

The Lived Experience of Pain: A Painful Journey for Medicine

Milton Cohen & John Quintner

Abstract

Although understandings of pain phenomena have benefited from theoretical developments such as the biopsychosocial model of illness, the clinical path of the person in pain presenting to a health professional still leads to frustration for both. The biopsychosocial model generated an "official" definition, two major conceptual frameworks, and three putative explanatory models. However, in the absence of a theory that embraces pain as an emergent and unpredictable phenomenon, these progeny have been unable to displace biomedical reductionism or to transcend dualism. The observer-dependent stance of the clinician results in marginalisation of the "patient," compromises therapy and confronts both parties with a potential clinical nemesis. The lived experience of pain reflects an *aporia*, a puzzle that denies us access to its secrets. However application of principles underlying the self-referentiality of living systems opens a path to integrating the traditional somatic dimension of biomedical analysis with dimensions of beliefs, emotions, behaviour and environment. Clinically, pain may be grasped anew through the engagement of autonomous self-referential living systems, creating the intersubjective or "third space" with each influenced by similar sets of humanist factors and confronted with the same *aporia*. This reframing generates new possibilities in assessment, treatment and policy.

Key Words: Biopsychosocial, reductionism, dualism, *aporia*, self-referentiality, autonomous systems, intersubjectivity, third space.

1. Introduction

This paper seeks to understand why what should be a basic function of the health professions - the management of pain - is not only unsatisfactory but also frustrating for both the person in pain and the health professional. How is it, we ask, that there is such a poor fit between the conventional medical approach to the person in pain and that of the lived experience of pain?

To address this conundrum, the basic tenets of (Western) biomedicine need to be appreciated, their inadequacies with respect to pain identified and the attempts to remedy this situation examined. A central issue remains - whether the "slipperiness" of defining and researching pain using the linguistic tools available limits any attempt to reframe the clinical encounter.

2. Western biomedicine and pain

The dominant discourse of Western medicine is characterised by two core philosophical tenets, each with two complementary aspects. Firstly, not only are body and mind conceptualised as different and distinct entities but also the physical ("body") dimension, being "objective" and therefore measurable, is accorded priority over the mental ("mind") dimension, which is "subjective" and therefore unmeasurable.¹ The second is the reductionist assumption, that all symptoms - including pain - are expressions of a discoverable disease process and that there is a predictable and reliable connection between pathological changes and clinical features. This is complemented by the assumption that knowledge about the properties of parts of a system is sufficient to understand the global behaviour of the whole system.

This primacy of structural pathology located in the body has been the major criterion for discovering disease in Western epistemology, eclipsing functional pathology and relegating to the background behavioural, psychological and social factors. When applied to the problem of clinical pain, biomedicine posits a predictable - if yet to be discovered - hard-wired relationship between identifiable tissue damage, consequent changes within the brain, and the report of pain.

The implications of this biomedical model of illness are profound. Firstly, the experience of deviation from wellness is demoted to the depersonalised expression of a (structural) disease process. Secondly, the process of clinical diagnosis becomes exclusively reductionist, the only phenomena of interest being those that constitute discrete, linear causal chains. Thirdly, the relationships between such chains are assumed to behave predictably, and to apply universally.²

In effect this model privileges the ostensibly "objective" view of the clinician over the sufferer's lived experience of pain. Thus if there is no discoverable and relevant nociception (the detection and signalling of "tissue damage", a concept integral to the "definition" of pain), there can be no "real" pain. The clinical encounter then resolves as either dismissal of the patient's complaint or an inference of "psychogenesis" by the clinician.³ This is in effect a reversion to the default tenet of body-mind dualism, defying and confronting the lived experience of pain, which is "simultaneously and interactively both physical and mental."⁴

3. The "biopsychosocial model of illness": an attempt to loosen the constraints of dualism and reductionism

To allow clinicians to embrace aspects of human illness that biomedicine proved unable to contemplate, psychiatrist George Engel (1913-1999) formulated the biopsychosocial model. Actually a framework through which to assess illness rather than an explanatory "model", Engel's proposition retained the assumptions of biomedicine but invited consideration that the experience of illness could be influenced if not determined by psychological, social, and cultural factors.^{5 6 7}

Engel's model was derived from General Systems Theory (GST) as formulated in the early 20th century by Viennese theoretical biologists, Ludwig von Bertalanffy and Paul Weiss.⁸ GST holds that organisms are highly organised hierarchical systems, embedded in their respective environments. A perturbation sufficient to cause changes in any one of these systems would evoke changes in any or all of the other systems. Most importantly, the organism itself would produce these changes "in an effort to stay constant with regard to its outside."⁹

