SOMATIC TO PSYCHOLOGICAL SYMPTOMS AND INFORMATION TRANSFER FROM IMPLICIT TO EXPLICIT MEMORY: A CONTROLLED CASE STUDY WITH PREDICTIONS FROM THE HIGH RISK MODEL OF THREAT PERCEPTION

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ABSTRACT

This is a case study of a patient presenting a variety of somatic symptoms in the absence of any identifiable pathophysiology or psychopathology. Testing with the High Risk Model of Threat Perception (HRMTP), autonomic monitoring and psychophysiological psychotherapy are associated with the retrieval and transfer of unconscious or implicit memories of sexual abuse (independently supported by court records) into explicit or conscious memory. This transfer of "repressed" memory appears to be associated with several powerful and theoretically salient consequences. First, an abrupt correlated remission of multiple somatic symptoms. Second, a correlated large increase in baseline negative affectivity, and third, large correlated changes in involuntary measures of physiological reactivity (e.g., heart rate, EDR, etc.) documented on 4 pre-post stress profiles. This inverse relationship between somatic and psychological symptoms plus the marked autonomic shift from a relatively parasympathetically dominant to a highly sympathetically reactive status raises profound theoretical questions regarding the nature and stability of the psychophysiological mechanisms implicated in the transduction of information and memory from physiological to psychological systems. Based on observations in this case study, several experimentally falsifiable predictive hypotheses derived from the HRMTP are presented.

INTRODUCTION

A well-documented case study that casts doubt on or proves an exception to current theories or empirical observations may contribute to scientific knowledge. The following case study is inconsistent with three major current hypotheses in psychopathology, memory, and psychophysiology. First, neuroticism or negative affectivity (N.A.), is hypothesized to be stable in adults (Costa & McCrae, 1986; Clark & Watson, 1991). N.A. is one of the "Big Five" in all personality taxonomies (Goldberg, 1993; Eysenck, 1991). It is one of the broad constructs underlying many specific clinical personality scales (e.g, MMPI, etc.). In fact, the re-test correlation coefficients of N.A. are .80, .60, and .64 respectively for 6 months, 1-5 years and 20 years (Clark & Watson, 1991). N.A. is partly genetically based as demonstrated by studies of monozygotic twins and dizygotic twins reared apart and reared together (Clark & Watson, 1991; Bouchard, Lykken, McGue, Segal, & Tellegen, 1990, Floderus-Myhred, Pederson, & Rasmussen, 1980; Tellegen et al., 1988). Second, the return of "repressed" memories are hypothesized to be mainly instances of the "false memory syndrome" (Loftus & Kaufman, 1992; Loftus, 1993). Third, psychophysiological stress profiles (Arena & Hobbs, 1993; Lacey & Lacey, 1962; Wickramasekera, 1976) and particularly cardiovascular stress profiles (Manuck, Kamarck, Kasprowicz, & Waldstein, 1993; Turner & Hewitt, 1992) are considered relatively stable across time and threats in adults and so is autonomic balance (Wenger & Cullen, 1972) and vagal tone (Porges, 1992; Cacioppo, 1994).

The present cognitive neuroscience literature discriminates between two classes of memory: explicit and implicit (Roediger, 1990; Schacter, 1987; Kihlstrom, 1987). Explicit memory requires conscious recall or recognition. Implicit memory does not require conscious reference to past experience, but instead assesses the impact of memory by measuring the effects of that experience on subsequent behavioral performance or psychophysiological measures (Schacter, 1987; Kutas, 1988; Kutas & Hillyard, 1984). Implicit memory can be called "Body Memory." The desynchrony between performance on the two types of memory tests can be seen most dramatically in brain-damaged amnesic patients (Schacter, 1987) whose memory is found to be significantly impaired or even absent when tested explicitly, yet whose memory appears to be less impaired when tested implicitly. The following case study illustrates the psychological, physiological and symptomatic consequences of the disruption of a hypothesized functional dissociation between implicit and explicit memory and its implications for certain hypothesized mechanisms of somatization (Wickramasekera, 1979, 1986, 1988, 1993).

CASE REPORT

The following case study, which manipulated neither surgery nor drugs, illustrates the profound consequences for N.A., memory, and the autonomic nervous system of the recovery of an *independently* documented "repressed" memory of sexual abuse. The "repressed" memories entry into consciousness or explicit memory (Roediger, 1990; Kihlstrom, 1987) appeared to be associated with large changes in typically stable psychological (N.A.) (Costa & McCrae, 1986; Clark & Watson, 1991; Bouchard et al, 1990; Floderus-Myhred et al., 1980; Tellegen et al., 1988; Eysenck, 1991) and involuntary physiology (Arena & Hobbs, 1993; Lacey & Lacey, 1962; Manuck et al., 1993; Turner & Hewitt, 1992; Wenger & Cullen, 1972; Porges, 1992; Cacioppo, 1994) measures like heart rate, blood volume pulse, peripheral skin temperature, electrodermal response, etc.

A 35-year-old white married engineer, mother of three children, was referred by her neurologist. She presented with episodic "vasovagal syncopy," dizziness, bilateral headaches, tinnitus, idiopathic episodic flushing, sweating, episodic blurred vision and episodic problems with sensory-motor coordination of sudden onset twelve months ago without any premorbid history. The clinical interview revealed that these symptoms became more frequent and intense both at work and home with apparently no relationship to any identifiable environmental or interpersonal stressors in her marriage (e.g., sex life), family (children, parents), or work situations. Multiple medical tests (MRI, endocrine test, EEG, spinal taps, etc.) and verbal interviews with specialists and sub-specialists (2 cardiologists, 2 neurologists, 2 gynecologists, 1 endocrinologist, etc.) were negative for pathophysiology or psychopathology. The referring neurologist had prescribed Bellergal, a drug containing a combination of phenobarbital, ergotamine tartrate, and belladonna alkaloids that is often prescribed for symptoms associated with "functional" somatic disorders, and is believed to restore the "balance" between sympathetic and parasympathetic autonomic activity. This regimen resulted in a 50% reduction in the symptom of syncopy, but did not affect other symptoms. Three months prior to the present referral the patient had tried to reduce the Bellergal,but the syncopy had increased, and remained worse for nearly one and one-half months.

