What Pain Asymbolia Really Shows

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Abstract

Pain asymbolics feel pain, but act as if they are indifferent to it. Nikola Grahek argues that such patients present a clear counterexample to motivationalism about pain. I argue that Grahek has mischaracterised pain asymbolia. Properly understood, asymbolics have lost a general capacity to care about their bodily integrity. Asymbolics' indifference to pain thus does not show something about the intrinsic nature of pain; it shows something about the relationship between pains and subjects, and how that relationship might break down. I explore the consequences of such a view for both motivationalism and the categorisation of pain asymbolia as a syndrome, arguing for a close link between asymbolia and various forms of depersonalisation.

1 Pain and Motivation

Pains motivate us. Must they? *Motivationalists* about pain say yes: motivational force is an intrinsic property of pains. Many disagree. The debate can be shaped by empirical facts. Find someone who is entirely unmoved by pain, and motivationalism is threatened. Fail repeatedly to find such a case, and motivationalism gains credence.

In a recent book, Nikola Grahek (2007) presents an apparent counterexample to motivationalism. This is the strange case of *pain asymbolia*. Pain asymbolia is a rare condition caused by lesions to the posterior insula (Berthier et al. 1988). Asymbolics say that they feel pain, but they are strikingly indifferent to it. In the first reported case, Schilder and Stengel note that:

The patient displays a striking behaviour in the presence of pain. She reacts either not at all or insufficiently to being pricked, struck with hard objects, and pinched. She never pulls her arm back energetically or with strength. She never turns the torso away or withdraws with the body as a whole. She never attempts to avoid the investigator.¹ (Schilder and Stengel 1928, p. 147)

 $^{^1{\}rm Quotations}$ from Schilder and Stengel 1928 are the author's own translation from the original German. Special thanks to Aleks Zarnitsyn and Mae Liou for their help with the translation.

Strange enough. But not only do asymbolics fail to react to such stimuli, they also appear to recognise what they feel *as pains*. Schilder and and Stengel continue:

Pricked on the right palm, the patient smiles joyfully, winces a little, and then says, 'Oh, pain, that hurts.' She laughs, and reaches the hand further toward the investigator and turns it to expose all sides ... The patient's expression is one of complacency. The same reaction is displayed when she is pricked in the face and stomach. (Schilder and Stengel 1928, p. 147)

As Schilder and Stengel note, the patient was in no way inattentive or unaware of the painful stimuli. Quite to the contrary: she was actively engaged with the investigators, and readily offered up new body parts to be poked and prodded (p. 148).

Asymbolics thus appear to feel pain without being motivated by it. Other counterexamples to motivationalism have been proposed, but pain asymbolia is arguably the cleanest. Stoics and masochists do not seem to react to the pains they feel. But plausibly, this is only because they have other, overriding motivations—duty or dignity for the stoic, self-control or submission for the masochist. Motivationalism claims only that pains are a part of our motivational structure, not that they are the only or strongest part. In contrast, there seems to be no overriding motivation that explains asymbolics' lack of response.

Lobotomised patients also appear indifferent to previously intractable pain. But as Melzack and Wall (1982, p. 131) point out, these patients still withdraw from pinprick, avoid walking on broken ankles, and generally react to pain as we do. The pain of the lobotomised thus retains its biologically basic biological motivational force; what has gone missing are the other emotions usually associated with strong pains. Brand and Yancy note that patients report feeling 'the little pain without the big pain' (quoted in Grahek 2007, p. 32), suggesting that what is missing are the secondary negative reactions to pain, not pain's core motivational import. The motivationalist can and should concede that emotions like fear, frustration, and anger are only contingently connected to pain. What pains necessarily motivate are actions that protect our bodily integrity; other negative affective states depend on a cognitive evaluation of the significance of pain. Asymbolics, on the other hand, do not protect their bodies when they encounter stimuli that cause them pain.

Finally, acutely injured patients given a dose of morphine will often say that they feel pain, but no longer care about it. They closely resemble asymbolics in this regard (and I will suggest that the difference is more than superficial). We usually take reports from patients on powerful narcotics with a grain of salt, however, and there are practical and ethical barriers to thorough experimentation in the emergency room. In contrast, there is a well-established tradition of using first-person reports of patients with lesions, and neurology permits detailed tests of their responses.

It is worth emphasising just how strangely asymbolics behave, even outside of laboratory settings. Schilder and Stengel (1931, p. 598) report a patient would readily stab herself with needles and jam objects into her eyelid. Berthier et al. (1988, p. 43) report an asymbolic patient who suffered a severe burn at home because he made no attempt to escape the danger. Hemphill and Stengel note of a patient:

The absence of any defence or withdrawal reaction was clearly shown when a strong, painful sensation was applied by surprise, e.g. when the examiner, standing behind the patient, suddenly pricked his hand or neck. When the patient was threatened with the first he made no effort to guard himself or to withdraw his head, nor did he show any instinctive combative reaction. (Hemphill and Stengel 1940, p. 256)

More generally, pain asymbolics seem willing to submit to ghastly batteries of tests, even though many of these tests are actually injurious.

So much for motivationalism? I say no. In what follows, I will argue that Grahek has misinterpreted pain asymbolia. Grahek treats asymbolia as a deficit of sensation. I will present an alternative view, on which asymbolics have lost a fundamental capacity to care about their bodies. The alternative view better explains the wide variety of phenomena associated with asymbolia. I will conclude by showing that this capacity-based view is compatible with a weak form of motivationalism, and suggest a story compatible with that weak motivationalism.

