

A CASE STUDY: ELECTROMYOGRAPHIC CORRELATES IN THE HYPNOTIC RECALL OF A REPPRESSED MEMORY

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ABSTRACT

This is a controlled case study of a 22-year-old female with a four-year history of episodic nausea, vomiting, tachycardia and social anxiety resistant to medical and psychiatric therapy. Systematic desensitization for social phobia with psychophysiological monitoring (electromyogram, skin temperature, skin conductance level (SCL), and heart rate) was associated with a decline in somatic symptoms, muscle tension (EMG), and subjective distress. At the tenth session, an EMG spike sustained for at least 33 minutes was associated with a forgotten traumatic dream from the previous night. Hypnotic recall of the traumatic dream during therapy was associated with an immediate collapse of the EMG spike and an abrupt increase in subjective distress. The dream contained recall of a repressed memory of a possible sexual exposure to HIV five years previously. This case study may illustrate the utility of psychophysiological monitoring (PPM) in the psychotherapy of somatoform disorders. It may also illustrate the value of electrophysiological and autonomic measures to identify repressed traumatic somatized memories and to demonstrate objectively the distinction between implicit and explicit memory.

INTRODUCTION

Recently, there has been renewed interest in studying unconscious processes within the framework of cognitive neu-

rosience (Greenwald, 1992). Kihlstrom (1987) suggests that unconscious processes and their relation to memory can best be understood within the cognitive distinction between implicit (unconscious) and explicit (conscious) memory. Wickramasekera (1988, 1993a) has suggested that applied psychophysiology may be a royal road to the unconscious, specifically in somatoform disorders.

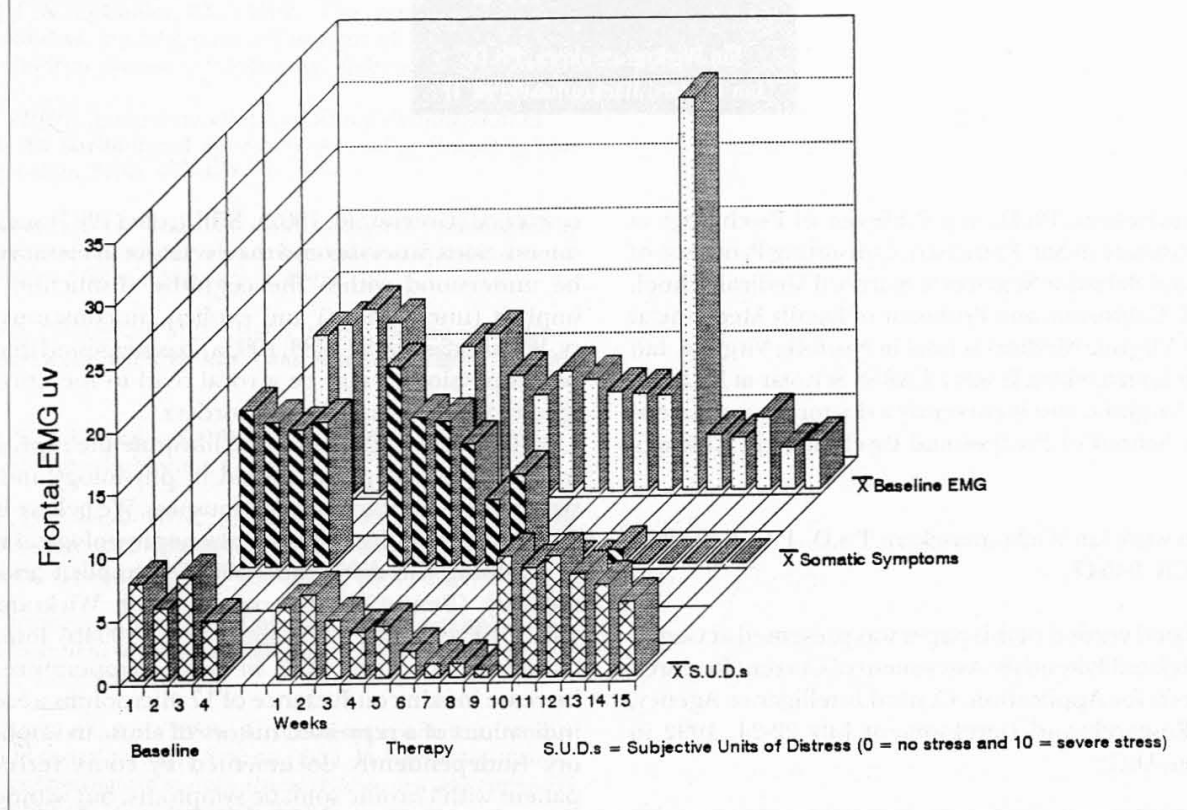
This case study appears to illustrate the repression of a traumatic memory, manifested in physiology and somatic symptoms but outside of consciousness. We believe it appears to demonstrate the utility of psychophysiological measures in assessing the distinction between implicit and explicit memory (Bentin & Moscovitch, 1990; Wickramasekera, 1994b). Previously, Wickramasekera (1994b) found that a contingent drop of 16°F in hand temperature and an increase in skin conductance of 17 microohms were the first indications of a repressed history of abuse in implicit memory (independently documented by court records) in a patient with chronic somatic symptoms, but without identifiable organic disease or psychopathology.

