Antecedents to Somatoform Disorders: A Pre- and Perinatal Psychology Hypothesis

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Full Text: Headnote ABSTRACT: The somatofonn cluster of behavioral disorders is the single most frequent class of unexplainable problems found in primary care medical settings today. What is known about these disorders is that there are physiological, social, and psychological variables that need to be considered. What is not known is how a person develops a propensity toward having physical symptoms as their primary complaint. The author suggests that human beings are classically conditioned when faced with intolerable emotional experiences in the womb or during birth. The residual feelings are laid down in the developing brain's neural pathways in an adaptative strategy of escape and avoidance (focusing on the body instead of the feelings), allowing the organism to survive. INTRODUCTION Somatization has been defined the most simply and intelligibly as: The expression of psychological pain through physical symptoms (Fauman, 1994). Physical pain is defined here as an unpleasant sensory experience that is associated with actual tissue damage (Benoliel, 1995). The somatoform disorders, according to the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders; APA, 1994), are a cluster of symptoms and behaviors with common features, primarily persistent or recurring physical complaints that are not supported by actual physical findings (Kirmayer &Taillefer, 1997). The somatoform cluster of behavioral disorders are the single most frequent class of problems in primary care medical settings (Mayou, Bass &Sharpe, 1995). Unexplained symptoms constitute from 25% to 60% of family medicine practices (Kirkwood, Clure, et al., 1982). These disorders cause clinically significant personal distress and/or impairments in social, occupational, or other areas of functioning (APA, 1994). Though the focus may be on bodily ailments, the research shows, there are also many more complex, underlying psychosocial and emotional antecedents, pain-related dysfunctions or disabilities, comorbid conditions, and pain-sustaining factors as well (Reid, Balis &Button, 1997). PSYCHOSOCIAL ANTECEDENTS The concept that psychosocial Stressors influence or exacerbate illness is certainly not new. Clinicians know that it is often possible to understand the complexity of present difficulties by examining the individual's life history. Many studies have noted that traumatic or stressful experiences seem to be qualitatively different from memories of ordinary events and are more emotional in nature (Teff, 1988; van der Kolk, 1994). Elevated life stress predicts a greater frequency of medical visits, strongest among patients with personality traits characterizing a tendency toward somatization (Miranda, Perez-Stable, Munoz, Hargreaves &Henke, 1991). It is a very difficult task determining the underlying components when diagnosing and treating a somatoform disorder because of the multitude of psychological factors that may contribute to the development, maintenance, or exacerbation of a somatic or physical complaint. Adding to the confusion is uncertainty as to the antecedents of the psychological pain. There are divergent views from scientists on this issue of origination as well. Before looking more closely at the hypothesis offered as an explanation of the antecedents, found in the earliest period of human development, an examination of the expression of physical complaints, within a cultural context will be offered. PAIN EXPRESSION AND CULTURE Every culture determines what are the accepted "help-seeking" behaviors for emotional pain and human suffering. If physical complaints are more socially acceptable than psychological distress, as is true in the United States, then somatic complaints are expressed (Chaplin, 1997). Western culture allows symptom relief when received from a physician who can determine the source of the pain or complaint and prescribe the appropriate medication. The cultural norm for pain expression in the United States is that a person will be given sympathy and attention with a physical ailment but can expect rejection with a mental one. UNDERLYING EMOTIONAL ANTECEDENTS Because all people in the U. S. who are subjected to