I have left two aspects of motivationalism purposefully general, and one temporarily vague. First, I have not said anything about *how* pains might motivate. Motivationalism is a big tent. It includes those who think that pains are reducible to other, more basic motivational states like attitudes, judgments, or evaluations (Nelkin 1986, 1994; Helm 2002). It also includes accounts that take pains (along with other bodily sensations) to have a *sui* generis, essentially motivating intentional content like an imperative or a command (Klein 2007; Hall 2008; Martínez 2010). It is compatible with accounts on which pain is simply a primitive qualitative experience of badness or the like. My initial argument will be pitched in such a way as to be available, in principle, to any of these positions. I will return in section 4 to evaluate these possibilities further.

Second, I have not said *what* pain motivates. That is again for the sake of generality. I will assume, however, that the biological function of pain is the preservation of bodily integrity, and that the phenomenology of pain has some tight connection to the fulfilment of that role. In normal cases pain motivates actions that help keep our bodies healthy and intact—to avoid injury, to nurse wounds, to favour wrenched joints, and so on.² Asymbolics do not seem to be motivated to protect their bodies. Hence the problem for motivationalism.

Third, I have not yet stated the motivationalist thesis with philosophical precision. My defence of motivationalism will, if successful, restrict and sharpen motivationalism. For now the intuitive idea—that pains are intrinsically motivating—will be enough to begin.

2 Two Models of Asymbolia

2.1 The Degraded Input Model

Here is one model of what has gone wrong in asymbolia. Pain is actually a composite mental state. It has (at least) two proper parts: a sensory part, perhaps representing something like tissue damage, and an affective/motivational part, which moves us to act. These two parts typically go together, and there is good biological reason for them to do so. Under the right conditions, however, one or the other can be absent.

Variants of this composite view of pain are popular among both philosophers and scientists (Dennett 1978; Hardcastle 1997; Price 2000). Grahek also endorses it. As he puts it,

... although pain appears to be simple, homogenous experience, is actually a complex experience comprising sensory-discriminative, emotional-cognitive and behavioural components. These components are normally linked together, but they can become disconnected and therefore, much to our astonishment, they can exist separately. (Grahek 2007, p. 2)

²One might worry that self-protection is not the *only* thing that pains motivate us to do: we might also be motivated to take an aspirin, see a doctor, complain, and so on. Motivationalism, as I understand it, is meant to be a view about what is common to all pains. The only plausible common motivational force is to protect one's body against actual or potential injury—this is the *immediate* import of pain. Further motivations, such as they are, are clearly shaped by idiosyncratic and cultural knowledge about the availability of other avenues of relief. See Wall 2000 for several arguments along these lines.

When the components of pain come apart, strange syndromes result. Asymbolia is a paradigm case. The pain of asymbolics, Grahek argues, has lost the affective/motivational component. As such, Grahek argues '[Pain] becomes a blunt, inert sensation, with no power to galvanize the mind and body for fight or flight. Such pain no longer serves its primary biological function' (2007, p. 73).

Call this the *degraded input* (DI) model of asymbolia. DI claims that asymbolics have a deficient sensation: their pain lacks the motivational push that our ordinary pains possess. This explains why asymbolics are indifferent to pain: the pain itself has changed. The DI model is incompatible with motivationalism. According to DI, the motivational force of pains comes from their affective/motivational component. That component can go missing, but the sensation remains a pain.³ So motivationalism is false.

Grahek argues that there is a double dissociation between the sensory and affective aspects of pain. Pain asymbolia provides one half of the dissociation: as he puts it, asymbolics feel pain without painfulness (where 'painfulness' refers to pain affect). The other half of the dissociation—painfulness without pain—depends on a case described in Ploner, Freund, and Schnitzler 1999 of a patient with a unilateral lesion to SI and SII. Laser stimulation to the left (contralateral) hand did not elicit pain sensation, but did produce in the patient a 'clearly unpleasant' feeling that he 'wanted to avoid' (p. 213). Grahek takes this as a case of pain affect preserved in the absence of pain sensation. We thus appear to have a double dissociation between pain affect and pain sensation.⁴ Double dissociation between two mental processes is

³Note that not all composite theories are incompatible with motivationalism. One could argue that pain must have both components to be deserving of the name, or that 'pain' properly refers to the motivational portion, not the sensory one. Armstrong (1962, p. 106ff) argues, for example, that pains are a combination of a tactile sensation plus an extreme dislike of that sensation. Grahek, drawing on Hardcastle, eschews this strategy (p. 95); I am happy to follow his lead for the sake of argument. As an anonymous reviewer pointed out to me, Grahek is not entirely consistent on this point: see his discussion of components of pain versus *real* pain (p. 111).

⁴Grahek never uses the term 'double dissociation', though his argument is obviously meant to be read as appealing to a double dissociation between pain affect and sensation. For a more explicit double dissociation argument for the same conclusion, see Hardcastle 1997. The point is not merely pedantic. *Paired* dissociations are crucial bits of evidence; As argued in Shallice 1988 (p. 35ff) single dissociations are hard to interpret, and provide weaker evidence for separability. I will argue shortly that Grahek misinterprets the putative dissociation provided by asymbolia. While the other half of the purported dissociation—painfulness without pain—is less relevant to the question of motivationalism, I am also suspicious of it. First, the patient described in Ploner, Freund, and Schnitzler 1999 *did* appear to feel pain in the hand contralateral to his lesion, albeit with a much higher threshold (see Fig. 2). Further, their patient described the sensation he was feeling

usually taken as evidence that they are only contingently related (even if they typically occur together). So the composite view of pain falls out directly, and DI appears to be well-motivated.