PATIENT

The patient is a 22-year-old white, single female who presented with a four-year history of episodic nausea, vomiting, tachycardia, and anxiety before or during social events. Extensive medical investigation by several internists, gastroenterologists, and neurologists had failed to identify any organic basis for her somatic symptoms. The patient had also experienced over 50 sessions of psychodynamic psychotherapy with two psychotherapists, and multiple trials of psychotropic medications (benzodiazepines and antidepressants) with two board certified psychiatrists. Psychodynamic psychotherapy with a prominent Freudian psychoanalyst and later with a Jungian analyst was associated with reduced psychological symptoms (anxiety and depression) but no change in somatic symptoms. Subsequent trials of psychotropic medication (precise medications unknown) with two biologically-oriented psychiatrists appeared to reduce her psychological symptoms further but significantly increased the frequency and intensity of her somatic symptoms.

The patient was an undergraduate college student who was raised by "loving parents" who were described as "hip-

FIGURE 1
Frontal EMG, Somatic Symptoms and SUDs



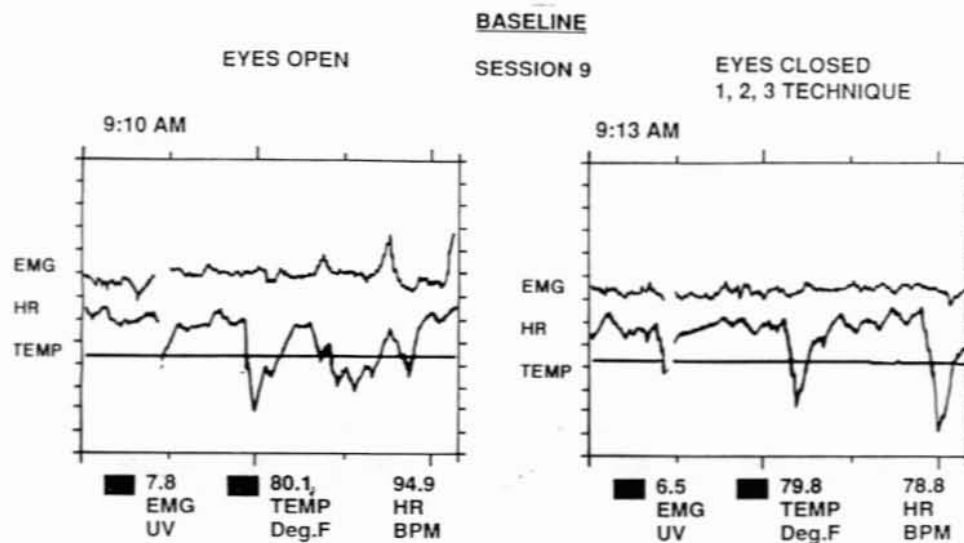
pies" in the 1960s. The patient and her only sibling, a younger brother, were raised on a private farm by a "bubbling brook in the woods" until approximately age eight. After her father became a successful banker, the family moved into the suburbs of a large city and into a conventional upper middle class lifestyle. Both parents were college graduates and her mother earned an M.A. degree while she taught high school. The patient denied any history of physical or sexual abuse. She also denied any pre-morbid history of psychological or psychiatric therapy and any history of significant medical problems. This lack of psychiatric and medical history was independently confirmed by both parents. Approximately at age 15 the patient started dating infrequently. She denied any sexual activity beyond kissing and holding hands up to date. She reported that she had never "gone steady" with any boyfriend. The patient was academically an average student and planned to study to become a teacher like her mother. Until the onset of her somatic symptoms, she had been active in sports such as track and basketball on a regular basis. Her brother was active in athletics and an outstanding student. The patient stated that her psychotherapy was helpful in terms of interpersonal support but had no apparent impact on her worsening somatic symptoms. Medication trials were

associated with some reduction in depression but a gradual increase in the frequency and intensity of her somatic symptoms. Her parents were gravely concerned about her somatic symptoms. Neither the patient nor her parents were aware of any psychological stress that could account for her multiple somatic symptoms and believed that her depression and anxiety were driven by the medically unexplained somatic symptoms that disrupted her academic and social life. At this point, the patient was referred by a neurologist for a trial of psychophysiological psychotherapy (Wickramasekera, 1988, 1993a) to the first author, who did all the testing and therapy in this case.

