PEDIATRICS PERSPECTIVES

Unintentional Symptom Intensification by Doctors

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Physicians at times are confronted with patients who present with vague yet disabling, nonprogressive symptoms for which they can offer no concrete medical explanation despite extensive evaluation. Many individuals who present with abdominal complaints, musculoskeletal pain, headaches, fatigue, dizziness, and other symptoms and/or their families believe that an undiscovered anatomic or biochemical aberration explains their symptoms despite having been informed that the symptoms may represent a "functional disorder."[1] They have difficulty believing that relatively common disorders such as functional abdominal pain,[2] fibromyalgia,[3] and chronic daily headache can cause the incapacitating symptoms that they or their children are experiencing. Their belief that a more specific "organic" explanation exists is rooted in the medical model, which implies that there is a specific cause for most symptoms that, when identified, will offer a path to cure.[4] The faith in this conviction is reinforced by reports of miraculous cures in the press, in medical dramas on television, and by material on the Internet that allows patients to search for uncurated information that might explain their symptoms and communicate with others confronting similar issues. Pediatricians may be particularly vulnerable to experiencing this phenomenon because of intense parental desire for a diagnosis that captures the symptoms that their child is experiencing.[5] As a result, many individuals believe that the solution to their or their child's suffering exists around the next investigative corner, and they bring this expectation to each physician encounter. Unfortunately, at the end of these visits, if no organic diagnosis emerges, patients are often disappointed and unsatisfied and physicians are often frustrated.

We perceive that physicians generally tend to fall into 1 of 3 camps in responding to persistent nonspecific symptoms in patients, although this observation does not preclude clinicians from changing philosophy as the evidence emerges or evolves: (1) those who continue to investigate symptoms despite a lack of "alarm" signs, even if no etiology emerges, occasionally motivated by liability considerations; (2) those who suggest that after "adequate evaluation" without emerging evidence of organic disease, the etiology is psychological and requires primarily psychological intervention; and (3) those who suggest that the explanation is either

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a controversial disorder or, more typically, a rare yet previously well-defined entity for which the traditional diagnostic boundaries have been stretched to accommodate the patient's presentation. Certainly, a clinical diagnosis of mast cell activation disorder, Ehlers-Danlos syndrome, postural orthostatic tachycardia syndrome, as well as a host of other syndromes, if present, may provide a tangible explanation and offer a specific treatment pathway and diagnostic home for the patient. It is equally likely, however, that these entities, even if present, have triggered or explain only a part of the puzzle and do not explain the entire multifactorial, individual clinical picture.

For any given patient, any of these strategies may be successful at alleviating his or her symptoms. However, they can also contribute to enormous costs, inappropriate labeling, and significant expansion of entities previously considered rare.[1] Although we recognize that, in fact, there are times when each of these approaches may be accurate, we would suggest that cumulatively they often create a situation in which there is unintentional intensification of these chronic symptoms, which may prolong the patient's problems and delay appropriate rehabilitative treatment. In any of these well-intended approaches, the patient may be subjected to unnecessary (and expensive) testing, unneeded psychiatric intervention, or assignment of a diagnostic label that may lead to identification with a disease that has expanded so significantly as to undermine attempts to meaningfully study it or lead to a diagnosis without corroborating evidence. These approaches may result in potentially harmful and/or expensive treatment and may yield inappropriate expectations for the future.

Consideration should therefore be given to the application of a previously well-described fourth pathway, the biopsychosocial model,[4] that assumes a different paradigm. With this model, it is suggested that the illness occurs in individuals in whom there are genetic or other biological vulnerabilities that, when triggered by environmental, biological, and psychological factors, ultimately impact the hypothalamic-pituitary-adrenal axis, immune system, microbiome, and nociceptive pathways to create peripheral and central sensitization, which, when triggered, produces the "hypersensitivity" responsible for this symptom cluster.[6] There is also a well-described bidirectional effect between psychological characteristics and responses to pain. In each case, the biological mechanism of the symptom may ultimately be similar, but the root causes may be significantly different. This allows for early adverse events, infections, inflammation, personality characteristics, past medical illnesses, and psychosocial stresses to be incorporated into any explanation for persistent symptoms. With increasing research on epigenetics, especially DNA methylation and neuroplasticity,[6] we can understand why similar triggers may evoke different responses in different individuals or in the same individual at different times in his or her life. Before our understanding of the complex multifactorial etiology of central sensitization, this category of disorders had been known as functional, which was often interpreted as "psychological." In the pending *International Classification of Diseases, 11th Revision*, these disorders, when pain is associated, are labeled as chronic primary pain disorders (a more-neutral term not laden with emotion and historic inaccuracy). In recent research, it has been shown that functional disorders can also occur in the setting of well-defined organic diseases that are well under control (sickle cell, inflammatory bowel disease, etc).[7]

What are the implications of this approach for clinical practice? First and foremost, it offers an explanatory model that is not reductionistic or dichotomous and allows for a continuum. The diagnosis is positive and not one of exclusion. It validates the legitimacy of symptoms without ascribing them to a specific, simple, treatable root cause. Finally, it offers a pathway (the rehabilitative model), which allows patients to take control of their symptoms, removes them from the investigative assembly line, and avoids the self-recrimination often associated with psychological explanations or the overidentification with a disease process that may or may not be accurate yet often has significant treatment implications. It also opens the door to psychological and physical interventions that are geared to symptom control and better functioning.

