OUT OF MIND IS NOT OUT OF BODY OR BEHAVIOR

The Interaction of Hypnotic Ability and Negative Emotions Can Drive Somatization and Organic Disease

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Somatization accounts for over 50% of patients seen in primary care medicine (Roberts, 1994 & DeGruy et al., 1987). Empirical mind-body therapy is expanding. In June 1996, Professional Psychology Research and Practice will print several “white papers” on mind/body therapy targeted at health policy administrators and legislators. Each paper will be written by leading (Ph.D./M.D.), teams documenting empirically effective protocols for diseases (vascular headache, irritable bowel syndrome, Raynaud’s disease) previously seen as strictly biomedical. But, this well controlled empirical research cannot specify the “active mechanisms” in these mind-body therapies which include biofeedback, behavior therapy, and hypnosis (Wickramasekera, 1976, 1985).

I have proposed (Wickramasekera, 1979, 1988) a multidimensional High Risk Model of Threat perception (HRMTP). The HRMTP states that the probability of somatization and eventual organic disease is related to the interaction between a set of (1) predisposers (high and low hypnotic ability, covert neuroticism, overt neuroticism, and catastrophizing), (2) triggers (major life changes and hassles), and (3) buffers (support system and coping skills) of threat perception. These 9 risk factors are operationally defined and measured. These risk factors are more or less unrelated, particularly hypnotic ability (Wickramasekera, 1995).

There are at least 3 “active mechanisms” (1. high and low hypnotic ability, and 2. covert neuroticism or “repression”) that can block psychosocial perceptions of “threat” from consciousness, and transduce threats into somatization and eventually into organic disease (Wickramasekera, 1979, 1993, 1996). I have theorized that all mind-body therapies “work” by changing the operation of at least these 3 “active mechanisms.” For example, it is documented that people who are high on hypnotic ability can through mechanisms, tracked by hypnotic angesia and post hypnotic amnesia, block acute surgical pain and fear from consciousness. The chronic maladaptive use of this cognitive inhibition mechanism is a risk factor for somatization. Another subset of people I call covert neurotics or “repressors” (Weinberger, 1990, Wickramasekera, 1988, 1994a) will on DSM-IV diagnostic verbal report interviews and objective tests (MMPI, SCL-90, etc.) appear like swans on the surface of lake (without psychopathology) when, in fact, they are kicking like hell underneath the water, as indicated by direct physiological measures (blood pressure, electrodermal response, muscle tension, etc.). Self-deception can put negative emotions out of mind and even out of body, but not out of body. Charcot observed this incongruence between language and physiology over 100 years ago. I have shown (Wickramasekera, 1976, 1988, 1994a,b) that distressing secrets kept from the mind will “out” in the body or in behavior. In fact, hypnotic ability in chronic pain patients shows a linear relationship to a pure measure of sympathetic reactivity (the electrodermal response) during math stress (negative affect) in the psychophysiology laboratory (Wickramasekera et al., 1996 In Press). But conscious verbal reports of distress (e.g., S.U.D.'s levels) are often incongruent with physiological measures of distress in high hypnotizables and covert neurotic patients. Hence, it is not hypnotic ability alone, but its interaction with negative emotions that can drive symptoms and disease (Wickramasekera, 1979, 1994a; Wickramasekera et al., 1996 In Press).

We predicted and found that patients who are low on hypnotic ability will produce mainly somatic symptoms and patients who are high will produce both somatic and psychological (depression, anxiety, etc.) symptoms (Wickramasekera, 1979, 1988, 1995). As predicted by the HRMTP, recent data shows that people who are low on a correlate of hypnotic ability (absorption) will present their stress related symptoms mainly in medical settings (Saxon, 1996; Lynch et al., 1996) or surgical settings (Price & Wickramasekera, 1994). The mechanisms of risk in lows is probably more nearly behavioral than physiological.

This research was started to increase hypnotic ability in normals with sensory deprivation (Wickramasekera, 1970) and biofeedback (Wickramasekera, 1977). But, I learned early that
both high and low hypnotic ability during trauma are mixed blessings in patients. I found that the measurement of hypnotic ability before therapy permits a rational and cost-effective matching of patients to specific therapy techniques (Wickramasekera, 1976, 1988). For example, low's are skeptics "from Missouri", and they find only objective, quantative therapies credible (e.g., drugs, behavior therapy, surgery). But highs are prone to "surplus pattern recognition" (read meaning into randomly distributed events) and to "permeable boundaries" in personal relationships. They get bored with slow linear procedures. Highs find complex psychodynamic and hypnotic techniques credible. But highs need concurrent monitoring of physiological and/or behavioral events during therapy. This objective feedback from monitoring the body or behavior can protect both patient and psychotherapist from "self-deception" and boundary problems (transference/countertransference). Physiological monitoring or "truth detection" can also limit our vulnerability to the diagnostic illusion of mental health (Shedler et al., 1993) in somatization. Finally, based on the HRMTT, Saxon (1996) found a promising psychometric marker of people at risk to pass from somatization (chest pain) to organic disease. Empirically, there never was a mind-body gap; it only existed in language and biomedical philosophy (Wickramasekera, 1988). Psychophysiology in the clinic illuminates this empirical continuity between mind and body (Davies et al., 1996).

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