2.2 The Lost Capacity Model

DI is not the only way to understand asymbolia. Here is another model: Asymbolics fail to react to pain because they no longer care about the physical integrity of their bodies. More precisely, they have lost the *capacity* to care about their bodies in whatever way is relevant to pain. They do not care about cuts and burns and scrapes, because they can no longer conceive of why such events are bad.

Call this the *lost capacity model* (LC) of asymbolia. Both LC and DI predict that asymbolics will be unmoved by pain. They differ, however, on the explanation of that fact. DI says that something has changed about the *person*, not the pain. LC says that something has changed about the *person*, not the pain. Further, LC predicts that the deficits in asymbolics should be relatively widespread. Asymbolics should be indifferent not just to pain, but to *any* immediate threat to their bodily integrity. Information about such threats can come from a variety of sources: sensation, language, beliefs, and so on. Caring about the integrity of your body requires hooking up sensation, cognition, affect, and behaviour in the right ways, regardless of how one comes to know about a threat. According to LC, asymbolics lack this integrative capacity, because their lesion has destroyed the neural substrate on which the capacity depends.

The Lost Capacity model faces an initial empirical complication that is worth addressing. If 'threat to bodily integrity' is understood so broadly as to include the threats that come from failure to eat or urinate, then LC looks empirically false. Schilder and Stengel's patient (1928, p. 152), for example, asked to eat and use the bathroom. If, as LC claims, asymbolics are not motivated to protect their bodies, what motivates them in these cases?

Distinguish between *immediate* and *distant* threats to one's body. Avoiding an immediate threat requires action now or in the very near future; avoiding a distant threat can typically be done in one's own time. Pains are associated with immediate threats. Hunger represents a distant threat:

as 'unpleasant' before he felt pain (p. 213). But there are many unpleasant sensations aside from painful ones. Why think that the patient felt the negative affect associated with *pain*, rather than just some other unpleasant sensation? Similar remarks apply to Hardcastle's interpretation (1997, p. 393) of tooth pulp stimulation under the influence of fentanyl. It takes explicitly casting Grahek's argument as a double dissociation, then, to make clear just what the problems with it might be.

failure to eat will eventually cause damage, but one typically has considerable time and flexibility in choosing how to meet that threat.⁵ Asymbolics' behavioural oddities, then, seem to be limited to direct threats. The lack of response to direct threats admits of several possible explanations. Here is the one I find most plausible.⁶ Our responses to distant threats are largely shaped by habit. Most of us eat at fixed times, for example, and just because it is time to eat. The difference in Asymbolics' responses to distant threats, then, may be accounted for by the retention of habits that promote bodily integrity even in the absence of the underlying capacity to care about bodily integrity. Responses to immediate threats are, for obvious reasons, much harder to shape and so much less dependent on habit. Hence a lack of care would show up most obviously in responses to immediate threats—like those associated with pains and the like.

While both DI and LC are psychological-level theories, a brief note about the brain is in order. Both Grahek and I accept that asymbolia results from damage to the posterior insula, a cortical region plausibly involved in integrating sensory and limbic signals related to pain (Craig 2003). We differ on how to interpret this functional consequences of this damage. Drawing on a proposal first put forth by Geschwind (1965), Grahek argues that pain represents a *sensory-limbic disconnection syndrome* (p. 52). On his view, damage to the insula in asymbolics prevents limbic processing from being incorporated with sensory processing. DI is motivated by this picture: there are two processing streams in normal folks, one of which has become a deadend in asymbolics.

Geschwind's model of disconnection syndromes has been criticised for assuming an entirely serial, feed-forward picture of the brain (Catani et al. 2005). On his view, each brain region performs a specialised function and

⁵The need to urinate even more so: one has to exert considerable force of will to be damaged rather than merely embarrassed. Further, eating and bathroom-going are likely to be tightly regulated in the institutional settings in which most asymbolics reside. In general, counterfactual analyses work reliably only for immediate threats. Were I to stop eating now I would end up in the hospital, not dead.

⁶It is not the only plausible story. Caring about one's body in the case of immediate threats might just dissociate from caring about one's body in the case of distant threats. More generally, the insula is a complex and functionally differentiated structure that underlies many different interoceptive functions (Ibañez et al. 2010). So it is possible that damage might spare some functions and not others. That is an empirical matter, one complicated by the fact that the insular damage is typically quite widespread and messy. Such dissociations would be surprising, but no more so than those found in other areas of neuropsychology. The conclusion would then be that asymbolics have lost the capacity to care for their bodies in some ways, but not in others. This possibility is empirically distinguishable from the story I suggest, though I find nothing in the actual clinical literature which would allow us to do so.

passes on the result to higher association centres, which in turn pass on their results to still further association centres, and so on. Earlier processes in the causal chain are entirely unaffected by later ones. DI embodies a picture like this: the sensory deficits of asymbolics are caused by a failure of limbic processing to be attached appropriately to sensory processing in some later stage. This simplistic model of brain function has fallen out of favour. The insula projects back to the limbic system, and receives input from a variety of frontal areas. Thus it seems to do more than simply composite together the results of earlier sensory processing stages—instead, it plays an active role in integrating multiple different cognitive processes, especially interoceptive and motivational ones (Singer et al. 2009). LC is partly inspired by this picture of the insula.

A final difference. DI treats the motivational force of pain (when present) as a brute fact about pain: some sensations just have the power to motivate, and pain is one. LC, in contrast, gives an explanation of just why pains motivate. Pains motivate because we care about our bodies. Were we to stop caring—something that is ordinarily impossible, for good biological reasons—then pains would not matter. Asymbolics are a realisation of this unusual possibility.

