Pain Asymbolia Is Probably Still Pain

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Abstract

Trevor Griffith and Adrian Kind argue that we should reject a standard interpretation of pain asymbolia, according to which asymbolics experience pain even though their pain lacks the affective-motivational element that typical pains possess. We make the case that Griffith and Kind's reasons for rejecting the standard interpretation are relatively weak. We end by arguing that debates between the standard interpretation and alternative interpretations cannot be resolved without addressing the issue of how we should taxonomize pain asymbolia as a neurological condition.

1. Introduction

Grahek (2007)'s *Feeling Pain and Being in Pain* introduced philosophers to so-called *pain asymbolia*. A rare consequence of brain lesions, usually in the region of the insular cortex (Bethier, Starkstein, and Leiguarda 1998), asymbolics appear to feel pains without being moved by them. As Grahek put it, asymbolia seems to be the only straightforward case "in which severe pain is not experienced as unpleasant at all, and in which there are no traces of any other aversive attitude toward it" (Grahek 2007, 38). Grahek also suggested that this might an example of "pure pain" (Grahek 2007, 37). Much of the subsequent philosophical interest in the literature has been around how to interpret reports of asymbolia, and what this shows about pain.

Griffith and Kind (2024) — henceforth 'G&K' — have recently argued that this entire literature rests on a shaky foundation.[1] The main goal of their paper is to reject what they call *the standard interpretation* of pain asymbolia, the origin of which they trace back to Grahek's work.

[1] For similar recent critiques, see Coninx (2020) and Park (2023).

The standard interpretation holds that "asymbolics who undergo noxious stimuli have a real and genuine pain experience, which nevertheless lacks some affective and motivational components of normal instantiations of pain" (G&K, 561). This is used in Grahek's work to defend the view that pain is ultimately a complex mental state comprising a separable sensory-discriminative component (the *what-it-is-like* to feel pain) and an affective-motivational element (which motivates us to take action). Grahek cites case reports of asymbolia as strong evidence for the view that the sensory-discriminative component can occur independently from the affective-motivational component.

Though we happen to favor a distinct interpretation of what is going on with asymbolic subjects,[2] our goal here is to claim that the reasons provided by G&K for rejecting the standard interpretation are relatively weak and that G&K have failed to capture some larger methodological issues in debates about pain asymbolia. To do so, we respond to two core moves that G&K make to reject the standard interpretation. We end by arguing that debates between the standard interpretation and alternative interpretations cannot be resolved without addressing the issue of how we should *taxonomize* pain asymbolia as a neurological condition.

2. Griffith and Kind's Mereological Argument

One of the core moves that G&K make is to argue that the standard interpretation commits a *mereological fallacy*. At the heart of their argument is the claim that the standard interpretation entails three inconsistent theses: "1) pain is complex, 2) PA [i.e., pain asymbolia] is not complex [because it only includes the sensory-discriminative element, not the affective-motivational component], 3) PA is pain" (G&K, 569). They add:

> But of course pain cannot be both simple and complex in the same way at the same time; if pain is *essentially* complex […], then its simples, including the phenomenal or sensory-discriminative elements as isolated in PA, cannot be pain; *either* asymbolics feel pain, or pain is complex. (G&K, 569, their emphasis)

As hinted in the second quote, and as G&K themselves explicitly emphasize (562), the argument focuses on versions of the standard interpretation that are developed in combination with

[2] See Klein (2015a; 2015b) and Klein and Duval (2023) for our own position.

essentialism about pain — i.e., the view that there are sufficient and necessary conditions for some entity being a pain.

Importantly, we think this raises a dilemma for G&K as to what the intended target of their mereological argument is really supposed to be. They claim early in their paper (562) that their official target is the combination of the standard interpretation with essentialism about pain. However, later in the paper (574), they seem to take their argument to show that we should reject the standard interpretation itself rather than the combination of the standard interpretation and essentialism about pain.[3] Hence the dilemma: either the intended target is the standard interpretation itself, or it is the combination of the standard interpretation *and* essentialism about pain. We argue that both options are problematic.

Let us start with the first horn: the target is the standard interpretation itself. The problem here is that there is a natural way of developing the standard interpretation without smuggling in an implicit commitment to essentialism and while satisfying all, or nearly all, the motivations of people like Grahek for adopting it. We maintain that proponents of the standard interpretation could do so by holding that the word 'pain' refers to a *homeostatic property cluster* (HPC) kind.

A HPC kind is a kind that is associated with a certain set of properties in virtue of homeostatic mechanisms that help entities belonging to the kind maintain the possession of these properties. For instance, Richard Boyd (1999a; 1999b) has argued that species terms (e.g., 'Panthera tigris', 'Homo sapiens') pick out HPC kinds. In support of this idea is the uncontroversial fact that there are homeostatic mechanisms that make it so that individuals of the same species have many properties in common — mechanisms like "gene exchange between certain populations and reproductive isolation from others [and] effects of common selective factors" (Boyd 1999a, 165).

