PAIN MEDICINE
Volume 9 • Number 7 • 2008

CLINICAL NOTE

Pain Medicine and Its Models: Helping or Hindering?

John L. Quintner, MB BS, FFPMANZCA,* Milton L. Cohen, MD, FFPMANZCA,†
David Buchanan, PhD,‡ James D. Katz, MD,§ and Owen D. Williamson, MB BS, FFPMANZCA¶

*Department of Rheumatology, Royal Perth Hospital, Perth, Western Australia, Australia; †Departments of Rheumatology and Pain Medicine, St Vincent's Campus, Sydney, New South Wales, Australia; ‡Department of Health Sciences and Nursing, Notre Dame University, Fremantle Campus, Western Australia, Australia; §Division of Rheumatology, George Washington University, Washington, DC, USA; ¶Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Victoria, Australia

ABSTRACT

Objective. To identify whether the biopsychosocial framework of illness has overcome the limitations of the biomedical model of disease when applied in the practice of pain medicine.

Design. Critical review of the literature concerning the application of biopsychosocial models to the praxis of pain medicine and the concepts of living systems.

Results. The biopsychosocial model of illness, formulated by Engel in 1977, has generated the International Association for the Study of Pain (IASP) definition of pain, two major conceptual frameworks in pain medicine, and three putative explanatory models for pain. However, in the absence of a theory that seeks to understand the lived experience of pain as an emergent and unpredictable phenomenon, these progeny of the biopsychosocial model have been caught in circular argument and have been unable to overcome biomedical reductionism or the perpetuation of body–mind dualism. In particular, the implication that pain can be a "thing" separate and distinct from the body bears little relationship to the lived experience of pain. Such marginalizing results when an observer attempts to reduce the experience of the pain of another person.

Conclusions. The self-referentiality of living systems (through their qualities of autopoiesis, noncentrality and negentropy) sees pain "emerge" in unpredictable ways that defy any lineal reduction of the lived experience to any particular "thing." Pain therefore constitutes an aporia, a space and presence that defies us access to its secrets. We suggest a project in which pain may be apprehended in the clinical encounter, through the engagement of two autonomous self-referential beings in the intersubjective or so-called *third space*, from which new therapeutic possibilities can arise.

Key Words. Biopsychosocial; Reductionism; Dualism; Aporia; Self-referentiality; Intersubjective; Third Space

Introduction

Although not always recognized by clinicians, their knowledge, experience, and praxis are imbued with theoretical underpinnings that arise out of the prevailing working conceptual models, in which they play a pivotal role:

> As observers we are also creators building complementary pictures of inexhaustible reality. As agents our simplest behaviour will have repercussions, probably unsuspected, in many systems which concern us. And, above all, whether as observers or agents, we are ourselves part of the systems we seek to understand and to change or sustain [1].

Conceptual models are no more than sets of ideas that shape the practice of scientific medicine. They are constructed from general theories and then used as learning tools or vehicles whose function is to explain and predict specific natural processes underlying bodily function [2,3]. They tend

Reprint requests to: Milton L. Cohen, MD, FFPMANZCA, St Vincent's Medical Centre, 376 Victoria Street, Darlinghurst, NSW 2010, Australia. Tel: (+61) 2 9361 5900; Fax: (+61) 2 9361 5061; E-mail: m.cohen@unsw.edu.au.

 1526-2375/08/$15.00/824 824–834 doi:10.1111/j.1526-4637.2007.00391.x

to mirror, duplicate or in some way illustrate the pattern of relationships of interest [4].

Scientific models undergo regular assessment and manipulation in the light of new data, ideas, and concepts. Then they are either modified or, should new evidence emerge that renders them insupportable, eventually discarded and replaced by more useful models.

The Biomedical Model of Disease

Biomedicine espouses that all symptoms—including pain—are expressions of a discoverable disease process and that there is a reliable connection between pathological changes and clinical features. This belief in the primacy of structural pathology has become the major criterion for discovering disease in Western societies, eclipsing functional pathology and relegating behavioral, psychological, and social factors to the background.

> As Foucault [5] suggested, this approach
> . . . gave to the clinical field a new structure in which the individual in question was not so much a sick person as the endlessly reproducible pathological fact to be found in all persons suffering in a similar way. (p. 97)

The task allotted to the clinician is to seek out the underlying cause of a symptom by artificially stripping down the body into separate identifiable elements and then searching each element, be it organ or system, to find an abnormality. The biomedical approach is in fact an explanatory tool, as it holds that there is a predictable relationship between change in bodily structure and a person's complaint.

The implications of the biomedical model are profound. First, the experience of illness is demoted to the depersonalized expression of a disease. Second, the process of diagnosis becomes reductionistic, whereby the only phenomena of interest are to be isolable, causal chains. Furthermore, important relationships can exist only between a small number of such chains, which are assumed to behave predictably and apply universally [6].