2.3 Evidence for a Lost Capacity

Both LC and DI predict the pain-related deficits of asymbolics. LC, however, predicts that there should be a general loss of appreciation for threats to bodily integrity. DI does not.

The clinical literature supports LC. First, asymbolics are not indifferent to pain alone. They also appear to be indifferent to any dangerous or threatening stimulus. Hemphill and Stengel's patient (1940, p. 256) was also 'quite disinterested' when matches were struck close to his face and eyes, and showed no response to unexpected loud noises or strong flashes of light. Schilder and Stengel report (1928, p. 149) that their patient also failed to respond to being threatened with a hammer, a knife, and a needle; to shrill whistles; and to a magnesium wire burned inches from her face.

Asymbolics' indifference is not limited to simple sensations. Berthier et al. report (1988, p. 43) that five of their six patients failed to respond to 'verbal menaces'. Schilder and Stengel note (1928, p. 154) that their patient 'shows no appreciation at all for threats of pain *or for any threats in general*'(my italics). Hemphill and Stengel's patient showed a unusually dangerous lack of reaction:

The patient was observed proceeding one morning along the main

road of the hospital. He made no effort to get out of the way of a lorry behind him in spite of the loud warning of the horn. That he heard the horn and recognised its character is certain, for he admitted as much with considerable heat when he was forbidden, for his own safety, to walk alone on the main road. (1940, p. 256)

LC handles these various phenomena well. It predicts that asymbolics should be indifferent to bodily threats *regardless* of modality.

What about DI? Grahek mentions these phenomena. He suggests that the relevant deficit is plurimodal, and does not discuss the issue further (p. 48). I can think of two readings of this suggestion, neither of which is terribly satisfying. First, Grahek could mean that that asymbolics have a conjunction of many specific deficits. That is, asymbolics fail to attach motivational force to pain, and auditory sensations, and visual sensations, and to written and spoken language, and so on. Any of these deficits could in principle occur on their own; in asymbolics they happen to occur together, perhaps because of the anatomical proximity of distinct functional substrates. This interpretation is possible, but it seems ad hoc. It posits a distinct and potentially dissociable deficit for every modality that experimenters have thought to test, with no further evidence that these are in fact distinguishable problems. Of course, the multiple-deficit version of DI might still be true; without further evidence it is not very convincing.

Second, Grahek could mean that there is a single deficit, which manifests itself across a variety of sensory modalities. This would presumably be a conduction deficit: that is, the failure of a linkage between the limbic system and higher association areas. This is more plausible. However, it still requires a certain degree of special pleading. Asymbolics' deficits seem to be limited within modalities as well: they are indifferent only to sensations conveying bodily threat, not to sensations generally. Schilder and Stengel's patient, for example, had a strong emotional reaction to being called a liar and a thief (Schilder and Stengel 1928, p. 150). So her deficit cannot be simply one in attaching emotional valence to sensation and language quite generally: it is only utterances that involve threats that are affected.

On either reading, DI faces a further difficulty. A thought experiment: Suppose I anaesthetised your arm and placed it out of sight. Suppose I then told you that I was pummelling it with a hammer. You would, I suspect, be motivated to act—to remove your arm, to flee, and to rethink your reasons for trusting me in the first place. *Why* would you be motivated? Not because of some sensation you are having: your arm is insensate and occluded. Instead, you would be motivated by a simple bit of practical reason: you care about your body, caring about your body means you should avoid needless injury to it, needless injury is happening, and therefore you have a reason to act. So we can be motivated to protect our bodies in two ways: directly, because of some sensation we are having, or indirectly, because we believe that our body is being harmed.

What about asymbolics? By all accounts, they seem to lack both ways of being motivated. They are not motivated by their pain. But they also are not motivated by the fact that their body is being damaged. That fact should be apparent to them—both because they retain the sensory, informative aspect of pain, and also because they appear to know what is happening to them. Again, asymbolics readily submit to actually injurious tests. Again, they are actually injured because of their condition. This is puzzling. If asymbolics lacked *only* the motivational aspect of pain, we should expect them to be otherwise like us when it comes to bodily damage. But they are not.

A useful comparison is with the congenitally insensitive to pain.⁷ From birth, the congenitally insensitive do not feel any pains at all. *A fortiori*, they do not have sensations with whatever affective/motivational component Grahek thinks is critical for pain behaviour. Yet they still learn to protect their bodies as best they can. That is, they learn what situations are injurious, and avoid these situations precisely because they do not want to be injured.⁸

If we accept Grahek's account, asymbolics' total lack of motivation is puzzling. Grahek claims that the pain of the asymbolic lacks the usual affective component, and that explains their lack of response. But if that was *all* that was missing, we would expect the asymbolic to be like the congenitally insensitive to pain: unmotivated by the sensation of pain, but still motivated to protect their bodies when they learn of threats. On the contrary, the asymbolic appears to to be entirely uninterested in the fate of their bodies, *however* they learn about an injurious situation. Hemphill and Stengel's patient who put himself in danger on the road did not react to the sound of the horn. But he also did not react to *the fact that a truck was bearing down on him*.

⁷Grahek, unlike many authors, correctly distinguishes asymbolia from congenital insensitivity; see (p. 98ff). What follows thus depends on a distinction that he ought to accept.