Because the patient and her neurologist were uncomfortable with an "empirical chemical therapy" that had not improved her other (headaches, tinnitus, etc.) somatic symptoms, she was referred to me for evaluation of "continuing ANS dysfunction etiology undetermined." Medical reports indicated that she denied any current distressing events at work, home, or in her marriage. Nor were there any distressing memories of stressors from the recent or distant past. My own intensive psychological interview found no evidence of *DSM-III-R* diagnosable psychopathology nor any evidence of current distressing events or memories of recent or remote distressing events. The patient reported only somatic symptoms. The patient was given a graph to record daily the frequency of all her somatic symptoms. The patient was scheduled for testing on the High Risk Model of Threat Perception (Wickramasekera, 1979, 1986, 1988, 1993), and psychophysiological stress testing (Wickramasekera, 1976, 1988, 1993) for risk factors for somatization.

I have found the High Risk Model of Threat Perception (HRMTP) a useful tool for the investigation of somatic complaints without identifiable pathophysiology or psychopathology (Wickramasekera, 1979, 1986, 1988, 1993). The HRMTP identifies a set of psycho-social factors that have been empirically associated with the risk of 1) transducing or 2) amplifying somatic complaints independent of pathophysiology (Wickramasekera, 1988, 1993). *Transducers* are patients who are hypothesized to present implicit threatening perceptions and/or memories as somatic symptoms, without any identifiable pathophysiological mechanisms. Symptom formation in transducers is hypothesized to be associated with a reduction in sympathetic nervous system baselines. In *Amplifiers*, patients who amplify, implicit or explicit perceptions and/or memories of threat are hypothesized to potentiate somatic symptoms which are based on pathophysiologic mechanisms. Amplifiers are predicted to show higher baseline sympathetic nervous system activity than transducers. Hence, the HRMTP may provide an approach to the differential diagnosis of somatization by inclusion, avoiding the serious problems (eg., inappropriate or insensitive biomedical tests) involved in diagnosis of somatization by exclusion (Wickramasekera, 1988, 1993; Smith, 1990) alone. The HRMTP identifies psycho-social factors in threat perception that are hypothesized to consciously or unconsciously drive somatic symptoms, without or with pathophysiology. These risk factors are: (1) high hypnotic ability marked by high (12-9) Harvard (Shor & Orne, 1962) scores, or low (0-4) Harvard scores; (2) cognitive catastrophizing measured with the Zocco scale (Zocco, 1984); (3) high overt (Eysenck > 75%) or high covert (Eysenck N < 25% and L > 75%) N.A. or neuroticism (Costa & McCrae, 1986; Clark & Watson, 1991; Bouchard et al., 1990; Floderus-Myhred et al., 1980; Tellegen et al., 1988; Weinberger, 1990) measured by the Eysenck Scale (Eysenck & Eysenck, 1968); (4) major life changes over 300 LCU (Holmes, 1981); (5) high hassles or microstressors (Kanner, Coyne, Schaefer, & Lazarus, 1981); (6) low number and satisfaction with social supports (House, Landis, & Umberson, 1988) measured by the Sarason, Levine, Bashan, & Sarason (1983) scale; and (7) low coping skills (Lazarus & Folkman, 1984; Rosenbaum, 1980). The High Risk Model has three components called *predisposers* (hypnotic ability, catastrophizing, neuroticism), *triggers* (major life change and hassles) and *buffers* (social support and coping skills). The predisposers are personality variables, the triggers are situational variables, and the buffers are attenuating constructs at the interface of personality and situations. The interaction of these predisposing, triggering, and buffering variables are hypothesized to account for the bulk of the variance in predicting the onset and stability of threat-related somatic and psychological symptoms. These risk factors are totally or partially orthogonal (Wickramasekera, 1988, 1993, in press) and their interaction is predicted to attenuate or potentiate somatic and psychological symptoms.

On pre-therapy testing this patient's profile on the

HRMTP was positive for only two of seven risk factors for somatization (see Table 1): (1) High Covert N.A. or "repressed" neuroticism (Jamner, Schwartz, & Leigh, 1988; Weinberger, 1990; Weinberger, Schwartz, & Kristeller, 1979) defined as L = 98%, and neuroticism = 3% (Eysenck & Eysenck, 1968) and (2) High hypnotic ability marked by a Stanford Form C=9 (Weitzenhoffer & Hilgard, 1962). But this patient did not initially show the elevated (Jamner et al., 1988; Weinberger, 1990; Weinberger et al., 1979) heart rate, skin conductance, or muscle tension found in non-clinical college student "repressors" (Jamner et al., 1988; Weinberger et al., 1979). It is hypothesized that the interaction of high hypnotic ability (e.g., the capacity to block the current perception or memory of surgical pain) and repression (low N.A., high L) can transduce threat perception into somatic symptom that can potently block even baselines sympathetic physiology. But when a patient is episodically symptomatic on a state or situation specific basis sympathetic reactivity and ANS balance will be altered. The pre-stress profile data (see Table 2) in this study was collected when the patient was not in a symptomatic episode and prior to the transfer from implicit to explicit memory. On the Stanford the patient had total post-hypnotic amnesia that was totally reversible. High hypnotic ability is associated with post-hypnotic amnesia and the capaci-

TABLE 1
Scores on High Risk Model of Threat Perception (HRMTP)

PREDISPOSERS	**Stanford Form C** Pre	**Harvard Form A** Post 1 (6 months)	Post 2 (12 months)
Hypnotic Ability	9	9	
Catastrophizing (Zocco, 1984)	24	40	
Neurotocism (Eysenck & Eysenck, Form A, 1968)	3%	27%	47%
Lie	98%	92%	83%
Extraversion	52%	1%	16%
TRIGGERS			
Major Life Changes (Holmes, 1981)			
Total	369.0	617.0	
1 year	205.0	373.0	
2 years	29.0	173.0	
3 years	135.0	71.0	
Hassles (Kanner et al., 1981)			
No.	23.0	36.0	
Intensity	1.0	1.5	
BUFFERS			
Support Systems (Sarason et al., 1983)			
No.	6.1	4.7	
Satisfaction	5.9	4.3	
Coping Skills (Rosenbaum, 1980)	77.0	58.0	

ty for other distortions of memory (Kihlstrom, 1987; Dywan & Bowers, 1983; Laurence & Perry, 1983). During hypnosis the patient reported the perception of "involuntariness" (Bowers, 1982), the current "litmus test" of hypnotic ability (Kirsch, Council, & Wickles 1990). Her Bowers (Bowers, 1982) "involuntariness" score was 46/60. The patient was surprised at the magnitude of her reversible amnesia response on the hypnosis test. Six months later on post-therapy hypnosis retesting her Harvard (Shor & Orne, 1962) test score was also 9. On the Harvard subjective involvement in hypnosis measure (Kirsch, Council, & Wickles, 1990) her score was 48/60, and on the Harvard measure of "involuntariness" (Bates, Dinges, Whitehouse, Orne, & Orne, 1991) her score was 94/120. The above multiple measures established that this patient had high hypnotic and amnesia ability, which is a marker of the ability to keep secrets from the "self" (Kihlstrom, 1987). The five other HRMTP factors were absent from her first test profile (see Table 1).