A central idea about HPC kinds is that, while entities belonging to the same HPC kind typically have many properties in common, there is generally no set of properties whose possession are jointly necessary and sufficient to be a member of the kind (Boyd 1999a). For instance, even though virtually all humans have kidneys, some entity can still be a human without having kidneys (if on constant dialysis).

[3] In particular, see this passage: "So far we have argued that there are three distinct reasons for rejecting the standard interpretation. […] The second is that the interpretation *itself* is guilty of the mereological fallacy" (574, our emphasis). The term 'essentialism' doesn't occur in this passage or any of the paragraphs surrounding it.

One reason to think that 'pain' denotes a HPC kind is that there is overwhelming evidence that pain experiences and behaviors are sustained by a suite of homeostatic mechanisms, including evolutionary (Sneddon et al. 2014), genetic (Mogil and Max 2006), and developmental (Baccei and Fitzgerald 2006) ones. And, if pain is a HPC kind, proponents of the standard interpretation can consistently hold the two following statements: (i) the *nature* of pain is that it is a mental state that (typically) comprises a specific kind of sensory-discriminative component as well as a specific kind of affective-motivational component; (ii) there are genuine instances of pain that fail to have either of these components. On the resulting view, the experiences of asymbolics could still be pains even if the subjects lack the relevant affective-motivational element. Hence, on the first horn of the dilemma, proponents of the standard interpretation can avoid the charge of mereological fallacy by turning to HPC kinds.

Let us turn to the second horn: the target of the argument is the combination of the standard interpretation *and* essentialism about pain. In that case, we believe that the argument is successful: the combination has to be false. But a new problem then arises: the argument cannot support the subsequent uses that G&K make of it — namely, recommending the rejection of the standard interpretation itself, motivating a new interpretation of cases of pain asymbolia, and maintaining that we should remove pain asymbolia from the arsenal of case studies in the philosophy of pain pending more evidence. After all, if it is possible to develop the standard interpretation without incurring any commitments to essentialism (as we have just argued by appeal to the notion of HPC kind), the standard interpretation could be true even if the combination of the standard interpretation with essentialism about pain is false, because essentialism itself is false.

In sum, we think that G&K's mereological argument is either unsound or it cannot be used for the purposes that G&K have in mind for it.[4] With this, we turn to the other core move that G&K make against the standard interpretation.

3. A Taxonomic Debate

G&K's other core move is to argue that there is no unequivocal or incontestable evidence that

[4] We believe that a similar strategy can help address "the argument from the collapse into subjectivism" that G&K (section 5) raise against the standard interpretation. Space does not permit full elaboration, but briefly: there is a dilemma that parallels the one raised here about the intended target of the argument, and proponents of the standard interpretation can avoid the collapse of their view into subjectivism by appealing to HPC kinds and endorsing (i) and (ii) at the same time.

asymbolic subjects exposed to noxious stimuli actually experience pain. To defend this view, G&K make two main claims: first, pain asymbolia is relatively rare, and second, the case reports of pain asymbolia are ambiguous.

We fully agree with these two claims. G&K also make what strikes us as many valid philosophical points against Grahek's analysis of the case reports. However, G&K move too quickly from rarity and ambiguity to the conclusion that we should avoid assuming that asymbolics are experiencing pain (at least until more scientific evidence is available). We discuss rarity and ambiguity in turn.

3.1. The Rarity of Pain Asymbolia

There is no doubt that pain asymbolia is a rare condition. There are only a handful of case reports of pain asymbolia — Grahek cites six papers —, each of which discusses a small number of patients. But we want to caution against drawing very strong conclusions from this fact alone.

One of the less obvious themes of Grahek (2007) is that pain asymbolia is meant to be one half of a neuropsychological double dissociation.[5] Asymbolia gives pain without painfulness. The other half — painfulness without pain — relies on a single report from Ploner, Freund, and Schnitzler (1999). It goes by quickly (Grahek 2007, 109-11), and it has not been terribly convincing (it strikes many as a report of poorly localized pain, rather than of untethered affect).

Even if one doubts the execution, however, the *logic* of the argument is important. Double dissociation is a venerable tool in neuropsychology (Caramazza 1984; 1986; Shallice 1988). A dissociation "occurs when a patient performs extremely poorly on one task — preferably way outside the normal range — and at a normal level or at least at a very much better level on another task" (Shallice 1988, 34). *Double* dissociations — a pair of patients who have tasks that dissociate in the opposite way — are evidence for partially distinct processing pathways. It is (as G&K rightly note) a further step to say that distinct processing means that we've found 'parts' of pain. It is also an optional step (double dissociations are often taken to show evidence about parts of *subpersonal* processes, and the relationship to the personal can be complex). But the core logic of double dissociation also does not demand that further, potentially problematic step.