⁸This care only goes so far—pain is still the most reliable spur to protect our bodies. Congenital insensitives have a drastically shortened lifespan, and would be worse off still without medical care. That said, a few take enough care to live into their thirties. Beyond childhood, they mostly avoid severe acute injury. Further, as Nash (2005) notes, some are laid low by conditions (appendicitis, ectopic pregnancy) of which pain is a symptom, but that would be untreatable before the modern era. Aside from this, adult insensitives have a twofold problem: they do a poor job of protecting minor injuries as they heal, and they fail to regulate their posture, leading to joint damage and subsequent osteomyelitis (Melzack and Wall 1982, pp. 18–19).

The point may be put in a slightly different way. The composite account of pain claims that asymbolics still have the sensory aspect of pain intact. What does that sensory aspect do? On most accounts, it informs about bodily damage or the like. (It could be a bare quale, but even then the presence of that *quale* is reliably associated with bodily damage, and so provides useful information). So according to DI, asymbolics should still know that they are being damaged. As per the bit of practical reason above, they should still be indirectly motivated to act. But they are not. Grahek, remember, says of the pain of the asymbolic that it is 'a blunt, inert sensory appearance with no power to galvanize the mind and body' (p. 73). But that would make the 'sensory-discriminative' aspect of pain unlike any other sensations we are familiar with. The sensation of seeing blue does not have (in ordinary cases) a motivational-affective dimension. But it still does something: it informs us that there is a blue thing nearby. On Grahek's story, the sensory-discriminative function of pain appears to be wholly epiphenomenal. It is there. We can make verbal reports about it. That is the only causal consequence it seems to have for our behaviour. That is deeply odd.

In contrast, LC gives a perfectly straightforward story about asymbolics' general lack of concern. Asymbolics do not care about the integrity of their bodies because they cannot. The capacity they lack applies to sensory evaluations of stimuli, to cognitive evaluations of threat, and indeed to any way in which we might normally learn that our physical integrity is jeopardised.⁹

Most work on downward modulation of nocioceptive pathways focuses on sensitisation.

⁹Asymbolics often fail to make even reflexive responses to stimuli they describe as painful; Schilder and Stengel report that their patient made only mild reflexive responses to extremely intense stimuli, and none at all to less intense manipulations. Several people (including an anonymous reviewer) have worried that a high-level explanation such as my own cannot account for this fact. Why would spinal-level reflexes be reliably suppressed by something like lack of care?

This objection depends on an inaccurate picture of spinal reflexes and their top-down control. All spinal reflexes are continually modulated by top-down signals from the cortex. In fact, some spinal reflexes *never* manifest in ordinary life—even slightly—after otherwise appropriate releasing stimuli. This is most obvious in the case of the so-called 'primitive reflexes' seen in infancy. The primitive reflexes are tonically suppressed by top-down signals in adults. They reappear after severe cortical damage, showing that the underlying spinal dispositions are constantly suppressed, rather than merely disappearing in adults (Schott and Rossor 2003; Plum and Posner 2007, p. 72). Further, many motor reflexes are altered in characteristic ways after frontal damage: stereotyped withdrawal reactions to injurious stimuli, for example, may become contextually inappropriate after severe brain injury (Plum and Posner 2007, p. 74; see also Berthier et al. 1988, p. 43 for a report of contextually inappropriate peripheral responses in asymbolia). Hence new stimuli do not provoke otherwise absent downward modulation—they merely change an ongoing modulatory process. Top-down modulation does not need to wait for signals to arrive from the periphery in order to be effective.

To conclude, there are two ways in which DI might be defended. The first is Grahek's, by arguing for a classic double dissociation between pain sensation and pain affect. Asymbolia is supposed to be one half of the dissociation, pain without painfulness. But asymbolia does not fit the classic double dissociation model. A dissociation requires severely impaired performance tasks involving one mental component and relatively preserved functioning on other tasks. Asymbolics, however, do not behave as we would expect someone with a mere sensory deficit to behave: the indifference they show runs deep. So there is no simple dissociation, and the argument fails.

Second, DI could be defended abductively—either as a neuropsychological argument, or as a more general species of inference to the best explanation. The strength of an abduction depends on the power of competing hypotheses. And, I argue, LC is a stronger explanation of asymbolia. Some phenomena it explains directly, while DI needs complex or *ad hoc* hypotheses to account for them. Other phenomena are explained by LC, but not by DI. So on balance, we have reason to prefer LC.

3 Modest Motivationalism

Let us suppose that the argument above is conclusive, and that LC is correct. Is LC compatible with motivationalism? Unsurprisingly, that depends on how we understand motivationalism. More surprisingly, the answer is yes. There is a philosophically interesting version of motivationalism to which asymbolia is no counterexample.

First, assume that all viable forms of motivationalism are hedged in the ways considered in section 1. That is, when we say that an agent is motivated

However, while relatively rare, some central abnormalities do seem to result in diminished or absent peripheral pain reflexes: the phenomenon is attested in cases of catatonia (Northoff 2002) and schizophrenia (Dworkin 1994). While speculative, I think the rough outlines of an explanation can be offered. We have known at least since Melzack and Wall 1965 that top-down modulatory input plays a crucial role in spinal pain processing, and that this can decrease as well as increase firing. Further, many serious injuries are initially painless, a fact that is likely due to adaptive central gating (Melzack et al. 1982). Spinal neurons are under descending control from brainstem nuclei that receive input from the amygdala, hypothalamus, and especially the periaqueductal grey. These in turn receive modulatory input from higher levels of cortex (Millan 2002; Price 2005). Asymbolics' lack of peripheral reflexes is likely due to a disorder in this complex system. The lack of care for one's body might directly cause abnormal tonic inhibition of reflexes. Reflex suppression might also be a mere side-effect: some insular region might inhibit a subcortical inhibitory region, and damage to that structure results in increased inhibition overall (Compare in this regard Sprague 1966). In any case, the lack of peripheral reactions to pain is consistent with other evidence on downward suppression of spinal-level reflexes.