When applied to the problem of clinical pain, the biomedical model posits a predictable and linear relationship between identifiable tissue damage and the report of pain, thereby presuming a neurobiologically hard-wired connection between the site of that damage and the brain. In this model, nociception is a necessary precondition for pain. That is, if there is no nociception, there can be no "real" pain. It follows that when a site of nociception cannot be localized, the patient's report of pain is at best doubted and at worst disbelieved. In effect, this privileges the ostensibly objective view of the clinician, which necessarily excludes the sufferer's expertise of their lived experience of pain. This defaults to either dismissal of the patient's complaint or to an inference of "psychogenesis" by the clinician.

When applied to psychiatry, the biomedical model reduces behavioral aberrations to diseases of the "mind," necessarily ignoring all (neuro)phenomena that fall outside this framework. In both medicine and psychiatry, this model has reinforced the prevailing doctrine of body/mind dualism. In short, by separating the mind from body, medicine marginalizes unnecessarily the lived experience of the patient.

Engel's Biopsychosocial "Model" of Illness

In response to what was seen by many clinicians as a crisis of interpretation inherent in the biomedical model, psychiatrist George Engel [7,8] proposed the biopsychosocial "model" of illness. His work is based upon a theory of open systems and its further generalization in General System Theory (GST), as proposed in the mid-20th century by biologists Paul Weiss [9,10] and Ludwig von Bertalanffy [11]. They independently argued that no biological system exists in isolation and that every system is influenced by the configuration of its environment, which comprises a hierarchical continuum of ever larger and more complex systems, extending from the level of the subatomic particle to the biosphere. According to GST, any perturbation sufficient to cause changes in any one system might *ipso facto* produce changes in any or all of the other systems in the hierarchical structure.

Engel's intent was to provide a scientific framework "within which can be conceptualised and related as natural systems all the levels of organization pertinent to health and disease" [12]. He wished to retain the scientific assumptions of biomedicine while at the same time enlarging its scope to include psychological and social factors [13]. Engel conceptualized health, disease, and disability in terms of the relative intactness and harmonious functioning of the components of systems operating on each level:

> Overall health reflects a high level of intra- and intersystemic harmony. Such harmony may be disrupted at any level, at the cellular, at the organ system at the whole person or at the community level. Whether the resulting disturbance is contained at the level at which it is initiated or whether other levels become implicated is a function of that system's capacity to adjust to change. [14]

In contrast to the biomedical model's "simple cause-and-effect explanations of linear causality" [12], Engel invited clinicians to entertain the proposition that patients' self-reports of illness could be influenced and sometimes even determined not only by biological but also by psychological, social, and cultural factors [14]. He hoped that clinicians would meticulously collect qualitative and quantitative data in each of these domains and incorporate them into the diagnostic and therapeutic processes, thereby arriving at a better understanding of the "human experience" of disease or, more correctly, of illness. Importantly, these meaningful transactions were to take place at the "two-person" system level, that is, within the doctor/patient relationship.

Pain Medicine Embraces Engel's Framework

Theoreticians in pain medicine were quick to adapt the biopsychosocial framework, as it seemed to accommodate the burgeoning recognition of pain as an expression of complex interactions between biological, psychological, social, and cultural factors [15–17]. The important contribution of such interactions to the lived experience of pain had already been predicted by the gate control theory of Melzack and Wall [18], which focused on the plasticity and modifiability of events at all levels within the central nervous system [19]. As conceptualized by Basbaum et al. [20], it is not only the nature of a particular stimulus but also the context within which it is experienced, together with associated memories, emotions, and beliefs, etc. that determine whether or not a person will perceive a particular stimulus as painful.

The application of the biopsychosocial framework to the discipline of pain medicine has been very useful in facilitating the evaluation of clinical pain, the generation of putative explanatory models and to some extent modes of therapy. The framework also promised to provide useful insights into enigmatic situations such as pain in the absence of nociception, the absence of pain in the presence of tissue damage, the variability and unpredictability of individual responsiveness to identical treatments, and the lack of a predictable relationship between pain, impairment and disability [17]. However, this promise has not been fulfilled, as the predicament of the person in pain without demonstrable nociception remains controversial [21]. Such patients are still seen by some purportedly independent observers as having a problem originating in the "sphere" of their mind rather than in their body [22–24].

In this review of the biopsychosocial framework a quarter of a century after its proposal, we find that the person in pain continues to be seen as an object of enquiry by the clinician according to the reductionist rules imposed by the biomedical model and that pain theorists have fallen into the trap of regarding pain as an objective "thing." This trap has not only prevented the formulation of a theory of pain, but also has perpetuated culturally entrenched body–mind dualism. Thus, the biopsychosocial initiative is better seen as another dualistic framework for analysis rather than as an explanatory model for the more complex neurophenomenology of emergent pain states. By contrast, as we have argued elsewhere, pain cannot be so reduced because the lived experience characterizes the mystery of an aporia, a space to which an observer is denied access [25]. In an attempt to accommodate this aporia, we suggest that there must be other theoretical and practical vantages that can be invoked in the practice of pain medicine.