[5] This is not obvious in Grahek except from the fact that he often uses the word 'dissociation' and related terms. It *is* explicit and clear in Hardcastle (1997; 1999), whom Grahek cites approvingly (2007, 37, 77).

On the austere versions of the neuropsychological approach, even a single rigorously demonstrated case is enough to establish a dissociation (Shallice 1988, Ch 2.4, Ch 10). Conversely, it is far more complex to draw specific conclusions based on *associations* between deficits, such as the comorbidity emphasized by G&K between pain asymbolia and aphasia. Lesions rarely respect functional boundaries, and there are known cases where postulated syndromes depended merely on anatomical proximity or shared vasculature. That is why dissociations carry more epistemic weight.

Now, it's always a bit tricky to find dissociations in the sorts of clinical reports that capture the philosophical imagination. But asymbolia is no worse off — certainly no more rare — than many other dissociations discussed in the philosophical literature. The fact of associated deficits like aphasias complicates things, but even within this small literature there are reports of pain asymbolia without aphasia (as G&K concede).

It's also worth mentioning that there are other interpretations of the case reports of pain asymbolia that completely avoid the neuropsychological approach while holding that asymbolics experience pain. Indeed, we suggest that Grahek's use of this approach is neither necessary nor even typical. The standard example of pain without painfulness before Grahek was a phenomenon known as *morphine pain* (Dennett 1985; Hardcastle 1997; 1999): patients given doses of morphine will often report for a bit that their pain persists, but they are no longer moved by it. Klein (2015a; 2015b) also argued that similar claims could be found in reports of depersonalization syndromes.[6]

As a philosophical example, morphine pain has disadvantages: it is transient, ethically difficult to study, and relies on reports from people who are very, very high on morphine. Yet if we're not thinking in dissociationist terms, phenomena like morphine pain and other dissociation syndromes provide a relatively large evidential base for thinking that pains might occur without the associated negative affect. For these cases, while messy, are far more common. Anecdotally, reports of morphine pain are quite common in emergency rooms. Moreover, by some estimates as many as 70% of people will have at least one instance of depersonalization (see Sierra 2009, 44ff for discussion). The core failure would then be not a loss of affect for pain in particular so much as a general breakdown of ownership or engagement with sensations. Others have suggested that similar phenomena, including effects on pain, might be found in schizophrenia (Bonnot et al. 2009), autism (Hoffman et al. 2023), and Cotard's delusion (Klein and Duval 2023).

[6] See Sierra (2009); Gerrans (2020) develops the connection further.

None of these are clean cases, but they need not be. On this latter approach, one looks not for purity but for commonalities. Klein (2015a), for example, emphasized the degree to which asymbolics don't seem to care about many other aspects of bodily safety and integrity. That kind of association is usually irrelevant for a pure neuropsychological approach, but it is central to an approach that explains asymbolia by connecting it to related phenomena. In any case, such interpretations can deal relatively easily with the fact that there are only a few reported cases of pain asymbolia. For, again, the messier cases are frequent enough to raise credence in the idea that affect and ownership might be eliminated to various degrees in these subjects' pain experiences.

3.2. The Ambiguity of the Case Reports

We have sympathy with much of what G&K say about ambiguity. To read the reports of pain asymbolia in full is to be struck by the oddity of what is presented. Moreover, G&K rightly emphasize two types of ambiguity: the ambiguity from comorbidities and the ambiguity of patient testimony. Because they raise different issues, we discuss them separately.

Let us start with the ambiguity from comorbidities. As G&K note, early case reports emphasize the comorbidity of pain asymbolia and various forms of what we would now call aphasia as well as apraxia (Schilder and Stengel 1928; 1931). At first sight, this could be taken to show that affected patients are not lacking an aspect of pain but "rather an inability to integrate certain perceptual information into higher-level cognitive processes" (G&K, 566).

However, more careful investigation shows that the existence of comorbidities doesn't fundamentally threaten the standard interpretation, or alternative interpretations that take it that asymbolic subjects experience. On the one hand, comorbidities are precisely what you would expect if you adopt a neuropsychological approach because, again, brain lesions rarely respect functional boundaries. On the other hand, proponents of alternative interpretations can point out that ambiguity comes with the territory because their interpretations *posit* associations between pain asymbolia and a variety of other symptoms related to lack of care about bodily safety and integrity. In particular, the fact that many reported cases of pain asymbolia "involved apraxia which became more severe with motor action concerning self-preservation" (G&K, 566) seems in line with what they would predict.[7] It follows that both types of interpretations of asymbolia can

[7] The association with language in aphasics is probably a case of anatomical proximity: the insula is located near important language centers, and the lesions that produce asymbolia are often large.

provide a natural explanation of the comorbidities, and so they remain relatively unscathed from such ambiguity.

