Wickramasekera, 1976, 1988, 1993) of 224 patients seeking evaluation for entry into a psychophysiological treatment program were evaluated in this study. All patients were referrals from physicians who had previously performed very extensive medical and neurological diagnostic testing on these patients for a variety of chronic pain complaints. All of these patients had previously been studied by multiple specialists and subspecialists (neurologists, neurosurgeons, internists, endocrinologists, cardiologists, OB-gyn, etc.) for chronic “stress-related” pain symptoms resistant to standard medical management. Extensive medical and neurological testing had identified no evidence of diabetes, scleroderma, lupus, or Raynaud’s disease in any of these patients. Some of these patients were still on analgesic, or psychotropic, medication except for a period of 24 hours prior to the present testing. None of these patients were on anticholinergic medications, which can alter SCL. All of these patients reported that psychosocial stress amplified their pain symptoms, which had not responded to standard medical-surgical intervention and/or physical therapy.

Procedure

The 20-minute stress profile was administered by a technician with over 6 years of experience with the protocol. All data collection was performed with the J&J physiologic monitor and recording system (Model 1330). Peripheral skin temperature response was measured in conjunction with electrodermal response, (SCL) in a temperature (78°F) controlled and electrically shielded psychophysiology laboratory. Each participant was seated in a comfortable chair during the stress profile. The SCL and temperature were measured by attaching transducers to the digits of the left hand, which rested on the arm of the chair isolated from any air currents. Peripheral skin temperature was recorded with a thermostor, affixed with surgical tape, on the ventral aspect of the fifth digit pulp. SCL was measured with the J&J computer system Model No. I-330, which monitors SCL and performs basic data reduction on the SCL signal. SCL is 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 333Ti 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, providing 60 averages for each 4-minute period of the cognitive stress profile. Averages of activity for each period or condition are also derived.

All patients were scheduled for testing at approximately the same time (9:00 a.m.–12:00 p.m.). The cognitive stress test protocol began once the patient was seated comfortably. Continuity of electrodes was verified by viewing the continuous screen on a TV monitor available only to the tester. The TV monitor was situated to prevent the patient from self-monitoring. The experimental procedure began with a 4-minute period of relaxation in which the subject was instructed to “sit quietly and relax with eyes open” (EO1). This was followed by a 4-minute period of relaxation instructions “sit quietly and relax with your eyes closed” (eyes closed one, EC1). Next, the patient was told with eyes open that
a timed mental arithmetic test of intelligence would be administered (S). This 4-minute, arithmetic challenge consisted of asking the patient to consecutively subtract seven from one thousand, out loud. Accuracy and speed were urged, and a conspicuous stopwatch was used to create performance pressure. Immediately after the cognitive stress period (S) the patient was told to relax with eyes open (EO2) and immediately afterwards with eyes closed (EC2) for consecutive periods of 4 minutes each.

**Data Analysis**

Contingency table analysis (chi-square) was used to compare the distribution of males and females among PTI and non-PTI patients. Paired t tests were used to compare change in temperature from one profile condition to another. Unpaired t tests were used to compare PTI and non-PTI temperatures at each profile condition. A Bonferroni correction for Type I error rate was made for each set of comparisons (i.e., differences in temperature between adjacent conditions for PTI and for non-PTI patients), and for comparison of PTI and non-PTI patients. Repeated measures analysis of variance was used to compare EDR over the 5 profile conditions for PTI and non-PTI patients. The impact of regression to the mean was analyzed by the method of Blomqvist (Blomqvist, 1977; Hayes, 1988).

**RESULTS**

The mean hand temperature of all pain patients regardless of PTI status was 83.5 + 5.57 degrees F at baseline eyes open. This was significantly lower (p < .0001) than a published sample of normal subjects with a mean hand temperature of 88.6 + 7.41 degrees F (Blanchard, Morrill, Witrock, Scharff, & Jaccard, 1989; Taub, 1977) collected under similar conditions.

Based on the above definition of PTI, there were 94 (42%) PTI patients and 130 non-PTI patients. There was no difference in the distribution of males and females among PTI and non-PTI subjects (p = .5430). The mean hand temperature of the men was 83.62 (SD = 5.46). The mean hand temperature of the women was 83.36 (SD = 5.63).

Figure 1 plots temperature means and 95% confidence intervals of PTI and non-PTI subjects for each condition. PTI subjects began with significantly lower temperature at baseline EO than non-PTI subjects (p < .05). PTI subjects then had a significant increase in temperature from baseline EO to baseline EC (p < .001), from baseline EC to Stress (as defined by PTI, p < .001) and from stress to post-Stress EC (p < .05). The mean increase in temperature from baseline EO to Stress was 1.68 degrees with an additional mean increase of .32 degrees from Stress to EC. On the other hand, non-PTI subjects had only a significant decrease in temperature from baseline EC to Stress (p < .001). Non-PTI subjects had a mean decrease in temperature from baseline EO to Stress of .54 degrees. At stress and after, PTI and non-PTI temperatures were very similar, and were not statistically significantly different.