Biopsychosocial Frameworks Extant in Pain Medicine

International Association for the Study of Pain Definition

In 1979, the International Association for the Study of Pain (IASP) defined pain, for clinical practice and research purposes, as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" [26]. This definition challenged the biomedical approach to pain, as it connoted that there were contributions other than nociception that may be relevant to the patient's presentation. From the perspective of the person in pain, the IASP definition had the potential for according legitimacy to their experience. Through breaking the nexus between actual tissue damage and the subjective experience, this definition has gained wide acceptance.

In the explanatory notes that follow the IASP definition, however, is the rider that "[M]any people report pain in the absence of tissue damage or any likely pathological cause; *usually this happens for psychological reasons*" [italics added]. Thus, without further elaboration, the IASP definition fell back into the circular trap of body–mind dualism, and has inadvertently sanctioned the efforts of clinicians to seek explanations for a variety of pain syndromes solely from psychosocial

factors, from which arises the untestable construct of somatization [27].

Notwithstanding the perhaps unforeseen consequence of this rider to the IASP's definition, theoreticians in pain medicine have responded to the challenge to formulate explanatory models for clinical pain using the biopsychosocial framework.

Loeser's Framework

Neurosurgeon John Loeser [28] proposed a framework consisting of four interacting hierarchical domains: nociception, pain, suffering, and pain behavior. Of these, only the last is observable, the interaction between the person in pain and the surrounding world. The clinician's task was to determine which of these four factors might be "playing significant roles in the genesis of the person's problem, and then to direct therapies at the appropriate aetiological factors" [28].

Despite its undoubted popularity, there are two major theoretical and practical problems with this approach. First, the framework suggests that pain, suffering, and pain behavior are somehow discrete phenomena, whereas in fact the clinician/observer is unable to make such a distinction. Second, nociception may be understood as a physiological (quantitative) process involving specific neuronal signals and pathways, but the other domains resist such a reductionist approach because they cannot be reflected in measurable physical properties (that is, they are qualitative). Furthermore, how do the physiological changes of nociception relate to these latter three domains, which are emergent properties of a self-organizing brain and therefore cannot be assigned to specific and predictable changes in the nervous system? To postulate a reliable link between the activity within in the nervous system and the various emergent phenomena constitutes a category error.

In effect, the "model" is not explanatory. Only three domains (nociception, suffering, behavior) are identified but the connection between them is undefinable and arbitrary. The framework implies that there are nonnociceptive influences on pain and behavior but it can neither escape dualism nor penetrate the unacknowledged aporia.

Waddell's Framework

The Glasgow Illness Model [29] was formulated to facilitate understanding by clinicians of the person presenting with chronic low back pain. This model postulates four domains of interest: physical problem, psychological distress, illness behavior, and social interactions. Its framework also implies contributions to the physical problem from nonsomatic factors.

Similar in operation to Loeser's framework, the Glasgow Illness Model obliges the clinician to differentiate the symptoms and signs of illness behavior from those of physical disease, as well as to detect any mismatch between the amount of demonstrable disease causing pain and the level of disability allegedly caused by the pain. How these crucial decisions are to be made is left entirely to the clinician's discretion, guided by the same arbitrary constructions constrained by dualism.

Although it takes Loeser's framework one step further by adding psychological distress, the Glasgow Illness Model also fails to explain the connection between these domains of interest, which leaves them as discrete nonconnected phenomenal domains, and as such becomes another nonexplanatory construct. Both frameworks are wholly observer dependent. That is, they privilege the "objective" view of the clinician when observing the "subjective" experience of the person in pain.

A Psychobiological Model

By contrast, Flor and Hermann [16] presented an explanatory model based on learning theory. In their view, pain is a multicomponent behavioral response to aversive stimuli. Through prior learning, operant and respondent conditioning, powerful pain memories are formed at all levels of the nervous system. Once established, such pain memories are said to be capable of maintaining pain even in the absence of peripheral nociceptive input.

The individual components (physiological, behavioral-motor, and subjective-verbal) of this response are said to reflect the dynamic and continuous interplay between various physiological and psychological factors. However, these same factors are also held to be responsible for the predisposition to and maintenance of chronic pain states, which constitutes again the circular reasoning of body–mind dualism.

However, when discussing the phenomenon of pain unrelated to trauma, Flor and Hermann [16] expand their view of pain from being a behavioral response (as a symptom) into an experience that may *cause* profound reorganizational changes within the nervous system, thus increasing an individual's susceptibility to all somatosensory input. Here, pain itself is conceptualized as an agent of change. In some way, this emergent aversive

response is capable of changing the very structure of the nervous system. Thus, the learned memory of pain perpetuates the reorganizational basis of its own genesis. This proposal accommodates all possibilities and thus not only constitutes a tautology but enshrines a circular argument.

Persistent Pain as a Disease

An echo of pain as a "thing" is seen in the attempt of Siddall and Cousins [30] to assign disease status to the clinical problem of persistent pain. These authors cite "a host of pathological changes" (labeled by them as "secondary pathology") within the nervous system, induced by altered sensory inputs from the periphery. These changes include anatomical reorganization in nociceptive pathways and the alterations in patterns of brain activation and cortical topography found in persistent pain states.

