A Model of People at High Risk to Develop Chronic Stress-Related Somatic Symptoms: Some Predictions

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Certain measurable high-risk factors that predispose people to develop functionally based somatic disorders are identified. These risk factors compose a multidimensional model that encompasses variables involved in the predisposition, the precipitation, and the buffering of stress-related symptoms. These high-risk factors are (a) high or low hypnotic ability, (b) habitual catastrophizing cognitions and pessimistic belief systems, (c) autonomic lability or neuroticism, (d) multiple major life changes or multiple minor hassles over a short period of time, and (e) a deficit in support systems or coping skills or both.

"Sometimes it is more important to know what kind of patient has a disease than what kind of disease the patient has."—Sir William Osler (Straus, 1968, p. 5)

Sir William Osler had to rely on intuition to identify patient features that can potentiate or attenuate either the symptoms or the etiology, or both, of a disease. My goal is to specify and quantify a promising set of empirically identifiable individual differences and also a set of situational events that increase the risk of developing stress-related physical symptoms. This model (Wickramasekera, 1979, 1980b, 1983) is based on clinical observations in an increasingly specialized clinical practice, on theoretical speculations, and on empirical data from several disparate lines of controlled research.

Today researchers distinguish psychophysiological or functional somatic disorders from physically based disorders almost entirely by excluding physical explanations of the patient’s somatic complaints. In this procedure one unrealistically assumes that physical and psychological processes that result in somatic symptoms must be mutually exclusive; that is, if one finds a physical process that explains the symptoms, then psychological factors must not be involved. If a physical process is not found, then psychological factors must be involved. This logic, by which one presumes a complete mind-body dichotomy, does not correspond to empirical facts.

This process of diagnosis by exclusion is practiced by default because there has been little systematic effort (Sternbach, 1966) devoted to the identification of specific experimentally testable positive psychological/psychophysiological findings that can account for physical symptoms independently of physical findings. General theories of predisposition to disease have been proposed, but they have been difficult to test in predictive and mechanism-oriented experimental studies (Alexander, 1950; Engel, 1968; Mason, 1971; Wolff, 1953). Recently, more operationally defined and experimentally testable risk factors such as Type A behavior (Friedman & Rosenman, 1974) and life change (Holmes & Rahe, 1967) have been proposed. The identification of such high-risk variables would have profound implications for diagnostic practice as well as for therapy and for primary prevention. For example, the presence of positive psychological findings in a patient can help the clinician to inhibit his or her tendency to submit the patient to extensive physical investigations that could increase the probability of identifying and then treating a false positive physical etiology and producing an iatrogenic condition. An example of this would be unnecessary back surgery for benign and self-limiting back pain (Fordyce, 1980). The coexistence of positive psychological and physical findings might suggest that complaints having a physical etiology are being exacerbated by psychological factors. Effective intervention would require attention to both aspects of the condition.

Basing the diagnosis of psychophysiological disorders mainly on the exclusion of physical findings is not a rational procedure because it is possible that the appropriate physical investigation was not done (Hall, Popkin, Devaul, Faillace, & Stickney, 1978). In fact, this approach implies that patients with physical complaints who lack positive and independent psychophysiological findings should receive the most complete and careful physical investigations.
Some physical complaints (e.g., headaches, backaches) can be accounted for either by positive physical findings (e.g., a brain tumor or a herniated disc) or, alternatively, by positive psychophysiological findings such as functionally based high levels of muscle tension (Flor, Turk, & Birbaumer, 1985) and neuroticism (Eysenck, 1983). It is hypothesized that the five high-risk factors alone, in the absence of physical findings, can independently account for stress-related physical symptoms. Patients who present physical complaints can be divided into four cells (see Figure 1) on the basis of the demonstrated presence or absence of identifiable physical findings from physical examination and laboratory tests or identifiable psychophysiological findings (from psychological and psychophysiological tests).

Cell 1 is an instance of both positive physical findings and positive psychophysiological findings. This patient presents with headaches and on investigation is found both to have a brain tumor and to be anxious and depressed. The anxiety and depression could be amplifying (Melzack, 1973) the sensory pain component that results from the brain tumor. The acute psychological symptoms (e.g., anxiety, depression) often clear up after the tumor is removed and the headache pain disappears. In certain people (identified by the high-risk model), however, the anxiety, the depression, and the pain may persist even after the brain tumor is removed. Physicians generally find patients in this group easy to manage if the acute psychological reactions disappear after surgery.

An instance of Cell 2 is the patient who presents chronic low back pain in the absence of a herniated disc or other appropriate physical findings, but who is depressed and anxious and sleeps poorly at night. These patients' therapeutic response frustrates the best diagnostic, surgical, and chemical efforts of physicians. Eventually, such patients become “doctor shoppers,” irritating and alienating many physicians (primary care doctors and specialists) because of their dissatisfied and demanding manner. Physicians find patients in Cell 2 the most difficult to manage.

