How Does Biofeedback Reduce Clinical Symptoms and Do Memories and Beliefs Have Biological Consequences? Toward a Model of Mind-Body Healing

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Changes in the magnitude and direction of physiological measures (EMG, EEG, temperature, etc.) are not strongly related to the reduction of clinical symptoms in biofeedback therapy. Previously, nonspecified perceptual, cognitive, and emotional factors related to threat perception (Wickramasekera, 1979, 1988, 1998) may account for the bulk of the variance in the reduction of clinical symptoms. The mean magnitude of these previously nonspecified or placebo factors is closer to 70% when both the therapist and patient believe in the efficacy of the therapy. This powerful placebo effect is hypothesized to be an elicited conditioned response (Wickramasekera, 1977a, 1977c, 1980, 1985) based on the memory of prior healings. These memories of healing are more resistant to extinction if originally acquired on a partial rather than continuous reinforcement schedule. High and low hypnotic ability in interaction with threat perception (negative affect) is hypothesized to contribute to both the production and reduction of clinical symptoms. High and low hypnotic ability respectively are hypothesized to be related to dysregulation of the sympathetic and parasympathetic arms of the autonomic nervous system. Biofeedback is hypothesized to be most effective for reducing clinical symptoms in people of low to moderate hypnotic ability. For people high in trait hypnotic ability, training in self-hypnosis or other instructional procedures (e.g., autogenic training, progressive muscle relaxation, mediation, CBT, etc.) will produce the most rapid reduction in clinical symptoms.

KEY WORDS: biofeedback; healing; hypnosis; beliefs; memory; placebo.

INTRODUCTION

In the early 1970s, biofeedback training captured the imagination of the public, clinicians, and scientists because of its claim to produce learned changes in typically involuntary and unconscious autonomic nervous system (ANS) functions (Brown, 1970; Green & Green, 1977). It promised to offer a learned alternative to drugs and surgery in the management of

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somatic symptoms and pain reduction. However, there appeared to be biological constraints on the magnitude of these learned involuntary and unconscious ANS changes and questions about the adequacy of baseline measurements (Paskewitz & Orne, 1973; Taub and School, 1978). Surprisingly, Taub (1977) and Taub and School (1978) showed that the efficacy of physiological change in the same biofeedback temperature training procedure was strongly related to the belief in the feasibility of temperature training and the nature of the social relationship (warm or cool) between the trainer and the trainee. For example, in a cool or impersonal relationship, only 2 of 22 subjects learned to warm their hands, but in a “warm” or informal relationship 19 of 21 subjects learned hand warming (Taub & School, 1978). Next, a series of studies (Qualls & Sheehan, 1981) showed that a personality variable absorption (Tellegen & Atkinson, 1974), that was a component of hypnotic ability, was negatively related to the acquisition of frontal EMG reduction training. In other words, high absorption ability retarded the rate of acquisition of frontal EMG reduction biofeedback training. An early theoretical review of biofeedback and hypnosis (Wickramasekera, 1976, 1977b) suggested that two personality variables (hypnotic ability and neuroticism) and two conditions (low physiological arousal and sensory restriction) under which instructions were delivered during biofeedback training could alter both the magnitude of physiological change and clinical outcome in biofeedback training. Hence, by the late 1980s, the simple statement that biofeedback could produce learned changes in involuntary and unconscious human physiological responses was muddied by several moderating factors: the social relationship between the trainer and trainee in biofeedback, personality variables in the acquisition of biofeedback skills, and the nature and credibility of instructional, baseline, and situational variables in biofeedback.

PHYSIOLOGICAL CHANGE AND CLINICAL CHANGE ARE WEAKLY RELATED

However, in the late 1970s and early 1980, a series of well-controlled clinical studies and a double-blind clinical study (Cohen, Graham, Fotopulos, & Cook, 1977) brought a new clarity to an important part of the field. These studies showed that neither the magnitude nor the direction of physiological change in biofeedback training was strongly related to the magnitude of the reduction of clinical symptoms. The reduction of clinical symptoms, was, of course, the major reason most clinicians and the public had flocked to biofeedback. Even today, however, the empirical demonstration nearly 20 years ago that the magnitude of physiological change in biofeedback is not strongly related to clinical outcome is the best kept secret in clinical biofeedback. This tacit conspiracy of silence between clinicians and research scientists is driven by many factors that are explored elsewhere (Wickramasekera, 1976, 1988, 1998).