Method

The patient was required to keep a daily record of the frequency of all her somatic symptoms for four weeks before therapy and also during the period of active therapy. Mean subjective units of distress (SUDs) reports were (see Figure 1) also collected before therapy (baseline) and during therapy (15 weeks). SUDs are simply self-ratings on a 0 (no distress) to 10 (extremely distressed) scale of subjective distress or tension. A baseline measure of forehead mean EMG (muscle tension) for three minutes with eyes open (EO) and with

FIGURE 2
Physiological Changes Induced by Self-Hypnosis



Sympathetic changes voluntarily induced in three minutes (9:00 a.m. - 9:13 a.m.) in resting eyes open baseline muscle tension (electromyogram microvolts), hand temperature (degree Fahrenheit), and heart rate (BPM or beats per minutes) by the eyes closed 1, 2, 3, self-hypnosis technique at session 9.

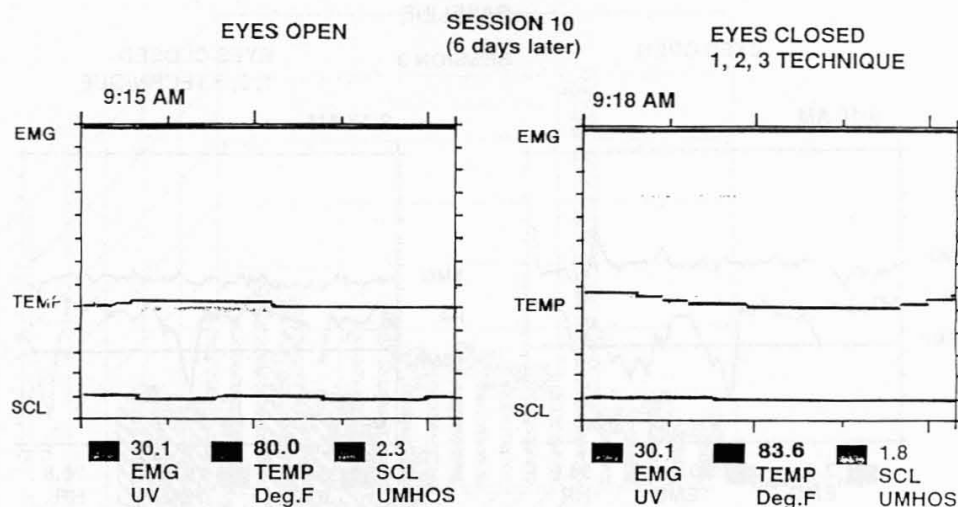
eyes closed (EC) is secured before each therapy session (see Figures 1, 2, and 3 from sessions nine and ten). Three other channels of physiology (heart rate, skin temperature, and skin conductance) are also continuously monitored before and during therapy to identify perceptions of threat outside of consciousness or SUDs. The High Risk Model of Threat Perception (HRMTP) hypothesizes that the following are risk factors for blocking threat from consciousness and inducing somatization: 1) high or low hypnotic ability; 2) high catastrophizing; 3) high overt; or 4) covert ("repressed") neuroticism (covert neuroticism is operationally defined as a Marlowe-Crowne [Crowne & Marlowe, 1960] score equal to or greater than 17); 5) major life change; 6) minor hassles; 7) low social support; and 8) low coping skills (Wickramasekera, 1979, 1986, 1993a, 1995, 1998). Testing on the HRMTP established that this patient had four of eight risk factors for somatization. The eight risk factors are operationalized with specific tests or known validity and reliability (Wickramasekera, 1993a, 1995). Her risk factors were: 1) high hypnotic ability (score of 11 out of 12 on the Harvard Group Scale of Hypnotic Susceptibility Form A [Shor & Orne, 1962]); 2) high catastrophizing (score of 66 out of 80 on the Zocco scale of catastrophizing [Zocco, 1984]); 3) high neuroticism (94% on the neuroticism scale of the Eysenck Personality Inventory Form A [Eysenck & Eysenck, 1968]); and 4) low coping skills (score of minus 23 out of 108 on the Rosenbaum

Self Control Scale [Rosenbaum, 1980]). All four risk factors are either totally or moderately unrelated and drive somatic symptoms, independent of organic disease (Wickramasekera, 1988, 1993a, 1994a, 1994b, 1995, 1998; Wickramasekera & Atkinson, 1993b; Wickramasekera, Pope, & Kolm, 1996).

Instruments

Electromyographic (EMG) activity was sensed with Ag-AgCl cup electrodes placed one inch above each eye, with a ground in the center of the forehead. Peripheral skin temperature measured in degree Fahrenheit and was sensed with a thermistor placed on the back of the middle phalanx of the middle finger of the non-dominant hand. Heart rate was sensed with a photoplethysmographic transducer placed on the pad of the middle finger of the non-dominant hand. Skin conductance (SCL) was sensed using Ag-AgCl disc electrodes, 12mm in diameter, attached to the distal phalanges of the second and third fingers of the subject's cleaned left hand according to the procedures described by Fowles et al. (1981). The EMG, skin temperature, pulse rate, and skin conductance signals were conditioned using the corresponding Coulbourn Instruments modules. The module signals were further processed using a Cyborg 91-I Integrated System for Automated Acquisition and Control (ISAAC) interfaced with an IBM PC/XT computer. The signal tracings were displayed in a