A patient in Cell 3 can present acute low back pain that is secondary to a herniated disc without enough time for psychological amplification of the back pain. Surgical repair is followed by rapid resolution of the physical complaint. Physicians manage patients in this group very rapidly and effectively. These are the patients whom they are specifically trained in their medical education to treat.

A patient in Cell 4 may present headache pain in the absence of positive physical or psychophysiological findings. These negative findings (Hall et al., 1978) may be attributed to insensitive or inappropriate physical and psychophysiological tests and examinations.

Five factors are hypothesized to increase the risk of stress-related physical symptoms. The first three risk factors, subject variables primarily related to the predisposition to illness, are (a) either high or low hypnotic ability, (b) autonomic lability (neuroticism), and more specifically, autonomic response specificity, and (c) the habitual cognitive tendency to catastrophize. The last two factors, situational variables primarily related to the precipitation of the illness, are (d) psychosocial stressors in the form of major life changes or minor hassles (or both) over a short period of time and (e) deficits in adaptive support systems or coping skills needed to manage the stressors.

Hypnotic Ability

The first risk factor is both extremes (i.e., high or low) of hypnotic ability (Wickramasekera, 1979, 1983). Hypnotic ability is a normally distributed, stable individual difference variable (Barber, 1969; E. R. Hilgard, 1965) that appears to be partly genetically based (Morgan, 1973; Morgan, Hilgard, & Davert, 1970). Hypnosis can be defined as a psychophysiological condition in which there is a relative reduction of peripheral awareness and critical analytic mentation, which may lead to major distortions in perception, mood, and memory that are sufficient to produce significant behavioral and biological changes. Hypnotic ability is measured via procedures of known reliability and validity, such as the Stanford and Harvard Scales (Barber, 1969; E. R. Hilgard, 1965). Current research suggests that hypnotic ability is best considered a mode of information processing (uncritical–holistic–visual–emotional) that can occur in a variety of situations (e.g., hypnotic induction, sensory restriction, transference) but particularly under conditions of high or low physiological arousal (Wickramasekera, 1971, 1972, 1976a, 1977a). It is most important to stop thinking of hypnosis as an event that occurs only during a hypnotic induction, in the same way that one does not think of intelligence as an event that occurs only during an intelligence test. About 10% of the population are able to very readily and profoundly obtain access to the hypnotic mode of information processing, and an equal percentage are almost never able to do so except under a set of special conditions that include high (fight or flight response) or low (hypnogogic or hypnopompic) physiological arousal (Wickramasekera, 1977a).

There are three features of both the high ability and the inability to use this mode of information processing that place people at high risk for developing somatic symptoms. Wickramasekera (1983) found that 85% of a sample of 103 patients with primary psychophysiological diagnosis scored either very high or very low on hypnotic ability on the
Stanford, Harvard, or Spiegel Scales. In a general way, it appears that people of high hypnotic ability can attend too much, amplifying even minimal unpleasant sensations in their bodies, whereas people of low hypnotic ability attend too little to the emotional–verbal correlates of physiological responses.

High Hypnotic Ability

The first feature of the high hypnotic ability that contributes to risk is the ability to hallucinate voluntarily. Unlike highly hypnotizable people, the psychotic person does not have voluntary control of his or her hallucinatory process. Factor-analytic studies of hypnotic behavior indicate that this capacity for generating rich images and fantasies is a major factor in hypnosis, accounting for close to 50% of the variance on standardized tests of hypnosis (E. R. Hilgard, 1982). Wilson and Barber’s (1982) report, in which they compared highly hypnotizable female subjects with a control group of moderate and low responders, provides numerous examples of these abilities. Highly hypnotizable people spend a great deal of time, up to 50% of waking time, in fantasy activity. The images produced in these fantasies are frequently reported to be indistinguishable from real events. Images could reach hallucinatory intensity in all sensory modalities. Recall of past events could achieve these same qualities. Actual external, nonfantasized events could be experienced intensely. For example, these people could “see,” “hear,” “feel,” and “smell” what was being talked about during simple social conversations.

The relevance of these fantasies for stress-related disorders is that they have corresponding physiological consequences. Beliefs, irrespective of their objective validity, are more likely to have biological consequences for the highly hypnotizable subject (Wickramasekera, 1979). Of Wilson and Barber’s (1982) highly hypnotizable subjects, 86% reported frequent experiences throughout their lifetimes of having illnesses or physical symptoms directly related to their thoughts, fantasies, or memories, whereas only 8% of the contrast group reported this. For example, 60% of the highly hypnotizable subjects reported false pregnancies and breast changes, abdominal enlargement, morning sickness, and fetal movements in response to the belief that they were pregnant; only 16% of the comparison group reported false pregnancy. It is hypothesized that the rich and convincing perceptual and cognitive experiences of highly hypnotizable people, together with the physiological consequences of these experiences, can provide a foundation for the development of more chronic symptomatology.