For example, Cohen et al. (1977) in a highly credible and carefully done double-blind EEG/EMG biofeedback study showed that the reduction of clinical symptoms during detoxification from methadone, verified by urinalysis, was unrelated to the magnitude of specific changes in an EEG/EMG biofeedback training procedure. Patients on contingent biological feedback demonstrated more control over EMG activity, but this control was unrelated to clinical outcome under double-blind conditions. Only 2 of 14 subjects in the noncontingent group believed they might be getting noncontingent feedback. In fact,
examination of patient logs indicated the presence of positive effects like “out of the body” sensations, loss of space and time perception, and a “high” that lasted beyond training for several hours equally in both contingent and noncontingent groups. Clearly, suggestion and expectation was the driving component in the verbal reports. In fact, the overall clinical efficacy under nonblind conditions was 67%, but under informed double-blind conditions, it was reduced to 34%. This indicates that double-blind conditions notably reduce the clinical success rate. This point will be discussed further later. Studies by Andrasik and Holroyd (1980) and Holroyd et al. (1984) showed that the reduction of headache pain (clinical outcome) was unrelated to changes in EMG levels. These findings confirmed earlier work by Cohen et al. (1977). A recent review of the literature of chronic pain by Arena & Blanchard (in press) also concludes that there is no strong relationship between the magnitude or direction of physiological change and clinical improvement. A study of EMG biofeedback for low back pain found that contingent EMG biofeedback was not essential for a positive clinical outcome and that high trait hypnotic ability was a significant predictor of positive clinical outcome in the placebo control group (Bush & Ditto, 1985).

Numerous studies, however, have shown that biofeedback training is reliably associated with the reduction of clinical symptoms (Hatch, Fisher, & Rugh, 1987; Wickramasekera & Kenkel, 1996a, 1996b). It appears that physiological change alone account for only, at best, a small portion (e.g., 20%) of the variance in clinical outcome. What factors then account for the bulk of the empirically demonstrated clinical efficacy of biofeedback? In other words, through what mechanism does biofeedback training reduce clinical symptoms? Current empirical evidence from controlled clinical studies suggest that cognitive and emotional nonspecific factors, may account for the bulk of the variance in the reduction of clinical symptoms, and not the magnitude of physiological changes.

Early in my clinical biofeedback practice (Wickramasekera, 1972a, 1972b, 1973a, 1973c, 1974; Wickramasekera & Truong, 1974) in the 1970s, I began to realize that in the bulk of my patients’ clinical progress was unrelated to the magnitude of physiological changes in biofeedback training (Wickramasekera, 1976, 1977a, 1977c). This observation and growing belief at first had a devastating effect on my confidence in biofeedback therapy and was associated with a sharp temporary drop in my clinical efficacy with biofeedback patients. Many of us in those early years of biofeedback believed that given sufficient specific and accurate biological feedback people would walk on water. Fortunately, for me personally, my earliest interest in biofeedback was not in the reduction of clinical symptoms. I was searching for a reliable and clinically acceptable procedure to induce mental relaxation (Wickramasekera, 1971, 1973b) and sensory restriction (Wickramasekera, 1969, 1970) that could, at least, temporarily increase a person’s openness to suggested changes in perception, or more specifically trait hypnotic ability (Hilgard, 1965). At a baseline level, there appear to be significant individual differences in the flexibility of instructional control of human perceptions, memories, and moods (Barber, 1969; Hilgard, 1965). I have found these individual differences very relevant to the prediction of clinical outcome (Wickramasekera, 1976, 1988). These stable baseline individual differences in the flexibility of human perceptions, memories, and moods have been most systematically and empirically studied under the label trait hypnotic ability.

I have stated (Wickramasekera, 1976, 1977a, 1977c, 1988) that the empirical efficacy of biofeedback in clinical symptom reduction is due to, at least, three mechanisms that deserve more empirical study in biofeedback and applied psychophysiology. First,

DOES BIOFEEDBACK INCREASE LOW TRAIT HYPNOTIC ABILITY AND OPENNESS TO CHANGE?