The biopsychosocial framework invited clinicians to collect qualitative and quantitative data in these psychological, social, and cultural domains and to incorporate them into diagnostic and therapeutic processes, thereby arriving at a better understanding of the "human experience" of disease or, more correctly, of illness. Importantly, these transactions were to take place at the "two-person" system level, that is, the doctor-patient relationship.¹⁰

Theoreticians in pain medicine were quick to adopt the biopsychosocial framework, as it seemed to fit the growing recognition of pain as an expression of complex interactions between biological, psychological, social and cultural factors.^{11,12,13}

Interestingly from the biological perspective, the Gate Control theory of Melzack and Wall (1965) had already predicted, based on the plasticity and modifiability of events at all levels within the central nervous system, that whether or not a person perceives and reports pain in response to a particular stimulus would be determined not only by the nature of that stimulus but also by the context within which it is experienced, together with associated memories, emotions and beliefs.^{14,15,16}

A major outcome of the application of the biopsychosocial approach was the "official" definition of pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage".¹⁷ This was an advance on the biomedical approach, as it could break the nexus between actual tissue damage and the subjective experience and thus accord legitimacy to the experience of the person in pain.

However the explanatory notes that accompany the IASP definition contain 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.*"¹⁸ [italics added] Thus, even this potentially enlightening definition fell back into the circular trap of body-mind dualism, and has inadvertently sanctioned the efforts to seek explanations for a variety of pain syndromes solely from psychosocial factors, from which arises the untestable construct of somatisation.¹⁹

Fink's critical examination of the IASP definition (this volume) exposes the ambiguous and puzzling nature of the term "pain", which can denote one or more of "a cluttered mixture of distinguishable concepts." He concludes that by reducing our everyday notion of pain to a scientific concept, the IASP definition fails to reflect the important moral and ethical dimensions of the pain experience: "Pain itself is not morally relevant in itself, but suffering is the morally relevant component."

Nonetheless the biopsychosocial framework generated a number of hierarchical "models" of pain. First among these was that of Loeser, who proposed four nested hierarchical domains: nociception, pain, suffering, and pain behaviour.²⁰ Waddell's Glasgow Illness Model was formulated similarly, postulating four domains of interest: physical problem, psychological distress, illness behaviour, and social interactions.²¹ Each "model" explicitly includes the moral dimension of "suffering" demanded by Fink, and in fact the only observable domain is the interaction between the person in pain and the surrounding world ("pain behaviour" in Loeser's model; "illness behaviour" and "social interaction" in that of Waddell). In each the clinician was to determine which of the four domains might be "playing significant roles in the genesis of the person's problem, and then to direct therapies at the appropriate aetiological factors".²¹

This exercise required the clinician not only to differentiate illness behaviour from physical disease but also 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 were to be made was left entirely to the clinician's discretion, guided by the same arbitrary constructions constrained by dualism.

These approaches pose major theoretical and practical problems. Firstly, the frameworks suggest that pain, suffering, and behaviour are somehow discrete phenomena, whereas in fact the clinician-observer is unable to make such a

distinction. Secondly, although nociception may be understood as a specific and quantitative physiological process, the other domains resist such a reductionist approach. Just as the connection between them is undefinable and arbitrary, so is the lived experience of pain not reducible to a neuroscientific explanation. Although these conceptual frameworks imply that there are non-nociceptive influences on pain and behaviour, they neither escape dualism nor solve the unacknowledged "puzzle of pain".²²

The next two decades saw a new trend emerging, with the focus on inferring overarching neurobiological mechanisms for the experience of pain. Flor and Hermann formulated a "psychobiological" model of pain, viewing it as a multi-component behavioural response to aversive stimuli. Through prior learning, operant and respondent conditioning, powerful pain memories are formed at all levels of the nervous system, reflecting the dynamic and continuous interplay between various physiological and psychological factors.²³ Once established, such pain memories were said to be capable of maintaining pain even in the absence of peripheral nociceptive input. That is, pain transmutes from being an emergent aversive response into a learned memory that may *cause* profound reorganisational changes within the nervous system, thus perpetuating itself.

By reconceptualising pain as a "homeostatic emotion", Craig pursued the theme of grounding pain in disturbed bodily function, as one of a number of emotional responses through which the integrity of the body (self) can be maintained in the face of conditions that threaten to disturb its homeostasis. He suggested that pain is not part of the exteroceptive somatosensory system but rather "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."²⁴

Craig's formulation was based on the known relationship in humans between activated C-fibres and the conscious perception of pain, and a proposal that slow ongoing spontaneous discharge in C-fibres functions to monitor the physiological condition (metabolic status) of the entire bodily tissues and could be responsible for an individual's subjective awareness of inner body feelings, including emotionality.²⁵

This claim raises two important issues. Firstly, 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. Craig is attempting to unify (old) specificity theory with (new) convergence theory, favouring the former. The second, and more profound, issue is that Craig's hypothesis depends on representationism whereby the nervous system is held to mirror an independent world,^{26,27} which includes a "thing" called pain and a sensory image of the physiological condition of the body.