A second feature of high hypnotic ability that can contribute to risk is hypersensitivity to psychological and physiological changes. The following loosely related but converging empirical features of the highly hypnotizable subjects’ learning process relate to the hypersensitivity hypothesis. One learning mechanism of symptom induction in high hypnotic ability subjects is a superior sensory memory and a superior ability to transfer information from sensory to short-term memory (Ingram, Saccuzzo, McNeill, & McDonald, 1979; Saccuzzo, Safman, Anderson, & McNeill, 1982). Studies of simple operant verbal conditioning (King & McDonald, 1976; Webb, 1962; Weiss, Ullman, & Krasner, 1960; Wickramasekera, 1970b), respondent conditioning (Das, 1958a, 1958b), and complex social–psychological influence procedures such as short-term psychotherapy (Larsen, 1966; Nace, Warwick, Kelley, & Evans, 1982) demonstrate that the subject with high hypnotic ability learns and conditions more rapidly than does the subject with low hypnotic ability. It is likely that highly hypnotizable people learn, remember, and incubate (Eysenck, 1968; Wickramasekera, 1970a) too well the experience of acute pain, permitting it too easily to become a chronic fear or chronic pain disorder.

A second learning mechanism of symptom induction is that the person with high hypnotic ability may be hyper-sensitive to sensory and social stimuli and may be a superior discriminator of visceral sensations (Hantas, Katkin, & Reed, 1984). Clinically, it is likely that without analgesic suggestions, people of high hypnotic ability are less tolerant of pain than are people of low hypnotic ability (Shor, 1964). It is known that people of high hypnotic ability have an unusual capacity for attention to and absorption in subjective events (Tellegen & Atkinson, 1974; Wilson & Barber, 1982) such as pain and fear, and perhaps this ability can be used to amplify their response to even minimal sensory and visceral stimuli. Results of a recent study suggest that subjects with high hypnotic ability may show an amplified physiological reactivity (frontalis electromyogram) to even transient stressors (Harsher-Towe, 1983). Because hypnotic ability is also positively correlated with standardized tests of creativity (Bowers & Bowers, 1979), it is possible that this creative ability is at times used to elaborate “maladaptive meanings,” thereby amplifying minimal sensations. It is also now well established that people of high hypnotic ability are much more likely to develop clinical phobias than are people of low or moderate hypnotic ability, and that an unexpected large number (48%–58%) of clinical phobics are highly hypnotizable (Frankel & Orne, 1976; Kelly, 1984; Perry, John, & Hollandar, 1982). It is also known that in a subset of highly hypnotizable people, the severity of childhood punishment (including abuse) is correlated positively with high hypnotic ability (J. R. Hilgard, 1979; Nash, Lynn, & Givens, 1984). The hypnotic ability may be developed in order to cope with the punishment. It is likely that this hypersensitivity to sensory and social stimuli provides the basis for the development of pain, anxiety, dissociative disorders (multiple personality), and phobic disorders.

It is known that people in the hypnotic condition or who have high hypnotic ability are more likely to report and believe that they are hypersensitive to extrasensory stimuli (Honorton & Krippner, 1969; Van De Castle, 1969). Patients who had high hypnotic ability were very likely to report psychic experiences (Wickramasekera, 1979). Wilson and Barber (1982) reported that a normal highly hypnotizable sample (92%; n = 27) was far more likely to report psychic experiences (precognition, telepathy, out-of-the-body experiences, or nonpsychotic hallucinations) than were low-hypnotizable controls (16%; n = 25). At least
some of these people can voluntarily and temporarily reset their perceptual filters outside the constraints of rational–logical–critical analytic brain functions, making themselves vulnerable to psychological pollution. I have found these patients to have resistant “medical” problems with multiple exploratory surgical histories. When one is given permission to discuss and to find meaning (“reframe”) or to assimilate (McReynolds, 1960) these experiences and to integrate them into everyday life, remarkable and durable symptomatic recovery is often observed. Today the mechanisms of this healing are obscure, but they may eventually have naturalistic explanations.

A third feature of people with high hypnotic ability is their ability to voluntarily alter their stream of consciousness (Evans, 1977) and memory functions (Kihlstrom, 1985). It is becoming clear that highly hypnotizable subjects have superior voluntary control of altered states of consciousness (Evans, 1977). This ability to alter their stream of consciousness may be a protective reflex developed to deal with their biological hypersensitivity to sensory and social stimuli. Many subjects of high hypnotic ability can voluntarily and easily initiate sleep during the day or night in multiple locations (e.g., sleep lab, work, classroom, on a plane or bus) and can wake up at a preselected time without an alarm. They can also learn during sleep (e.g., during REM sleep) without waking up, and they demonstrate state-dependent retention of simple information several days, several weeks, or 6 months later (Evans, 1977).