Several studies have shown that biofeedback works better for low rather than high trait absorption people (Qualls & Sheehan, 1981) and absorption is a modest correlate of trait hypnotic ability. The first mechanism through which biofeedback reduces clinical symptoms is by temporarily increasing trait hypnotic ability and several empirical studies support this conclusion (Engstrom, 1976; Wickramasekera, 1971, 1973b, 1977b). Biofeedback probably increases the patient’s openness (Costa & McCrae, 1986) to alternative explanations of clinical symptoms. Biofeedback alters the attribution process in clinical symptoms. The importance of this temporary increase in hypnotic ability or perceptual flexibility is that low hypnotic ability, in fact, may be a risk factor for stress (threat) related disease and the clinical symptoms that bring patients into therapy (Wickramasekera, 1979, 1986, 1988, 1998). For example, low trait hypnotic was related to delayed recovery from cardiac surgery in the intensive care unit (Greenleaf, Fisher, Miaskowski, & Duttamal, 1992). The authors (Greenleaf et al., 1992) did not, however, comment on the fact that there were significantly more low hypnotic ability (N = 19) than moderate (N = 8) or high hypnotic ability people (N = 5) in their sample of cardiac surgery patients. However, they noted that both “high” and “lows” took longer to recover (P < 0.01) in the ICU than moderates. In the primary care setting, we found that low-trait absorption (a modest correlate of low hypnotic ability) characterized significantly more patients (P < .001) complaining of nonspecific chest pain (Saxon & Wickramasekera, 1994). Harris, Porges, Clemenson Carpenter, & Vincenz (1993) found, in a normal population, that “low” trait hypnotic ability people had higher baseline heart rates and lower cardiac vagal tone than highs. DeBendittis, Cigada, & Bianchi (1994) found that during hypnosis, lows were less likely to increase vagal tone (a physiological measure of relaxation) than highs. A study of morbid obesity (Wickramasekera & Price, 1997) found that the significantly more morbidly obese patients (P < 0.001), who were candidates for gastric bypass surgery, were low on trait absorption (a correlate of low hypnotic ability), and it is suspected that parasympathetic dysregulation may be implicated in morbid obesity (Wisen et al., 1987). The above findings of higher heart rate and lower vagal tone (Porges, 1992) are consistent with my hypothesis that parasympathetic dysregulation and the repressive cognitive style of low trait hypnotic ability people contributes to their risk for stress (threat) related disorders and clinical symptoms (Wickramasekera, 1988; Wickramasekera & Price, 1997; Wickramasekera, 1998). We also found that “lows” have poorer subjective perception of a stressor (P < .001) than highs (Pomerantz & Wickramasekera, 1988) and also have lower sympathetic (electrodermal) reactivity to a cognitive stressor than “highs” (Wickramasekera, Pope, & Kolm, 1996).
Sympathetic hyperreactivity has been hypothesized to be one of the mechanisms that place high trait hypnotic ability people at risk for stress-related disorders (Wickramasekera, 1979, 1988, 1998; Wickramasekera, Pope, & Kolm, 1996). Hence, highs are hypothesized to be sympathetically and lows parasympathetically prone to autonomic dysregulation (Wickramasekera & Price, 1997; Wickramasekera, 1998). Frankel, Apfel-Savitz, Nemiah, & Sifneos (1977) found that “lows” are likely to be alexithymic and are less likely to acknowledge and describe their feelings. Clinically, I have found that the cognitive style of the “lows” is rigidly skeptical, rational, and analytic. “Lows” are more responsive to being “shown” rather than told about external, mechanical, and physical explanations of natural phenomena. Hence, they profit more from biofeedback than “highs.” We found the mean trait absorption ability (a correlate of low hypnotic ability) of two different samples of medical students (N = 200) to be significantly lower than national norms for absorption (Wickramasekera, Davies & Davies, 1996b; Wickramasekera, 1998). If this finding of low trait absorption ability is replicated at other medical schools, particularly those that train medical educators (Harvard, Stanford, and others), it will not be surprising why “bottom-up” (Sperry, 1980) and not “top-down” approaches dominate biomedical research and therapy. Our health care services and system is largely limited to and controlled by a “bottom-up” biomedical reductionism. Within this system, “top down,” subtle psychosocial, and cognitive-emotional variables are relegated to the trivial status of epiphenomena. I hypothesize that biomedical reductionism is in part a direct consequence of the low-trait absorption cognitive styles (low hypnotic ability) that dominate health care research and therapy in the western world (Wickramasekera, 1988, 1998).