FIGURE 3
High and Stable Muscle Tension and Physiological Dissociation



At session 10, the 1, 2, 3 self-hypnosis techniques induces changes in hand temperature (80.0 - 83.6 degrees Fahrenheit) and SCL or skin conductance level (2.3 micromhos to 1.8 micromhos) in three minutes (9:15 a.m. - 9:18 a.m.) but no changes in very high and stable forehead muscle tension (30.1 microvolts electromyogram) in this patient. The above incongruity or dissociation in physiological changes during this self-hypnosis technique indicates that dissociation may have physiological correlates.

scrolling graphical format on the computer monitor with momentary numerical values also displayed on-screen, which could be printed.

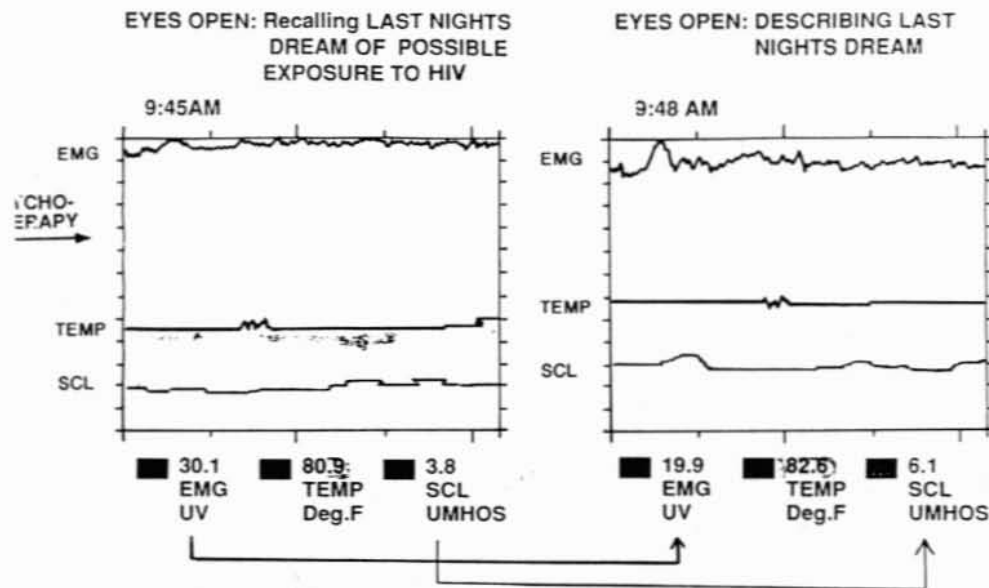
Intervention and Results

The mean frequency of somatic symptoms (nausea, vomiting, tachycardia, etc.) per week before intervention (See Figure 1) was 11.5 episodes per week. The patient could recall no period when she was totally free of somatic symptoms for even one week in the previous two years. Figure 1 shows baseline mean frontal EMG, mean somatic symptoms and mean SUDs ratings at baseline (pre-therapy) and during 15 therapy sessions. Our standard protocol for psychophysiological psychotherapy (Wickramasekera, 1988, 1993a) for somatization begins at each therapy session with printing physiological baseline data at the end of a three-minute Eyes Open (EO) and three-minute Eyes Closed (EC) procedure (see Figures 2 and 3). Psychophysiological psychotherapy (Wickramasekera, 1988, 1993a) includes the following four components: 1) baseline recordings of physiological measures with EO and EC prior to psychotherapy intervention as above; 2) psychotherapy exploration/interpretation with concurrent physiological monitoring to identify threat perception outside of consciousness; 3) systematic desensitization of threat perceptions with concurrent physiological mon-

itoring; and 4) training in the eyes closed (EC) self-hypnosis 1, 2, 3 technique (Wickramasekera, 1988, 1993a) adapted from Spiegel and Bridges (1970) for the self-soothing of stress.

Systematic desensitization of social phobia was started at therapy week one (see Figure 1). The somatic symptoms responded rapidly to nine sessions (once per week) of systematic desensitization and psychotherapy focused on reducing in imagination the phobia of social situations. Adjunctive training to self-soothe with self-hypnosis, called the 1, 2, 3 eyes closed technique (Wickramasekera, 1988), enabled the patient to reliably reduce sympathetic activation voluntarily (see Figure 2). For example, at session nine (baseline), during 1, 2, 3 EC technique mean frontal EMG dropped from 7.8 uv (9:10 am) to 6.5 uv (9:13 a.m.), and mean heart rate dropped from 94.9 bpm to 78.8 bpm. Note (see Figure 2) that during the 1, 2, 3 EC period, all the above physiological measures are in the normal range, except for peripheral hand temperature, which is 79.8°F. The mean and standard deviation for hand temperature in normal age matched controls is 88.62°F and 7.41°F (Blanchard, Morrill, Wittrock, Scharff, & Jaccard, 1989). This low hand temperature suggests some residual threat perception in the vascular system despite a reduction in somatic symptoms and subjective distress (SUDs). Note (see Figure 1) that by the ninth session,