Maladaptive and aversive physiological responses, such as muscular bracing (Whatmore & Kohli, 1974), can be learned in states of hyperarousal (sexual trauma, automobile or industrial accident) or states of hypoarousal (sleep state, nightmares) that are not accessible to verbal analysis in states of moderate arousal (e.g., everyday consciousness). It is now known that highly hypnotizable subjects, as opposed to poorly hypnotizable subjects, can alter the content of their night REM dreams by simply instructing themselves to do so before sleep (Belicki & Bowers, 1982; Stoyva, 1965; Tart, 1964). Perhaps their waking negative–aversive expectations can alter the content of REM dreams and establish maladaptive patterns of muscular and vascular response in sleep. Often patients will wake up from sleep with a sudden onset of chronic pain or severe muscular or vascular headache. E. R. Hilgard (1977) cogently documented the ability of highly hypnotizable subjects to process information outside of their own awareness to an extent that the poorly hypnotizable subjects cannot. E. R. Hilgard (1977) termed this phenomena dissociation.

Clinically, it appears that the high physiological arousal of sexual trauma or near-death industrial accidents (e.g., railroad, mining) can induce “dissociative states” (hypnotic) in which overlearned and incubated abnormal muscular responses can be acquired (Eysenck, 1968; Wickramasekera, 1970a). Phenomena such as incidental learning and false or constructed memories (Dywan & Bowers, 1983; Laurence & Perry, 1983) acquired in unrecognized hypnotic states induced by high physiological arousal are particularly likely in subjects of high hypnotic ability who present chronic pain or other somatic symptoms. Hence the person of high hypnotic ability can very rapidly learn fear and pain response and be unaware of what was learned and where (source amnesia) it was learned.

Pettinati, Horne, and Staats (1985) showed that over 50% of bulimics had high hypnotic ability and 0% had low. Also, a subset of anorectics who used purging as opposed to abstinence from food also had high hypnotic ability. It is likely that a subset of some eating disorders may be learned and enacted in a dissociated state. Because of the phenomenon of state-dependent learning, the perceptions and cognitions that trigger some forms of bulimia may be relatively insulated from interventions (e.g., conventional psychotherapy) initiated outside of their state-dependent conditions of acquisition and maintenance.

It is known that a feature of high hypnotic ability that can contribute to development of symptoms is an amnesic ability and an ability to create memories (Dywan & Bowers, 1983; Kihlstrom, 1985; Laurence & Perry, 1983). Factor–analytic studies have revealed that the second major factor in hypnotic ability is the capacity to make the mind blank and that this factor is orthogonal to the fantasy factor (E. R. Hilgard, 1982). This amnesic ability may be used to avoid or delay the recognition of organically based somatic stimuli in the acute phase of a disease, which results in the postponement of treatment until the chronic phase. Amnesic ability may also result in disconnection of verbally mediated consciousness from the motor or autonomic response systems, which results in conversion symptoms (Bendefeldt, Miller, & Ludvig, 1976).

Low Hypnotic Ability

There are three features of low hypnotizability that are hypothesized to increase risk. These are hyposensitivity to psychological and physiological changes, a tendency to deny psychological causation of behavior, and a tendency to delay seeking medical investigation. Much less is known about these individuals, so these remarks should be considered as more speculative. Basically, the subject with low hypnotic ability is vulnerable to stress disorders because he or she is relatively insensitive to or deficient in attention to relations between psychological (verbal–emotional) states and physiological (interoceptive or visceral) states. The low-hypnotizable person’s psychological insensitivity to changes in mood and feelings may be a liability from the viewpoint of preventive health care because studies have suggested that changes in mood and feelings can precede the onset of even established infection (Canter, 1972; Hall et al., 1978). Low-hypnotizable people are nearly always limited to a skeptical, critical, and analytic mode of information processing; hence they tend to negate (deny) or attenuate minimal sensory cues from their bodies. They are unwilling or unable to use verbal fantasy and imagination. It also appears that they prefer to think in concrete and discrete terms. Biofeedback instruments, such as meters, are helpful to low-hypnotizable people because they put their “insides” on the “outside” in observable, concrete, amplified, quantitative forms. These instruments track the physiological cor-
relates of psychological changes in ways that are harder to deny and dispute.

The low-hypnotizable person’s hypothesized hyposensitivity to psychological and physiological changes overlaps with the concept of alexithymia. *Alexithymia* is defined as lacking “words for moods” (Sifnios, 1972) and it was first identified in individuals with psychosomatic disorders. Frankel, Apfel-Salizit, Nemiah, and Sifnios (1977, N = 32) found that most people labeled as alexithymic are actually people who have low hypnotic ability. For the present, I prefer to identify alexithymia with hypnotic ability measures. Hypnotic ability is well defined and reliably measured, whereas alexithymia is poorly defined and no reliable measure of it has been developed (Lesser & Lesser, 1983).