Not only is this claim untestable, it requires that the organism step outside itself in order to observe its own condition which, as it leads to an infinite regress, would appear to be impossible.²⁸ Furthermore, it would also require what the philosopher Daniel Dennett calls a Cartesian Theater, an obscure place in the mind/brain "where it all comes together."²⁹

Another example of pain as a "thing" was the attempt of Siddall and Cousins to assign disease status to the clinical problem of persistent pain.³⁰ These authors cited "secondary pathology" within the nervous system induced by altered sensory inputs from the periphery, including anatomical reorganisation in nociceptive pathways and the alterations in patterns of brain activation and cortical topography found in persistent pain states. In the light of recent knowledge of brain plasticity this proposition was, in itself, unexceptional. However, to then state "persistent pain does give rise to its own secondary pathology"³¹ again elevates pain to the status of a causative "thing" and thus constitutes the circular argument that pain causes itself.

These authors went further by adducing environmental factors as the "tertiary pathology" of pain and thus asserted that pain is "an environmental disease". Such tertiary pathology was said to include "factors such as genetic makeup, level of spinal inhibition, psychological status and the societal litigation system."³² In effect, this proposition is no different from identifying that non-nociceptive factors may be relevant to the pain experience. However to label this as "tertiary pathology" carries the implication that these factors can somehow contribute to a permanently altered nervous system. At the same time, by including "psychological status" as part of this tertiary pathology, and therefore external to (and acting upon) 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"?

This proposition appeared to be concordant with the biopsychosocial framework but the attempt to portray pain itself as a causative "thing" represented a dramatic return to the linear causality of the biomedical model. Moreover, Siddall and Cousins failed to distinguish between their "discovery" of the "pathology" of chronic pain and any process that might lead to justification of its validity.³³

4. Critical evaluation of the biopsychosocial framework of pain

The application of the biopsychosocial framework to the evaluation of pain has been useful in clinical assessment, in generating putative explanatory models and to some extent influencing 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.

However, in the absence of a theory that seeks to understand how the different domains might interact with each other, these attempts have been caught in circular argument and have been unable to transcend 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 emphasises the inherent problem that arises when an observer attempts to reduce the experience of the pain of “the other” to predictable parameters.

In this initiative, pain theorists continued to adopt the traditional observer-dependent stance of the clinician, which not only marginalises the patient but also ignores the important role the observer plays in the clinical encounter and its outcome. The lived experience of pain cannot be reduced to its neurophysiological correlates, nor can it be unambiguously conceptualised as a disease, an emotion, or a memory, although it may contain elements that fit with each of these concepts. Thus the biopsychosocial initiative is better seen as another reductionistic and dualistic framework for analysis rather than as an explanatory model for the complex phenomenology of pain states.³⁴

5. A new theoretical framework for Pain Medicine

Why are pain and its ethical treatment so elusive of theory? To address this question we identify the clinical encounter as the pivotal transaction in pain medicine: the presentation of a person distressed because of a profound threat to their bodily integrity to another person reputed to be learned in the arts (if not also the sciences) of healing.

In searching for a new theoretical perspective to understand this encounter, we will draw on three vantage points:

- The central concept of pain as an *aporia* that cannot be accounted for adequately by the biomedical model or in a biopsychosocial framework;
- The nature of humans (and indeed other biological systems) as autonomous self-organising autopoietic units that have different perspectives and needs based on the demands of their particular circumstances (which includes the societies in which they are embedded);
- Inter-subjectivity or empathy as the natural ground for rapprochement between the world of the clinician and the world of the person in pain in the clinical setting.

5.1 Pain as an *aporia*

The lived experience of being human is not linear and indeed is beyond body-mind monism or dualism.³⁵ Pain is not only difficult to express in language but also ultimately not communicable in these terms: too complex to be apprehended from linear determinism or from a desire to make sense of it. Our attempts to develop explanatory models bounce off a metaphysical brick wall, which constitutes the *aporia* of pain.