Table 2 shows her pre-post therapy psychophysiological testing. I use the J and J computer system model no. I-330 which monitors and performs basic data reduction of physiological responses. Psychophysiological stress testing con-

TABLE 2
Psychophysiological Stress Profile (Wickramasekera, 1976, 1988)

		EO		EC		STRESS		EO		EC	
		x	sd	x	sd	x	sd	x	sd	x	sd
TEMP	**Pre**	91.92	0.40	92.02	0.16	91.02	0.91	91.69	0.52	92.16	0.19
	1 Post	81.52	0.67	80.23	0.12	80.08	0.12	79.15	0.31	81.71	1.32
	2 Post	71.49	0.04	71.63	0.13	72.51	0.26	72.78	0.19	73.93	0.11
	3 Post	77.72	0.59	81.66	1.07	82.62	0.50	80.28	0.69	79.68	0.91
HR	**Pre**	75.43	3.76	76.06	3.12	79.36	4.77	72.66	2.76	72.79	2.49
	1 Post	92.10	3.59	95.49	3.86	114.64	10.21	91.70	5.10	91.30	3.84
	2 Post	91.16	3.21	91.83	4.19	97.03	6.01	86.20	3.49	87.11	4.24
	3 Post	76.05	3.18	77.33	4.11	85.14	4.84	78.59	3.84	78.06	3.38
EDR	**Pre**	4.37	0.26	4.19	0.14	7.99	1.29	6.52	0.79	5.35	0.36
	1 Post	24.47	0.69	24.09	1.50	32.99	1.09	33.20	1.03	32.27	0.87
	2 Post	4.95	0.29	4.64	0.33	8.52	0.43	7.10	0.26	6.23	0.64
	3 Post	3.00	0.16	1.98	0.24	6.95	1.52	6.03	0.98	4.00	0.51
EMG	**Pre**	1.10	0.41	1.27	0.34	2.96	0.93	1.12	0.28	1.42	0.31
	1 Post	2.83	0.53	3.49	2.12	18.47	6.02	2.94	0.60	3.36	0.51
	2 Post	3.74	1.52	3.56	0.82	13.75	5.89	2.80	0.65	3.08	1.17
	3 Post	1.67	0.58	1.72	0.22	7.19	3.54	1.37	0.23	1.73	0.28
BVP	**Pre**	34.51	4.57	33.65	2.08	15.98	5.56	31.52	2.16	30.56	4.08
	1 Post	14.82	3.59	19.84	3.18	11.25	1.41	14.55	2.65	16.45	3.90
	2 Post	11.04	2.53	15.31	4.16	8.88	1.25	13.84	3.39	12.27	3.28
	3 Post	14.91	4.35	18.20	2.92	5.94	1.80	7.29	1.25	14.55	4.33

The three Post tests are approximately 6, 12, and 24 months after the Pre-therapy test.

sists of 5 components of 4 minutes each of physiological computer monitoring of 5 channels of physiology in a temperature regulated, sound attenuated, and electrically-shielded psychophysiology laboratory. The channels are (1) peripheral skin temperature or TEMP, (2) heart rate or HR, (3) skin conductance or EDR, (4) frontal EMG, and (5) blood volume pulse or BVP). All standard procedures were used in adapting the patient to the lab and in attaching transducers (Andreassi, 1989). Each of the 5 components is constituted of 4 minutes (mean of 60 data points) each of physiological monitoring under conditions of eyes open (EO), eyes closed (EC), stress (standardized cognitive mental arithmetic stress), and return to baseline EO and EC. Pre-therapy psychophysiological testing shows a general pattern of parasympathetic dominance (Wenger & Cullen, 1972) or high vagal tone (Porges, 1992; Cacioppo, 1994) reliably across five separate measures of relatively low and stable sympathetic activation before, during and after cognitive stress (see Table 2). Table 3 shows her pre-post psychological testing on conventional tests like the SCL-90-R (Derogatis, 1977), Marlowe Crowne (Crowne & Marlowe, 1960), Beck Depression Inventory (Beck, 1972), and NEO (Costa & McCrae, 1989). This pre-therapy psychological testing reveals a remarkably normal patient. All of her pre-therapy SCL-90-R scores are in the average or low range. Her pre SCL-90-R provides no basis for a *DSM III-R* diagnosis of any kind (General Severity Index or GSI<63 and all primary clinical dimensions are <63). This supports the impression of a lack of explicit memory for psychopathology from the multiple prior clinical interviews. The problems with *DSM-III-R* diagnosis of somatization

TABLE 3

	T-SCORE	
SCL-90-R, VERSION 2.1	**Pre**	**Post**
1 Somatization	51	49
2 Obsessive-compulsive	62	50
3 Interpersonal sensitivity	50	68
4 Depression	46	62
5 Anxiety	37	57
6 Hostility	54	48
7 Phobic anxiety	44	44
8 Paranoid ideation	41	60
9 Psychoticism	44	65
General severity index	49	60
Positive symptom distress index	54	55
Positive symptom total	47	58
MARLOWE-CROWNE (Measure of social desirability or "self-deception.")	17	13
BECK DEPRESSION INVENTORY	4	13
NEO (Costa & McCrae, 1989)		
Neuroticism	30	92
Extraversion	125	103
Openness	133	129
Agreeableness	52	49
Conscientiousness	58	62

TABLE 4
Protocol for Psychophysiological Psychotherapy
(Flow Chart)