In several early nonclinical studies, I found that EMG and theta EEG biofeedback done by me, at least, temporarily increases human suggestibility or trait hypnotic ability (Wickramasekera, 1971, 1973b, 1977b). I was not troubled when it was reported (Spanos & Bertrand, 1985) that novice undergraduate EMG biofeedback trainers were unable to replicate my findings. Because my finding that EMG and EEG theta biofeedback, at least, temporarily amplified hypnotic ability had been independently confirmed with EEG biofeedback by several investigators (Engstrom, 1976; Engstrom, London, & Hart, 1970; London, Hart, & Leibovitz, 1968), the current rash of multiple EEG biofeedback clinical applications also supports this conclusion. I showed that low arousal biofeedback (relaxation) training temporarily amplifies trait hypnotic ability perhaps by blurring the boundaries between perceptual events and by increasing the plasticity of human perceptions, memories, and moods (Wickramasekera, 1977b). Increased openness (Costa & McCrae, 1986) to cognitive-emotional changes may permit alterations in the core cognitive and emotional assumptions about the causes of a patient’s clinical symptoms (Wickramasekera, 1988). Current verbal report and behavioral measures of these two important constructs (a) openness and (b) hypnotic ability are poorly correlated (Gliky, Tataryn, Tobias, & Kihlstrom, 1991). But correlations between verbal report measures of openness and physiological measures of hypnotic ability (Graffin, Ray, & Lundy, 1995) have not been explored or reported. Because low arousal biofeedback only temporarily (a state change) increases trait hypnotic ability, a time sensitive physiological measure of state hypnotic ability like amplified frontal and temporal theta EEG (Graffin et al., 1995) is probably preferable to a less time-sensitive behavioral measure of trait hypnotic ability, such as the Harvard or Stanford Scales. Changes in a patient’s core cognitive and emotional assumptions about their clinical symptoms are crucial to the reduction of their suffering and clinical symptoms, particularly with transparently psychosocial providers rather than medical methods of therapy (e.g., cognitive behavior therapy,
hypnosis, psychotherapy). The Trojan Horse Role Induction (Wickramasekera, 1988, 1993) can increase the openness of skeptical low hypnotic ability patients to recognize unconscious threat perceptions driving their somatic symptoms (e.g., diarrhea, nausea, vomiting). For such somatic symptoms, psychosocial interventions typically have zero face validity. In the final analysis, beliefs and cognitive styles powerfully constrain the range of operant behaviors and problem-solving behaviors explored when skeptical people are confronted with biological (e.g., chronic disease) and environmental constraints. In the face of threat, deficits, in cognitive style and in the credibility of approach coping beliefs, can be more immobilizing than the absence of an arm or leg.

DO BIOMEDICAL INSTRUMENTS USED IN BIOFEEDBACK ELICIT A CONDITIONED PLACEBO RESPONSE BASED ON THE MEMORY OF PRIOR HEALINGS?

The second hypothesized mechanism through which biofeedback reduces clinical symptom is by eliciting a conditioned placebo response. Biomedical instruments used in biofeedback training are hypothesized to elicit a therapeutic mind-body learned placebo response based on memories of prior healings that stimulate current hope and reduces current anxiety (Wickramasekera, 1977a, 1977c, 1980, 1985). This is a rapidly elicited response in some patients, based on a prior reinforcement history of healing with biomedical instruments that were associated with vivid memories of previous healings and the credible expectation from biomedical instruments of present and future healing. This automatic and probably unconscious Pavlovian response can inhibit fear and pain responses and reduce uncertainty and doubt about clinical symptoms. Mobilizing this placebo effect of biomedical instruments, can boost the credibility and efficacy of cognitive reconstructions of the causes of present clinical symptoms. This reconstruction of belief is particularly important for patients who are locked into a strictly organic explanation of their symptoms, and, are therefore, unable to become motivated to actively participate in a psychosocial therapy. A psychophysiological therapy (Wickramasekera, 1988) targets the reduction of threat perception. Psychophysiological therapy regards threat perception and its psychophysiological and behavioral consequences as major factors maintaining or amplifying clinical symptoms.