An *aporia* (from the Greek meaning “lacking a path, a passage or a way”) is a mystery or puzzle, encompassing the dual problems of not knowing how one has arrived here and not knowing where to go next.^{35a} As it is unlikely that pain can ever be known objectively, the clinician’s encounter with the *aporia* of the patient is often one of uncertainty, discomfort and doubt. The patient in pain, currently encountering the *aporia*, presents his/her body to the clinician for investigation and treatment with the quite reasonable expectation that the clinician will be able to “ground” the lived experience. But the clinician may also be “lost”, so that both parties lack knowledge and understanding, which leads to a crisis of choice, of action and of identity.^{35b} As Bodwell argues (this volume) the clinician’s dilemma can then lead to loss of empathy and even to feelings of resentment towards the person in pain.

When confronted with the clinician’s dilemma, the patient too is forced to share the same doubt and uncertainty, compounding their discomfort, and with potentially negative epistemic and moral implications for the therapeutic relationship, including stigmatisation within the health care system. Nielsen (this volume) explores the relationship between chronic pain and stigma in society and argues that the devastating consequences of stigmatisation (“social suffering”) need to be addressed at both practice and policy levels.

5.2 Self-referential biological systems

A critical insight from GST is the capacity of a living organism to change itself in response to perturbations in its environment. Chilean biologists Maturana and Varela 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". They defined an "autopoietic unit" as a particular type of homeostatic system capable of being self-sustaining by virtue of an inner network of reactions (its organisation) that regenerate all the system's components (its structure).

Autopoiesis is the continual production by a network of the very components that comprise and sustain the network and its processes of production.³⁶ In other words, such a system is said to be self-referential (capable of examining itself) and self-organising (capable of changing and ordering its internal structure according to local rules).^{37-38,39}

The critical variable of a living system is its own organisation. This is highly negentropic (operating far from thermodynamic equilibrium): loss of the system's organisation results in its death. By contrast its structure (its various components) changes constantly as the system continues to adapt itself to both predictable and unpredictable disturbances caused by environmental changes.

In higher animals, the nervous system, including ultimately the brain, forms part of this autonomous 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, which enables it to participate in the process by which the organism and its environment remain in a continuous inseparable relationship. In this formulation, the brain is not simply a hard-wired computer-like machine that only processes an externally referenced environment. Rather the brain is both the object of interpretation and the interpreter: its own self-organising reference.⁴²

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 unique unitary domain of communication.⁴⁰ Importantly, during such interaction each organism conserves its own autopoiesis and compatibility with its environment. However the result of that interaction is not determined by a stimulus external to the organism but only by the aggregate state of each organism itself at a given moment.⁴¹

We argue that the key to understanding pain is found in the role of the self-referential brain embedded in an autopoietic living system. When that living system is a human reporting the experience of pain, we – if we are to be empathically engaged observers – must infer that an important change has occurred in that person's nervous system arising out of an attempt to maintain its autopoietic organisation. Bendelow (this volume) makes the important point that sufferers endeavour to elicit from clinicians whatever is needed to assist this process.

5.3 Empathy

Although clinician and patient are both excluded from the same *aporia* (the pain of the "other"), they interact as two autopoietic autonomous entities, simultaneously observer and observed. Both come together in a process of exploration. Each is invited to take on the other's questions, problems and *aporia* as their own. The domain of this "level playing-field" interaction allows the emergence of the phenomena of *intersubjectivity* and *empathy*.

There is a potential neurobiological basis for these phenomena, building on the observation that the knowledge that someone else is currently in pain is sufficient to evoke activity in brain regions associated with the experience of pain. Specifically such a relationship might be built upon primary involuntary activity of deeply embedded cortical sensorimotor "mirror" neurons in the observer and the observed.^{43,44} As Favareau has argued:

For at the mirror neuron level of organization, the distinction between seer and doer, action and reaction, identity and alterity is – like the "reflection" one finds oneself presented with in front of a full length mirror – a distinction which is impossible to maintain.^{45,46}

Clearly that is not a sufficient explanation but it shows what might happen when neuroscientific findings are interpreted from the point of view of the subject rather than by an observer seeking "objective" evidence.

The realisation of intersubjectivity takes place within what Winnicott termed the "third space".⁴⁷ In relation to play, the "third space" is that in which children are able to construct a relevant culture. In the empathetic clinical encounter, clinician and patient seek to carve out a communal public space of signs and understandings created by their respective actions.⁴⁸

Kalman and Scheman (this volume) offer a valuable discussion of the ways in which this inter-subjective space can be inflected when it is pervaded by the pain of "the other". We suggest that both clinician and patient can bring into this

space a host of “baggage” items along with the issue at hand, attempting to make sense of the patient’s lived experience of pain. Some of these items are personal, in terms of beliefs, fears and emotions, but others can be antipathetic, stemming from the culture in which both are deeply embedded. At least so far as Western society is concerned, they are likely to share the belief and expectation that Medicine can provide a firm ground upon which they can both stand during their engagement.