by pain, we mean they are disposed to perform certain actions to protect the integrity of their physical body (Though that disposition can be overridden, and need not result in further negative affective states.) Given that, here are three ways to understand motivationalism:

- **Ambitious Motivationalism** Necessarily, if an agent feels pain they are motivated by it
- Modest Motivationalism Pains motivate in virtue of some property p, and pains intrinsically and necessarily have p
- Lazy Motivationalism If a typical agent in normal circumstances feels pain, they will be motivated by it

Lazy motivationalism is not threatened by asymbolia. LC tells us that asymbolics are not typical agents in typical circumstances. So there is at least one way of understanding motivationalism on which it is compatible with asymbolia. That is an unsatisfying victory. I suspect that few have been tempted to deny lazy motivationalism, and fewer still for good reasons. Grahek's position, note, is entirely compatible with lazy motivationalism. Lazy motivationalism is too weak to capture a real debate. Most importantly, while lazy motivationalism might be descriptively accurate, it sheds very little light on pain itself: in particular, it says nothing about why normal circumstances and typical agency matter. Let us put it aside, and try for something stronger.

Ambitious motivationalism *is* philosophically interesting. Grahek denies it, and it does place some strong constraints on our theories of pain. It is arguably the most intuitive way of cashing out the motivationalist thesis. But Asymbolia is also clearly a counterexample, even if we accept LC. LC does not deny that asymbolics feel pain, nor that they are unmotivated by it. Ambitious motivationalism says that is impossible. So ambitious motivationalism is false.

That leaves only the carefully hedged modest motivationalism. Like ambitious motivationalism, the modest variety is incompatible with Grahek's view, so it is *prima facie* philosophically interesting. Unlike lazy motivationalism it makes a strong claim about the nature of pain: however pains motivate, they always have the property in virtue of which they do so.

Modest motivationalism, however, does not claim that pains *always* motivate: just that they always have the property in virtue of which they motivate. The key claim is that motivation is a two-place *relation* between a sensation and an agent: my pains motivate *me*. Modest motivationalism says that this relationship can fail to hold. If it does, however, it fails in virtue of a change in the *agent*, not because of a change in pain itself. An analogy. Both lit matches and chlorine trifluoride are ignition sources: they have the power to start fires. Chlorine triflouride will start fires (nearly) anywhere and on anything.¹⁰ Lit matches, by contrast, start fires only if certain background conditions are in place: there must be oxygen and dry tinder, the air cannot be too humid, and so on. Given these conditions, and a lit match, a fire will start. We are happy to attribute to lit matches the property of being an ignition source despite this. This is because matches have the right sort of intrinsic property that causes fires to start; that distinguishes them from other things (bricks, donuts, puppies) that do not. If a struck match*fails* to ignite, we usually blame conditions, not the match.

Ambitious motivationalism views pains as a bit like chlorine trifluoride: they light the fires of action come what may. Modest motivationalism, in contrast, says that pains are like matches. They always have an intrinsic power to motivate, but that power manifests only if circumstances are appropriate.

Of course, modest motivationalism runs the risk of collapsing back into lazy motivationalism: without saying more about the necessary background conditions, modest motivationalism does no more than dig a dark hole in which counterexamples can be hidden. This is where the LC model of asymbolia comes in handy. The LC model suggests that the relevant background condition is the capacity to care about the fate of your body. This is a substantial empirical and philosophical claim. First, it claims that there is a unified capacity for caring about your body in the right way. The care we have for our bodily integrity is not just caring about pains, and *also* caring about sudden noises, and *also* acting appropriately when you believe you are being injured, and so on. All of these more particular states are manifestations of a single general capacity, and so must stand and fall together. That in turn has empirical consequences. Grahek claimed that pain asymbolia was a specific dissociation between pain and motivation. It is not. If LC plus modest motivationalism is true, there cannot be any such specific, simple dissociation. Instead, any agent who is indifferent to felt pain should be as asymbolics actually are: possessed of a collection of deficits that manifest in many different but related ways. That in turn makes strong, falsifiable empirical predictions.¹¹

¹⁰More precisely, chlorine trifluoride is hypergolic and an extremely strong oxidiser, and so will start fires in the absence of oxygen and in materials not normally thought of as flammable—sand, concrete, asbestos, water, and so on (Clark 1972, p. 73).

¹¹For the connoisseur of the neuropsychology literature, an aside. One might object that the above story is built on evidence from association of deficits. Shallice (1988) has argued that associated deficits are a weak foundation for neuropsychological inference (p. 32ff, p. 226ff). Two points are worth noting. First, Shallice's argument is strongest against syndromes posited on the basis of probabilistic generalisation over groups of patients,

4 Motivation and command

Modest motivationalism is thus compatible with pain asymbolia. Maintaining modest motivationalism requires, however, that pain have some intrinsic property p that motivates under ordinary circumstances and that persists, without motivating, in cases of asymbolia. The argument I gave was deliberately abstract, and gives different types of motivationalism an opportunity to slot in their preferred property for p. Modest Motivationalism ultimately depends on whether there is some appropriate property, and many otherwise plausible candidates do not fit the bill. Property p arguably cannot be the property of *representing* facts about damage (or other bodily states). The property of representing bodily damage does not seem to have the correct direction of fit to be motivating *per se*: if I come to learn that I have been damaged through some other route, I may or may not be motivated to do anything about it. Nor does the property of being a desire, emotion, or other affective state seem to be a candidate for p. Asymbolics feel pain, but do not appear to have any of the ordinary affective states associated with it. So even if typical pains *also* motivate in virtue of some associated affective states, asymbolics can recognise sensations as pains without those states. They are thus not candidates for an *intrinsic* properties in virtue of which pain motivates. These objections are not decisive, and the clever motivationalist may find ways around them. A motivationalist could also treat p as some sui generis property with just the features required, though I suspect such a move will be philosophically unsatisfying.