Is there any empirical evidence to support the above theory that biomedical instruments elicit placebo effects? Several empirical studies show that biomedical instruments per se can have placebo effects. For example, Hashish, Feinman, & Harvey (1988) found that an ultrasound instrument was effective at reducing both pain and swelling (a local response to tissue damage) whether it was turned on or not, provided both the patient and therapist believed it was emitting sound. In the above study, the pain and edema was an acute response to tooth extraction. Placebo responses to medical and surgical tools are well-documented (Long, Uematsu, & Kouba, 1989; Schwitzgebel & Traugott, 1968). The affective component of low back pain reduction with Tens (Marchand, Charest, Li, Chenard, Lavignolle, & Laurencelle, 1993) may be a placebo response. Sox, Margules, & Hill Sox (1981) showed that the use of medical diagnostic tests alone were an independent predictor of clinical recovery from non-specific chest pain. Hence, there is empirical evidence that some part of the initial reduction in clinical symptoms in biofeedback training may be a conditioned placebo response to biomedical instruments (Wickramasekera, 1977a, 1977c, 1980, 1985).
As previously mentioned, the first well-controlled study of EEG/EMG clinical biofeedback found that the placebo effect was 67% under nonblind conditions even in the reduction of severe chronic detox symptoms of opiate addicts. Under double-blind conditions with the same type of patients, however, the placebo rate was reduced to 34%, and it was found that physiological changes in EEG and EMG were unrelated to clinical outcome. What is the magnitude of the placebo effect in double-blind drug studies? The mean placebo effect in double-blind drug studies is reported to be 33% + 2%. The actual placebo rate in published studies ranges widely from 10% to 90% (Turner et al., 1994) and true blindness may not occur (Fisher & Greenberg, 1989). Hence, the mean magnitude of the placebo effect in double-blind drug and biofeedback studies is almost identical (34% vs. 33%). A recent review of nonblind drug and surgical studies encompassing 6,931 patients found that the mean placebo rate was 70% (Roberts, Kewnan, Mercier, & Hovell, 1993). This is similar to the Cohen et al. (1977) finding of a of a stronger placebo rate (67%) obtained under nonblind conditions in biofeedback training. In actual clinical work, treatments are seldom used unless both the patient and therapist have some reason to believe they work. Roberts et al. (1993) reviewed two medical (glomectomy for bronchial asthma, and gastric freezing for duodenal ulcer) and three drug therapies (levamisole for herpes simplex virus [HSV], photodynamic inactivation for HSV infection, and organic solvents for HSV). These five therapies are now abandoned by the medical profession as ineffective. However, under nonblind conditions (routine in actual clinical practice) or when both the patient and the doctor believed in the treatment, the mean clinical efficacy rate was a high 70%: 40% of patients got excellent clinical results, 29.6% got good results, and 30.3% got poor clinical results. Hence, under more naturalistic or ecologically valid conditions of both high patient and therapist expectations (nonblind), the placebo effect leaves only 30% of the variance in clinical outcome to be accounted for by germs and genes. This is very consistent with potential magnitude of “top down” effects in biology anticipated by the Nobel laureate Roger Sperry (1980, 1988). Predicting and controlling the elicitation of the psychophysiology of healing memories or the placebo effect (Wickramasekera, 1980, 1985), is likely to be one of the more important events in health care in the 21st century.