FIGURE 4
Electromyographic Correlates of Recall and Verbalization of a Traumatic Dream



Changes in muscle tension (30.1 microvolts electromyogram - 19.9 microvolts electromyogram) skin temperature (80.9 to 82.6 degrees Fahrenheit) and skin conductance level (3.8 to 6.1 micromhos) during (9:45 a.m. - 9:48 a.m.) the recall and verbalization of a traumatic dream. Note reduction in forehead muscles tension but increase subjective anxiety indicated by an increase in skin conductance and paradoxical increase in skin temperature (Wickramasekera, et al., 1998) on recall and talk of traumatic dream. Paradoxical skin temperature increase may be a physiological marker of dissociation (Wickramasekera & Pope, 1998).

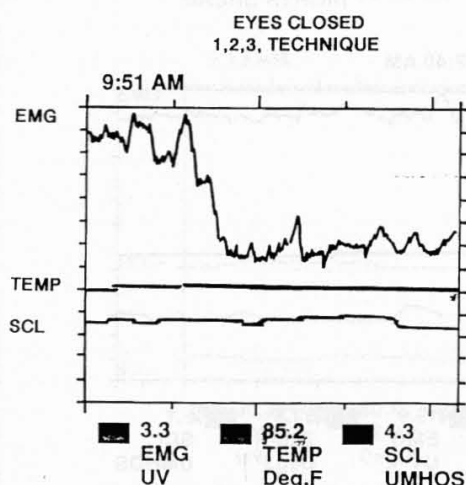
the patient was totally free of all prior somatic symptoms for three weeks and her SUDs level was at the lowest recorded level. This was reported to be the longest period she had been free of somatic symptoms in four years, in spite of exposure during the last three weeks to multiple social situations (public speaking, attendance at a party, etc.) that had previously triggered nausea, vomiting or tachycardia. This incongruity between low baseline hand temperature indicating threat perception but reduced somatic and subjective symptoms indicating clinical improvement, illustrates a typical psychophysiological dissociation between clinical symptoms and peripheral autonomic nervous system (ANS) physiology.

Figure 3, from her tenth therapy session, shows her baseline physiology collected at approximately the same times (i.e., 9:15 a.m. EO and 9:18 a.m. EC) a week later. Note that, at approximately the same time, but only six days later, both her EO and EC forehead EMG are very significantly higher and stable at 30.1 uv than during the previous ninth session (EO EMG = 7.8 uv and EC EMG = 6.5 uv). This stable spike in EMG level (30.1 uv) is more than 100% higher than six days ago at the ninth session (see Figure 2). Also there

is no evidence of the typical EMG drop on entry into the 1, 2, 3 EC self-hypnosis procedure at 9:18 a.m. Her baseline skin temperatures, EO (80.0 degrees F.) and EC (83.6° F.), are not significantly different from those six days earlier and in fact even show a modest temperature increase on entry into self-hypnosis at 9:18 a.m. Hence, under the two standard baseline conditions, pre-therapy muscle tension remains locked in at EMG 30.1 uv. Her SCL shows a small drop on entry into self-hypnosis consistent with the small increase in hand temperature. If the elevated EMG baselines at 9:15 a.m. and 9:18 a.m. in the tenth session indicate some significant perception of threat, this threatening perception is now most notably specific to the musculo-skeletal response system (EMG) and not observable in temperature or SCL. This is another psychophysiological dissociation or incongruity. SCL and temperature, in fact, demonstrate a paradoxical modest reduction in sympathetic activation consistent with relaxation (see Figure 3).

During this 17-minute period of 9:18 a.m. to 9:35 a.m. session, psychotherapeutic inquiry was focused on exploring what the patient was consciously feeling and thinking that could drive and keep her frontal EMG locked in at 30.1

FIGURE 5
Physiological Correlates of Reframing
and Anxiety Reduction



Use of 1, 2, 3 self-hypnosis technique to reframe and reduce fear of possible HIV infection four years ago.

Note consequent integrated or non-dissociated changes in muscle tension (microvolts electromyogram), skin temperature (degrees Fahrenheit) and skin conductance level (micromhos) indicating anxiety reduction and in integrated reduction in sympathetic activation.

uv. She reported mild somatic "nausea" and "scared, weird and awkward feelings" on waking that morning. She denied any other somatic symptoms except nausea. I printed and showed her the atypical, but stable, high frontal EMG signal from her forehead on the computer screen. I invited her to speculate on what might be causing this unusually high and very stable forehead EMG. She denied feeling muscle tension in her head or feeling bilateral or unilateral headache pain. She was not frowning or squinting her forehead or face. I did a careful immediate check of the EMG instrument. I removed the electrodes from the patient's head and substituted a standard calibrating EMG signal generating device for the patient's frontalis muscles. This calibrating device and electrode check indicated no malfunction of the EMG system or electrodes. When the electrodes were replaced on the patient's head the EMG signal was still 30.1 uv.