TECHNICIAN:		
1.	Instrumentation	4 minutes
2.	B^1 (eyes open)	3 minutes
3.	B2 (eyes closed)	3 minutes
THERAPIST:		
4.	S1 Psychotherapy	25 minutes
5.	R1 Hypnosis (to soothe)/ Biofeedback (to self-soothe) to reduce physiological arousal	15 minutes
6.	S2 inquiry about spontaneously disinhibited cognitions, images or emotions during low arousal	10 minutes
TOTAL:		60 minutes

disorders (Katon et al., 1991; Smith, 1990) which are based on explicit or objective verbal report measures (clinical interviews and verbal report psychological tests) alone have been discussed elsewhere (Wickramasekera, 1988, 1993, 1994a). There is no evidence on the SCL-90 from her Positive Symptom Total (P.S.T.), or Positive Symptom Distress Index (P.S.D.I.) of conscious or explicit "faking good" (PST=14). The patient's pre-therapy "Big Five" NEO (Costa & McCrae, 1986) (Table 3) scores are all in the normal or positive range and her level of neuroticism on both the NEO (score of 30) and Eysenck (3%) are very low. (See Table 2). In a large sample (N=189) of similar somatizing patients (Wickramasekera, 1988, 1990, 1991, 1993, in press) it was found that hypnotic ability, neuroticism and even major life change are orthogonal.

Therapy

This is an abbreviated description of a complex clinical tool described elsewhere called psychophysiological psychotherapy (see Table 4) designed to investigate mind-body interaction (Wickramasekera, 1988, 1989, 1990, 1991, 1993). Psychophysiological psychotherapy consists of a six-component procedure (see Table 4) with concurrent physiological monitoring. It is targeted to reduce the autonomic correlates of threat-perception, to increase cognitive creativity, and to inhibit cognitive stereotypy. Cognitive creativity is hypothesized to be enhanced by placing a period of low physiological arousal between psychotherapy and psychotherapeutic inquiry. This period of self-soothing low arousal is hypothesized to (1) increase cognitive creativity and cognitive reappraisal in perceiving old conflicts, and to (2) increase input from the cognitive long-term memory and the unconscious mind (Wickramasekera, 1988, 1990, 1991, 1993; Kihlstrom, 1987). Its goals are (1) to disrupt cognitive-emotional stereotypy in threat-perception by using hypnosis or biofeedback to reduce autonomic nervous system correlates of threat perception; (2) to disinhibit relevant long-term memories, images, and emotions to amplify the probability of fresh or creative cognitive reappraisals of relevant threat perceptions.

This description of the patient's therapy is greatly abbreviated. In the first session of therapy the patient was trained in self-hypnosis and her self-hypnosis was first focused on hand-warming. In the second session of psychophysiological psychotherapy the patient's baseline hand temperature dropped from 91°F to 75°F when she was asked to describe in detail the circumstances under which her somatic symptoms started. Her somatic symptoms started while enrolling her oldest son in a nursery school program 12 months ago. While verbally reporting the above events her electrodermal response jumped from 3 micro ohms to 20 micro ohms and there was marked constriction of blood volume pulse (BVP dropped from 37 to 4). There was no change in environmental factors or in the patient's overt motor activity during this episode that could account for these temperature, EDR, and BVP changes. The above physiological changes indicate "on-line" sympathetic activation or the perception of "threat." I hypothesized this sympathetic activation to index stimulation of an unconscious or implicit state-specific memory of threat which was not observable to me in her overt behavior or verbal report. I printed a record of her temperature, EDR, and BVP and handed it to her without comment. The patient reported surprise on seeing the printed data on her physiology and stated that she had not perceived this sympathetic activation. This procedure appears to create cognitive dissonance and introspection in the patient. During the hypnosis/biofeedback or fifth component (see Table 4) of this therapy session I instructed her to return in hypnosis (time regression procedure) to the previously mentioned nursery school registration situation 12 months ago, but to observe the situation as a calm detached spectator. She exited this hypnosis phase with a spontaneously occurring memory image of herself as "a sad child in nursery school looking out of a window at other children playing in the yard, but confined to the classroom by a scary male principal."

During this limited time regression hypnotic procedure the sympathetic activation continued and her heart rate ranged

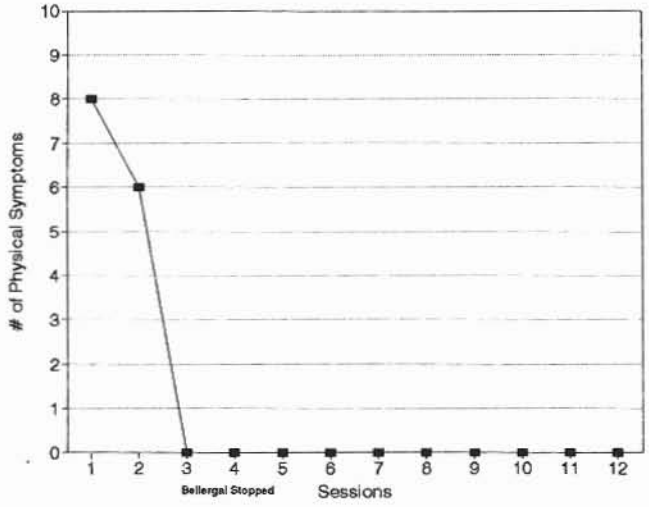
between 90-112 BPM. At the termination of her second therapy session, I suggested outside of hypnosis (component 6: inquiry phase, Table 4) that "other memories of her childhood might return during the coming week and she should record them and bring them to her next session."

The patient returned to the third (7/16) session (see Figures 2 and 3) very anxious and sympathetically activated (episodic heart rates of 95-135) with a written record of several "involuntary" or intrusive vivid memories of sexual abuse by a male nursery school principal. Repeated use of the self-hypnosis procedure in the psychophysiological laboratory and at home was associated with more implicit memories of sexual abuse becoming explicit. After her third therapy session (7/16) these implicit memories of sexual abuse started to intrude involuntarily into routine consciousness, for example, while at work, while cooking, and while dressing her children. She recalled the abuser's anger with her for "bleeding" and "making a mess" during their "games." She reported a lifetime history of her visual fields becoming blurred or destructured for several seconds whenever she first entered a room with a bathtub. This visual phenomenon terminated after she recalled the principal holding her head under water in a bathtub, to block her crying while he washed out her bleeding vagina. She recalls him "sealing" her by telling her she was a "bad girl" and that "her parents would not like or love her" if she told them about their "games."