When both patient and therapist believe in the efficacy of a therapy, what is the mechanism of this powerful placebo effect? Evans (1974) has proposed that the placebo works by reducing anxiety. Barber (1959) has proposed that the placebo works by mobilizing the suggestibility of the patient. Strobel & Glueck (1973) and Kirsch (1978) have proposed expectancy mechanisms. Wickramasekera (1977a, 1977b, 1980, 1985) has proposed a memory or conditioned response mechanism of the placebo effect. Active treatments (e.g., morphine, penicillin) and active diagnostic procedures are unconditioned stimuli (UCS) and the vehicles in which they are delivered (e.g., pills, capsules, syringes, biomedical instruments) are conditioned stimuli (CS). The medical treatments and diagnostic procedures that people experience during their lives constitute conditioning trials during which these vehicles (e.g., capsules, syringes, biomedical apparatus) are paired with active ingredients (e.g., morphine, penicillin, accurate and valid medical information). These pairings (Ader, 1988, 1995; Wickramasekera, 1977a, 1977c, 1980, 1985) endow the pills, capsules, syringes, and biomedical instruments with the capacity to elicit the memories of therapeutic clinical effects as conditioned responses (CRs). The conditioned response (CR) model of placebo responding (Wickramasekera, 1977a, 1977b, 1980, 1985) involves the elicitation of implicit (unconscious) and explicit (conscious) psychophysiological memories of actual prior healing experiences in childhood or adolescence and the credible expectation of future
healing. Central to CR model is the hypothesis that the elicitation through memory of the behavioral, autonomic, neuroendocrine, and immune components of prior healings, is the crux of the placebo effect. Conditioning is probably the most reliable and powerful method of installing and eliciting complex mind-body memories and expectations in humans and animals.

The CR model states that the placebo is a learned complex mind-body response. It predicts that placebo learning, for example, based on partial reinforcement (UCS present on 50% of trials), will produce greater resistance to extinction than placebo learning acquired on continuous reinforcement (UCS present on 100% of trials). The CR model of the placebo effect can account for at least some of the clinical efficacy of biofeedback in both rehabilitation training and low arousal (relaxation) biofeedback training. Historically, the children of Israel who were on continuous reinforcement schedule in the desert rapidly lost their faith when Moses (CS) withdrew from them (extinction schedule) for a few days. Partial reinforcement during acquisition of faith will produce stronger memories of healing during periods of extinction or loss of both UCS and CS.

Is there any data to support the conditioned response model of placebo learning and more specifically the partial versus continuous reinforcement prediction of resistance to extinction of placebo learning or faith? Ader & Cohen (1982) paired saccharin (CS) and cyclophosphamide (UCS) on a 100% and 50% reinforcement schedule and showed that saccharin (CS) could delay the onset of proteinuria and death in New Zealand mice genetically prone to lupus. Several other controlled animal studies (Ader, 1995; Schuster & Thompson, 1969) and human studies (Suchman & Ader, 1992; Voudouris, Peck, & Coleman, 1985, 1989, 1990) are also supportive of the CR or memory model of the placebo effect. In fact, a clinical case study (Olness & Ader, 1992) of a child with lupus showed that pairing a conditioned stimulus (rose perfume and cod liver oil) with cytotoxin (UCS) during chemotherapy sessions provided a clinically successful outcome with 50% less drug (UCS).

If we examine the Pavlovian (Das, 1958a, 1958b) and operant conditioning data (King & McDonald, 1976; Wickramasekera, 1976; Webb, 1962), we find large individual differences in how rapidly people acquire conditioned responses. Trait hypnotic ability appears to be an important determinant of the rate of acquisition (r = .5) and extinction (r = .4) of Pavlovian eyelid conditioning (Das, 1958a, 1958b). Hence, it appears that neutral stimuli (CS) through conditioning can come to elicit complex placebo (CS) healing neuroendocrine and immune function changes, and trait hypnotic ability may be a significant determinant of the rate of acquisition and extinction of such conditioned responses. Theories of hypnosis that recognize the importance of trait hypnotic ability have stressed the automatic, respondent or elicited nature of hypnotic behavior under standardized induction conditions (Wickramasekera, 1988).

Individual differences in trait hypnotic ability can be operationalized with several behavioral tests of known high validity and reliability (Barber, 1969; Bates, 1993; Hilgard, 1965) like the Stanford (Forms A, B, C), Harvard Group Hypnotic Ability Test (Form A), and the Barber Suggestibility Test (Barber, 1969). Hypnotizability appears to be normally distributed in the general population, and there appears to be no significant sex differences in trait hypnotic ability (Hilgard, 1965). Trait hypnotic ability is very stable r = .71 (Piccione, Hilgard, & Zimbardo, 1989) over 25 years and appears to be partially genetically based (Morgan, Hilgard, & Davert, 1970). When hypnotic ability tests are used to sort people into high and low trait ability groups, it appears that approximately 15% of the population are at both ends of the distribution of test scores.
DOES BIOFEEDBACK RECRUIT AND REVERSE THE DIRECTION OF ACTION OF A RISK FACTOR (HIGH OR LOW TRAIT HYPNOTIC ABILITY) FOR STRESS DISORDERS?