An example of this societal expectation may be seen in the way in which systems of personal injury compensation place great emphasis on medically assessable impairment, as opposed to the broader concept of disability. Dubin (this volume) shows how this expectation can be imposed upon both patient and clinician with the former being thrust on to the “roller-coaster trajectory of chronic pain” and the latter being drawn into adversarial processes of litigation.

Another impediment to the engagement is the time-based medical consultation, which does not allow patients to tell their stories to an empathic clinician (see Bodwell, this volume).

6. Summary

As clinicians it is necessary to engage the *aporia* of pain because of our moral and ethical obligation to the person in pain. The clinician and the person in pain share two outstanding characteristics: they are simultaneously observer and observed, locked in a dance that defines the impossibility of objectivity, and haunted by the spectre of self-reference.⁴⁹ Thus the clinical encounter of the other in pain is the engagement of two self-referential organisms constructing a unique narrative.

This framework has the advantage of escaping dualism by linking the self-referential brains of both clinician and patient through underlying neurobiological processes without privileging either participant in the clinical engagement. It allows the clinician to infer a change in the autopoietic organisation of the patient and the patient to come to appreciate that such a change might have occurred. Thus it has the potential to accommodate moral and ethical considerations, at a phenomenological level now and probably at a neurobiological level in the future.

In overcoming the linear reductionist thinking of the biomedical model, which has been perpetuated in biopsychosocial iterations to date, this framework does not supplant the quest for clinical identification of mechanisms or causes or factors but rather invites integration of that approach with a biologically informed substrate that embraces the complexity of pain.

New therapeutic possibilities that may emerge from the arena of this intersubjective engagement include developing unique narrative (especially but not only verbal language, as there are other creative modes of expression), reframing “cognitive-behavioural” approaches and more limited but targeted use of agents that might modify distress without compromising nervous system function.

To give Dennett the last word:

... I recommend 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.⁵⁰

Acknowledgement: The authors are grateful to Dr Pamela Lyon for her helpful suggestions.

Notes

¹ M. Crowley-Matoka, S Saha, SK Dobschka & DJ Burgess, Problems of quality and equity in pain management: exploring the role of biomedical culture. *Pain Medicine*, vol. 10, 2009, pp. 1312-1324.

² W. Gray, ‘Ludwig von Bertalanffy and the development of modern psychiatric thought’ in W Gray & ND Rizzo (eds), *Unity Through Diversity: a Festschrift for Ludwig von Bertalanffy, Part 1*, Gordon and Breach Science Publisher, New York, 1973, pp.169-83.

³ JL Quintner, ML Cohen, D Buchanan, J Katz & O Williamson, Pain medicine and its models: helping or hindering? *Pain Medicine*. 2008, pp. 824-834.

-
- ⁴ M. Crowley-Matoka et al., op. cit.
- ⁵ GL Engel, The need for a new medical model: a challenge for biomedicine. *Science*, vol. 196, 1977a, pp. 129-136.
- ⁶ GL Engel, The care of the patient: art or science? *Johns Hopkins Medical Journal*, vol.140: 1977b, pp. 222-32.
- ⁷ D. Greaves, Reflections on a new medical cosmology, *Journal of Medical Ethics*, vol. 28, 2002, pp. 81-85.
- ⁸ M. Drack & W Apfalter, Is Paul Weiss' and Ludwig von Bertalanffy's system thinking still valid today? *Systems Research and Behavioral Science*, vol. 24, 2007, pp. 537-546.
- ⁹ PA Weiss, Tierisches Verhalten als 'Systemreaktion'. Die Orientierung der Ruhstellungen von Schmetterlingen (Vanessa) gegen Lic Schwerkraft. *Biologica Generalis*, vol. 1, 1925, p.125.
- ¹⁰ GL Engel, 1977b.
- ¹¹ GM Aronoff, RM Gallagher & JB Feldman. 'Biopsychosocial evaluation and treatment of chronic pain' in PP Raj (ed), *Practical Management of Pain*, 3rd edn, Mosby, St Louis, 2000, pp. 156-65.
- ¹² H. Flor & C Hermann, 'Biopsychosocial models of pain' in RH Dworkin & WS Breitbart WS (eds). *Psychosocial Aspects of Pain, Progress in Pain Research and Management, Vol 27*, IASP Press, Seattle, 2004, pp. 47-75.
- ¹³ F. Andrasik F, H Flor H & DC Turk. An expanded view of psychological aspects in head pain: the biopsychosocial model. *Neurological Science*, vol 26, 2005, pp. S87-S91.
- ¹⁴ R. Melzack & PD Wall, Pain mechanisms: a new theory, *Science*, vol. 150, 1965, pp. 971-979.
- ¹⁵ R. Melzack, From the gate to the neuromatrix, *Pain*, Suppl. 6: 1999, pp. S121-126.
- ¹⁶ A. Basbaum, MC Bushnell & M Devor, 'Pain: basic mechanisms' in DM Justins (ed), *Pain 2005 - an Updated Review: Refresher Course Syllabus*, IASP Press, Seattle, 2005, pp. 3-9.
- ¹⁷ H Merskey & N Bogduk, *Classification of chronic pain*, IASP Press, Seattle,1994, p. 210.
- ¹⁸ Ibid.
- ¹⁹ AJ Barsky & JF Borus, Somatization and medicalization in the era of managed care. *Journal of the American Medical Association*, vol. 274, 1995, pp.1931-1934.
- ²⁰ JD Loeser, 'Perspectives on pain' in P Turner P (ed) *Proceedings of the First World Congress on Clinical Pharmacology and Therapeutics*, Macmillan, London, 1980, pp. 313-16.

