

# What Pain Asymbolia Really Shows\*

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## 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 is partly empirical. 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, Nikolai Grahek presents an apparent counterexample to motivationalism [Grahek, 2007]. 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 behavior 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

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away or withdraws with the body as a whole. She never attempts to avoid the investigator. ([Schilder and Stengel, 1928] 147)<sup>1</sup>

Strange enough. But not only do pain asymbolics fail to react to painful stimuli, they also appear to recognize the stimuli *as pains*. Schilder and Stengel go on to describe:

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. Pricked on the soles of the feet, she begins to smile, openly titillated.<sup>2</sup> (ibid)

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 ([Schilder and Stengel, 1928] 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 don’t 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,

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<sup>1</sup>Quotations from Schilder and Stengel 1928 are translated from the original German. Special thanks to Aleks Zarnitsyn and Mae Liou for help with the translations.

<sup>2</sup>The incongruous reaction of laughter is also striking, and puzzling. Ramachandran gives an intriguing explanation, arguing that it comes from the asymbolics’ perceived incongruity between typical responses and their own [Ramachandran, 1998]. In support of this explanation, other asymbolics seem to feel the need to rationalize their responses. An asymbolic described by Hemphill and Stengel rationalized 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” ([Hemphill and Stengel, 1940] 256).

there seems to be no overriding motivation that we could appeal to to explain asymbolics' lack of response. Lobotomized patients often appear curiously indifferent to previously intractable pain. But as Melzack and Wall point out, these patients still withdraw from pinprick, avoid walking on broken ankles, and generally react as we react to pain ([Melzack and Wall, 1982] 131). The pain of the lobotomized thus retains its biologically basic motivational force; what has gone missing are the other emotions usually associated with strong pains.<sup>3</sup> 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, don't even protect their bodies when they encounter painful stimuli. 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'll suggest that the difference is more than superficial). We usually take the first-person reports of people 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 lesioned patients, and neurology permits detailed tests of their responses.

It is worth emphasizing just how strangely asymbolics behave, even outside of laboratory settings. Schilder and Stengel's patient would readily stab herself with needles and jam objects into her eyelid ([Schilder and Stengel, 1931] 598). Berthier et al. report an asymbolic patient who suffered a severe burn at home, but who made no attempt to escape the injurious stimulus ([Berthier et al., 1988] 43). 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

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<sup>3</sup>Brand and Yancy note that "Patients report feeling 'the little pain without the big pain'" (quoted in [Grahek, 2007] p31), suggesting that what is missing is secondary negative reactions to pain, not its core motivational import.

his head, nor did he show any instinctive combative reaction.  
([Hemphill and Stengel, 1940] 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'll argue that Grahek has misinterpreted pain asymbolia. Grahek treats asymbolia as a deficit of sensation. I'll 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'll then argue that this capacity-based view is compatible with a weak form of motivationalism.

I have left two aspects of motivationalism purposefully general, and one temporarily vague. First, I haven't 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, Nelkin, 1994, Helm, 2002]. It also includes positions 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. I will not take a stance here about the correct position. I intend my defense to be available, in principle, to any of these positions.

I will assume, however, that motivationalism is a *personal*-level thesis: that is, it makes a claim about whole persons and their relationship to the pain that they feel. Sub-personal stories about the brain can serve, at best, to make plausible a personal-level interpretation of asymbolics and their behavior. My disagreement with Grahek can be adjudicated without recourse to stories about the brain (though see footnote 8 for a brief discussion).

Second, I haven't said *what* pain motivates. That's 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 fulfillment of that role. In normal cases pain

motivates actions that help keep our bodies healthy and intact [Wall, 2000]. So, for example, pains at least motivate us to avoid injury, to nurse wounds, to favor wrenched joints, and so on. Asymbolics don't 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 defense of motivationalism will, if successful, restrict and sharpen motivationalism. For now, however, the intuitive idea—that pains are intrinsically motivating—will be enough to get us started.