The proposition that persistent nociception may induce pathoanatomical and pathophysiological changes in the nervous system is in itself unexceptional in the light of recent knowledge. However, to then state "persistent pain does give rise to its own secondary pathology" is unacceptable as it elevates pain to the status of a causative "thing" and thus constitutes a circular argument. These authors then go further by adducing environmental factors as the "tertiary pathology" of pain and thus that pain is "an environmental disease." Such tertiary pathology includes "factors such as genetic makeup, level of spinal inhibition, psychological status and the societal litigation system."

In effect, this proposition is no different from identifying that nonnociceptive factors may be relevant to the pain experience. To label this as "tertiary pathology" is more than a terminological sleight-of-hand, as it carries the implication that these factors can in some unspecified ways contribute to an altered nervous system. But at the same time, by including "psychological status" as part of this tertiary pathology, and therefore external to the body, the argument defaults to the familiar circle of body–mind dualism. Furthermore, what is the pathological basis of the influence, for example, of the "societal litigation system"?

Siddall and Cousins's [30] proposition appears to be concordant with the biopsychosocial framework but their attempt to portray pain itself as a causative "thing" represents a dramatic return to the linear causality of the biomedical model. Moreover, they fail to distinguish between their "discovery" of the "pathology" of pain and any process which might lead to justification of its validity [31].

Pain as a Homeostatic Emotion

Craig [32–34] pursues this theme of grounding pain in disturbed body function by reconceptualizing it as a "homeostatic emotion." Influenced by the argument of William James [35] that emotion is a consequence of bodily change rather than vice-versa, Craig suggests that pain is one of many possible human emotions through which the integrity of the body (self) can be maintained in the face of conditions that threaten to disturb its homeostasis. He cites recent neurobiological findings that indicate that pain is not part of the exteroceptive somatosensory system but rather "is represented in an unforeseen, novel pathway in humans that is part of a hierarchical system of interoception subserving homeostasis, the sense of the physiological condition of the body (interoception) and the subjective awareness of feelings and emotion."

Craig notes that, although the conscious perception of pain in humans is associated with the summated activation of C-nociceptors (as demonstrated microneurographically), there is also slow ongoing spontaneous discharge in C-fibers, which is not consciously perceived. He suggests that this discharge may reflect current tissue metabolic status: that is, these C-fiber afferents constitute part of the lamina I spino-thalamo-cortical pathway the function of which is to monitor the physiological condition of the entire body [32]. This pathway of presumed interoception (also termed cenesthesia), which is visible only in primates and well developed only in humans, could be responsible for an individual's subjective awareness of inner body feelings, including emotionality [34]. He also proposes that the dorsal margin of the insular cortex contains a primary sensory representation of the activity contained in this pathway [32,33]. If so, this cortical area would constitute an "interoceptive image" of activity in "homeostatic afferents" responsible for a myriad of bodily feelings and, importantly, for their associated emotions, which reconceptualizes pain as but one of many possible responses to disturbed homeostasis [36].

This claim raises two issues. First, the particular disturbance of homeostasis that gives rise to pain is quite specific, being signalled only through the summated activation of nociceptors, that is, by tissue damage. This is an attempt to unify (old)

specificity theory with (new) convergence theory, favoring the former. The second, more profound, issue is that Craig's hypothesis depends on representationism (objectivism) whereby the nervous system is held to mirror an independent world (which includes a "thing" called pain and a sensory image of the physiological condition of the body). Not only is this untestable, but it requires that the organism step outside of itself in order to observe itself, which is an impossibility.

Critique of the Biopsychosocial Framework as a "Model"

The biopsychosocial model for understanding illness has generated the IASP definition of pain, two simpler conceptual frameworks, and three explanatory schemata for pain. However, in the absence of a theory that seeks to understand how the different domains interact with each other, these attempts have been caught in circular argument and have been unable to transcend either biomedical reductionism or the perpetuation of body–mind dualism. In particular, the implication that pain is a "thing" separate and distinct from the body not only bears little relationship to the lived experience of pain, but also emphasizes the inherent problem that arises when an observer attempts to reduce the experience of the pain of "the other" to predictable parameters [25].

Philosopher Daniel Dennett [37] arrived at the same conclusion in his essay "Why You Can't Make a Computer that Feels Pain":

> . . . I recommend giving up incorrigibility altogether, in fact giving up all "essential" features of pain, and letting pain states be whatever "natural kind" states the brain scientists find (if they ever do find any) that normally produce all the normal effects . . . .These will be discoveries based on a somewhat arbitrary decision about what pain is, and calling something pain does not make it pain . . . whether or not one is in pain is a brute fact, not a matter of decision to serve the convenience of the theorist. (p. 228)

In the absence of a testable theory of pain, attempts to model it as an emotion, a memory, a disease, or an illness have to date been flawed as they have been made within a theoretically insupportable metaphysical and therefore arbitrary philosophical methodology. The question that needs to be asked is, why does pain and its ethical treatment defy the methodology?

Pain as an Aporia

The biopsychosocial framework for understanding the nature of illness in general and the problem of pain in particular has been useful in broadening the purview of the clinician. The opportunity to be freed from the shackles of the biomedical approach has generated a rich literature, the multidisciplinary approach to pain management and a degree of validation of the predicament of the person in pain.