-
- ²¹ G Waddell, M Bircher, D Finlayson & CJ Main. Symptoms and signs: physical disease or illness? *British Medical Journal*, vol 289, 1984, pp. 739-741.
- ²² R Melzack & PD Wall, *The Puzzle of Pain*, Penguin Books, Harmondsworth, 1973.
- ²³ Flor & Herman, 2004. op. cit.
- ²⁴ AD Craig, How do you feel? Interoception: the sense of the physiological condition of the body, *Nature Reviews Neuroscience*, vol 8, 2002, pp. 655-66.
- ²⁵ AD Craig, Human feelings: why are some more aware than others? *Trends in Cognitive Sciences*, vol 8, 2004, pp. 239-41.
- ²⁶ A Bullock & S Trombley, eds. *The New Fontana Dictionary of Modern Thought*, Harper Collins, London, 1977, p. 749.
- ²⁷ WG Lycan, In: N Bunin & EP Tsui-James, eds, *The Blackwell Companion to Philosophy*, Blackwell Publishers Ltd., Oxford, 1996, pp. 167-197.
- ²⁸ G Edelman, *Bright Air, Brilliant Fire*, Penguin Books Ltd., London, 1992, pp. 73-80.
- ²⁹ D Dennett, *Consciousness Explained*, Penguin Books Ltd., London, 1993, pp. 21-42.
- ³⁰ PJ Siddall & MJ Cousins, Persistent pain as a disease entity: implications for clinical management, *Anaesthesia Analgesia*, vol.99, 2004, pp. 510-20.
- ³¹ Ibid.
- ³² Ibid.
- ³³ P Medawar, *Induction and intuition in scientific thought*, Methuen, London, 1969.
- ³⁴ CC Butler, M, Evans, D, Greaves & S Simpson. Medically unexplained symptoms: the biopsychosocial model found wanting. *Journal of the Royal Society of Medicine*, vol 97, 2004, pp. 219-212.
- ³⁵ OD Williamson, DA Buchanan, JL Quintner & ML Cohen, Pain beyond monism and dualism. *Pain*, vol.116, 2005, pp. 169-170.
- ^{35a} NC Burbules, Aporias, webs, and passages: Doubt as an opportunity to learn. *Curriculum Inquiry*, Vol. 30 No. 2 (2000): 171-187.
- ^{35b} Ibid.
- ³⁶ HR Maturana & FJ Varela, *Autopoiesis and Cognition: the Realization of the Living*, Reidel Publishing Co., Dordrecht, 1980, pp. 5-58.