Alexithymic persons’ thinking is relatively unresponsive to psychological events and, I predict, to symbolic or conditioned stimuli (CS), but their concepts are very responsive to unconditioned stimuli (UCS) that are concrete and objective. They either lack or fail to use a rich vocabulary to label and discriminate among their feelings and moods. They tend to attribute psychological changes to external physical (UCS) changes (the “weather,” “something I ate”) and to express psychological states (e.g., depression) in somatic language (e.g., pain). When the low-hypnotizable person is exposed to a traumatic event (e.g., auto or industrial accident) that causes physiological arousal, he or she is very likely to verbally inhibit or deny his or her feelings (fear, terror, rage, anxiety, depression) associated with the trauma. There is now some fresh evidence (Pennebaker, 1985) that verbal and behavioral inhibition of trauma is associated with higher levels of physiological arousal, somatic complaints, and even physical diseases (e.g., cancer and hypertension). Hence very often for the low-hypnotizable person, somatic complaints are the final common pathway for unverbalized psychosocial conflicts. I hypothesize that external stimuli (UCS) through receptors may directly and reflexively change autonomic nervous system (ANS) functions and motor responses in low-hypnotizable subjects, bypassing consciousness (central nervous system) and the opportunity for the symbolic mediation (CS) and potential attenuation through procedures such as “reframing” of these visceral or motor changes. These people present their psychological conflicts in somatic forms, and they present them in medical, not psychiatric, settings.

Low-hypnotizable people are conditioned more poorly in both the operant (King & MacDonald, 1976; Webb, 1962) and respondent (Das, 1958a, 1958b) modes and probably are slow in forming conditioned anticipatory responses. It is not the sight (CS) or sound (CS, rustling of leaves) of the tiger that is dangerous to the deer, but the tiger’s teeth (UCS) and claws (UCS). The low-hypnotizable person has to wait until he or she feels the canines (UCS) in his or her jugular before he or she responds. The ability to easily form conditioned anticipatory defensive responses (CR) to neutral stimuli (CS, rustling leaves) is self-preservative and biologically adaptive up to a point (this point is exceeded by the highly hypnotizable persons). The low-hypnotizable persons’ slow respondent conditioning may be an ANS correlate of the inability to readily alter their states of consciousness. Ader (1981) showed the immune system to be sensitive to psychological events and in fact subject to respondent conditioning. The respondent conditionability of the immune system (Ader, 1981) is probably an adaptive function that is relatively sluggish in people of low hypnotic ability. If the mechanism of the placebo response is immunopotentiation by way of respondent conditioning (Wickramaasekera, 1977b, 1980a), then low-hypnotizable persons, because of their poor ability to be conditioned, will be poor placebo responders. The experience of aversive emotions and stress can inhibit the immune system, can facilitate disease, or can potentiate the immune system (thereby protecting one from disease; Levy, Herberman, Maclish, Schlien, & Lippman, 1985; Sklar & Anisman, 1981).

A deficit in hypnotic ability may also inhibit the neurogenic resetting of dysfunctional (hypothalamic-pituitary-adrenal) feedback systems when the stress abates (e.g., after an auto or industrial trauma). The ability to enter altered states of consciousness, as in hypnosis, may facilitate the use of cognitive mechanisms (central nervous system), such as suggestion, to reset dysfunctional peripheral (ANS) feedback systems. For example, neurogenic regulation of blood pressure (Kezdi, 1967), perhaps through resetting baroreceptors, restores the body after stress to a state of homeostasis (Cannon, 1932).

People of low hypnotic ability are less aware of psychological distress and deny the role of psychological causes of physical dysfunction. These patients are apt to deny recognizing the psychological distress that may come before established infection (Carter, 1972; Carter, Cluff, & Imboden, 1972; Imboden, Carter, & Cluff, 1961) or tissue damage (unconditioned response, or UCR) and they delay seeking medical help. Physicians tend to attribute their somatic complaints to undetected organic pathology and tend to overinvestigate these patients in ways that add iatrogenic complications to the original somatic complaint.

Presently I measure the hypnotic ability construct in three modes. Behaviorally, hypnotic performance is measured via the Harvard Scale (Shor & Orme, 1962), which is an established screening instrument of known high reliability and validity. It is measured verbally-subjectively with a self-report scale called the Absorption Scale (Tellegen & Atkinson, 1974), and the physiological potential for hypnotic behavior is measured with the conjugate lateral eye movement test (Bakan, 1969).

