Response

Convergence in observation and philosophy if not methods and constructs

Ian Wickramasekera

I am delighted that Professor Sivik’s clinical observations, theory, and psychometric research has independently converged on conclusions similar to my own theory and empirical research. This is noteworthy in view of the fact that she appears to have known nothing about my work in the mind–body domain, and I have definitely been unaware of her important work. I want to apologize for my ignorance in this regard. The observational convergence is particularly impressive because she has used psychometric scales that appear to be very different from my own theoretical concepts and empirical methods in the last 25 years.

My goals for the three risk factors mentioned in my paper are more modest than the grand vision of “curing medicine” that she proposes. However, my preliminary data appear to indicate that medical schools, in the United States at least, attract an unusually large number of students who have low absorption scores (Saxon & Wickramasekera 1994) and high Marlowe Crowne scores (Palsson et al. 1997; Saxon & Wickramasekera 1994). I have used low absorption scores and high Marlowe Crowne scores as independent operational definitions of the repression of negative affect that predisposes people to somatoform and/or psychophysiological disorders. In other words, it appears that a significant number of medical students are at risk for blocking negative affect from consciousness and...
may be psychophysically challenged. Since the tests relate to cognitive-affective mechanisms that operate unconsciously, it is likely these mechanisms will impair future clinical judgment of and empirical research into the investigation of the whole patient.

My goal in my paper was simply to encourage primary care physicians to study the sensitivity and specificity of the three risk factors in their patients who initially present somatic symptoms with minimal or no pathophysiology. I am hopeful that when primary care physicians discover that the risk factors contribute to somatic symptoms and may later contribute to organic disease, they will become curious enough to investigate the risk factors in themselves.

My clinical work builds on the concepts of Charcot and Freud. It attempts to define empirically the parameters and boundaries of the phenomena of hypnosis and the mechanisms of transduction of threat perception into physiology. This work is empirically constrained by laboratory and clinical observations in the above domains in the last 100 years. The empirically specified parameters of high and low hypnotic ability (shape of the distribution, sex differences, age differences, relationship to psychopathology, etc.) as risk factors for the transduction of threat perception is the province of my modest clinical psychophysiological goals. My purpose is simply to get primary care doctors to start looking more systematically at the possibility of diagnosing somatoform disorders by inclusion criteria (however primitive at first) rather than strictly by exclusion, as is the current DSM-IV practice.

I fully agree that concepts like somatization and somatoform disorders are problematic and that they are temporary constructs on which we stand as we extend, deepen, and differentiate our conceptual and empirical tools in the domain of clinical psychophysiology.

It is clear that both top-down (through autonomic nervous system, neuroendocrine, and immune mechanisms that cognitive neuroscience is specifying even as we write) and bottom-up causation operates in this domain. The evidence is also clear that psychosocial factors do not only amplify physical symptoms but actually contribute in a dose response manner to biologically verified viral infections (Cohen et al. 1991). Hence, I believe that the three risk factors can influence not only somatoform disorders but also all physical diseases. My High Risk Model of Threat Perception (Wickramasekera 1979, 1988, 1995) includes several other risk factors (catastrophizing, high overt or covert negative affect, major life change, density of minor hassles, social support, and coping skills) that, though not discussed in my paper, may also directly or indirectly (through health behaviors) amplify or reduce somatic symptoms and physical disease. Hence, I do not pretend that the three risk factors are the only contributing pathways to clinical psychophysiology and pathophysiology.

In an ideal world, I believe that organic factors and psychosocial factors will be investigated concurrently in all presenting chronic disease and psychophysiological disorders. I agree that in an ideal world a psychosomatic perspective should be brought to every patient, even those presenting with acute medical problems. For now, I would like to see a psychophysiological study (at least three risk factors), including a psychophysiological stress profile (Evron et al. 1996; Kamack et al. 1997; Palsson et al. 1998; Wickramasekera 1998; Wickramasekera et al. 1996a; Wickramasekera et al. 1996b), performed routinely with at least all patients at risk for cardiovascular disease.