-
- ³⁷ H von Foerster, 'On self-organizing systems and their environments' in M Yovits & S Cameron (eds), *Self-Organizing Systems*, Pergamon Press, London, 1960, pp. 31-50.
- ³⁸ N Luhmann, *Social Systems*, Stanford University Press, Stanford, 1995.
- ³⁹ PL Luisi, Autopoiesis: a review and a reappraisal, *Naturwissenschaften*, vol. 90, 2003, pp. 49-59.
- ⁴⁰ Maturana & Varela, op. cit.
- ⁴¹ P Lyon, Autopoiesis and Knowing: Reflections on Maturana's biogenic explanation of cognition. *Cybernetics and Human Knowing*, vol. 11, 2004, pp. 21-46.
- ⁴² M Arbib, P Erdi & J Szentagothai J, *Structure, Function and Dynamics: An Integrated Approach to Neural Organisation*, MIT Press, New York, 1988.
- ⁴³ D Favareau, Beyond self and other: on the neurosemiotic emergence of intersubjectivity, *Sign System Studies*, vol. 30, 2002, pp. 57-100.
- ⁴⁴ For a fuller discussion of mirror neurons, see Rizzolatti et al., 1999; Rizzolatti & Sinigaglia, 2007; Catani & Rizzolatti, 2009.
- ⁴⁵ Favareau 2003, op cit.
- ⁴⁶ T Singer & C Lamm, The social neuroscience of empathy. *Annals of the New York Academy of Sciences*, vol. 1156, 2009, pp. 81-91.
- ⁴⁷ DW Winnicott, *Playing and Reality*. Tavistock Publications, London, 1971.
- ⁴⁸ Favareau 2003, op. cit.
- ⁴⁹ Quintner et al, 2008, op. cit.
- ⁵⁰ D Dennett, *Brainstorms: Philosophical Essays on Mind and Psychology*, Penguin Books, London, 1997, pp. 190-229.

Bibliography

- Andrasik, F, H Flor & DC Turk, An expanded view of psychological aspects in head pain: the biopsychosocial model, *Neurological Science*, vol 26, 2005, pp. S87-S91.
- Arbib, M, P Erdi & J Szentagothai J, *Structure, Function and Dynamics: An Integrated Approach to Neural Organisation*, MIT Press, New York, 1988.
- Aronoff, GM, RM Gallagher & JB Feldman, 'Biopsychosocial evaluation and treatment of chronic pain' in PP Raj (ed), *Practical Management of Pain*, 3rd edn, Mosby, St Louis, 2000, pp. 156-65.

-
- Barsky, AJ & JF Borus, Somatization and medicalization in the era of managed care, *Journal of the American Medical Association*, vol. 274, 1995, pp.1931-1934.
- Basbaum, A, MC Bushnell & M Devor, 'Pain: basic mechanisms' in DM Justins (ed), *Pain 2005 - an Updated Review: Refresher Course Syllabus*, IASP Press, Seattle, 2005, pp. 3-9.
- Bullock, A & S Trombley (eds), *The New Fontana Dictionary of Modern Thought*, Harper Collins, London, 1977, p. 749.
- Burbules, NC, Aporias, webs, and passages: Doubt as an opportunity to learn. *Curriculum Inquiry*, vol 30, 2000, pp. 171-87.
- Butler, CC, M Evans, D Greaves & S Simpson, Medically unexplained symptoms: the biopsychosocial model found wanting, *Journal of the Royal Society of Medicine*, vol 97, 2004, pp. 219-212.
- Cattaneo, L & G Rizzolatti, The mirror neuron system, *Archives of Neurology*, vol 66, 2009, pp. 557-560.
- Craig, AD, How do you feel? Interoception: the sense of the physiological condition of the body, *Nature Reviews Neuroscience*, vol 8, 2002, pp. 655-66.
- Craig, AD, Pain mechanisms: labelled lines versus convergence in central processing, *Annual Reviews of Neuroscience*, vol 26, 2003, pp. 1-30.
- Craig, AD, Human feelings: why are some more aware than others? *Trends in Cognitive Sciences*, vol 8, 2004, pp. 239-41.
- Craig, AD, Forebrain emotional asymmetry: a neuroanatomical basis? *Trends in Cognitive Sciences*, vol.12, 2005, pp. 566-71.
- Crowley-Matoka, M, S Saha, SK Dobschka & DJ Burgess, Problems of quality and equity in pain management: exploring the role of biomedical culture, *Pain Medicine*, vol. 10, 2009, pp. 1312-1324.
- Dennett, D (1978), *Brainstorms: Philosophical Essays on Mind and Psychology*, Penguin Books, London, 1997, pp. 190-229.
- Dennett, D, *Consciousness Explained*, Penguin Books Ltd., London, 1993, pp. 21-42.
- Drack, M & W Apfalter, Is Paul Weiss' and Ludwig von Bertalanffy's system thinking still valid today? *Systems Research and Behavioral Science*, vol. 24, 2007, pp. 537-546.
- Edelman, G, *Bright Air, Brilliant Fire*, Penguin Books Ltd., London, 1992, pp. 73-80.
- Engel, GL, The need for a new medical model: a challenge for biomedicine, *Science*, vol. 196, 1977, pp. 129-136.
- Engel, GL, The care of the patient: art or science? *Johns Hopkins Medical Journal*, vol.140: 1977, pp. 222-32.
- Favareau, D, Beyond self and other: on the neurosemiotic emergence of intersubjectivity, *Sign System Studies*, vol. 30, 2002, pp. 57-100.
- Flor, H & C Hermann, 'Biopsychosocial models of pain' in RH Dworkin & WS Breitbart WS (eds), *Psychosocial Aspects of Pain, Progress in Pain Research and Management, Vol 27*, IASP Press, Seattle, 2004, pp. 47-75.
- Foerster, H von, 'On self-organizing systems and their environments' in M Yovits & S Cameron (eds), *Self-Organizing Systems*, Pergamon Press, London,1960, pp. 31-50.