In view of this patient's high hypnotic ability and therefore her capacity for memory distortion and creation (Dywan & Bowers, 1983; Laurence & Perry, 1983) it is particularly appropriate to be skeptical about these verbal reports of sexual abuse and to seek independent confirmation of them. First, after the transfer of the traumatic memories from implicit to explicit memory there were very large (e.g., 10-20 point changes in baseline EDR, hand temperature, and heart rate) and reliable changes in her autonomic baselines and cognitive stress reactivity on her first post stress profile (Table 2). These changes are indicative of an elevated perception of threat or "fight and flight" response. Also during therapy reviews there was a marked change in her heart rate and EDR (Figures 2 and 3) across sessions. Reliable baseline changes of this magnitude in ANS function are typically related only to chemical or surgical intervention and not to changes in cognition and memory. Second, support for the patient's "recollections" came from her mother's independent testimony that two years after the patient had left nursery school and was in elementary school, the principal of her prior nursery school was convicted, according to a newspaper report, of child molestation. Hence the abuse apparently occurred at age four years. Her mother explicitly denied mentioning this report to her husband or children and it is unlikely that a six-year-old would have read this newspaper report or understood a television report of molestation. I saw a copy of the 29-year-old newspaper report and the court records documenting a conviction based on the independent testimony of three female children. The conviction of this patient's principal was published many years prior to the present epidemic of child molestation allegations. The patient's mother also confirmed at least two lifetime episodes of abnormal psychological symptoms in the patient that were only investigated medically and had remitted spontaneously. These incidents were (1) a school phobia at age five years, and (2) a period of severe nightmares at age six or seven.

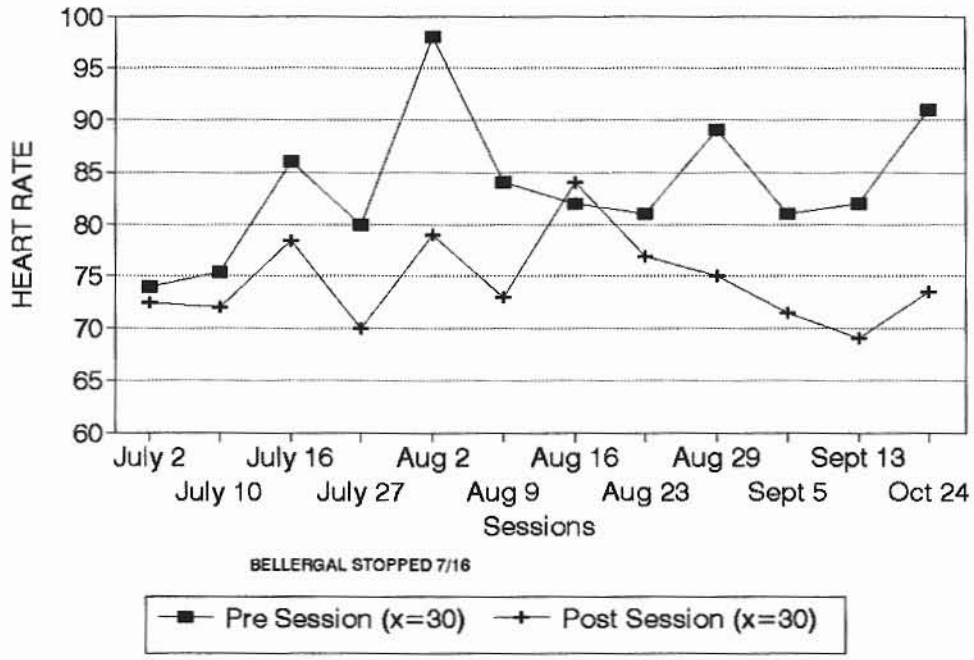
During intensive therapy (see Figures 2 and 3, 7/2-10/24) the patient's perspective on her whole life to date changed. Specifically, she became critical of her husband's and her parents' refusal to listen to or to discuss her fearful and/or angry feelings about her new memories and her efforts to assimilate them into her present life situation on a cognitive basis. She was particularly angry because her mother, a medical professional, has failed to recognize the physical trauma from the sexual abuse. She perceived pressure from her husband and mother to "stuff" her anxiety, anger, and depression, to look "chipper," and to "go on with her life as wife and mother." They insisted that she "put the past behind." She considered divorcing her husband and felt that he has "used" her as a "housekeeper and sex partner." She is considering quitting her engineering job and applying for doctoral training. Within three sessions of starting to unload and process these childhood memories, the patient terminated the Bellergal (7/16) without any relapse of syncopy or her other somatic symptoms (see figure 1). In this connection it is significant to recall that the pre-therapy effort to stop the Bellergal had been associated with an amplification of her somatic symptoms that persisted for one and one-half months even after the resumption of the medication.

FIGURE 1
Changes in the Number of Somatic Symptoms
Reported per Session
(Over 20 sessions of psychophysiological psychotherapy)