Yet, this sense of progress is illusory. Our examination of the conceptual proposals generated within the biopsychosocial framework reveals that there has been no resolution of how the different domains of analysis relate to each other, let alone explain the phenomenon of pain. The exercise reflects our desire for sense-making rather than in fact making sense.

Despite the best of intentions, the proposals arising out of the biopsychosocial framework have imposed the world view of the clinician-observer at the ethical and clinical expense of the patient. Much knowledge is communicated through narrative, but the *de facto* narrative of the observer historically has dominated, thereby ignoring that the observer is part of the very system being observed. As a result, the body–mind dichotomy has been perpetuated arbitrarily and wilfully upon the person in persistent pain. All the different approaches essentially apprehend a default of responsibility to the patient's "mind" and invite a clinical judgement of psychosocial susceptibility.

Ironically, culturally trapped as we are in the format of linear (biomedical) and body-or-mind (biopsychosocial) thinking, we have succeeded only in developing a series of circular arguments. No matter what line of enquiry we pursue, we come up against questions that defy our accessibility to answers. The lived experience of being human is not linear and indeed is beyond body–mind monism or dualism. We have to accept that some things are likely to be insuperable, too complex to apprehend from an embedded linear determinism or from an *either/or* desire to make sense of pain. That is to say, our attempts to develop explanatory models bounce off a metaphysical brick wall, which constitutes the aporia of pain. From the Greek meaning "without a path, a passage or a way," an aporia is a mystery, a space to which we are denied access. *Presences* like love, God, death, and pain are spaces to which we cannot gain access but, paradoxically, from which we cannot escape [38]. Above all, they seem to escape any truth claim proclaimed over them. So we must be careful not to do the very things we say cannot be done. Under such claims, they have already escaped *elsewhere* [39].

Yet, as clinicians, it is necessary to engage the aporia of pain because of our ethical obligation to the person (as "*the other*") in pain. We do know some things about pain: it is not an illness, not a disease, not an emotion, not a memory, but rather all these things although not susceptible to any one such reduction. By contrast, the only persons who expertly know pain are the persons "in pain"; yet, their efforts to communicate such expertise is limited by the lack of language to express their pain. Though as Scarry [40] pointed out: *When pain does find a voice it is always through the creative frame* (p. 13).

The clinician and the person in pain share two outstanding characteristics. First, they simultaneously are both observer and observed, locked inextricably in a dance that defines the impossibility of objectivity. Second—and as the basis for a new theory for pain—they are haunted by the ghost, the spectre of self-reference, which is a fundamental characteristic of living systems, as will be described below. Thus, the clinical encounter of *the other* in pain is the engagement of two self-referential organisms always teasing out the *modus communicandi* of their unique narrative which is the creative frame of pain.

Basis for a Creative Frame

We see three challenges to be met in this project: to accept that the pain of another person is irreducible to its neuronal correlates, as argued above; to acknowledge all the principles which characterize autonomous biological systems (autopoiesis, noncentrality, and negentropy), to follow below; and to allow a rapprochement between the world of the clinician and the world of the person in pain. This last necessarily requires an apprehension of both the limitations and affirmations of the narratives each carries and the real difficulties language has in expressing them in the intersubjective or *third space* [41]. To know what exactly happens in this third space, in terms of a scientific model or indeed, even to try and express it in the simplest of language, is difficult to pin down, as Winnicott explained:

> What, for instance, are we doing when we are listening to a Beethoven symphony or making a pilgrimage to a picture gallery or reading Troilus and Cressida in bed, or playing tennis? What is a child doing when sitting on the floor playing with toys under the aegis of the mother? What is a group of teenagers doing participating in a pop session? It is not only: what are we doing? The question also needs to be posed: where are we (if anywhere at all)? We have used the concepts of inner and outer, and we want a third concept. Where are we when we are doing what in fact we do a great deal of our time, namely, enjoying ourselves? ([42], p. 187)

The Problem of Language

The IASP definition of pain is orientated to the clinician, whose task becomes to decide to what extent pain is somatically or psychologically engendered, as well as whether its severity matches the extent of demonstrable or presumed tissue damage. The definition implies that the clinician and person in pain can negotiate in a shared language, which communicates what it is like to have sustained tissue damage or pain. However, clinicians have limited language that is not steeped in dualism. Meanwhile, people in pain have no language with which to express their pain and are constrained to use metaphor in their creative frame, as Scarry [40] has shown. Indeed, clinical language itself is problematic, as exemplified by the current frustrations concerning the denotation and connotation of neuropathic pain. For "neuropathic" is the adjective to the noun "neuropathy," which is not the meaning conveyed by the IASP for the former term (namely, pain initiated or caused by a primary lesion or dysfunction in the nervous system) [26]. This concern is highlighted by Le van Quyen [43]: "Despite a growing body of evidence . . . our understanding of these large scale brain processes remains hampered by the lack of theoretical language for expressing these complex behaviours in dynamical terms" (p. 67).

It follows that the assumption that the experience of pain is communicable to an observer is untenable.