## 2 Two Models of Asymbolia

### 2.1 The Degraded Sensation Model

Here is one model of what's 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.<sup>4</sup> Grahek also endorses it. As he puts it,

...pain, although appearing to us as simple, homogenous experience, is actually a complex experience comprising sensory-discriminative, emotional-cognitive and behavioral components that commonly go together, but may well be disconnected and thus exist, to our great astonishment, separately. ([Grahek, 2007] 7).

When the components of pain come apart, strange syndromes result. Asymbolia is a paradigm case. The pain of asymbolics, Grahek argues, has lost

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<sup>4</sup>See for example [Dennett, 1985, Hardcastle, 1997, Price, 2000].

the affective/motivational component. As such, “[Pain] becomes a blunt, inert sensory appearance with no power to galvanize the mind and body for flight or fight, and doesn’t serve anymore its primary biological function.” ([Grahek, 2007] 66)

Call this the *degraded sensation* (DS) model of asymbolia. DS claims that asymbolics have a sensory deficit: 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 DS model is incompatible with motivationalism. According to DS, the motivational force of pains comes from their affective/motivational component. That component can go missing, but the sensation remains a pain.<sup>5</sup> So motivationalism is false.

Grahek argues that there is a double dissociation between the sensory and affective aspects of pain.<sup>6</sup> 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 by Ploner, Freund, and Schnitzler of a patient with a unilateral lesion to SI and SII. Laser stimuli 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” [Ploner et al., 1999]. Grahek takes this as a case of pain affect preserved in the absence of pain sensation. We thus appear to have a classical double dissociation between pain affect and pain sensation. Double dissociation between two mental processes is usually taken as evidence that they are only contingently related (even if they typically occur together). So the composite view of pain falls

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<sup>5</sup>Note that not all composite theories are incompatible with motivationalism. One could argue on conceptual grounds 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. Grahek, drawing on Hardcastle, forswears this sort of conceptual jiggery-pokery ([Grahek, 2007] 84); I’m happy to follow his lead. More prosaically, however, one could think that as an empirical matter both portions are necessary for something to be called pain. Armstrong argues, for example, that pains are a combination of a tactile sensation plus an extreme dislike of that sensation ([Armstrong, 1962] 106ff). If the dislike went missing, one would just have a tactile sensation, not pain. Asymbolia is, presumably, meant to be a counterexample to this variety of composite view.

<sup>6</sup>Grahek never explicitly uses the language of double dissociation, but his argument is most charitably interpreted as doing so. For a more explicit version of the same argument, see [Hardcastle, 1997]

out directly, and DS appears to be well-motivated.

## 2.2 The Lost Capacity Model

DS is not the only way to understand asymbolia. Here is another model: Asymbolics don't 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 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 DS predict that asymbolics will be unmoved by pain. They differ, however, on the explanation of that fact. DS says that something has changed about the sensation of 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* threat to their bodily integrity.<sup>7</sup> 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 behavior 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.<sup>8</sup>

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<sup>7</sup>A complication: If 'threat to bodily integrity' is understood so broadly as to include the threats that come from failure to eat or urinate, then LC is empirically false. Schilder and Stengel's patient, for example, asked normally to eat and use the bathroom ([Schilder and Stengel, 1928] 150). I will use 'threat to bodily integrity' to mean immediate threats to the physical body. That lack of appreciation of such threats is possible while still appreciating other sorts of threats is ultimately an empirical matter, and one I think that has been secured by asymbolia.

<sup>8</sup> While both DS and LC are personal-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]). Drawing on a proposal first put forth by Geschwind, Grahek argues that pain represents a *sensory-limbic disconnection syndrome* [Geschwind, 1965]. On his view, damage to the insula in asymbolics prevents limbic processing from being

A final difference. DS 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’s nearly impossible, for good biological reasons—then pains wouldn’t matter. Asymbolics are a realization of this abstract possibility.

### 2.3 Evidence for a Lost Capacity

Both LC and DS predict the pain-related deficits of asymbolics. LC, however, predicts that there should be a general loss of appreciation for threats to bodily integrity. DS 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 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 ([Hemphill and Stengel, 1940] 256). Schilder and Stengel report 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. ([Schilder and Stengel, 1928] 149).