this same socialization process do not have a physical symptom focus, we need to look still deeper. There is general acknowledgment among professional therapists that affect regulation and biological patternings are closely related (Schore, 1994; LeDoux, 1993; Thayer, 1989). This would seem intuitively correct as emotions give human beings a distinctive readiness to act. LeDoux (1993), for example, has looked at precisely this emotion-to-action question within the amygdala (limbic system) in the architecture of the brain. His research found that inputting sensory signals travel into the brain to the thalamus first (previously known), then across a single synapse to the amygdala, giving it the ability to begin to respond first (previously unknown) before the cerebral cortex does. This is essentially saying that the amygdala is ready to act when the thinking brain is still coming to a decision on how to respond. The role of the emotions in psychological development has been studied substantially. Currently there are detailed descriptors on how each emotion prepares the body for a very different kind of response (Goleman, 1995). In fact, anxiety can actually produce physical pain when the prolonged muscle tension associated with anxiety may trigger other points and induce vasoconstriction (Carlson, 1995). THEORY AND RESEARCH AROUND PAIN In the last several decades there has been a swing from the entirely mechanistic biological view to a complexity that includes individual, environmental, and cultural characteristics. An article of this length can not cover the advances in theory in all these areas adequately, so two specific views will be looked at around physical pain symptoms: 1) where emotions are a factor, and 2) from an evolutionary perspective where survival is the motivator. EMOTIONS In the last 15-20 years, a number of new theories on the nature of emotion have been proposed (Bower, 1981; Lang, 1984; Leventhal, 1984) and attempts at investigating and explaining emotionally-driven behaviors have appeared (Leventhal &Tomarken, 1986). These have shown relevance to somatoform pain behaviors in clinical treatment. For example promising research, such as the disclosure of emotions paradigm (Dominguez, et al., 1995) is helping to clarify the psychological variables that increases a patient's risk for developing a somatic pain problem. These patients are said to be psychologically stuck in the past with its consequential depression. Unless they express the emotion and focus on the present they remain past-focused and pain-prone. There is also literature which shows that the impact of any emotionally stressful event can be significantly influenced by how a person appraises the situation and/or copes with it (Gatchel, Baum, &Krantz, 1988). This in turn influences how individuals differ in then emotional and physiological responses. For example, one person may have an increased heart rate and blood pressure but no muscle tension, whereas another will have primarily increased muscle tension. The research examined the particular physiologic symptom or organ which was identified as more stressed. However, this systematic research has yet to determine the predictive validity of the vunerable organ becoming the future somatoform focus. The diathesis-stress model of illness (Levi, 1974) proposed simply that psychosocial, environmental, genetic, and physiological elements should be considered interactive. A physiological predisposition toward a certain illness (genetic weakness or biochemical imbalance), psychosocial stimuli (stress and how a person responds to and copes with it), and previously experienced environmental conditions jointly determine many disease states. Animal research has supported a diathesis-stress model of various psychophysiological disorders (Gatchel, et al., 1988). EVOLUTIONARY VIEW An evolutionary perspective would say that physical symptoms and pain are a way the body signals something is wrong and that other activities should cease until the damage is stopped (Neese &Williams, 1994). Memory of a painful event, for example, teaches the individual to avoid the same situation in the future. Even when there is a medical condition such as an injury or surgery to remove an organ, the pain is adaptive because continued use of damaged tissue may compromise the effectiveness of the functional portions of the body that remain. Thus pain is fundamentally essential for survival. Unpleasant emotions can be thought of as a defense analogous to pain from an evolutionary perspective. Just as the capacity for physical pain has evolved to protect human beings from tissue damage, the capacity for anxiety has emerged to protect us against future dangers and other kinds of threats. Another example of evolutionary protectiveness is our experience of fatigue to keep us from a state of overexertion. Evolutionary psychologists might say that to be able to respond to every situation and