Next, I specifically asked her if anything had occurred that morning (e.g., heavy traffic, increase in cups of coffee, conflict at home that morning, etc.), last evening or last night, or during the previous week to make her "nauseous" and "scared." She denied any stressful experiences in the last six days or the previous day. She also denied any relapse of her

somatic symptoms in the prior week, except for the nausea and anxiety she felt when she arose from sleep that morning. I asked her if she felt uneasy anticipating the end of her therapy. She denied feeling any anxiety about termination or coming to this specific therapy session. However, she stated that earlier that morning she had a "very scary" dream, but she had absolutely no memory of the content of the dream. After 17 minutes of this unproductive careful investigation of her high level of muscle tension (her EMG remained on the ceiling at 30.1 uv or above from 9:15 a.m. to 9:35 a.m.), I asked her to "change channels" with the 1, 2, 3 self-hypnosis technique and to enter the hypnotic mode of information processing. I suggested, after she closed her eyes, in this hypnotic self-soothing mode that she might recall the previous night's "scary dream" as if she were seeing it with detachment on a TV screen. The patient entered the hypnotic mode at 9:36 a.m. and exited it at 9:45 a.m. (a total of nine minutes in self-hypnosis) as indicated by the event recorder on the psychophysiological computer monitor. Upon opening her eyes at 9:45 a.m. the patient spontaneously, very rapidly, and very emotionally started to verbalize the contents of the "very scary dream" she had just recalled in self-hypnosis (see Figure 4). In this dream she recalled an actual three-month relationship she had over five years previously with a lover. She rapidly "fell in love" with a young man and became sexually (vaginally and orally) active with him, until he stated that he was bisexual. His statement panicked her, and she immediately dropped the relationship without any explanation and agonized about getting an HIV test. She was afraid to find out if her lover had infected her with HIV. She did not get the HIV test but remained in a very high state of anxiety for several weeks. During the panic she was afraid to confide in any friends or family because of fear of rejection and abandonment. She stated that until today she had completely "forgotten" about the need for an "HIV test, her lover, and the AIDS panic." She was uncertain precisely when the "forgetting" started. She reported no recall of the dream on awakening that morning until she used the self-hypnosis procedure at my suggestion to enhance dream recall between 9:36 a.m. and 9:45 a.m. in the tenth session. The patient stated that she had not told her parents, friends or any of her prior therapists about this "AIDS panic" episode because she had no memory of it until today. She stated that she had somehow "forgotten" about "the AIDS panic" when she started therapy for her somatic symptoms.

It is remarkable (see Figure 4) that even as the patient started to verbalize explicitly, very rapidly and emotionally (9:45 a.m. to 9:48 a.m.) this "memory" of her dream, her frontal EMG started to drop. It had remained locked at 30.1 uv through the entire baseline, psychotherapy, instrumentation check, and hypnosis session from 9:15 a.m. to 9:45 a.m., until she began to verbalize her dream at 9:45 a.m. (see Figure 4). Note that EMG is always scaled logarithmically on this instrument so that there is high resolution of the EMG sig-

nal in the low EMG range of the scale and low resolution at the high EMG range.

At 9:45 a.m., her skin temperature started to increase from 80.9°F. up to 82.6°F. and her SCL started to increase from 3.8 umhos to 6.1 umhos as she verbalized her recall of the dream and the disturbing "memory." We have previously observed and reported this paradoxical temperature increase (PTI) with fear in stress-related disease (Wickramasekera & Atkinson, 1993b; Wickramasekera et al., in press). It is worth noting that this PTI occurred in association with a drop in EMG and increase in SCL. During PTI, the patient stated that her fear was specifically related to the possibility of HIV infection and death.

To re-induce self-control and cognitive reframing of the memory, at 9:51 a.m., I suggested that the patient switch back into the 1, 2, 3, self-hypnotic mode of information processing. She resumed self-soothing in self-hypnosis while I quietly reminded her that she had developed no obvious symptoms of HIV infection in spite of multiple medical tests and that there had been some progress in effectively treating the HIV-related disease in the past five years. When the 1, 2, 3 self-hypnotic technique was used this time after recall, it was associated with a further drop in frontal EMG from 19.9 uv to 3.3 uv, a further increase in skin temperature up to 85.2°F. and a drop in SCL to 4.3 umhos (see Figure 5). Incidentally, these changes in EMG, temperature, and SCL are now in normal congruity or association. This increase in skin temperature, drop in EMG, and reduction in SCL suggests an integrated physiological reduction of sympathetic drive. At the end of the session the patient reported that she felt relatively "calm" and free of the previous "nausea, weird, and awkward feelings." In subsequent psychotherapy sessions the patient's fear and chronic avoidance coping (Moos, 1993) declined to the point that she was able to take an HIV test, which proved negative. We used the Gestalt "empty chair technique" (Perls, 1969) to verbalize, process, and close her guilty feelings about "dropping" her lover without any explanation. She also realized that her unconscious or implicit fear of the possibility of HIV infection was possibly increased by the prospect of each social intimacy, and it may have driven her social avoidance in the last five years. With guidance, she speculated that her symptoms of nausea, vomiting, and tachycardia may be 1) unconscious guilt driven efforts to purge the repressed memory of oral sex, 2) the implicit incubating social fear of another risky social intimacy, or 3) the fearful prospect of infecting another person with her own "diseased" love.