I believe that with chronicity, the probability of psychosocial impact on all organic diseases increase through Pavlovian (respondent) and operant conditioning mechanisms (Wickramasekera 1980, 1988, 1993). In loose primitive forms, the high hypnotizability (hysteria) type was anticipated by Charcot, Freud, Janet, and others. However, they did not know that it was not hypnotizability per se, but its interaction with negative affect that drove symptoms and psychophysiological disease (Wickramasekera et al. 1996b). However, the high Marlowe Crowne type may represent a new construct known to be empirically orthogonal to hypnotic ability. It is an independent pathway into the blocking of negative affect from consciousness that was first studied in normal college students in a more complex system (Schwartz 1990; Weinberger 1990; Weinberger et al. 1979).
The relationship between hypnotic ability and electroencephalogram measure of lateralization mentioned by Sivik is complex and controversial. However, it appears that the best evidence to date indicates that even at a baseline level (prior to hypnotic induction) surplus theta frequency in the frontal and temporal areas are the best discriminators between high and low hypnotic ability in normal college students (Graffin et al. 1995). This finding is very consistent with my early electromyographic and theta biofeedback studies documenting at least temporary increases in hypnotic ability (Wickramasekera 1977) in normal people. Because of the special role of interactions with negative affectivity (Watson & Clark 1984) in psychopathology and clinical psychophysiology (Wickramasekera et al. 1996b), it remains to be seen if these theta electroencephalogram correlates of hypnotic ability will hold for clinical samples (Wickramasekera 1998).

In summary and conclusion, I am delighted by the convergence between Professor Sivik's work and mine in clinical psychophysiology. I suspect that my ignorance of her work is partly owing to the fact that my efforts during the last 30 years have been largely focused on the fields of biofeedback, hypnosis, and behaviour therapy (Wickramasekera 1976, 1988). More recently, I have become involved with the empirical study of the biological basis of spiritual belief as the preeminent coping skill in spontaneous remission from terminal disease (Levin et al. in press). I have also begun the investigation of cognitively and affectively unassimilated anomalous perceptions (for example, extrasensory perception) as risk factors for somatoform disorders (Wickramasekera 1986, 1989). Hence, in a broader philosophical sense, I perceive a kindred spirit in Professor Sivik's work, and I hope we will meet some day.

REFERENCES


Wickramasekera IE 1976 Biofeedback, behavior therapy and hypnosis: potentiating the verbal control of behavior for clinicians. Chicago: Nelson Hall.


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**Charles F. Stroebel 1936 – 1998**

Exchange

More on somatization

The following exchange continues the discussion in the Spring 1998 issue of Ian Wickramasekera’s article “Secrets Kept From the Mind But Not the Body or Behavior: The Unsolved Problems of Identifying and Treating Somatization and Psychophysiological Disease.” The article sets out a model to help primary care physicians determine whether patients who present physical symptoms apparently without physical causes are somatizers – or, in the more technical language of the Diagnostic and Statistical Manual of Mental Disorders – IV, “somatoform patients.” Wickramasekera argues that the presence of either high or low hypnotic ability or high scores on the Marlowe Crowne scale (which measures the capacity for blocking aversive perceptions, memories, and moods from consciousness) is a strong indication of somatization. Tatjana Sivik, MD, PhD, MA, here comments on Wickramasekera’s paper. Sivik heads the Institute for Psychosomatic Medicine in Göteborg, Sweden, and is affiliated with the Department of Primary Health Care at the University of Göteborg. Wickramasekera, PhD, is Professor of Psychology at Saybrook Institute, San Francisco, CA; Professor of Family Medicine at Eastern Virginia Medical School, Norfolk, VA; and is visiting Professor of Psychiatry and Behavioral Science at Stanford Medical School, Palo Alto, CA (USA).

Since we have both body and mind, we are all psychosomatic

Tatjana Sivik

Dr Wickramasekera’s paper evokes ambivalent feelings. The issue he discusses – the differential diagnosis of patients seeking medical help at primary health care centers – is indeed a very important one. Since the late 1970s, I myself have been engaged in this problem, and several of Wickramasekera’s findings are in accord with those of my colleagues and myself. On the other hand, I do not fully agree with his background description of the problems, and I wonder about his own ambivalence regarding the relations between body and soul. In addition, there are a few questions regarding definitions of concepts, methods, and not least, his relations to and anchoring in other psychosomatic theories, and his familiarity with similar research in the field.

Definitions, concepts, and methods

The first issue I want to raise is the use of the term “somatization.” In my view, this label can be misleading and may conserve an old-fashioned and over-simplified view of mental interactions. At the time when Lipowski coined this concept, there was a fairly good reason for doing so, but with the knowledge we have today about the constantly ongoing interaction process between all possible factors involved in the preservation of life and health, it is no longer appropriate. These interacting factors – genetic, neuro-endocrine-immunological, societal, (inter)personal, physical environment, cultural, psychosocial, existential, religious, etc. – are always involved in the