Psychologic Versus Somatic? Is It a Pertinent Alternative?

It is common knowledge that psychosocial and behavioral factors strongly influence chronic pain. Almost 20 years ago, 3 or 4 groups[1,2] of chronic pain patients could be identified on the basis of scores for pain intensity, life interference, emotional distress, life control activity, and social support. The individualized groups were called dysfunctional, interpersonally distressed, repressors, and adaptive copers. It was suggested later that psychologic factors were more important than the actual disease entity in terms of management and outcomes.[3] In addition, a low correlation was reported between somatic signs and symptoms and psychologic factors. Accordingly, Dworkin and LeResche[4] proposed considering 2 separate axes, an Axis I, linked to somatic signs and symptoms, and an Axis II, linked to psychologic factors, each receiving a separate diagnosis and requiring separate treatment. This rationale is also supported by the results of a cluster analysis that, using signs and symptoms as variables, showed a totally distinct location of the somatic versus the psychologic signs.[5] This is in full agreement with the statement of Turk and Rudy,[3] which can be summarized as follows: A depression should be treated as such without considering whether the subject is also suffering from, for example, stomatodynia or arthromyalgia.

While the recognition of the affective and cognitive dimensions of chronic pain has been an important advance, the aforementioned emphasis put on dichotomized presentation of clinical cases may also constitute a pitfall. Although an individual cannot be split into psyche and body, the basic concepts underlining the therapeutic responses are quite separated. To respond to Axis I diagnoses, treatments are prescribed by a dentist or physician using mostly pharmacologic and mechanistic tools. In a parallel way, Axis II diagnoses are addressed by different tools handed out by psychologic specialists. In the best-case scenario, these 2 groups of professionals are gathered in a pain center where they communicate with one another. In some other cases, probably more frequently, the dentist or physician notes that his or her patient cannot be cured by somatic techniques and that he or she should be treated by psychologic techniques, but psychologic treatment is not available or affordable, and in the end, no psychologic treatment is carried out. In a third scenario, the patient receives the old and terrible statement that "her pain is in her mind." Actually, the gap between soma and psyche, which was introduced in diagnoses and therapeutic techniques, also exists in our therapists' minds. The crucial question is, is there a gap inside the patient's brain?

Obviously the response is no. The individuality of anyone's psyche is a direct consequence of both genetic makeup and the impact of personal life experiences on neuronal functioning. The implication is that certain environmental stimuli may influence brain affective and cognitive functions. Physiology and neurochemistry of pleasure, sexual drive, passion, addiction, and fear fill book chapters but are not as pertinent for orofacial chronic pain as physiology and neurochemistry of anxiety and response to stress can be. The response to a stressful event is an indispensable adaptation aimed at maintaining the homeostatic balance. The increase in the circulating levels of corticosteroids, in particular cortisol, in response to hypothalamic-pituitary-adrenal (HPA) activation, is the major physiologic response to acute physical or psychologic stress, as also noted in the article by Bertoli et al[6] in this issue of the *Journal of Orofacial Pain*. Under normal conditions, the HPA axis is controlled by an autoregulated feedback system which controls and tempers the response and rapidly drives the response to the background level. There is evidence, however, that sustained functional impairment of the HPA axis may occur in the presence of certain kinds of environmental stimuli. Changes in the control of cortisol have been shown during chronic stress as well as depression and anxiety.[7,8] As noted in this issue of the *Journal* by Bertoli et al,[6] post-traumatic stress disorders developed by women or men who experienced sexual and/or physical abuse in early childhood or following exposure to an extreme traumatic stressor are other examples of disruption of normal control of adrenals by the hypothalamus and pituitary gland.[8] These changes may result in both hypo- or hypercortisolism[8,9] and in dysregulation of many other steroids.

Chronic stress, post-traumatic stress, depression, and chronic anxiety are prevalent states in what have been called *functional pain conditions*.[10] This group of disorders includes fibromyalgia, chronic fatigue syndrome, chronic pelvic pain, irritable bowel syndrome, atypical depression, atypical facial pain, myofascial pain, temporomandibular joint and masticatory muscle disorders, stomatodynia, and post-traumatic stress disorder. Although they form a major health problem with a rising prevalence, they are devoid of any obvious organic signs, their etiology is still unknown, and treatments are mainly symptomatic. In addition to being putatively associated with some kinds of psychologic distress, all these conditions are also characterized by a marked gender effect; they are more prevalent among women.[10] This is probably best exemplified in stomatodynia, which displays a female-male ratio of about 10 to 1.[11]

A general explicative hypothesis for the symptoms seen in patients suffering from functional conditions could rely on long-standing and simultaneous modifications in the main sources of steroid hormones. Permanent changes in the HPA axis, resulting in changes in the levels of gluco-

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corticoid levels that have already been evoked, together with constant modifications in the levels of gonadal steroids, could be the basis for the triggering of the pain. That these changes in steroid concentration are related to symptoms implies that permanent changes in steroid concentration have consequences for the neural cells. There is already much evidence of such steroid-induced changes in both the peripheral and central nervous systems. For example, while basal release of stress steroids mediates positive functions in the brain,[12] both high and low cortisol levels have been described as damaging to neurons or to the brain.[13] Similarly, neuroprotection appears to be a major effect of estrogen and testosterone steroid receptor activation.

This hypothesis may also explain the following paradox: Although the different functional conditions share many common symptoms and are often associated in the same individuals,[5,9,10] they have also been described as separate entities.[5,14] The old debate between researchers who believe that the functional conditions are almost all the same and those who think that they are distinct entities could be the direct consequence of the variability observed in the changes of both gonadal and adrenal steroids from one condition to another. Indeed, the direction of gender effect differs from one disease to another. For instance, the reproductive period is a risk factor for myofascial pain,[15] although stomatodynia is mostly seen in the menopausal and postmenopausal periods.[11,16] Similarly, patients often have symptoms and/or history of anxiety or depression in stomatodynia,[16] while post-traumatic chronic stress is frequently associated with fibromyalgia.[17] Therefore, the many possible different forms of change in HPA axis and gonadal production could be the causes of the different groups of symptoms that individualize the different functional diseases.

Steroids are obviously not the only factor by which aggressive environmental stimuli modify the physiology of the nervous system, but because of the influences of these steroidal substances in the gender factor and in the anxio-depressive context seen in epidemiologic studies, they surely stand among the top candidates to link the concepts of psyche and soma of persons with functional pain. The identification of any specific imbalance in steroids of these functional entities that is hypothesized here could be a key to understanding better the pathophysiology for this functional entity and therefore to proposing an adapted treatment.

The approach that has been briefly noted in this editorial may be profitable to the patient, since it could eventually lead to new therapeutic solutions. Also, it is quite different to tell a patient that he or she is suffering because of anxiety, and to explain that this anxiety is causing a disturbance in the level of steroids somewhere inside the body, than to tell the patient that the pain is a symptom of psychological perturbation.

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