Valerie Hardcastle’s book is a wide-ranging and thorough account of pain, covering philosophical, neuroscientific, and psychiatric literatures. It is driven by an over-arching biological model of pain processing, pain inhibition and pain sensations, which is tested against and illustrated by many appropriate examples and case studies (including an astonishing and literally painless account by David Livingstone of what it feels like to be mauled by a lion). Hardcastle makes three main claims: (1) pain is not to be exclusively identified with a subjective (i.e. phenomenological) state of mind, (2) there is no such thing as a psychogenic pain, and (3) the biological model of pain put forward should serve as a paradigm for the analysis of other psychopathologies. She argues convincingly for (1), showing how the phenomenological aspect of pain is just one component within a wider pain system (which she points out is more complex than is usually recognized by philosophers); but, I shall argue, claims (2) and (3) are problematic. I shall first give a brief overview of the book, and then try to give an assessment of its main claims.

1. Overview

Chapter one sets out the aims of the book and gives a brief history of theories of pain from Galen onwards. Chapter two reviews the clinical literature on psychopathological pains (i.e. those chronic pains which seem to be caused by psychological rather than physical trauma) and concludes that no research has established firm causal links between psychopathology and pain and that ‘in all likelihood, all pains are physical in origin’ (p. 31). Thus, there is no such thing as a ‘psychogenic’ pain, namely a pain that has a mental or psychological cause.

In chapter three, Hardcastle deals with the distinction between mental and
physical and reviews some of the literature in philosophy of mind concerning the problems of mental causation and of naturalizing content. Although she takes a firm materialist position, she does not rule out mental causation per se, and agrees that the sciences of psychology, psychiatry and biology should, at least at the present time, make use of the distinction between mental and physical in terms of different explanatory theories or different levels of explanation (but not as ontologically distinct entities). Chapter four reviews two different perspectives in neuroscience and how each one conceptualizes pain.

Chapter five addresses the nature of pain and tests philosophical accounts against what is known biologically about pain. She distinguishes several views of pain held by philosophers, including ‘objective’ views—e.g. pain is intrinsic to the injured body part (Armstrong, 1981), or is a set of behavioural reactions (Wittgenstein, 1953); and ‘subjective’ views—e.g. pain is a subjective state which is essentially private and mysterious (McGinn, 1983). Hardcastle then sketches the biology of the pain system, which involves two subsystems, each with a distinct physiological pathway. The first pathway, dealing with the ‘affective-motivational’ properties of pain (e.g. unpleasantness) leads ultimately to the frontal cortex and motor activity. The second, dealing with the ‘sensory-discriminative’ aspects of pain (e.g. location and intensity) leads to the somatosensory cortex and the localizing of the pain. She then diagnoses why there is so little agreement among the philosophical theories. They have all identified pain with just one of the components in one of the two pain subsystems: for example, the behaviourist view has identified pain only with its motor aspect, and the subjective view with, according to Hardcastle, its somatosensory aspect. In chapter six, Hardcastle aims to show that we have two independent pain-related systems. The first is the pain processing system, of which the two subsystems mentioned above are a part. The second is a pain inhibiting system, and she uses this latter system to explain the puzzle of self-injurious behaviour (and also why David Livingstone felt no pain when mauled by a lion).

Chapter seven focuses on pain sensations and argues for a version of eliminativism about our pain language. She outlines four dimensions of pain sensations: location, somatic quality, feeling of suffering, and negative reaction to the feeling of suffering, and points out that we often get one aspect of the pain sensation without the others (e.g. diazepam reduces suffering, but the locational and somatic aspects of the pain remain the same). This means that the everyday language we use for pain sensations is very crude: it tends to lump together different and separable things under the single term ‘pain’. Hardcastle then argues for a kind of partial elimination of pain talk. She says ‘we should eliminate our current pain talk and replace it with something more psychologically and biologically accurate’ (p. 171), but since she also regards our beliefs about our pain sensations to be an important component of pain processing, she makes one exception to the elimination programme: namely the folk psychological notion of belief.

Chapter eight is concerned with the treatment of pain. Perhaps surprisingly,
the most effective pain-relieving technique (according to research cited by Hardcastle) is hypnosis. The reason for this is analysed by Hardcastle as being that hypnosis appears to have a global effect on the brain and thus modifies many more dimensions of our complex pain system than other treatments, which each alter only one or two components of the pain system. The end of the chapter deals with pain in infants and children, and Hardcastle argues that we must prevent infants’ pain—whether or not they consciously experience it—because of evidence of the negative effects of early intense activation of the pain system on later development.