Self-referential Biological Systems

The principle of self-referential systems is that the elements of such a system are compatible with each other [44]. Self-reference is given when these elements generate themselves again and again [45]. The three characteristics of self-referential biological systems are autopoiesis, noncentrality, and negentropy [46,47].

Chilean biologists Maturana and Varela [48] argued that the unique property possessed by living organisms is a particular circular mechanism of spontaneous autonomous activity contained within a semipermeable boundary, a process which they called *autopoiesis*, from the Greek, meaning self-producing. In 1932, Cannon [49] had postulated the existence of a self-regulatory adaptive mechanism that allowed organisms to maintain themselves in a state of dynamic balance in the face

of changing conditions. For this mechanism, he coined the term homeostasis. Maturana and Varela [48] defined an "autopoietic unit" as a homeostatic system capable of being self-sustaining by virtue of an inner network of reactions (i.e., its organization) that regenerate all the system's components (i.e., its structure) [50]. These components are ceaselessly regenerated and the system always contains the very network that produces them.

The critical variable of a living system is its own organization, which determines both the identity and general configuration (structure) of the system. Loss of the system's organization results in its death. By contrast, its structure changes constantly as the system continually adapts itself to perturbations (unpredictable disturbances) caused by environmental changes. In order to be autonomous, living systems need to obtain resources from the environment in which they live. Thus, they are at the same time autonomous and dependent systems [51]. This paradoxical condition, which is a characteristic feature of living beings, is not amenable to linear analysis but by contrast requires a way of thinking that allows an apprehension of the self-organizing and dynamic relationships between its parts [51].

The second characteristic of living systems is noncentrality. This refers to the ability of autopoietic systems to be complexly organized and interconnected in the production of their own components so that these components are continuously decentered in their reorganization. That is, there is no "pain center" in the brain, no separation between producer and product, and no locus that determines or derives such production [48]. As Faingold [52] points out: "The complexity of the brain has placed a seemingly insurmountable constraint on our ability to understand with any degree of precision how the functional mechanisms of the brain are organized to perform even the simplest tasks" (p. 57).

The third characteristic of self-referential living systems is negentropy [53], which refers to their propensity to gain in energy or information so that input to this enormously complex autopoietic and noncentered system does not equal its output. From the thermodynamic viewpoint, such systems can be said to maintain themselves in a state of high statistical improbability of order and organization. They spontaneously develop to states of greater heterogeneity and complexity by taking in energy available from their environment. Without such energy transfer, living systems would rapidly decay into the inert state of "equilibrium."

The Self-referential Nervous System

The brain "is controlled and also controls, learns and teaches, processes and creates information, organizes its environment, and is organized by it" [54]. That is to say, the brain is not simply a hard-wired computer-like machine that only processes an externally referenced environment. Specifically, it does not merely reflect the phenomenal world. Rather, the brain is both the object of interpretation and the interpreter. Another way to say this is that the brain is its own self-organizing, noncentered, and negentropic reference. This perspective emphasizes not only structural organization (neural specificity) but also functional interactions (distributed networks), and dynamical processes (self-organization). As has been described elsewhere, the brain is "like a gigantic mass of interneurons that interconnects them in an ever-changing dynamics" [55]. Hence, not only is the brain characterized by neural specificity with temporal entrainment, but it possesses a recursive property to the parallel distributed processes that forms the substance of brain function. Clearly, to conceive of the brain as a "central processor" fails to account for the fact that the brain necessarily becomes the medium that impacts its own dynamical structure; that is, it is self-referential.

The nervous system forms part of an autonomous unity in which every state of activity leads to another state of activity in the same unity, because its operation is circular, or in operational closure. Being an embedded system in continuous structural change, the nervous system has the property of plasticity. This makes it possible for the system to participate in the process by which the organism and its environment remain in a continuous inseparable relationship. This leads to the proposition that the key to understanding pain is to recognize the role of the self-referential brain embedded in an autopoietic living system.

Toward a Rapprochement Between Clinician and Person in Pain

When the organism's environment includes another living organism, each triggers changes in the other's structure; such a congruent reciprocal relationship continues for as long as they remain engaged. During that time, the lived body-environment of each becomes a unitary structure that emerges through their reciprocal interaction. Importantly, each organism conserves its own autopoiesis and compatibility with its environment.

When applied clinically, this model can be conceptualized as two autopoietic human beings with embedded nervous systems that are engaged within a shared environment, the intersubjective *third space*, from which new therapeutic possibilities can arise. Metaphorically speaking, "the nervous system of each is one of a group of players engaged in jazz improvization, and the final result emerges from the continued give and take between them" [56].

The engagement which takes place between the clinician and the patient in pain does so in the linguistic domain—here used to describe a "world brought forth" engendered out of human interaction but not observing pre-existent and static universal truths. The verbal and nonverbal narratives of languaging beings may interact in ways that creatively frame not a universal experience but a particular and uniquely self-organizing one [55].

From such linguistic engagement arise new phenomena of empathy, intersubjectivity, and interbeingness, which is—whether taken from the purely scientific vantage or a literary metaphysic—the problem of "the other" [57].