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incorporated with sensory processing ([Grahek, 2007] 52). DS is motivated by this picture: there are two processing streams in normal folks, one of which has become a dead-end in asymbolics.

Geschwind’s model of disconnection syndromes has been criticized for assuming an entirely serial, feed-forward picture of the brain [Catani et al., 2005]. On his view, each brain region performs a specialized function and passes on the result to higher association centers, which in turn pass on their results to still further association centers, and so on. Earlier processes in the causal chain are entirely unaffected by later ones. DS 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 favor. The insula projects back to the limbic system, and receives input from a variety of frontal areas [Singer et al., 2009]. In short, the insula seems to do more than simply composite together the results of earlier sensory processing stages. Rather, 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.

Asymbolic' indifference is not limited to simple sensations. Berthier et al. report that five of their six patients failed to respond to "verbal menaces" ([Berthier et al., 1988] 43). Schilder and Stengel note that their patient "shows no appreciation at all for threats of pain *or for any threats in general*" ([Schilder and Stengel, 1928] 154, 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 recognized 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. ([Hemphill and Stengel, 1940] 256).

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

What about DS? Grahek mentions these phenomena. He suggests that the relevant deficit is plurimodal, and does not discuss the issue further ([Grahek, 2007] 45). 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 DS might still be true; however, 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] 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.

Either reading faces a further difficulty. A thought experiment: Suppose I anesthetized your arm, and placed it out of sight. Then, suppose I told you that I was pummeling 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're having: your arm is insensate and occluded. Instead, you'd 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're 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 aren't 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's 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.<sup>9</sup> From birth, the congenitally insensitive don't feel any pains at all. So, *a fortiori*, they don't have sensations with whatever affective/motivational component Grahek thinks is critical for pain behavior. Yet they still learn to protect their

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<sup>9</sup>Grahek, unlike many authors, correctly distinguishes asymbolia from congenital insensitivity; see ([Grahek, 2007] 87ff).

bodies as best they can. That is, they learn what situations are injurious, and avoid these situations precisely because they don't want to be injured.<sup>10</sup>

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'd 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 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*.

We may put the same point 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 DS, 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 aren't. 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" ([Grahek, 2007] 66). But that would make the 'sensory-discriminative' aspect of pain unlike any other sensations we're familiar with. The sensation of seeing-blue doesn't 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

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<sup>10</sup>This care only goes so far—pain is still the most useful 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 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 [Nash, 2005]. 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] 18-19).

epiphenomenal. It's there. We can make verbal reports about it. That's the only causal consequence it seems to have for our behavior. That's deeply odd.

In contrast, LC gives a perfectly straightforward story about asymbolics' general lack of concern. Asymbolics don't care about the integrity of their bodies because they can't. 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 jeopardized.

To conclude, there are two ways in which DS 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.<sup>11</sup> 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'd 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, DS 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 DS needs complex or *ad hoc* hypotheses to account for them. Other phenomena are explained by LC, but not by DS. So on balance, we have reason to prefer LC.

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<sup>11</sup>The other half of the dissociation—painfulness without pain—is less relevant to the question of motivationalism. I'm also suspicious of it. First, the patient described by Ploner, Freund, and Schnitzler *did* appear to feel pain in the hand contralateral to his lesion, albeit with a much higher threshold (see [Ploner et al., 1999] figure 2). Further, their patient described the sensation he was feeling as 'unpleasant' before he felt pain. 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 of tooth pulp stimulation under the influence of fentanyl ([Hardcastle, 1997] 393).

### 3 Motivationalism defended

Let's 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 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 aren't typical agents in typical circumstances. So there's at least one way of understanding motivationalism on which it is compatible with asymbolia. That is pretty unsatisfying, though. I suspect that few have been tempted to deny lazy motivationalism, and fewer still for good reasons. Grahek's position is also entirely compatible with lazy motivationalism. There is a real debate here, though, and lazy motivationalism seems to be too weak to capture it. 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's 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's 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's *prima facie* philosophically interesting. Further, 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.