In chapter nine, Hardcastle proposes to show how her biological model of pain should serve as a paradigm for how to understand psychopathologies in general. The paradigm includes: focusing on the neurophysiology of a disorder, to help define the illness and to prevent covert dualism; and being aware of the complexity of nonconscious brain processing to decrease our dependence on unreliable subjective reports. She gives some pointers as to how this approach might deal with autism, drug addiction, and obsessive-compulsive disorder.

2. Evaluation: What Happened to the Psychological Level?

Hardcastle deserves praise for her ambitious cross-disciplinary coverage of a large set of topics in pain research. She has succeeded admirably in creatively bringing together diverse work on pain from psychology, philosophy and neuroscience. The ultimate success of her project, however, depends on the soundness of her three main claims. One general comment I would like to make is that Hardcastle’s ambivalence towards the psychological level of explanation adds confusion to the assessment of her claims: the psychological level is present throughout much of the detailed reasoning of the book, but it disappears just at the moment conclusions are drawn, as we shall see.

2.1 Claim 1: Pain is not to be Exclusively Identified with a Subjective State of Mind

This is the most convincingly established of the three claims. In chapters 5–8, Hardcastle demonstrates the biological complexity of the pain system, and the varied components of which it is comprised. With regard to infant pain, Hardcastle demonstrates that the physiological and behavioural activation of the pain system in infants can have negative effects upon later development, whether or not infants have pain experience. For example, premature babies given analgesics during an operation show fewer hormonal responses and have fewer circulatory complications than those not given analgesics (Anand, Sipell and Aynsley-Green, 1987). This description shows the negative physiological effects of intense activation of the pain system, but says nothing about phenomenal experience. Hence ‘pain’ is not simply an experience and what is nega-
tive about pain is not merely its phenomenology. Though this case is well-made, it does not follow that pain phenomenology is only a minor component of the pain system, a position to which Hardcastle seems to be drawn. She sometimes appears to treat pain phenomenology as an ‘optional add on’ feature of the pain system. However, a strong case can be made for the functional importance (as well as the obvious personal importance) of the conscious experience of pain (e.g. see Balleine and Dickinson’s, 1998, account of the link between hedonic experience and intentional action in mammals).

2.2 Claim 2: There are no Psychogenic Pains

This is the claim that no pains have a mental cause and all pains have a physical, somatic cause. Now, according to a physicalist view of the mind all mental events ultimately have a physical basis, so what Hardcastle could be saying here is simply that any putative mental cause for a pain is really a physical cause, since every mental event is ultimately really a physical event. However, it is important to realize this is not what Hardcastle is saying. She explicitly does not reject mental causes per se. Rather, she specifically rejects them for the case of pain on empirical grounds—her review of the literature on psychogenic pains in chapter two leads her to this conclusion. However this means that the issue of psychogenic pains is not an ontological one about the metaphysical status of a particular cause of pain, but is rather a theoretical and empirical one about what is the best explanation for a particular pain. Insofar as Hardcastle’s attack is against clinicians who use the diagnosis of psychogenic pain to mean that the pain exists ‘only in the mind’ and nowhere else (or that it exists only at the mental level and not at all at sub-personal functional level, or neurophysiological level) then her attack, according to the physicalist project, is completely justified, and she would be correct that these kinds of psychogenic pains do not exist. She provides examples that suggest that some clinicians do indeed take the ‘only in the mind’ view and in these cases she is right in saying that there is a ‘pernicious dualistic mind/body distinction lurking in psychopathology’ (p. 7). But insofar as her attack is against clinicians who merely think that the most appropriate explanatory theory of a particular pain involves mental terms rather than biological terms, it is less clear her attack is justified and indeed she seems to contradict herself.

Before we illustrate this for pain, let us consider a state for which many people would agree the best explanatory account is sometimes psychogenic and sometimes somatogenic: namely, nausea. Sometimes nausea is caused by eating a noxious food, for example bad meat. At other times nausea is caused by particular thoughts and beliefs, for example the thought ‘I will die if I get on that plane’, or the belief that ‘everyone is going to judge my performance in the presentation I am about to give’. In psychogenic nausea, the thoughts and interpretations activate the autonomic nervous system (according to standard emotion theory) and thereby have an effect on internal organs including the stomach and bowels. In both psychogenic and somatogenic nausea, saying
the cause is mental or physical means, in the present context, ‘the best explanation of the nausea, all things considered, is that is was caused by [mental event x/somatic event x]’.

Are pains radically different from nausea in this respect? Do pains never have mental causes? It is, initially, hard to think of particular thoughts which might cause people physical pain. An individual might complain that thinking very hard about a stressful or difficult topic gives them ‘a headache’, but often this is metaphorical. However, to give just one example, abdominal pain associated with irritable bowel syndrome can certainly be triggered by anxious thoughts, via the well-established connection between emotional appraisal and autonomic arousal. For example, there is evidence that acute experimental stress can enhance the motor activity of the gut and cause pain (Almy and Tulin, 1947). Of course these pains are mediated by somatic events. But that is not to say that they do not have psychological causes, if the most appropriate explanation in the immediate chain of events leading to the abdominal pain is that the person had a worrying thought rather than, say, that they were punched in the gut.