Habitual Catastrophizing (Panicking) Cognitions and Pessimistic Belief Systems

Several large-scale prospective longitudinal studies (Hinkle, 1961; Stewart, 1962; Valliant, 1978) have shown that pessimism, self-doubt, passivity, and dependency are modest predictors of subsequent complaints of psychosomatic illness. There have, however, been methodological problems in the assessment of these constructs. Ellis (1962) elucidated the role of habitual catastrophizing cognitions in
the acquisition and maintenance of psychopathology. Catastrophizing can be defined as becoming intensely and frequently absorbed in a negative psychological or sensory event and talking to oneself about it in ways that potentiate its aversive properties. For example, panic ("Oh my God, I am dying") during a myocardial infarction can potentiate the event through inducing vasoconstriction that increases cardiac load. It is likely that the cognitive tendency to catastrophize is at least partly based on either a generalized or situation-specific pessimistic or nihilistic belief system. It is likely that catastrophizing also plays a major role in attending to symptoms, altering sensory thresholds, and escalating the levels of sympathetic arousal in stress-related disorders. When catastrophizers encounter a negative experience, they typically and reflexively think thoughts such as "I can't stand this," "This is killing me," or "This should not be happening to me; this is not fair." These thoughts amplify their misery and increase sympathetic activation.

Catastrophizing has at least two response components. One is to keep the attentional focus on the sensory or visceral events that are antecedents or consequences of symptoms; the second is to remember or anticipate a wide range of negative physical and psychosocial consequences and antecedents of the aversive or symptomatic event. There are a number of studies relating catastrophizing to somatic complaints. At present, the assessment of catastrophizing is limited to interjudge agreement and ad hoc self-report scales. Chaves and Brown (1978) found that dental patients could be divided into catastrophizers and copers during an injection or extraction. Catastrophizing ideation was reliably associated with higher levels of distress and pain in the dental situation. Brown (1979) replicated this clinical finding with experimentally produced pain. Brown and Chaves (1980) found that the bulk of chronic pain patients (low back pain and headache) are catastrophizers. Catastrophizers have significantly higher pain ratings than do copers (Spanos et al., 1979). Eighty-six percent of catastrophizers were prescribed antianxiety or antidepressant medication, whereas only 12% of copers were on this type of medication. Copers can be defined as people who use pleasant or positive cognitive distractions to attenuate their response to unpleasant sensory events. Spanos, Brown, Jones, and Horner (1981) found that catastrophizing (exaggerating) self-statements increased pain reports in experimental pain situations. Presently I measure catastrophizing with the Zocco (1984) Scale. This scale was developed to enable one to measure catastrophizing in phobic patients. It is not a totally adequate measure of catastrophizing in patients presenting physical symptoms. I also use a clinical rating of low, moderate, and high catastrophizing.

Autonomic Lability and Neuroticism

High neuroticism (N) is a self-report dimension of personality that is based on autonomic lability (Eysenck, 1983; Wenger, 1948, 1966) or, more specifically, the degree of reactivity of subsystems of the sympathetic division of the ANS. Like hypnotizability, neuroticism seems to have a genetic basis (Shields, 1962) and is known to decline with age. The literature linking high self-report N scores to the limbic system is still ambiguous because of methodological and other problems (Eysenck, 1983). Initially elevated baselines and measures of delay in returning to baseline after stressful stimulation appear to be the most promising physiological correlates of high N scores (Eysenck, 1983). Several retrospective studies reviewed by both Jenkins (1971) and Steptoe (1981) reveal an association between coronary artery disease and neuroticism. Two large-scale (12,000 men) prospective studies (Ostfeld, Lebovits, Shekelle, & Paul, 1964; Medalie & Goldbount, 1976) revealed a strong relation between neuroticism and the later development of angina pectoris but not myocardial infarction. Neuroticism appears to be related to the number of physical complaints reported (Costa & McCrae, 1985) and to the tendency to report negative or aversive feelings across numerous places and times (Watson & Clark, 1984). I use the neuroticism scale from the Eysenck Personality Inventory (Eysenck & Eysenck, 1968) to take a brief paper-and-pencil assessment of neuroticism.

Clinically, the most promising aspect of sympathetic reactivity is autonomic response specificity (ARS; Lacey, 1967; Sternbach, 1966). This refers to the frequent observation of a stable profile of sympathetic response regardless of variations in the character of the stressor (e.g., mental arithmetic or cold pressor). This phenomena may have clinical implications (Sternbach, 1966). For example, people who show maximum reactivity in the cardiovascular system may be at high risk for angina pectoris, myocardial infarction, or stroke (Krantz & Manuck, 1984), whereas those who show the strongest response on an electromyogram measure may be at greater risk of tension headache or low back pain. The physiological system that is most strongly reactive (latency, elevated baseline, delay in returning to baseline) can be termed the person's window of maximum vulnerability, or the organ system in which he or she will develop clinical symptoms when under stress.