Modest motivationalism, however, doesn't claim that pains *always* motivate: just that they always have the property in virtue of which they motivate when they do. The key insight 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 trifluoride will start fires (nearly) anywhere and on anything.<sup>12</sup> Lit matches, by contrast, start fires only if certain background conditions are in place: there must be oxygen and dry tinder, the air can't 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 the need for background conditions. 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 don't. Put another way, if lit matches *don't* start fires, we explain this fact by blaming conditions, not the match.

Ambitious motivationalism views pains as a bit like chlorine trifluoride: they

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<sup>12</sup>More precisely, chlorine trifluoride is hypergolic and an extremely strong oxidizer, and so will start fires in the absence of oxygen and in materials not normally thought of as flammable—concrete, asbestos, water, and so on.

light the fires of action come what may. Modest motivationalism, in contrast, says that pains are like matches. They do 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 leave a big hole into which counterexamples can be tossed. This is where the LC model of asymbolia comes in handy. The LC model suggests that the relevant background condition for pains to motivate 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 isn't just caring about pains, and *also* caring about sudden noises, *and* also acting appropriately when you believe you're 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's 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 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.<sup>13</sup>

One might object that modest motivationalism doesn't really capture the motivationalist intuition. I'm not inclined to argue about who has had which intuitions. But, I submit, motivationalism isn't interesting *just* because it puts some philosophical clothes on a bare intuition. Motivationalism is in-

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<sup>13</sup>For the connoisseur of the neuropsychology literature, an aside. One might object that the above story is built on evidence from association of deficits. In his influential work, Shallice has argued that associated deficits are a weak foundation for neuropsychological inference ([Shallice, 1988] p32ff, 226ff). Two points are worth noting. First, Shallice's argument is strongest against syndromes posited on the basis of probabilistic generalization over groups of patients, 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 which 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.

interesting and worth debating precisely because of what we can get out of it. One thing—probably the primary thing—that motivationalism offers is insight into the nature of pain itself. Modest motivationalism gives this. It rules out some theories of pain. Composite views of pain like Grahek’s are the most obvious example. However, it also rules out models on which pains have a direct and inevitable motivational force. That includes views on which pains are primitive motivational states, or can be reduced to states (desires, judgments, etc) that are themselves taken to be directly motivating. Again, modest motivationalism says that ‘motivates’ is a relational predicate, and that the details of the agent side of the relation matter as much as the pain side. I’ve left the account general enough to accommodate many ways of cashing out this relation. But if you want to understand how pain motivates, you also need a story about the agent being motivated.

## 4 Asymbolia as Depersonalization

A brief recap. Asymbolics are indifferent to pain. I considered Grahek’s model of this indifference, on which asymbolics had deficient sensory input. I found this model lacking: asymbolics don’t behave like people who are merely indifferent to sensory pain. I instead posited that asymbolics lacked a fundamental capacity to care about the integrity of their body, and showed that the evidence best supported this model. Finally, I concluded by showing that this model of asymbolia is compatible with a modest version of motivationalism. The modest motivationalist can maintain that pains might fail to motivate, but only under situations where the agent is severely impaired. That in turn has consequences for the right theory of pain.

Here is a looser, more suggestive way of putting the same argument. Motivationalism says that pains always motivate. That doesn’t mean that all pains motivate everyone, of course. It means that *my* pains motivate *me*, your pains motivate you, and so on. The asymbolic feels pain. But it has ceased to be, in some important sense, *their* pain. They can no longer link pains (or any threat) to anything that they care about. That is why the pains of the asymbolic appear inert and blunt: they are sensations that no longer have a proper owner, and so there is no one who can be motivated by them. The

situation of the asymbolic is a bit like the unperceptive man who hears the police officer shout “Stop or I’ll shoot!” He can recognize the utterance as a command, and think that whoever it is addressed to has a very good reason to stop, all without realizing that it is *he* who should stop.