Indeed Hardcastle herself gives a perfect example of mental causation of pain later in her book (and which she endorses). Regarding the triggers for phantom limb pain, she writes: ‘Some cognitive or emotional event activates a conscious schema associated with painful experiences. A remembered pain, then, becomes real once again. The brain creates the experience of pain from available cognitive and emotional schemata and other associations’ (p. 119). This seems like a textbook example of psychogenic pain—the immediate cause of the pain is some cognitive or emotional event which activates a conscious schema. The explanatory account here is at the level of psychology, not physiology.

A second excellent example of psychogenic pain occurs on p. 171. Hardcastle reports, in a positive light, a study carried out by Silverman et al. (1997), in which irritable bowel syndrome patients underwent PET scans and were told that a rectal balloon was being inflated even though it wasn’t. At the fictitious point of onset of balloon inflation the patients ‘claimed to feel pain in their rectal areas, and these felt pains correlated with activations in prefrontal cortex’ (p. 171). In this case a belief or expectation has caused a pain; and again we have a case of psychogenic pain. Hardcastle seems to accept the interpretation just given of these two examples and therefore she contradicts the conclusion of chapter two, that there are no psychogenic pains.

### 2.3 Claim 3: A Biological Paradigm for Psychopathology?

In the final chapter, Hardcastle argues that her biological model of pain should serve as a paradigm for the philosophical analysis of other psychopathologies, such as autism, addiction, and obsessive-compulsive disorder. Two of the key lessons of her approach are (1) to focus on the neurophysiology of a disorder, and (2) to decrease our reliance on subjective reports. Applying these lessons to autism, she first recounts Simon Baron-Cohen’s (1995) description of the
illness: ‘The key features of …autism… include lack of normal eye contact, lack of normal social awareness or appropriate social behaviour, “aloneness”, one-sidedness in interaction, and inability to join a social group’ (Baron-Cohen, 1995, pp. 62–3). Hardcastle then writes:

As long as descriptions have remained on the level of the behavioural or the cognitive, autism has remained a syndrome of bizarre and inexplicable symptoms. However, instead of thinking of autism as a cluster of features…, we might instead start thinking about autism as something like atrophied vermis of the cerebellum. This underlying neurobiological structure provides a good peg on which to hang the diverse symptomology… (p. 202).

It is not clear to me why thinking about autism in this way makes the symptoms less ‘bizarre’. Indeed, in Baron-Cohen’s account the diverse symptoms are most definitely not ‘bizarre and inexplicable’. He explains them as all following from underlying cognitive deficits in ‘shared attention’ and ‘theory of mind’ mechanisms. These functional mechanisms are tentatively located by Baron-Cohen in the superior temporal sulcus, and the orbito-frontal cortex respectively. Similarly, Frith (1992), in a multi-levelled model of autism, considers the causal pathway between underlying biological causes, brain abnormalities, a cognitive deficit, core problems, and observed symptoms. The core problems and observed symptoms (including all those recounted above from Baron-Cohen) are explained as due to an underlying cognitive deficit in mentalization.

Even if Baron-Cohen’s and Frith’s accounts turn out to be incorrect, they both explain the disorder much more clearly than Hardcastle’s proposed single-level account. Frith’s model includes and encompasses Hardcastle’s (in that it includes the neurobiological level), but includes more explanatory levels of analysis. Surely Hardcastle’s model requires these extra functional layers otherwise how are we to describe what the vermis of the cerebellum does? Even if we identify the neurobiological structures responsible for autism, this identification in itself explains nothing—by which I mean it provides no conceptual explanation, only a limited causal one. But it is precisely the conceptual explanation which can render the symptoms less ‘bizarre’ (in Baron-Cohen’s account the lack of normal eye contact, the aloneness, etc. are all explained conceptually by the deficits in shared attention and theory of mind mechanisms).

In chapter seven Hardcastle writes admiringly that ‘the best clinicians attend to historical, environmental, and interpersonal influences on pain perception, as well as the more standard cognitive, emotional, and behavioural dimensions. They believe that understanding the total experience of pain necessitates understanding the physiology, subjective experiences, behavioural responses, and psychosocial environment of the subject’ (p. 148). This sounds like an
excellent multidimensional, multifactorial model of pain. I thought this was going to be Hardcastle’s model in her final chapter. Instead, in the final chapter we get a much cruder model in which the psychological and social levels have vanished and which, by her own earlier arguments and analyses of pain, is insufficient to explain either the diverse phenomena of pain or the complexity of psychological disorders.

References