The Psychophysiological Stress Profile (Wickramasekera, 1976a, 1976b), which is based on the ARS concept, is a standardized testing procedure developed to enable one to directly measure the magnitude and duration of a patient's physiological response to a standardized psychosocial stressor. An on-line computer collects, reduces, and prints data (range, number of data points, mean, and standard deviation) on heart rate, blood pressure, frontalis electromyogram, skin conductance, respiration, and peripheral skin temperature under three conditions. The first condition is a 15-min habituation period; the second for patients with somatic presentations is a 1-min period of stress (mental arithmetic problems); and the last is a 15-min recovery or instructed return-to-baseline period. The patient is asked to give a subjective (on a visual analogue scale) rating of his or her level of muscle tension, on a subjective unit of disturbance scale (SUDS) ranging from 0 to 10 units before actual physiological (integrated electromyogram) monitoring. In patients with chronic stress-related disorders (headaches, back pain, etc.), there is nearly always a marked
discrepancy between the verbal report measure (much lower) and the frontalis physiological measure (0–50 microvolts electromyogram). This suggests that these patients have psychologically habituated to a physiologically abnormal state.

Presently I measure autonomic lability/neuroticism in two channels: a verbal report measure with the Eysenck Personality Inventory (Eysenck & Eysenck, 1968) and the psychophysiological stress profile (Wickramasekera, 1976b).

Major Life Changes and Daily Hassles

Major life changes (a new job, a divorce, the birth of a new child, etc.) can be potent sources of psychosocial stress and precursors of somatic illness (Holmes & Rahe, 1967). In modern industrialized society, psychosocial stressors are probably the primary class of stressors that activate the “fight or flight response” and the “general adaptation syndrome” (Selye, 1956). As Mason (1971) suggested, both physical (e.g., hunger) and psychological stressors may operate through a common psychological mechanism: the perception of threat to the well-being of the animal. In fact, his research shows that in the absence of the perception of threat, biological changes (hypothalamic–pituitary–adrenal axis) may not occur in spite of physical stressors (Mason, 1971). Because the perception of threat is a learned response, it can occur chronically and intermittently in response to conditioned stimuli (CS) such as cognition, images, and so on, in the total absence of any UCR (tissue damage).

Psychosocial stressors such as a problem child, an unhappy marriage, the death of a spouse, an unpleasant job, an aging parent who resides with one, and so on, produce certain unique and different features from physical stressors. First, psychosocial stressors commonly elicit both avoidance and approach tendencies either sequentially or simultaneously; for example, a divorce after many years of marriage can be both a relief and a regret. Second, the sources of psychosocial stress are often nebulous and difficult to recognize and even harder to define, unlike the threat from a saber-toothed tiger. Third, psychosocial stressors tend to be intermittent, chronic, and resistant to rapid or final resolution by primitive defenses such as either fighting or flight. For example, the problems posed by an adolescent or an aging parent who lives with one cannot be resolved by either physical attack or flight, and they tend to linger. In summary, then, ambivalence, ambiguity, and chronicity are three special features of psychosocial stressors that interact with the special features of people at high risk, potentiating the probability of somatic disorders and disease.

A massing of major life event changes at one time appears to be associated with a higher probability of illness onset. A method of assessing the impact of situational stress on health is the measurement of major life changes (Dohrenwend & Dohrenwend, 1978; Holmes, 1981; Holmes & Rahe, 1967). The major weakness of this method is the empirical finding that the relation between life event change scores and health outcomes is too weak for individual prediction (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982). Major life changes are infrequent events and are confounded with other variables (Rabkin & Struening, 1976).

I have supplemented the major life change procedure with the Hassles Scale (Kanner, Coyne, Schaefer, & Lazarus, 1981). The Hassles Scale enables one to assess the ongoing daily stresses and strains of everyday life (e.g., getting caught in rush-hour traffic; running out of gas; noise; work overload; unexpected company). The research of Kanner et al. (1981) and DeLongis et al. (1982) demonstrates that a massing of daily hassles is strongly related to somatic health outcomes and that this effect remained even after the effects of major life events was statistically removed (DeLongis et al., 1982; Zarski, 1984). Patients who have in the last 6 months experienced several life changes (e.g., divorce, death of a parent or sibling, job change, change in residence) or multiple minor hassles may be at higher risk for becoming physically symptomatic. For example, they may develop complaints of recent back pain, headaches, stomach distress, or chest pain, and these complaints should be evaluated in this situational context. Identification of stressors is crucial, of course, for the therapeutic management of the individual.

Social Support Systems and Coping Skills

Social support (Caplan, 1974) is the comfort, help, and information that one gets through formal or informal enduring contact with individuals or groups. The impact of a massing of major life event changes or minor hassles, or both, will depend not only on the degree of the perception of psychological “threat” (Mason, 1971) provoked by life changes and hassles, but also on access to and the effective use by the patient of support systems and coping skills (J. Cassel, cited in Struening & Guttentag, 1975; Medalie & Goldbount, 1976). Support systems are essentially psychological resources (spouse, siblings, psychotherapist, church, friends) on which the patient can lean and with whom he or she can react to cushion the impact of stressors. The perceived availability and satisfaction with social support can buffer the impact of stressors.