We need to play close attention not only to the new language that is used to cope with this expansion of our neurophenomenal world, but also to the lack of language that is currently available to cope with this new phenomenal context.

Conclusion

We have argued that, although it may have helped the practice of pain medicine escape from biomedical monism, the utility of the biopsychosocial framework has now stalled and stands as a hindrance. Biopsychosocial "models" remain inside Descartes' circle of self-imposed certitude: any body–mind apprehension of pain is condemned to a circular tautology of, at best, clinical insupportability and, at worst, highly evidenced obsolescence. Thus, we still lack an explanatory model for pain.

We have formulated here a project to accept that pain is an aporia and, at the same time, to develop a theory of pain for use in the clinical context. The epistemological and ontological constraints of the aporia that is pain are underpinned by our inability to access the secrets of the self-referential brain and by our difficulty in using our languaging systems to express such complex phenomena. Yet these very theoretical understandings of pain are essential in the clinical situation of the undeniable ethical, epistemological, moral, and humanitarian responsibility we have toward the other in pain.

Biologists Maturana and Varela [55] proposed that mind and matter are not separate but represent two bits of language that seeks to signify what is looking more like inseparable aspects of our beingness and the phenomenal world of our self-organizing nervous systems. The neurobiologist Szentagothai [46] also said such neural self-organization and autopoeisis was a challenge for professional philosophers. We can but agree. And it is an invitation to break out of the circle of teleology and to accept the inherent uncertainty which attends the operation of the self-referential brain and its unlikely emergent pain states.

Now is not a time for professional hubris or the proclamation of truth by a few (least of all ourselves); rather, in the face of the universe that is our neuro-*beingness*, it is a time for reflection by all those who would assert clinical truth over such complex phenomena. This article is a call to move forward to explore the engagement of self-referential beings through the creative frame of language, in which the narrative of neither the clinician nor the patient is dominant but the way they touch and grow in understanding, empathy, and shared outcomes is paramount.

References

1 Vickers G. Some implications of systems thinking. In: Open Systems Group, eds. Systems Behaviour, 3rd edition. London: Harper and Row, Publishers; 1981:19–25.
2 McLaren NA. A critical review of the biopsychosocial model. Aust N Z J Psychiatry 1998;32:86–92.
3 Radden J. The philosophy of mind and psychiatry. Curr Opin Psychiatry 1999;12:589–92.
4 Reber AS. The Penguin Dictionary of Psychology. Harmondsworth: Penguin Books Ltd.; 1985:738–9.
5 Foucault M. The Birth of the Clinic: An Archaeology of Medical Perception. Translated by Alan Sheridan. London: Routledge; 1973.
6 Gray W. Ludwig von Bertalanffy and the development of modern psychiatric thought. In: Gray W, Rizzo ND, eds. Unity Through Diversity: A Festschrift for Ludwig von Bertalanffy, Part 1. New York: Gordon and breach Science Publisher; 1973:169–83.
7 Engel GL. The need for a new medical model: A challenge for biomedicine. Science 1977;196:129–36.
8 Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry 1980;137:535–44.
9 Weiss PA. Self-differentiation of the basic patterns of coordination. In: Jeffress LA, ed. Cerebral