cope with the challenges of life, adaptive ways of functioning (e.g., internal psychological regulation based on past experiences) were necessary for human survival. In this capacity, pain clearly functions as a protective survival mechanism. However, evolutionary processes do not always follow a completely logical track. Consider that our immune system is able to recognize and attack a million foreign proteins, yet we still get pneumonia. Is a survival-oriented, yet perhaps unseen limit reached here also, as in fatigue? What might happen when too much emotional pain is experienced which predictably could lead to a serious psyehopathological state? If for example, an individual's early traumatic experiences were so overwhelming that a realization of them would make life appear unbearable, an individual might have another coping mechanism that would take the (emotional) overload completely out of the anxious state and into the physical body focus. A parallel illustration for this concept might be when a student recognizes that he/she is failing a course and feels anxious. Doesn't he/she tend to drop the class (not facing the failure which could be devastating) and try another subject (or even quit school)? Not failing is equated with emotional survival in this case. Withdrawing is the way that the pain of failing (not surviving) functions to limit an individual's range of responses, whether that is an intolerable emotional feeling, or a physically painful experiences. This makes sense clinically where somatoform patients are observed as having severe limits in their own capacity for absorbed attention, processing information, and goal-directed behaviors. HYPOTHESIS OF A PRE- AND PERINATAL ANTECEDENT FOR SOMATOFORM PAIN COMPLAINTS If the psychological components of the DSM-IV somatoform pain disorders emerge somehow from emotional stress or trauma in the absence of physical findings, how the affective antecedent is transformed into physical symptoms should be of interest to researchers attempting to learn about, and help, this difficult patient population. Returning to the evolutionary psychology viewpoint, the reason the emotional pain may become perceived physical pain is an adaptation of escape and avoidance from stress or trauma allowing the organism to survive. This transformation may occur through the process of classical conditioning where the early experience is extremely agonizing-often a life or death event. Kessler and his colleagues (1996) have stated that the effects of childhood adversities on later adult psychopathology are largely due to powerful effects at first onset rather than later events or conditioning. We also know neurophysiologically that bundles of nerves, connected together, learn by repeating highly emotive experiences (van der Kolk, 1989). Researchers are calling for clarification of the antecedents of psychopathology, subjective emotional conditions and experiences, and physiological activity (Behavioral Science Task Force of the National Advisory Mental Health Council, 1995). It appears that the entrenched, but adaptive, somatoform behaviors originated and remain in the preverbal neural pathways laid down in the pre- and perinatal time frame of life. When taken in total with later reinforcing and socializing experiences, these illusive origins of later symptoms often remain unreachable to the clinician in the somatoform patient. Emerson (1996) has indicated that the earliest (pre- and perinatal) "traumas shape how subsequent events will be perceived and experienced." Frank Lake (1987,1981) also pointed to the period of gestation for the origin of somatoform complaints. According to Lake, psychosomatic equivalents of chronic resentment and hostility are early emotional states ("persecutory womb experiences") that produced high blood pressure, ulcerative colitis, asthma, rheumatoid arthritis, peptic ulcers, dermatitis, chronic colds or sinus infections, and chronic nasal congestion. Lake's supposition was that prenatal trauma is repressed and then transferred into other areas of the body until it later manifests in a dysfunction or disease. The immediate critique to a pre- and perinatal psychology paradigm has been that for the most part psychologists do not believe the idea that people have the ability to retain early traumatic experiences (Bauer, 1996). The objections are based on the lack of solid evidence that early experiences are related to later mental processes and the general conviction that the preverbal brain is not sufficiently developed to record early experience (Riedlinger &Riedlinger, 1986). But hopefully, this view is becoming antiquated with the medical innovation of obstetrical monitoring devices (ultrasound and fiber optic cameras) that allow us to view prenates as active, and reactive (physically and emotionally) when they can be seen to strike out against the invasiveness of an amniocentesis needle. These new technologies are now added to the overwhelming evidence that the physical and sensory