The concepts of coping and coping skills originates in both the animal learning and psychoanalytic ego psychology literature. Lazarus and Folkman (1984) discussed the complexity and clinical value of these concepts. Coping skills (religion, projection, intellectualization, humor, sublimation, escape through fantasy or reading, work, jogging, recreation, relaxation, meditation, etc.) can also be used to distort the patient, change ("reframe") the aversive meaning of events, and lower the level of physiological arousal during both the acute and chronic phases of the stressor's impact. Patients who lack coping skills or social support systems are at much higher risk of ego fragmentation and clinical symptoms.

Currently, I assess support systems with the Social Support Questionnaire (Sarason, Levine, Basham, & Sarason, 1983) and assess coping skills with a standardized clinical
interview and the Ways of Coping (revised) Scale (Lazarus & Folkman, 1984).

All of the aforementioned five risk factors are quantified and profiled on each patient, as illustrated in Figure 2.

**Discussion**

This multidimensional model is composed of quantifiable components that separately may be weak predictors of clinical outcome but when considered together are potent predictors by which one recognizes the complexity of interactions between mind and body in real clinical situations. The model accounts for the observation that some people with clear physical findings who get specific medical remediation can continue to have symptoms. The model provides broad targets for psychophysiological therapy in general (e.g., increase or decrease hypnotic ability; decrease catastrophizing and increase coping skills and support systems) and particularly for the patient who has not responded to the specific but exclusively medical intervention. The model is experimentally testable with pre–post measures of the five high-risk variables.

Clinical observation suggests that the impact of multiple major life changes or multiple minor hassles will depend not only on personality traits (high or low hypnotic ability, autonomic response specificity/neuroticism, and habitual catastrophizing ideation), but also on the patient’s access to and effective use of social support systems and personal coping skills. For example, Nuckolls, Cassel, and Kaplan (1972) found that 90% of women with high life change scores but low social support scores had one or more complications in pregnancy, whereas only 33% of women with equally high life change scores but with high social support scores had any complication in pregnancy. The patient at greatest risk is the one who is positive for all the predisposing features, is deficient in support systems and coping skills, and has experienced a massing of multiple major life changes and hassles. Even when there are positive physical findings, the identification of two or more of these risk factors can be regarded as likely to potentiate an illness that is due to a pathogen or to tissue damage.

Clinical observations over the last 15 years have directed my attention to and confirmed the importance of these high-risk factors. I have attempted clinically to assess or quantify them with procedures of increasing validity and reliability. Hypnotic ability is adequately measured today, but ANS lability, catastrophizing, major life changes/minor hassles, coping skills, and social support systems are either poorly conceptualized or inadequately assessed via available scales. These five risk factors have clarified difficult diagnoses, enhanced the prediction of clinical outcome, and, most importantly, have provided broad targets for heuristic diagnostic investigation and clinical intervention. After further

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**Figure 2.** Patient profile for high-risk model (Wickramasekera, 1979). (L = left, R = right, AMB = ambidextrous; CLEM = conjugate lateral eye movements, S = straight; Wickram = Wickramasekera; $\overline{X}$ = mean.)
refinement and validation, these five risk factors may someday be the focus of primary prevention efforts starting in childhood or adolescence. For example, psychologists may need to reduce hypnotic ability in some people and increase it (Wickramasekera, 1977a) in others (e.g., alexithymics). They may need to reduce some patients' catastrophizing verbalizations (Ellis, 1962) and increase their probability of coping verbalizations.

Some Predictions According to the Model

1. The bulk of people who present with chronic physical symptoms without physical findings or with only marginal physical findings will be found to have either low (Harvard Hypnotic Scale 0-4) or high (Harvard Hypnotic Scale 8-12) hypnotic ability.

2. Those who have high hypnotic ability will make both medical and psychological symptom presentations in either medical or psychiatric settings, but those who have low hypnotic ability will make mainly physical presentations and almost exclusively in medical settings (e.g., medical centers).

3. People with both high hypnotic ability and high neuroticism scores (ANS lability) will respond most strongly and recover most slowly from stressful stimulation, and they will be found to have lower sensory thresholds for aversive stimulation.

4. People with low hypnotic ability will respond slowly (if at all) but consistently to stress-management therapies (e.g., biofeedback, relaxation therapy, systematic desensitization), as opposed to highly hypnotizable people, who will respond rapidly and, if highly neurotic, inconsistently to psychophysiological therapy (Wickramasekera, 1976b).

5. A simple somatic checklist will show that nonpatients over age 35 with high or low hypnotic ability will have a higher incidence of somatic complaints than will people of moderate hypnotic ability. The number of somatic complaints reported by low hypnotic-ability people may need a K-correction factor for denial.

References


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