Mechanisms in Behavior—The Hixon Symposium. New York: Wiley; 1951:89–91, 140–2.
10 Weiss PA. The basic concept of hierarchic systems. In: Weiss P, ed. Hierarchical Organized Systems in Theory and Practice. New York: Hafner; 1971:1–43.
11 von Bertalanffy L. The model of open systems: Beyond molecular biology. In: Breck AD, Yourgran W, eds. Biology, History and Natural Philosophy. New York: Plenum Press; 1967:17–30.
12 Engel GL. The biopsychosocial model and the education of health professionals. Ann N Y Acad Sci 1978;310:169–81.
13 Greaves D. Reflections on a new medical cosmology. J Med Ethics 2002;28:81–5.
14 Engel GL. The care of the patient: Art or science? Johns Hopkins Med J 1977;140:222–32.
15 Aronoff GM, Gallagher RM, Feldman JB. Biopsychosocial evaluation and treatment of chronic pain. In: Raj PP, ed. Practical Management of Pain, 3rd edition. St Louis: Mosby; 2000:156–65.
16 Flor H, Hermann C. Biopsychosocial models of pain. In: Dworkin RH, Breitbart WS, eds. Psychosocial Aspects of Pain, Progress in Pain Research and Management, Vol. 27. Seattle: IASP Press; 2004:47–75.
17 Andrasik F, Flor H, Turk DC. An expanded view of psychological aspects in head pain: The biopsychosocial model. Neurol Sci 2005;26:S87–91.
18 Melzack R, Wall PD. Pain mechanisms: A new theory. Science 1965;150:971–9.
19 Melzack R. From the gate to the neuromatrix. Pain 1999;6(suppl):S121–6.
20 Basbaum A, Bushnell MC, Devor M. Pain: Basic mechanisms. In: Justins DM, ed. Pain 2005—An Updated Review: Refresher Course Syllabus. Seattle: IASP Press; 2005:3–9.
21 Hadler NM, Greehalgh S. Labelling woefulness: The social construction of fibromyalgia [editorial]. Spine 2005;30:1–4.
22 Lucire Y. Neurosis in the workplace. Med J Aust 1986;145:323–7.
23 Awerbuch M. Repetitive strain injuries: Has the Australian epidemic burnt out? Intern Med J 2004;34:416–9.
24 Shorter E. Sucker-punched again! Physicians meet the disease-of-the-month syndrome. J Psychosom Res 1995;39:115–8.
25 Williamson OD, Buchanan DA, Quintner JL, Cohen ML. Pain beyond monism and dualism [letter]. Pain 2005;116:169–70.
26 Merskey H, Bogduk N. Classification of Chronic Pain. Seattle: IASP Press; 1994:210.
27 Barsky AJ, Borus JF. Somatization and medicalization in the era of managed care. JAMA 1995; 274:1931–4.
28 Loeser JD. Perspectives on pain. In: Turner P, ed. Proceedings of the First World Congress on Clinical Pharmacology and Therapeutics. London: Macmillan; 1980:313–6.
29 Waddell G, Bircher M, Finlayson D, Main CJ. Symptoms and signs: Physical disease or illness? Brit Med J 1984;289:739–41.
30 Siddall PJ, Cousins MJ. Persistent pain as a disease entity: Implications for clinical management. Anaesth Analg 2004;99:510–20.
31 Medawar P. Induction and Intuition in Scientific Thought. London: Methuen; 1969.
32 Craig AD. How do you feel? Interoception: The sense of the physiological condition of the body. Nat Rev Neurosci 2002;8:655–66.
33 Craig AD. Pain mechanisms: Labelled lines versus convergence in central processing. Ann Rev Neurosci 2003;26:1–30.
34 Craig AD. Human feelings: Why are some more aware than others? Trends Cogn Sci 2004;8:239–41.
35 James W. What is an emotion? Mind 1884;9:188–205.
36 Craig AD. Forebrain emotional asymmetry: A neuroanatomical basis? Trends Cogn Sci 2005;12:566–71.
37 Dennett D. Brainstorms: Philosophical Essays on Mind and Psychology. London: Penguin Books; 1997.
38 Derrida J. Aporias. Translated by Thomas Dutoit. Stanford, CA: Stanford University Press; 1994.
39 Derrida J. Writing and Difference. Translated by Alan Bass. London: Routledge, Keagan and Paul; 1978.
40 Scarry E. The Body in Pain: The making and the Unmaking of the World. New York: Oxford University Press; 1985.
41 Scannell K. Writing for our lives: Physician narratives and medical practice. Ann Intern Med 2002;137:779–81.
42 Winnicott DW. The Maturational Processes and the Facilitating Environment. New York: International Universities Press; 1965.
43 Le Van Quyen M. Disentangling the dynamic core: A research program for neurodynamics at a large scale. Biol Res 2003;36:67–88.
44 von Foerster H. On self-organizing systems and their environments. In: Yovits M, Cameron S, eds. Self-Organizing Systems. London: Pergamon Press; 1960:31–50.
45 Luhmann N. Social Systems. Stanford, CA: Stanford University Press; 1995.
46 Szentágothai J. Self-organisation: The basic principle of neural function. Theor Med 1993;14:101–16.
47 Buchanan DA. Persistent unexplained pain and the language of the uncanny in the creative neurophenomenal reference. PhD thesis. Edith Cowan University, Perth, Australia, 2006.
48 Maturana HR, Varela FJ. Autopoiesis and cognition: The realization of the living. Dordecht: Reidel Publishing Co.; 1980.

49 Cannon WB. The Wisdom of the Body (revised ed.). New York: Norton; 1939.
50 Maturana H. Neurophysiology of cognition. In: Garvin P, ed. Cognition: A Multiple View. New York: Spartan; 1970:3–23.
51 Mariotti H. Autopoiesis, Culture and Society. Available at: http://www.oikos.org/mariotti.htm (accessed June 26, 2005).
52 Faingold CL. Emergent properties of CNS neuronal networks as targets for pharmacology: Application to anticonvulsant drug action. Prog Neurobiol 2004;72:55–85.
53 Schrödinger E. What is Life?: The Physical Aspect of the Living Cell with Mind and Matter and Autobiographical Sketches. Cambridge: Cambridge University Press; 1944.
54 Arbib M, Erdi P, Szentagothai J. Structure, Function, and Dynamics: An Integrated Approach to Neural Organisation. New York: MIT Press; 1988.
55 Maturana HR, Varela FJ. The Tree of Knowledge: The Biological Roots of Human Understanding, revised edition. Translated by R. Paolucci. Boston, MA: Shambhala Publications Inc; 1998.
56 Chiel H, Beer R. The brain has a body: Adaptive behavior emerges from interactions of nervous system, body and environment. TINS 1997; 20:553–7.
57 Avenanti A, Bueti D, Galati G, Aglioti SM. Transcranial magnetic stimulation highlights the sensorimotor side of empathy for pain. Nat Neurosci 2005;8:955–60.