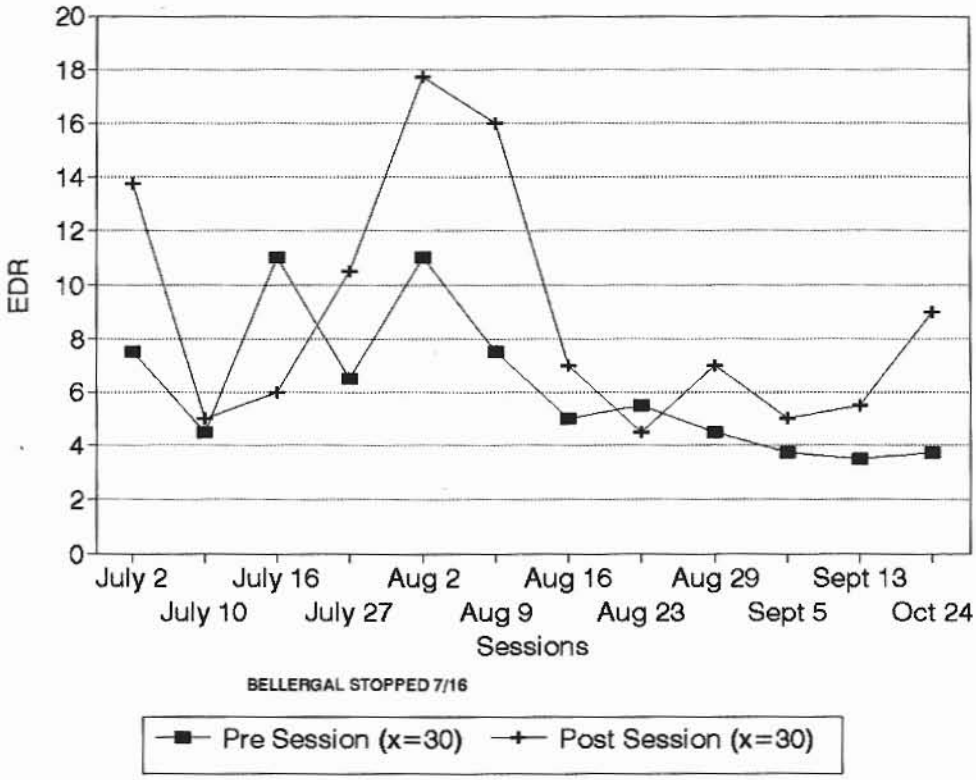


TABLES 2 & 3
35 Year Old WMF with Vasovagal Syncope. Implicit (Unconscious) Perception of Threat Triggered by Recall of Traumatic Childhood Memories of Sexual Abuse from Age Four.

Amplified Attention to Internal Events? Explicit Threat Perception Increasing?



Implicit (Unconscious) Threat Perception Decreasing?



Two Year Follow-Up

The patient has been seen in long term psychophysiological psychotherapy which focused on issues of her self-esteem, vocational plans, and the intrusive images of the sexual abuse occurring during sexual arousal and intercourse with her husband. There has been only one episode of syncopy since this symptom stopped during psychotherapy about two years ago. This one-minute episode of total syncopy occurred during the intrusion of a very aversive memory of sexual abuse. During episodes of marital conflict the patient has had mild to moderate bilateral headaches of 2-8 hours duration which respond well to the use of her self-hypnosis training for hand warming. In spite of stopping the Bellergal almost two years ago, the somatic symptoms (tinnitus, idiopathic flushing, sweating, blurred vision, and impaired sensory motor coordination) have not returned. No new somatic symptoms have been observed. Her baseline (EO, EC) heart rate, EMG, and skin conductance (EDR) have returned to pre-therapy levels (see Table 2).

At first her psychological symptoms increased, but more recently they have reduced in intensity. The Eysenck Personality Inventory (see Table 1) shows extroversion (positive affectivity) initially reduced, but currently increasing. Her level of negative affectivity on the Eysenck has steadily climbed during the course of therapy, suggesting an increase in introspection (Costa & McCrae, 1986; Clark & Watson, 1991). Her Lie Score has declined only modestly across therapy sessions. Her Lie Score, her Marlowe-Crowne or high self-deception score, (see Table 3) and her high hypnotic ability score (Table 1) have remained fairly unchanged, suggesting that she has two stable mechanisms for transducing psychosocial stress into somatic symptoms (Wickramasekera, 1988, 1993, 1994a). But peripheral skin temperature and blood volume pulse (BVP) have not returned to her pre-therapy baselines. This suggests some relatively permanent changes in her pattern of peripheral circulation and an actual paradoxical increase in skin (PTI) temperature during cognitive stress (Wickramasekera, 1979; Wickramasekera, Kolm, Pope, unpublished research) which we have observed in approximately 52% of chronic pain patients and 31% of the grossly obese.

We have repeated her psychophysiological cognitive stress profile (see Table 2) approximately every six months since the original pre-therapy profile. Table 2 shows some reduction in both her baseline sympathetic tone and sympathetic reactivity to cognitive stress in heart rate and, particularly in EDR which is purely sympathetically innervated (Boucsein, 1992). But moderate or no return to her pre-test baseline reactivity to cognitive stress in terms of skin temperature, heart rate, EMG or blood volume pulse.

DISCUSSION OF RELEVANT THEORETICAL ISSUES

Note that threatening information transfer from implicit to explicit memory is associated with increased N.A. or neuroticism on the Beck, Eysenck, The SCL-90 and the NEO (see Tables 1 and 3). In this study there was no demonstration of implicit memory for sexual abuse in a strict cognitive laboratory sense. Current cognitive science technology does not permit testing this specific hypothesis. But multiple careful independent clinical interviews, multiple verbal reports, and psychometric test data of known validity and reliability provide an analogue of explicit cognitive science laboratory tests. Several pre-therapy explicit psychometric tests failed to find even indirect evidence (e.g., depression, anger, anxiety, etc) of sexual abuse. These tests found no explicit or conscious recollection of memories of sexual abuse prior to the hypnotic "time regression" procedure. Prior to the "time regression" procedure no effort was made to suggest or elicit memories on any specific type, much less memories of sexual abuse. In fact because of the current rash of childhood sexual abuse charges and the patient's high hypnotic ability I was particularly cautious and skeptical about even her later intrusive memories during work and housekeeping. I explicitly instructed the patient to avoid the deluge of popular books on abuse. In spite of Loftus' disagreement (1993), there is some empirical evidence (Ceci & Bruck, 1993) that older children are able to encode and recall accurately personally meaningful information from childhood. Enrolling her child in a nursery school triggered an unconscious situation-specific somatized post-traumatic stress disorder in this patient based on her protective maternal instincts. This account is also consistent with an identification with her daughter's vulnerable situation. The shift on the NEO neuroticism scale (see Table 3) from 30 to 92 is particularly noteworthy. A "Big Five" personality construct like neuroticism is believed to be a stable (Costa & McCrae, 1986; Clark & Watson, 1991) and partly genetically based personality trait (Bouchard et al., 1990; Floderus-Myhred et al., 1980; Tellegen et al., 1988) that is resistant to change in adult life (Costa & McCrae, 1986; Clark & Watson, 1991) unlike specific MMPI clinical scales.