Put this way, asymbolia isn’t as odd as it might appear. It can be understood as a variety of *depersonalization syndrome*. The DSM IV defines depersonalization 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 anesthesia, lack of affective response, and a sensation of lacking control of one’s actions, including speech, are often present ([APA, 2000] §300.6).

The DSM also notes that fleeting depersonalization is a relatively common experience; depersonalization disorder proper is diagnosed when these feelings become severe and persistent.

That damage to the insula might produce depersonalization 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 calls *interoception*: that is, awareness and reflection on the state of one’s body [Craig, 2002]. Damage to the insula can produce a variety of deficits of bodily self-awareness [Ibañez et al., 2010]. As Karnath and Baier note, 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) ([Karnath and Baier, 2010] 414ff). In short, the insula seems to play a crucial role in identifying bodily sensations as our own.

Further, the feelings of disengagement typical of depersonalization can extend to the sensation of pain. In a recent book, Mauricio Sierra notes the similarities between asymbolics and the utterances of patients with de-

personalization disorder ([Sierra, 2009] 150-151).<sup>14</sup> One such patient remarked that while he felt pain, "...it is as if I don't care, as if it was somebody else's pain." ([Sierra, 2009] 49). Another patient, upon being pricked with a pin said that the sensation was "as if it were being done to another person" ([Sierra, 2009] 150). Depersonalization is also a symptom of other psychiatric diseases, including schizophrenia. Schizophrenics have long been noted to be indifferent to pain, sometimes to the point of self-mutilation. Many authors have assumed that this phenomena shows that some schizophrenics are simply insensitive to pain.<sup>15</sup> The roots of this phenomena are likely to be complex. However, recent 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 inappropriate than 'insensibility' to pain" ([Guieu et al., 1994] 255). Finally, and perhaps most intriguingly, Wylie and Tregellas have recently noted consistent evidence that abnormalities in the insula are often associated with schizophrenic depersonalization symptoms, and suggest that the phenomena may be understood as parallel to pain asymbolia ([Wylie and Tregellas, 2010] 98).

Treating asymbolia as a species of depersonalization 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's handy: asymbolics themselves are rare and difficult to study.<sup>16</sup> In turn, we might find analogues of depersonalized 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 depersonalization. Con-

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<sup>14</sup>Relevant to the present discussion, Sierra also notes that purely sensory theories of depersonalization have long since fallen out of favor ([Sierra, 2009] 10-11).

<sup>15</sup>Including Grahek; see ([Grahek, 2007] 107ff).

<sup>16</sup>Asymbolics often have severe language deficits. That is probably a neurological accident: the insula is located near important language centers, and the lesions that produce asymbolia are usually large.

versely, patients with depersonalization disorder have compared it to the effect of morphine.<sup>17</sup> We might thus understand morphine pain as a variety of drug-induced depersonalization: patients are indifferent to pain not because the pain has changed, but because they no longer care about what it might signify.<sup>18</sup>

Finally, the depersonalization account of asymbolia might be relevant to current debates about the unity of consciousness. The asymbolic, and the depersonalized more generally, feels sensations that they do not take to be *theirs*. 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 realize it or not.<sup>19</sup> However, it does show that there is another sense in which our sensations may be unified: as sensations over which we have a feeling of ownership. Asymbolia, and depersonalization 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.<sup>20</sup>

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<sup>17</sup>See for example Noyes and Kletti's patient who remarked "I would compare it to a morphine high; I once had morphine after an operation" ([Noyes et al., 1977] 378).

<sup>18</sup>As someone who has actually experienced morphine pain, I can testify to the accuracy of this account. I received an injection of morphine after a bad fracture of my ankle. The pain persisted for a bit. I didn't care about it. Or, more precisely, I felt pain, but I couldn't really see what it had to do with *me*—there was pain in my foot, but what was going on all the way down there didn't seem especially relevant. Of course, this is a self-serving, 20 year old anecdote about a drug experience; the reader may want to take it with a grain of salt.

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

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