systems are functional during the preand perinatal time frame. Further, with the addition of LeDoux's valuable research it has been shown that there is a functional neural pathway for fast-acting, emergency-type responses other than only through the cortex, as was previously thought. As long as the antecedents of somatic pain complaints remain unknown, these patients remain difficult and costly to treat. What is known is that there are physiological, social, and psychological variables that need to be considered when treating these disorders. What is not known is what gives a person a propensity or premorbidity toward having physical pain as their primary symptom complaint. Human beings' emotional and physical defenses are the (primary) armament they have when they are at the earliest preverbal developmental stage of life. These sensory and bodily coping mechanisms that worked early on were laid down in the developing brain's neural pathways which-after cultural and environmental reinforcement, continue to direct the behaviors in the present. This behavior occurs over and over in the present when a classically conditioned emotional stressor is triggered by a life circumstance, The psychological stress factor reverberates through the system, then defaults to the physical pain focus, as it did originally. An everyday example of this is when a person pulls his/her hand away from a hot surface instinctively. This strategy is superior to taking the time to evaluate if it is in our best interest to withdraw. References REFERENCES American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, D.C.: Author. Bauer, P. J. (1996). What do infants recall of their lives? Memory for specific events by one- and two-year-olds. American Psychologist, 51(1), 29-41. Behavioral Science Task Force of the National Advisory Mental Health Council (1995). Basic Behavioral Science Research for Mental Health: A National Investment: Emotion and Motivation. American Psychologist, 50(10), 838-845. Benoliel, J. (1995). Multiple meanings of pain and complexities of pain management. Nursing Clinics of North America, 30(4), 583. Bower, G. H. (1981). Mood and memory. American Psychologist, 31, 129-148. Carlson, N. R. (1994). Physiology of behavior. Boston, MA: Allyn and Bacon. Chaplin, S. L. (1997). Somatization. In W. Tseng &J. Streltzer (Eds.), Culture &psychopathology: A guide to clinical assessment (67-86). New York: Brunner/Mazel. Dominguez, B., Valderrama, P., de los Angeles Meza, M., Perez, S. L., Silva, A., Martinez, G., Mendez, V. M. &Olvera, Y. (1995). The roles of disclosure and emotional reversal in clinical practice. In J.W. Pennebaker (ed.), Emotion, disclosure, &health. Washington, DC: American Psychological Association. Emerson, W. R. (1996). The vulnerable prenate. Pre- and Perinatal Psychology Journal, 10(3), 125-142. Fauman, M. A. (1994). Study Guide to DSM-IV. Washington, D.C.: American Psychiatric Press, Inc. Gatchel, R. J., Baum, A., &Krantz, D. S. (1988). An introduction to health psychology (2 d ed.). New York: McGraw-Hill. Goleman, D. (1995). Emotional Intelligence. New York: Bantam Books. Kessler, R. C., Nelson, C. B., McGonagle, K. A., Lieu, J., Swartz, M., &Blazer, D. G. (1996). Comorbidity of DSM-111-R major depressive disorder in the general population: results from the U. S. National Comorbidity Survey. British Journal of Psychiatry, 169, 17-30. Kirkwood, C. R., Clure, H. R., Brodsky, R., Gould, G. H., Knaak, R., Metcalf, M., &Romeo, S. (1982). The diagnostic content of family practice: 50 most common diagnoses recorded in the WAMI community practices. Journal of Family Practice, 15(3), 485-492. Kirmayer, L. J. &Taillefer, S. (1997). Somatoform disorders. In S. M. Turner, &M. Hersen (Eds.), Adult psychopathology and diagnosis (3d Ed.). New York: John Wiley &Sons, Inc. Lake, F. (1987). Clinical Theology. New York: Crossroad. Lake, F. (1981). Tight Corners in Pastoral Counselling. London: Darton, Longman and Todd. Lang, P. J. (1984). The cognitive psychophysiology of emotion: Fear and anxiety. In A. J. Tuma &J. D. Maser (Ed.), Anxiety and anxiety disorders (pp. 130-170). Hilldale, NJ: Erlbaum. LeDoux, J. (1993). Emotional memory systems in the brain. Behavioral and Brain Research, 58, 24-39. Leventhal, H. (1984). A perceptual-motor theory of emotion. In L. Berkowitz (Ed.), Advances in experimental socialpsychology (pp. 117-182). New York: Academic. Leventhal, H., &Tomarken, R. (1986). Emotion: Today's problems. Annual Review of Psychology, 37, 565-610. Levi, L. (1974). Psychosocial stress and disease: A conceptual model. In E. K. Gunderson &R. H. Rahe (Eds.), Life stress and illness. Springfield, IL: Charles C. Thomas. Mayou, R., Bass, C., &Sharpe, M. (1995). Overview of epidemiology, classification, and etiology. In R. Mayou, C. Bass &M. Sharpe (Eds.), Treatment of functional

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