Follow-Up

Independent investigation with the patient's parents indicated that five years previously the patient had had a brief but intense relationship with a young man. The parents denied any knowledge of the details of the relationship but stated that it ended abruptly and that the patient appeared

very distressed when it terminated. She had refused to talk about the relationship and its termination. Further independent verification of this apparently repressed memory with the "young man" appeared clinically contraindicated in this case at follow-up. There appears to have been a tacit conspiracy of silence in the family about any temporal association between the patients' symptoms and this relationship. It is hypothesized that in such repressed but close relationships anomalous communication continues about repressed topics among family members (Wickramasekera, 1991).

A follow-up interview at approximately three months and telephone interviews at 12 and 24 months detected no evidence of relapse of any of the prior somatic symptoms or any new somatic symptoms. A second HIV test, which she took at eight months post-therapy, was reported to be associated with intense anxiety and depression, but there was a rapid remission of all the psychological symptoms on learning that the test was negative. During the 24-month telephone follow-up interview, the patient denied any recurrence of the old somatic symptoms or any new somatic symptoms. She reported episodic depression and anxiety that seemed to be situation-specific. She had graduated from college, was living an active social life, and was engaged to be married.

DISCUSSION

Memory and Psychophysiological Data

Possible exposure to potentially lethal HIV infection can be regarded as a traumatic event. Review of both laboratory and field studies conclude that "highly negative emotional events are relatively well retained, both with respect to the emotional event itself and with respect to the central, critical detail information of the emotion-eliciting event (Christianson, 1992). The problem then for this case study is to account not for the memory but the forgetting (repression). There is empirical evidence that independently verified traumatic events may be repressed or inhibited from consciousness (Coons, 1994; Coons & Milstein, 1986; Kluft, 1995; Williams, 1994) as proposed by Freud and Janet through active repression or passive disconnection or dissociation. Hypnotic recall appears to be a more implicit-like memory retrieval process (Spiegel, 1994). There is evidence that depression can impair both explicit and implicit memory (Elliott & Greene, 1992), and this patient was clinically depressed for at least four years as indicated by her medical records. There is evidence that hypnosis can have enhancing effects on recall in spite of the occurrence of memory distortions and pseudo memories (McConkey, 1992). Hypnosis used neutrally and non-suggestively may be a useful tool to recall traumatic memories (Kluft, 1995; Spiegel, 1994). It appears from independent verification with the parents that the main events occurred but the details could not be verified. This case study confirms both Freud's and Janet's concepts of a functional inhibition of recall.

This case study may be heuristic in illustrating an EMG correlate of an implicit or repressed traumatic memory made explicit with hypnotic recall and supports a previous study (Wickramasekera, 1994b) demonstrating the utility of psychophysiological measures to identify functionally (repressed) inhibited somatized memories. There is previous clinical theory and observation (Eich, 1990; Reik, 1949; Jacobson, 1938) and some experimental data that EMG may reflect encoded cognition (McGuigan, 1978). McGuigan (1978) received empirical evidence that covert linguistic processes have EMG correlates. EMG measures are higher in psychiatric patients with unsolvable than solvable concept identification in experimental tasks (Pishkin & Shurley, 1968). Without EMG monitoring, both the patient and I would have been unaware at the tenth session of her high frontal EMG, when subjectively (SUDs) and symptomatically she appeared "cured." I might not have questioned her so closely about her feelings that morning and discovered the return of the mild nausea, etc., and the "forgotten" early morning dream had I not noticed that her frontal EMG was 30.1 uv.

Her low peripheral skin temperature also indicated sympathetic activation because it was approximately 9°F lower than the norms for a person her age (Blanchard et al., 1989). This suggests residual threat perception in at least two peripheral physiological channels even though she was symptomatically, electromyographically, and subjectively improved. Her hand temperature increased on the tenth session during fear. Her hand temperature response (PTI) may have been driven by some implicit threatening cognitive intrusion during the 1, 2, 3 hypnosis technique. PTI may be a psychophysiological dissociation or incongruity. Normally, integrated peripheral autonomic nervous system responses, may become dissociated due to chronic threat repressed from consciousness (Wickramasekera, 1988, 1993a; Wickramasekera & Pope, 1996). We had not observed this in the prior (ninth) session. We have observed PTI in 50% of all male and female somatizers when they are cognitively threatened (e.g., mental arithmetic stress), particularly if they have a low baseline hand temperature. Others (Cooke, Creager, Osmundson, & Shepherd, 1990) have also observed PTI to mental arithmetic stress in both men and women with cold hands. Barlow and Cerny (1988) reported a PTI in some panic disorder patients during a "relaxation induced" panic attack. This PTI or vascular relaxation associated panic attack may be a defense against the intrusion of more repressed or implicit aversive memories (Wickramasekera, 1976, 1988, 1993a; Wickramasekera et al., in press).