The patient's four pre-post Psychophysiological Stress Profiles show large changes (See Table 2). Note the large magnitude changes in skin conductance, blood volume pulse, frontal EMG, heart rate, and peripheral skin temperature at baseline, during and after cognitive stress. It is worth noting that these measures were collected many months apart. There has been some prior controversy regarding the stability of some measures on the psychophysiological stress profile, recently (Arena & Hobbs, 1993; Manuck et al., 1993) this has been resolved by the use of the correct statistic (e.g., intra class correlations) and methodolgy (Manuck et al., 1993; Turner & Hewitt, 1992). These measures are known to be relatively stable (Lacey & Lacey, 1962; Manuck et al., 1993; Turner & Hewitt, 1992; Wenger & Cullen, 1972; Morales-Ballejo, Eliot, Boone, & Hughes, 1988; Cacioppo, 1994). It is possible that major shifts in autonomic nervous systems balance may be a promising independent index of the transfer of traumatic memories from implicit to explicit storage.

This patients post-therapy elevated N.A. baseline and her chronic hyper-vigilant sympathetic status may be associated with the transfer of implicit or unconscious memories of threat into chronic explicit or conscious memories of threat. It is hypothesized that as the unconscious threatening memories of childhood moved "out of her body" and into her

conscious mind, her level of sympathetic hyper-vigilance and psychological anxiety and depression increased, driving up her level of N.A. or neuroticism. It is surprising that such large and reliable changes in autonomic physiology and N.A. occurred with only changes in "software" (cognition and memory). More generally this is a clear demonstration of large and stable changes in autonomic physiology secondary to changes in perception, memory and mood. The profound change in autonomic balance (Wenger & Cullen, 1972) or vagal tone (Porges, 1992; Cacioppo, 1994) was associated with and perhaps driven by a shift of N.A. from repression (Weinberger, 1990; Weinberger et al., 1979) in implicit memory to consciousness or explicit memory (see changes in Tables 1,2 & 3). Note (Tables 1 and 3) associated changes in catastrophizing, major life change, hassles and depression (Beck scores), plus drops in coping skills and support systems secondary to information transfer from implicit to explicit memory. But note only modest reductions in her L% score (98% to 92%) and Marlowe-Crowne (M-C) scores (17 to 13). Her Marlowe-Crowne score and Eysenck Lie scores are measures of social desirability, self-deception, or repression (Jamner et al., 1988; Weinberger, 1990; Myers & Brewin, 1994) and elevations on the Marlowe-Crowne have been shown to be related to low lifetime prevalence of psychological symptoms (Lane, Merikangas, Schwartz, Huang, & Prusoff, 1990). This is supportive of the position that social desirability measures reflect substance and not style (McCrae & Costa, 1983). There may be a positive correlation between somatization and Marlowe-Crowne scores during cognitive threat. These self-deception measures changed only modestly in spite of major changes in physiology, and N.A. (depression, anxiety, paranoid ideation, psychoticism, etc.). Her somatic symptoms (See Figure 1) were replaced by psychological symptoms (higher N.A.) but her trait of self-deception changed only slightly. This shift from somatic to psychological symptoms associated with "insight" is consistent with predictions from Freudian theory. Note that her hypnotic ability also remained stable in spite of the alteration in N.A. and autonomic physiology. As her explicit or conscious memories of prior sexual abuse and "damage" expanded, her explicit suffering increased (N.A.) but at least two aspects of her personality remained stable (social desirability and hypnotic ability). Hypnotic ability (Stanford Form C or Harvard Form A) and social desirability (Marlowe-Crowne scores) have been empirically shown to be orthogonal measures (Wickramasekera, 1990, 1993; Palsson, 1992; Remler, 1990) and stable features of human personality that are hypothesized to predispose people to somatization (Wickramasekera, 1979, 1988, 1993, 1994; Jamner et al., 1988; Weinberger, 1990). The probability of transducing psychosocial threat into somatic symptoms may remain a permanent feature of these patients when they are under implicit or explicit threat (Wickramasekera, 1988, 1990, 1991, 1993). Because "out of mind may not be out of body" (Wickramasekera, 1988, 1993, 1994). Freud notwithstanding, information and not energy transfer between physiological and psychological systems may be the only constant in the universe of psychobiological events.

Figures 2 and 3 show the means for pre and post heart rate (BPM) and skin conductance (EDR) for each session of psychophysiological psychotherapy, across the first 12 therapy sessions. They represent means of the first and last 30 data points at the start and end of each therapy session. Pre-session heart rate appears to increase across sessions while pre session EDR drops across sessions. Pre-session EDR increases erratically across therapy sessions but at the sixth session (8/9) begins to drop across sessions. But within sessions EDR increases in approximately 75% of the sessions. This shift within and across sessions in skin conductance is consistent with Pennebaker's Model of Self-Disclosure (Pennebaker, Hughes, & Heeron, 1987). This pattern of change in heart rate and skin conductance is hypothesized to mark the most significant sessions of cognitive-affective processing and transferring of information from implicit to explicit memory. The general drop in mean pre-session EDR across 12 sessions (7/2-10/24) is hypothesized to mark reductions in the patient's need for the chronic autonomic work of repression (Pennebaker, Hughes, & Heerson, 1987). Each series of data, pre-session and post-session BPM and EDR, were analyzed by time series regression analysis. For pre-session heart rate, there was no significant trend over the 12 sessions ($p = .2174$). The series is best described as a stationary series with a mean of 83.92 + 2.02 (standard error). For post-session heart rate, there was also no significant trend over the 12 sessions ($p = .8503$). This series has a mean of 74.25 + 1.25 (standard error). For pre-session EDR, there was a significant linear trend (monotonically decreasing) over the 12 sessions ($p = .0240$). The regression coefficient was -.45 which means that EDR decreased at a rate of .45 units per session. The regression equation was: EDR = 8.997 - .45 (sessions). For post-session EDR, there was no significant linear trend over the 12 sessions ($p = .4839$). Tests for a non-linear trend were also non-significant ($p = .5499$). Thus, the post-session EDR series is best described as a stationary series with a mean of 8.42 + 1.43 (standard error).

