He suggests that careful patient selection based on clinical guidelines of best practice can have economic benefits but must be supported by the reimbursement climate. His paper is a model for the evidence that is needed to support our practices in pain medicine, the kind of evidence that Dr. Steig calls for in his insightful review of the state of financing for pain treatment.

This first issue also presents a compelling first look at a conceptual model that attempts to carefully and logically explain, through the lens of neuroscience, the biopsychosocial clinical phenomenology of chronic pain. The Rome's paper is meticulously researched and beautifully written; it should be digested by all pain clinicians and researchers.

Why a new journal? The Academy can take great pride in the successful development of its former journal; but without ownership, the Academy, its members, and the field of pain could not benefit from its prior investment, leadership and success, and the journal's further growth and development. The need for AAPM's journal to support our academic mission, the desire to continue its multidisciplinary focus, and the desire to develop electronically and globally, in concert with the evolution of the field, were paramount considerations in the decision to begin our own journal. Our new publisher, Blackwell Science, is a partner dedicated to that vision, and is superbly positioned with its sophisticated electronic publishing and global network to help us achieve our goals. You can receive PAIN MEDICINE electronically and on paper, too.

The journal will reflect the breadth of our field and the interests and needs of our multidisciplinary audience, as the content of this first issue attests. Of course, research papers will be the core of the journal and we need you to submit your good work to us, without hesitation. We will present important conceptual papers, such as by Howard and Jeffrey Rome, and scholarly reviews of diverse topics, such as by Steig, by Marbach and Raphael, and by Shoemaker and Ashburn. Case conferences, case reports of promising treatments, and timely, helpful, entertaining book reviews will round out original manuscripts. We desire a lively intellectual exchange in our Letters to the Editor, so please respond to our articles or to issues that you believe are important for our discussion and debate. I will facilitate their publication. Our dedicated, experienced board promises timely, expert, and helpful reviews without inordinate delays, reviews that will sharpen your scholarship as well as facilitate publication. Our editors are very willing to discuss your ideas for a paper, so do not hesitate to write, call, or e-mail (easiest) me or other board members.

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the human experience. Our minds and hearts together can find their fullest expression in the personal encounter between patient and physician.

Pain as both neurophysiological event and as human suffering is a core dialectic of this physician experience. Pondering when in the course of evolution the experience of pain became particularly human, one must consider its personal qualities, the reverberation of pain throughout consciousness, or suffering. Suffering is at once profoundly personal and private, but also a poignant interpersonal state. Empathy, an interpersonal aptitude we consider most human, implies a capacity to understand, and even to experience, suffering in others. Empathy is an instinct that draws us powerfully to the role of healer. Nowhere in the practice of medicine is empathy more needed, but so difficult to sustain, than in caring for patients with unrelenting pain and suffering and associated negative emotions and behaviors.

Pain also engages our inquiring, scientific intellect and its product, technology. The neurophysiology and molecular biology of pain and the pain experience challenges many of our best minds, and the response is magnificant. We now possess a detailed knowledge of the physiology and pharmacology of acute pain, and are unraveling the molecular neurobiology of persistent pain, particularly the peripheral somatic aspects of nociception and pain transmission [1]. Constructs such as neuroplasticity and sensitization are now established in our concept of a dynamic system that subserves pain perception and modulation and their behavioral correlates [2]. As these processes are revealed through our science, the notion of the separation of neurobiology of the mind, brain, and body represents a cognitive construct, a formerly convenient heuristic that is no longer scientifically tenable or clinically useful. Body, brain, and mind are complexly reciprocal, with cognition, emotion, and pain different but related neuromatrix functions. The questions remain: how do emotions exert their effects on pain, and vice versa? How do we explain the saliency of depression in chronic pain? What is the function, role, and impact of experience? Despite the delineation of some aspects of central discrimination and emotional processing [3,4], a theory that enables us to integrate the phenomena of emotion and pain with experience has remained elusive.

The paper by Howard Rome and Jeffrey Rome carefully musters the argument for a coherent theory of chronic pain that enables us to conceptualize these relationships through the lens of molecular biology and behavioral neurophysiology. They present a strong argument for the influence of “kindling (phenomena) and related models of neuroplasticity” on the development of a “sensitized corticolimbic state,” based in gene regulation and expression modified by experience. They aptly label this “Limbically Augmented Pain Syndrome,” and describe a typical case with which pain clinicians are all too familiar. Their argument is convincing, making sense intuitively as well as empirically.

Any theory that promises to explain the complexities of human nature is immediately suspect. Yet the Romes have marshaled a cogent argument for fundamental processes that may enable us to explain much of what we see clinically and experimentally. For example, the data from Cano’s paper suggest that a spouse’s negative reactions to pain complaints may lead to decreased marital satisfaction and increased pain severity, and then to depressive symptoms. Are increased pain symptoms the first manifestations of emotional distress, followed by the negative mood state? Does this state then contribute to affective, experiential learning, described in neurobiological terms such as “kindling” and “sensitization”? Do these phenomena cause a vulnerability to pain flare-ups in response to interpersonal or even cognitive triggers that might seem insignificant at face value? Does this explain the finding that it may be the stress of living with chronic pain, not hereditary risk, that leads to depression in chronic pain patients [5]?

Full circle from the peripheral stimulus and central processing are the environment and the physician-patient encounter. Just as stress and negative emotions may sensitize the processes of pain activation from either the periphery or centrally, can the therapeutic interpersonal encounter desensitize these processes? Rainville and colleagues [3] have shown that hypnosis manifests therapeutic effects in the anterior cingulate gyrus, a locus for the unpleasantness aspects of pain [4]. And bedside manner has long been thought to effect the outcome of treatment creating a positive physiologic state in the patient that may modulate pain and its negative emotions. Many of us have noticed that when doing procedures, an effective bedside manner seems to correlate with better outcomes.

The power of a theory is judged ultimately by its utility. The Gate Theory of pain provided the field with a major conceptual breakthrough that led to an explosion of new research and much of the knowledge that defines our field today. Although the Romes’ thesis is constructed on a strong empirical foundation of basic and clinical science, the hard work of testing the utility of this and other
neurobiological models of biopsychosocial medicine challenges us all.

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