A 2 (PTI vs. non-PTI) × 5 (profile conditions) repeated measures analysis of variance (ANOVA) was performed to determine if PTI and non-PTI patients also had different SCL profiles. There was no significant main effect for PTI (p = .9423) nor was there a significant PTI × condition interaction (p = .6208). Figure 2 plots the SCL means of PTI and non-PTI patients at each condition. As can be seen, the profiles were virtually identical.
Paradoxic Temperature Increase in Chronic Pain

Fig. 1.

Fig. 2.
A possible explanation of the PTI phenomenon is regression to the mean, the fact that subjects with initial high temperatures will tend to have lower temperatures at a later condition, and subjects that have low initial temperatures will tend to have higher temperatures at a later condition just as a result of measurement error. The within-subject variability from the 60 measurements at a given profile condition provided an estimate of measurement error. Using the method of Blomqvist (1977), an adjustment for measurement error was made to the slope of the regression line of change in temperature at stress to baseline temperature at EO. Figure 3 plots these data. The regression line had a statistically significant, negative slope ($-0.0415$, $p = .0003$). After adjustment for measurement error, the slope remained statistically significantly different from zero ($-0.0347$, $p = .0025$). Thus, changes in temperature for PTI and non-PTI patients cannot be attributed primarily to regression to the mean.

DISCUSSION

The predominant findings of this study were the following:

1. Chronic pain patients, as predicted by the HRMTP (Wickramasekera, 1979, 1988, 1998), appear to have colder mean digit temperature than nonpatients.
2. There was no significant difference in baseline digit temperature between these male and female patients.
3. 42% of these chronic pain patients were PTI responders, and this response was specific to the peripheral vascular system during cognitive threat perception.
4. There was no significant difference in the probability that men (49.4%) or women (42.6%) would be PTI responders.
5. PTI subjects began with significantly lower baseline (EO1) digit temperature than non-PTI subjects.
6. In terms of an SCL definition of threat perception, both PTI and non-PTI patients were equally cognitively stressed, but only the PTI group at T demonstrated a significant paradoxical temperature increase.
7. This PTI response cannot be explained primarily by regression to the mean.

That chronic pain patients compared to normal controls (Blanchard et al., 1989; Taub, 1977) have significantly colder hands is of some theoretical salience (Wickramasekera, 1979, 1988, 1998). In retrospect, it may have been better to do other analyses of this large and complex database. However, the data was not available for reanalyses because the first author has relocated. It could be said on an a priori basis that the baseline was not stable or long enough and that the increases in the PTI group would have occurred without any stress intervention. This hypothesis remains open to empirical study. The fact that chronic pain patients and PTI patients, in particular, have colder hands at baseline is consistent with predictions from the HRMTT’s threat amplifying or repressing (blocking from consciousness) psychosocial risk factors that drive sympathetic activation and ANS dysregulation (Wickramasekera, 1979, 1983, 1988, 1993, 1994, 1998). PTI is hypothesized to be driven by implicit or unconscious chronic sympathetic activation, and, hence, predicted to be associated with a greater number of conscious occluding risk factors than in non-PTI patients (Wickramasekera, 1998). The incidence of PTI in nonpatient is presently unknown. Prior extensive medical testing reduces the probability of undiagnosed medical diseases in these patients. Chart review indicated no systematic differences in prior medication use that could account for the PTI. At this time, the data is not available to correlate PTI and non-PTI with medication status.

Temperature increase can occur during sympathetic withdrawal, increased vagal tone (Jennings & McKnight, 1994), or cognitively mediated relaxation (Taub, 1977). Learned unconscious or implicit cognitive disengagement or dissociation from immediate explicit threat perception (Wickramasekera, 1979, 1988, 1993, 1998) is predicted to be marked by peripheral vascular relaxation during threat. In other words, peripheral vascular relaxation during threat perception may be a peripheral physiological marker of unconscious disengagement from threat perception. The fact that this unconscious disengagement is specific to the vascular system may mean that the peripheral vascular system is more vulnerable to unconscious learning and conditioning (e.g., primary hypertension) than SCL. The increase in SCL during the backward 7-digit task indicates that the task effectively induced threat perception. We have some preliminary clinical data supportive of the hypothesis that some highly hypnotizable normal people who can abolish threat from consciousness during the stress of hypnotic surgical analgesia also demonstrate PTI. Clinically, we have found that PTI is abolished posttherapy in patients who learn through low arousal temperature biofeedback (Taub & Emurian, 1976; Wickramasekera, 1976) or self-hypnosis training (Wickramasekera, 1976, 1988, 1993) to bring unconscious threat perception into consciousness. PTI may be a psychophysiological marker of trauma blocked from consciousness. It has been proposed (Wickramasekera, 1998; Wickramasekera & Wickramasekera, 1997) that PTI is a psychophysiological marker and example of what was called “incongruence” (Rogers, 1951) or dissociation (Hilgard, 1977; Spiegel et al., 1993) in the domain of psychopathology.
In summary, a significant number of patients (42%) with chronic pain symptoms and cold hands, without obvious pathophysiology or psychopathology displayed a paradoxical increase in digit skin temperature in response to a cognitive stressor.

REFERENCES