It appears possible to experimentally install memories for events that never occurred in normal people of high hypnotic ability (Laurence & Perry, 1983) to a point that is tantamount to a destruction of evidence. In this case, care was taken not to suggest any specific dream content beyond enhanced recall for "whatever was dreamed the previous night." But we cannot be certain that the "memories" report-

ed by this patient actually occurred, particularly in view of the negative HIV tests.

The changes observed in the EMG may be correlates of self-generated aversive fantasy suggested by a prior movie or novel. The patient denied exposure to any movie, novel, or trauma between her ninth and tenth sessions. The careful check of the EMG instrument and electrodes with a standard EMG signal generator reduces the probability of instrument failure. Replacement of the electrodes on the patient's forehead found the same EMG level without observable evidence of any voluntary contraction of her forehead (e.g., frowning). The sustained (recorded for 33 minutes) EMG contraction spike collapsed contingent on recall of the dream. We have previously reported on a similar contingent autonomic nervous system phenomena (Wickramasekera, 1993a, 1994b). Since the relationship between the EMG spike and the verbal report of an aversive dream is correlational, no conclusions about causality can be drawn from this data. The possibility of measurement artifact is remote.

Negative Affect, Hypnotic Ability, and Dissociation

Controlled studies (Ohman & Soares, 1994) have found that phobic people experience fear as indicated by physiological measures even to phobic stimuli that they cannot consciously identify. It has been found that unexplained physiological arousal (e.g., heart rate, respiration rate, etc.) in people with high hypnotic ability is most frequently coded negatively and starts a search for causes of the negative state (Zimbardo, LaBerge, & Putten, 1993).

This patient may have self-hypnotically blocked from mind but not from body, the threat of HIV infection five years ago. The chronic blocking of threatening information or guilty memory (Mowrer, 1964; Pennebaker, Hughes, & O'Hearon, 1987; Wickramasekera, 1976) from consciousness (i.e., short term memory) is "extra work" for the autonomic nervous system. It is hypothesized that as a threatening unconscious (implicit) memory is unloaded from the body (e.g., EMG drops) and deposited in explicit memory (consciousness), the inhibitory work of muscle contraction, vasoconstriction, and SCL is reduced (Pennebaker et al., 1987; Wickramasekera, 1976, 1988, 1994b).

It appears that chronic unconscious or repressed negative memories are associated with somatic symptoms (nausea, vomiting, etc.), and with autonomic dissociation (e.g., cold and/or moist hands). But conscious negative memories may be more likely to be associated with psychological symptoms like anxiety and depression and with relatively less ANS dissociation or dysregulation (Wickramasekera, 1988, 1993a, 1994a,b, 1995). Information transfer from implicit to explicit memory appears to be associated with a shift from somatic to psychological symptoms (Wickramasekera, 1994b).

People high on hypnotic ability are at greater risk for somatoform disorders as proposed by Charcot over 100 years

ago (Wickramasekera, 1988, 1995). But this is not because of high hypnotic ability per se as proposed by Charcot, but because of the interaction of high hypnotic ability and high negative affect (Wickramasekera, 1988, 1995; Wickramasekera et al., 1996). I have theorized (Wickramasekera, 1988, 1993a,b, 1994a,b; Wickramasekera et al., 1996) that the coincidence of hypnotic ability and the unrelated trait high negative affectivity or neuroticism (Wickramasekera, 1988, 1993a, 1994a, 1995) is the core of what is today clinically described as "dissociation" or "dissociative" phenomena. This patient was high on both hypnotic ability (Harvard = 11) and neuroticism (94%). The interaction of hypnotic ability and negative affectivity (Wickramasekera, 1988, 1995; Wickramasekera et al., 1996) is hypothesized to very significantly amplify the probability of dissociative symptoms and somatization during trauma (Wickramasekera et al., 1996). Trauma (aversive sympathetic activation) appears to temporarily increase baseline hypnotic ability in all people (Wickramasekera, 1976, 1988, 1993a). But after trauma, people of low or moderate hypnotic ability return to their pre-trauma baseline hypnotic ability levels. For example, we have empirically shown that even automobile trauma can temporarily alter the perception of time, memory, and mood perhaps driving negative affect into somatization and autonomic dysregulation or dissociation (Wickramasekera, 1993a). ■

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