The patient's pre-session heart rate appears to paradoxically increase across sessions (7/2 = 75 BPM - 10/24 = 91 BPM), but post-session heart rate (BPM) appears to drop within sessions. This mean change of 16 BPM pre-session from session 1 to session 12 is substantial. This progressive increase in heart rate across sessions may be a situation-specific response marking positive transference, and/or indicating growing anticipation and/or skill in attending to internal events or introspection (Lacey & Lacey, 1978), that is trained by psychophysiological psychotherapy (Wickramasekera, 1988, 1989, 1993). This pre-session increase in heart rate appears to occur in spite of the fact that each prior therapy session ends with a low arousal induction phase and demonstrates an empirically documented drop in heart rate at the end of 91% of therapy sessions (see Figure 2: heart rate difference between pre- and post-session). Prior basic empirical research in psychophysiology (Lacey & Lacey, 1978) suggests that attentional focus on internal events (cognitive work or introspection) is associated with increases in heart rate but attention to external events (external auditory or visual stimuli) is associated with drops in heart rate. The changes in EDR, heart rate and skin temperature and frontal EMG on pre-post stress testing support a hypothesis of greater vigilance or enhanced attention to both inter-

nal and external events (59) Transducers are defined to be people who present somatic symptoms in the absence of any identifiable pathophysiology to account for the somatic symptom and who have transduced psychological threat into somatic symptoms (Wickramasekera, 1988, 1993). It is our clinical observation that only the "transducing" or "repressing" (Wickramasekera, 1988, 1993) type somatizer shows lower heart rates at the start of psychophysiological psychotherapy and will show higher relative left hemisphere activation (Tomarken & Davidson, 1994) and higher vagal tone or parasympathetic dominance (Wenger & Cullen, 1972; Porges, 1992). This parasympathetic dominance or high vagal tone in "transducers" (Wickramasekera, 1993) is probably the physiological basis of what Charcot called "la belle indifference" in hysteria. We reliably observe an increase in heart rate and drop in EDR across therapy sessions in the transducing-type of somatizer (Wickramasekera, 1993) whose symptoms remain in remission as a function of long-term psychophysiological psychotherapy (Wickramasekera, 1988, 1989). We do not observe the above progressive increases in heart rate in somatizing patients who drop out of therapy after their symptomatic "fires" remit but before learning verbal introspective skills to process emotions. Heart rate increases only in those patients who not only put out the symptomatic "fires" but also develop introspective skill at finding "the matches."

In the above case, apparently the memory of sexual abuse was first represented indirectly in the psychological symptom of school phobia at ages four or five and nightmares at ages six or seven. But these symptoms were treated non-specifically or medically, and not specifically or psychologically (Wickramasekera, 1985) with psychotherapy for trauma. "Non-specific" medical-drug therapy, effectively a placebo therapy (Wickramasekera, 1985), was associated with the predicted repression (avoidance-reinforcement) of this childhood threat. Hence my conditioned response theory of the placebo effect (Wickramasekera, 1985) would predict that future psychological activations (26 years later) of this long-term repressed implicit memory of threat are likely to be presented somatically or medically. It is possible that the partial success of the prior non-specific drug therapy (effectively a placebo therapy) of her psychological trauma (sexual abuse) reinforced somatization tendencies in the patient.

It is worth noting that this particular patient at baseline demonstrated two orthogonal mechanisms, high hypnotic ability, and high social desirability, that have been empirically shown to be able to insulate threat in implicit memory and perception from explicit memory and explicit perception. It is hypothesized that these separate mechanisms and particularly their interaction with high covert N.A. or neuroticism (Wickramasekera, 1988, 1993, 1994) can generate somatic and psychological symptoms. The reliable drop in heart rate within sessions is consistent with an interpretation of internal information processing of threat to the point of the extinction of explicit conscious threat within the session. The recurrent pattern of increasing EDR within therapy sessions and reducing EDR across sessions is consistent with confronting and extinguishing unconscious threatening implicit memories and perceptions across therapy sessions. Heart rate may reflect a desensitization of implicit memory within sessions and a sensitization of explicit memory across sessions, indexing the retention of introspective skills. EDR may reflect a sensitization effect of explicit memory within sessions and a desensitization effect (of implicit memories) across sessions. EDR and heart rate are known to be driven by sub-cortical regions adjacent to those implicated in short and long term memory (Fukai, Motomara, Kobyashi, & Asaba, 1990; Whishaw, Flannigan, & Schallart, 1982; Boucsein, 1992). The erosion of insulation between memory systems may permit new cognitive associations to be made to previously unconscious or implicit memories and perceptions, so that recursive cognitive-affective neurotic perceptual loops, or what Luborsky (1976) calls "core conflicts" may extinguish or be amenable to assimilating new cognitive-emotional information. Ethically we have felt constrained to tell all of our new somatizing patients that while putting out the somatic "fires" with psychophysiological skills may be pleasant, looking for the "matches" may be associated with some temporary psychological discomfort and autonomic changes.

PREDICTIVE HYPOTHESES

1. Hypnotic ability and social desirability are stable orthogonal traits (Wickramasekera, 1993; Palsson, 1992; Remler, 1990) which are independent risk factors for somatization. The apparent interaction of these traits as in this case study can generate the most potent transduction ("transducers") of psychological threat into somatic symptoms.

2. Hypnotic ability and Marlowe-Crowne scores are stable aspects of personality which are highly resistant to change and likely to reinduce future somatization during explicit or implicit threat perception.

3. The interaction of high hypnotic ability and high Marlowe-Crowne scores will be associated with parasympathetic dominance (Wenger & Cullen, 1972) or higher vagal tone (Porges, 1992; Cacioppo, 1994) in patients as opposed to non clinically symptomatic college students. In transducing type of somatizing patients paradoxically somatic symptom formation is predicted to reduce sympathetic baselines.

4. Neuroticism or "negative affectivity" (NA) scores will rise as a function of threatening information transfer from implicit to explicit memory across therapy sessions, in people who have transduced psychosocial threat perception into somatic symptoms (Wickramasekera, 1988, 1993, 1994a).

5. Sympathetic activation will rise as a function of

the disruption of inhibitory personality mechanisms (high hypnotic ability and/or high Marlowe-Crowne scores) in transducers who are covertly high on N.A.

6. There will be a tendency for EDR to increase within but drop across therapy sessions with transducers.

7. Mean pre-session heart rate in "transducing" somatizers without psychological symptoms (threat in implicit perception and memory) will tend to be lower than in patients presenting with psychological symptoms (threat in explicit memory and perception).

8. Mean pre-session heart rates will tend at first to increase across therapy sessions as a function of (a) threatening information crossing from implicit to explicit memory and/or (b) expanding attention to processing threatening information.

9. Mean pre-session measures of EDR in transducers will drop across therapy sessions.

10. Mean post-session heart rate will tend to drop within sessions as a function of the extinction of the explicit memory of threat.

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