The third, hypothesized mechanism through which biofeedback alters clinical symptoms is by recruiting and reversing the direction of activity of the infrequent phenomena, high- or low-trait hypnotic ability (Wickramasekera, 1979, 1986, 1988, 1998). Spiegel and Spiegel (1978) have theorized about the relationship between hypnotic ability, personality and psychopathology. Trait hypnotic ability per se is known empirically to be unrelated to psychopathology (Hilgard, 1965). I have hypothesized, however, that people high or low on trait hypnotic ability are at greater risk during threat perception (stress) of dysregulation of the sympathetic or parasympathetic arms of the ANS (Wickramasekera, 1988, 1998). Hence, psychopathology and eventually pathophysiology are hypothesized to be partly driven by the interaction of hypnotic ability and negative affect (stress). In general, this is because the “highs” are emotionally hypersensitive to the perception of threat, and the lows are emotionally hyposensitive to the perception of threat. This hypersensitivity or hyposensitivity in processing threatening emotions can be associated with a measurable incongruence between implicit (e.g., behaviors, physiological responses) and explicit (conscious verbal report measures) measures of threat perception that can drive psychopathology, somatic symptoms, and, eventually, pathophysiology. This incongruence between implicit and explicit measures of threat perception can produce an increasingly wider disconnection between verbal report (phenomenological), behavioral, and physiological measures of threat perception driving dysregulation of the ANS. Low trait hypnotic ability people appear to be a greater risk for dysregulating the parasympathetic nervous system and “highs” are at greater risk of dysregulating the sympathetic nervous system (Wickramasekera and Atkinson, 1992; Wickramasekera, 1998).

Wickramasekera (1987) and Wickramasekera et al. (1996) showed that “highs” during threat perception (cognitive stress) react sympathetically (electrodermal response) more strongly and take longer to recover from stress than moderates or lows.

Consequently, highs have been shown to be at greater risk during stress for phobias (John, Hollander, & Perry, 1983), bulimia and substance abuse (Pettinati, Horne, & Staats, 1985; Pettinati et al., 1990), anticipatory nausea and vomiting in chemotherapy (Wickramasekera & Saxon, 1988), EEG defined primary insomnia (Perlstrom & Wickramasekera, 1998; Wickramasekera, Ware, & Saxon, 1992), asthma (Wagaman, 1996), chronic urticaria (Shertzer & Lookingbill, 1987) chronic pain (Remler, 1990; Stam, McGrath, Brooke, & Cosire, 1986), moderate obesity (Wickramasekera & Atkinson, 1993), post-traumatic stress disorder (Spiegel, Hunt, & Dondershine, 1988; Stutman & Bliss, 1985) and delayed recovery from cardiac surgery (Greenleaf et al., 1992).