There is one candidate for p, however, that I think is both philosophically satisfying and compatible with motivationalism. This is to treat pains as *imperatives*: that is, as states which have commands as their intrinsic intentional content. The pain of a broken ankle on such an account is an imperative with a content like 'Protect your ankle by keeping weight off of it!' In ordinary circumstances one is moved by the command, and so has a reason to take only those actions that do not involve putting weight on your ankle. Different pains command different sorts of protective action, and the biological function of pain in general is to motivate appropriate actions towards actual or potential injury.

which is not at issue here. Instead, the prediction is that distinct tests of the *same* construct will show similar patterns of impairment: that is, there is only one psychological deficit that manifests itself in various ways on various tests. Second, Shallice argues that associations of deficits are evidentially shaky, as they can always be overturned by dissociations observed in the future. That is true, but also a virtue of the present account: it is empirically riskier, and so easier to falsify.

I have defended such a view elsewhere on independent grounds.¹² I argue that the property of possessing imperative content is also a viable candidate for property p.

First, imperative content would explain why and how pains are motivating in ordinary cases. The purpose of issuing a imperative is to motivate actions. Commands thus have a direction of fit more like desires than beliefs: they have satisfaction conditions rather than truth conditions (Hamblin 1987). Further, in cases where we accept the issuer as authoritative, a command motivates directly and without further deliberation. The willing private who is ordered to do pushups by his drill sergeant is thereby motivated to do so. That motivation comes quite independently of the private's antecedent desires for pushups. In terms popularised in political philosophy by Hart (1982), authoritative commands give *content-independent* reasons for action. Those reasons might ultimately be overridden by other reasons, of course, and we often override pain for the sake of some further end.¹³ Note, however, that even when we override pain we are still motivated by it: the intrinsic motivating force is always present.

Identifying p with the right kind of imperative content also clarifies how motivation might break down in cases like Asymbolia. Being motivated by a command requires accepting its source as an authority. That is usually done for some reason or other. Once one *has* accepted a source as an authority, those reasons need not enter into further deliberation. But if the reason for accepting an authority breaks down, commands from that authority will cease to be motivating without a change in their content.¹⁴ In the case of pains, the issuing authority is the (phenomenal) body. The reason why we accept commands is because we care about our bodily integrity. In treating our body as an authority, we accept that it might sometimes make mistakes.

 $^{^{12}}$ In Klein 2007 and 2012. Note that there are several possibilities for cashing out imperative content, and the version I sketch here differs from my previous attempts in focusing on commands to protect, rather than negative commands to cease movement. I used to think that negative commands were necessary to deal with cases like asymbolia; the present account provides what I now think is a more satisfying solution.

¹³The parallel with the political is thus complicated by the fact that pains do not appear to give *exclusionary* reasons in Raz's (1986) sense. A full story would require delineating more precisely the domains over which the body is taken to be authoritative, and the conditions under which pains are taken to give practical rather than merely epistemic reasons for action. Settling these issues is inessential for the present story.

¹⁴Assuming, plausibly, that the content of a command does not contain anything about the sources of of its legitimacy. We are sometimes required to make the authority of a command explicit—'I'm your father, that's why!'—which would be odd if commands carried such information intrinsically. On general considerations against the reduction of commands to threats or other legitimacy-carrying representations, see Parsons 2012.

Indeed, the non-deliberative nature of motivation by pain implies that we will continue to be motivated by bodily commands even when we *know* that a mistake has been made (as in the case of phantom limbs). However, if we cease to care about our bodily integrity entirely, then the authority of the body would be undermined. Pains would cease to motivate. That failure, however, would occur without any change in the imperative content of pain.

This, I suggest, is what has happened in cases of pain asymbolia. The asymbolic recognises pains, because their ordinary imperative content has not changed. However, they have ceased to treat such bodily commands as binding, and so have ceased to be motivated by them. The situation of the asymbolic is thus a bit like the unperceptive man who hears a police officer shout 'Stop or I'll shoot!' He can recognise the utterance as a command, and think that whoever it is addressed to has a very good reason to stop—all without realising that he has a good reason to stop.

The resulting picture is a form of modest motivationalism: it says that pains possess an intrinsic property (imperative content) in virtue of which they ordinarily motivate, while admitting that pains may in fact fail to motivate given suitable changes to the *agent*.

5 Asymbolia and Depersonalisation

A full defence of imperativism about pain is beyond the scope of this paper. Nevertheless, there is at least one viable candidate for p that would preserve modest motivationalism. Further, I think this story might shed some light on the phenomenology of asymbolia and related phenomena. I conclude by discussing briefly, then, what it might be like to *be* asymbolic.

Asymbolics lack the capacity to care about bodily integrity. That does not need to manifest as an occurrent belief about lack of care. Instead, it may manifest itself more as a type of *indifference*. One's body becomes, as it were, just another object in the world. An odd object, perhaps, that still commands you to care for it—but not an object that you have any deeper reason to care for than anything else around you.