For such a model to work, however, a number of conditions must be met. Generally acceptable algorithms for what constitutes an adequate evaluation of specific symptoms need to be developed, validated, endorsed by relevant organizations, and promoted to the practicing community. This should include the identification of alarm signs or biomarkers that would demand additional investigation and need to be constantly updated to keep up with new discoveries. Such a model has been developed for pediatric functional gastrointestinal disorders.[2]

How the symptoms are explained is critical. An individual's understanding of his or her condition has been linked to compliance with recommendations, level of disability, satisfaction with care, and with ultimate outcome. The research of Moseley and others[8,9] has demonstrated the importance of this "neuroeducation," and the use of metaphors to help children and adolescents understand their condition is especially beneficial. For this approach to be successful, the clinician also needs comfort with

symptom control, independent of etiology. Sophisticated management targeting specific neurologic, psychological, musculoskeletal, and gastrointestinal symptoms is often helpful. Another essential feature is the concept of "watchful waiting" while instituting treatment focused on rehabilitation of function. By scheduling frequent appointments, patients can be assured that their symptoms are being closely monitored and will not be ignored. Although it has been strongly suggested in studies that evaluation and treatment by a multidisciplinary team results in fewer hospitalizations, reduced reliance on medication, and generally better functioning, access to such teams is severely limited. Ultimately, however, the relationship between the patient and provider is the most important variable in determining outcome. It has been suggested in research that in adults seeking treatment of chronic pain, for example, satisfaction correlated more with the quality of the patient-provider relationship than with the reduction of pain.[10]

We recognize that there are some patients and their families who will remain fully entrenched in the traditional medical model as there will be clinicians who will continue to assign specific diagnoses to their patients despite the limited support in the literature for those assumptions. These contingencies are inevitable. However, we do feel that reframing symptoms from a medical to a biopsychosocial model will reduce the promotion of unnecessary investigation, attribution of vague symptoms to stress or other psychological origin, and provision of false hope or false pessimism. Such a model may reduce some of the heated controversies and patient frustrations often associated with these diagnoses, promote the continued research into mechanisms that will allow us to understand and target more directly these complicated conditions, and diminish some of the unfortunate labeling and suffering that surround them.

REFERENCES

1. Mayer E, Bushnell MC. Functional Pain Disorders: Time for a Paradigm Shift. In: Mayer ME, Bushnell MC, eds. *Functional Pain Syndromes: Presentation and Pathophysiology*. Seattle, WA: IASP Press; 2009:pp 531–565
2. DiLorenzo C, Nurko S. *Rome IV: Pediatric Functional Gastrointestinal Disorders*, 1st ed. Raleigh, NC: Rome Foundation; 2016
3. Sluka KA, Clauw DJ. Neurobiology of fibromyalgia and chronic widespread pain. *Neuroscience*. 2016;338:114–129
4. Engel GL. The need for a new medical model: a challenge for biomedicine. *Science*. 1977;196(4286): 129–136
5. Riley B, Palermo T, Walco G, et al. Persistent Pain in Children. In: Ballantyne JC, Fishman AC, Rathmell J, eds. *Bonica's Management of Pain*, 5th ed. Philadelphia, PA: LWW; 2018:pp 509–529
6. Arnedt-Nielsen L, Morilon B, Perrot S, et al. Assessment and manifestation of central sensitisation across different chronic pain conditions. *Eur J Pain*. 2018;22(2):216–241
7. Bromberg MH, Schechter NL, Nurko S, Zempsky WT, Schanberg LE. Persistent pain in chronically ill children without detectable disease activity. *Pain Manag*. 2014;4(3):211–219
8. Gallagher L, McAuley J, Moseley GL. A randomized-controlled trial of using a book of metaphors to reconceptualize pain and decrease catastrophizing in people with chronic pain. *Clin J Pain*. 2013;29(1):20–25
9. Coakley R, Schechter NL. Chronic pain is like…The clinical use of analogy and metaphor in the treatment of chronic pain in children. *Pediatric Pain Letter*. 2013;15(1):1–8
10. Hirsh AT, Atchison JW, Berger JJ, et al. Patient satisfaction with treatment for chronic pain: predictors and relationship to compliance. *Clin J Pain*. 2005;21(4):302–310