Paradoxically, high trait hypnotic ability increases the efficacy of both experimental and clinical pain reduction with hypnosis (Hilgard & Hilgard, 1975). There is good empirical evidence that people high on trait hypnotic ability can block from consciousness (verbal report) the perception of even surgical pain and negative affect, and that they can also block aversive memories as in post-hypnotic amnesia from consciousness (Hilgard, 1977). It is their natural sympathetic hypersensitivity coupled with their probably short-term adaptive compensatory capacity for blocking aversive sensations and memories from consciousness, but not necessarily from behavior and physiology, which places them at risk for long-term dysregulation of ANS, neuroendocrine, and immune systems (Wickramasekera, 1988, 1998).
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This capacity of “highs” for cognitively and emotionally blocking or reframing aversive sensations and memories, in ways that can temporarily alter their physiology and behavior, can be a liability but also an asset. Whether it is an asset or liability depends on what (e.g., CBT) recruits it, and the direction (e.g., self-soothing) in which it is mobilized. For example, a recent meta-analysis of several cognitive behavior therapy (CBT) studies of obesity found that simply adding the label “hypnosis” rather than “relaxation” doubled the efficacy of cognitive behavior therapy or CBT (Kirsch, Montgomery, & Sapirstein, 1995; Kirsch, 1996). Highs appear to be a greater risk for moderate obesity in the first place (Wickramasekera & Atkinson, 1993). Hence, it is likely that CBT recruited and reversed the direction of activity of high trait hypnotic ability amplifying the therapy response of these moderately obese patients. The CBT studies that used the word “hypnosis” probably recruited the patient’s hypnotic ability and cognitively reframed their perceptions and memories of food. It probably taught them to cope with their hypersensitivity to aversive emotions (threat) by using self-soothing self-hypnosis skills rather than catastrophizing and using food or drink to soothe themselves during episodes of threat (Wickramasekera, 1988, 1998). High hypnotic ability and sympathetic hyper-reactivity (strong EDR response to cognitive stress) were found to be risk factors for moderate obesity (Wickramasekera & Atkinson, 1993). Hence, it is likely that CBT simply mobilized one of the mechanism (e.g., high trait hypnotic ability causing hypersensitivity to threat) contributing to moderate obesity and reversed its direction of activity to reduce sympathetic hyperreactivity (e.g., taught patients self-soothing hypnotic coping skills). Klein & Spiegel (1989) showed that highs, but not lows, could with hypnosis cognitively, increase and reduce gastric acid secretion. Whorwell, Prior, and Faragher (1984); Whorwell, Prior, and Colgan (1987); and Whorwell, Houghton,
Taylor, and Maxton (1992) showed that hypnotherapy was more effective than a control procedure (psychotherapy and placebo drug) in the therapy of severe chronic irritable bowel syndrome. Whorrell’s work is another demonstration of how self-regulation procedures (biofeedback, self-hypnosis) can alter the direction of activity of a risk factor (high- or low-trait hypnotic ability) transforming hypersensitivity into analgesia, and painful memories or fantasies can be reframed or desensitized with post-hypnotic amnesia techniques. People high on trait hypnotic ability are at greater risk for developing many stress related disorders (Wickramasekera, 1979, 1986, 1988, 1998). Biofeedback, hypnosis, and CBT probably mobilize this risk factor for stress disorders and reverses its direction of activity so that a liability like hypersensitivity that amplifies threat perception can become a therapeutic asset like in hypnotic analgesia or post-hypnotic amnesia (Wickramasekera, 1976, 1988).

PREDICTIONS FROM THIS MODEL OF HEALING

The onset and offset of psychophysiological clinical symptoms is in large measure hypothesized to be controlled by the interaction of nine risk factors, only three of which are discussed here (Wickramasekera, 1979, 1988, 1998). The above model makes several empirically verifiable predictions: (a) changes in specific measures of the perceived cause or causes of somatic symptoms will correlate more highly with the reduction of clinical symptoms than any measure of physiological change per se; (b) “warm” low arousal biofeedback training can temporarily increase the mean measured “openness” to experience and hypnotic ability of biofeedback patients of moderate to low hypnotic ability; (c) immediate biological feedback will be most effective with people of low to moderate hypnotic ability; (d) “delayed biological feedback” (Wickramasekera, 1988; Zilmer & Wickramasekera, 1987) rather than “immediate” feedback will be most effective in altering physiological responses in high hypnotic ability people. Biofeedback information provided after several minutes of self-hypnosis training can be used to shape and confirm the efficacy of self-hypnosis training. “Delayed feedback” should be used to objectively validate or confirm the natural ability of “highs” to alter their physiology in a respondent manner, that is, in response to antecedent cognitions and images. “Lows” have to learn to alter their physiology in an operant or trial and error manner; (e) biomedical instruments used credibly in therapy will amplify our ability to alter the “core cognitive-emotional assumptions” of patients regarding factors causing and/or maintaining their symptoms; (f) biomedical instruments used in the monitoring mode can provide identification and assessment information about unconscious or implicit threat perception. This implicit information can be used to design therapy interventions that amplify the reduction of clinical symptoms (Wickramasekera, 1988, 1993, 1998); and (g) The placebo effect of biomedical instruments (CS) originally associated with continuous reinforcement (UCS present 100%) will have less resistance to extinction than biomedical instruments originally associated with partial reinforcement (UCS present 50%).

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