If this is right, the phenomenology of asymbolia might resembles a kind of *depersonalisation syndrome*. The DSM IV defines depersonalisation as 'a feeling of detachment or estrangement from one's self', and notes that

The individual may feel like an automaton or as if he or she is living in a dream or a movie. There may be a sensation of being an outside observer of one's mental processes, one's body, or parts of one's body. Various types of sensory anaesthesia, lack of affective response, and a sensation of lacking control of one's actions, including speech, are often present. (APA 2000, Sect. 300.6)

Perhaps, then, the asymbolic's experience of pain is an experience of a certain kind of detachment from that pain. They recognise it as pain, but in some important sense it has ceased to be something worth caring about. It thus has the feel of a sensation which they can no longer identify with as their own.¹⁵

That damage to the insula might produce depersonalisation is not surprising. There is a growing consensus that the insula plays a complex and active role in maintaining representations of the body, especially facts about homeostatic needs. One crucial function it plays seems to be in supporting what Craig (2002) calls *interoception*: that is, awareness and reflection on the state of one's body. Damage to the insula can produce a variety of deficits of bodily self-awareness (Ibañez et al. 2010). As Karnath and Baier note (2010, p. 414ff), this can include asomatognosia (the feeling that a patient's limbs do not belong to them) or somatoparaphrenia (the feeling that a patient's limbs belong to someone else). Damage to the insula thus seems to interfere with identification of sensations as our own.

Further, the feelings of disengagement typical of depersonalisation can extend to the sensation of pain. Mauricio Sierra (2009, p. 150–1) notes the similarities between asymbolics and the utterances of patients with depersonalisation disorder.¹⁶ One such patient discussed by Sierra remarked that while he felt pain, '... it is as if I don't care, as if it was somebody else's pain' (p. 49). Another patient, upon being pricked with a pin said that the sensation was 'as if it were being done to another person' (p. 150). Depersonalisation is also a symptom of other psychiatric diseases, including schizophrenia. Some schizophrenics are indifferent to pain, sometimes to the point of self-mutilation. Many authors assume that this phenomenon is due to simple insensitivity to pain (including Grahek; see p. 124ff.) However, re-

¹⁵This might also explain why many asymbolics appear amused or befuddled by their pains. Ramachandran (1998), noting frequent reports where asymbolics laugh in the face of pain, argues that they recognise the incongruity between typical responses and their own. In support of this explanation, other asymbolics seem to feel the need to rationalise their responses. An asymbolic described in Hemphill and Stengel 1940 rationalised his absence of reaction to pain by saying 'I am used to that because I have worked on the road' and 'Labourers are always hurting themselves; we don't take any notice of it' (p. 256). All of these appear to be reasonable responses to an unusual depersonalisation experience—for asymbolics can certainly remember that they *used* to be motivated by their pains, and that most reasonable people are, and be struck by this incongruity without being able to explain it.

¹⁶Relevant to the present discussion, Sierra (2009, pp. 10–11) also notes that purely sensory theories of dependence of the long since fallen out of favour.

cent reviews of the literature have noted that schizophrenics appear to have the same pain threshold as normal subjects, and that this effect is present even in unmedicated schizophrenics (Singh et al. 2006; Bonnot et al. 2009). Guieu et al. thus argue that, for schizophrenics, 'the term of "indifference" to pain may be more appropriate than "insensibility" to pain' (Guieu et al. 1994, p. 255). Finally, and perhaps most intriguingly, Wylie and Tregellas (2010, p. 98) have recently noted consistent evidence that abnormalities in the insula are often associated with schizophrenic depersonalisation symptoms, and suggest that the phenomenon may be understood as parallel to pain asymbolia.

Treating asymbolia as a species of depersonalisation disorder is thus an intriguing possibility. For one, it means that asymbolia is not a *sui generis* deficit. It is instead a specific and severe form of a more common disorder, and one that those interested in pain might study more readily. That is handy: asymbolics themselves are rare and difficult to study.¹⁷ In turn, we might find analogues of depersonalised pain in even more prosaic situations, including those that have long intrigued philosophers writing on pain.

One such case, mentioned in section 1, is that of morphine pain. Patients given an acute dose of morphine often say that they are indifferent to their pain. Morphine can produce powerful feelings of depersonalisation. Conversely, patients with depersonalisation disorder have compared it to the effect of morphine.¹⁸ We might thus understand morphine pain as a variety of drug-induced depersonalisation: patients are indifferent to pain not because the pain has changed, but because they no longer appreciate it as a command worth following.

Finally, treating asymbolia as a species of depersonalisation might be relevant to current debates about the unity of consciousness. The asymbolic, and the depersonalised more generally, feels sensations that they are estranged from—that they do not take to be *theirs* in the sense that we normally do. This may not threaten some forms of the unity of consciousness thesis: there is another important sense in which the pain is their sensation whether they realise it or not.¹⁹ However, it does show that there is another sense in which

¹⁷Asymbolics often have severe language deficits. That is probably a neurological accident: the insula is located near important language centres, and the lesions that produce asymbolia are usually large.

¹⁸See Noyes and Kletti's patient who remarked 'I would compare it to a morphine "high"; I once had morphine after an operation' (Noyes and Kletti 1977, p. 378).

¹⁹For example, it does not threaten something like what Bayne (2010) calls Phenomenal Unity or what Rosenthal (2005) dubs the Thin Immunity Principle (though for worries about the latter in the related phenomenon of somatoparaphrenia, see Liang and Lane 2009).

our sensations may be unified: as sensations over which we have a feeling of ownership. Asymbolia, and depersonalisation more generally, shows that this sort of unity may fail. Its failure comes not from a change in the sensations we feel, but in the sort of agents we are. These syndromes show that failures of this kind of unity are not just real, but have grave consequences.²⁰

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