-
- Gray, W, 'Ludwig von Bertalanffy and the development of modern psychiatric thought' in W Gray & ND Rizzo (eds), *Unity Through Diversity: a Festschrift for Ludwig von Bertalanffy, Part I*, Gordon and Breach Science Publisher, New York, 1973, pp.169-83.
- Greaves, D, Reflections on a new medical cosmology, *Journal of Medical Ethics*, vol. 28, 2002, pp. 81-85.
- Loeser, JD, 'Perspectives on pain' in P Turner (ed), *Proceedings of the First World Congress on Clinical Pharmacology and Therapeutics*, Macmillan, London, 1980, pp. 313-16.
- Luhmann, N, *Social Systems*, Stanford University Press, Stanford, 1995.
- Luisi, PL, Autopoiesis: a review and a reappraisal, *Naturwissenschaften*, vol. 90, 2003, pp. 49-59.
- Lycan, WG, Philosophy of Mind, in N Bunin & EP Tsui-James (eds), *The Blackwell Companion to Philosophy*, Blackwell Publishers, Ltd., 1996, pp: 167-197.
- Lyon, P, Autopoiesis and Knowing: Reflections on Maturana's biogenic explanation of cognition. *Cybernetics and Human Knowing*, vol. 11, 2004, pp. 21-46.
- Maturana, HR & FJ Varela, *Autopoiesis and Cognition: the Realization of the Living*, Reidel Publishing Co., Dordrecht, 1980, pp. 5-58.
- Medawar, P, *Induction and intuition in scientific thought*, Methuen, London, 1969.
- Melzack, R, & PD Wall, Pain mechanisms: a new theory, *Science*, vol. 150, 1965, pp. 971-979.
- Melzack, R, & PD Wall, *The Puzzle of Pain*, Penguin Books, Harmondsworth, 1973.
- Melzack, R, From the gate to the neuromatrix, *Pain*, Suppl 6: 1999, pp. S121-126.
- Merskey, H, & N Bogduk, *Classification of Chronic Pain*, IASP Press, Seattle, 1994, p. 210.
- Quintner, JL, ML Cohen, D Buchanan, J Katz & O Williamson, Pain medicine and its models: helping or hindering? *Pain Medicine*, 2008, pp. 824-834.
- Rizzolatti, G, L Fadiga, L Fogassi & V Gallese, Resonance behaviors and mirror neurons, *Archives of Italian Biology*, vol. 137, 1999 100.
- Rizzolatti, G & C Sinigaglia, Mirror neurons and intentionality, *Functional Neurology*, vol. 22, 2007, pp. 205-210.
- Ryle G, *The Concept of Mind*, The University of Chicago, Chicago, 1949, pp. 15-16.
- Siddall, PJ, & MJ Cousins, Persistent pain as a disease entity: implications for clinical management, *Anaesthesia Analgesia*, vol.99, 2004, pp. 510-20.
- Singer, T & C Lamm, The social neuroscience of empathy, *Annals of the New York Academy of Sciences*, vol. 1156, 2009, pp. 81-96
- Varela, FJ, E Thompson & E Rosch, *The Embodied Mind: Cognitive Science and Human Experience*, MIT Press, 1991, pp. 133-145.
- Waddell, G, M Bircher, D Finlayson & CJ Main, Symptoms and signs: physical disease or illness? *British Medical Journal*, vol 289, 1984, pp. 739-741.

Weiss, PA, Tierisches Verhalten als 'Systemreaktion'. Die Orientierung der Ruhstellungen von Schmetterlingen (Vanessa) gegen Licht und Schwerkraft. *Biologica Generalis*, vol. 1, 1925, 165-248.

Williamson, OD, DA Buchanan, JL Quintner & ML Cohen, Pain beyond monism and dualism. *Pain*, vol.116, 2005, pp. 169-170.

Winnicott, DW, *Playing and Reality*. Tavistock Publications, London, 1971.

Milton Cohen is a Consultant Physician in Rheumatology and Pain Medicine, St Vincent's Campus, Sydney, New South Wales, Australia.

John Quintner is a Consultant Physician in Rheumatology and Pain Medicine, Pain Medicine Unit, Fremantle, Western Australia, Australia.