Now consider the ambiguity of patient testimony. G&K rightly point out that some asymbolic patients in the papers cited by Grahek say that they are *not* in pain or that they are *not* hurting while undergoing procedures that reliably induce pain in normal subjects. For instance, G&K bring attention to the following quote: "Two patients stated repeatedly that the pinprick did not hurt even after prolonged application and to the point of drawing blood" (Rubins and Friedman 1948, 565). Moreover, interpreting the speech of some patients in response to the experimenter's questions is also complicated by the specific comorbidity of aphasia, which involves issues understanding sentences. Even in Schilder and Stengel (1928)'s first case report, the patient reported that a prick on the hand actually did hurt a bit, though only after reporting that she didn't quite know what it was, or where it ought to be localized.[8]

That said, there are simple ways to handle this ambiguity. To begin, it is worth emphasizing that this ambiguity doesn't pertain to all asymbolic subjects — some patients are free from aphasia and verbally describe their experiences as pains. Moreover, putting aphasic patients aside, variability in verbal reports is to be expected. Patients know that their experiences differ in important ways from their pre-lesion experiences of pain; some confusion about how to report what is going on is probably normal. It would be more worrying if these patients *systematically* refused to label any experience whatsoever as 'pain'. But, in many of the papers discussed by Grahek, it seems that the experimenters were able to elicit some verbal or non-verbal response equivalent to an admission of pain *in at least some experimental contexts* from nearly every subject (e.g., the pain threshold experiments reported in Berthier, Starkstein, and Leiguarda 1998).

In the case of the few subjects that never displayed any such response, there is also a more radical response available. G&K, for their part, seem to think that the existence of such subjects undermines the credibility of the verbal reports from *all* asymbolic subjects. But faced with a small group of patients who never label any experience as 'pain', another response is to say that these

[8] "Stiche auf der linken Hand. (Hat es weh getan?) Es rut schon weh, aber ich weiß nicht, was das war eigentlich ist. Vielleieht tut es beim. Herzen weh oder was. (Greift sich an die Brust.) Mitunter sagt Patientin: Es tut schon ein bisserl weh." (Schilder and Stengel 1928, 151) To translate the last expression as "It hurts indeed," as G&K do, strikes us as a bit of a stretch. It appears to be a truncation of Grahek's slightly clunky translation; in the original, the "schon" arguably reads more naturally as kind of a mild surprise than as a strong emphasis. Thanks to Julia Staffel for helpful discussion of the subtleties.

patients should *not* have been categorized as having pain asymbolia in the first place (cf. Bain 2014). As Grahek emphasized, 'asymbolia' was sometimes incorrectly applied to patients who were simply insensate to pain. Mistakes in categorization occur, but they do not undermine the category itself.

3.3. Coming Back to Taxonomy

We have argued that the standard interpretation, as well as alternative interpretations that assume that asymbolic subjects experience pain, have relatively natural responses available to deal with the rarity and ambiguity of the case reports of pain asymbolia. So, it seems premature to recommend their rejection based on rarity and ambiguity, as G&K do.

It is worth highlighting at this point the connection with debates about the *taxonomy* of pain asymbolia. As we show in other work (Klein and Duval 2023), the two strategies for interpreting asymbolia mentioned above correspond to two distinct traditions in the clinical literature on pain without affect, both of which stretch back to the 19th century. On the one hand, we have a view, inspired by the German tradition, of pain asymbolia as a very rare, specific deficit in the processing of the meaning of pain signals. This view posits a neuropsychological dissociation, and it aligns with the standard interpretation. On the other hand, we have a view, inspired by the French clinical tradition, on which pain asymbolia is one striking symptom among many other symptoms that often cluster together. This view fits well with the alternative interpretations of authors who treat asymbolics as the most severe tip of an iceberg of relatively common experiences. Interestingly, G&K seem to end their paper by endorsing a version of the latter view. They conclude that "[since] PA arguably never occurs in isolation from other neuropsychiatric syndromes, it is at least prima facie plausible to conclude that PA is not a syndrome at all, but is merely a symptom which sometimes occurs as a consequence of other clinical conditions" (575).

Which approach is correct? That remains the big question. But stepping back a bit, we would suggest that the debate about how to understand pain asymbolia cannot be separated from the debate about how to taxonomize it. If the taxonomy *were* settled, we suggest that G&K's objections would be sapped of much of their force. That is, if we were sure pain asymbolia should be treated as a single dissociation, then rarity and ambiguity would come with the territory. On the other hand, if we thought asymbolics were the philosophically clean cases at the end of a big

spectrum of messy ones, then we'd have already decided that the phenomenon of pain asymbolia was primarily a symptom. Hence, the apparent complexity and difficulty of the clinical literature on asymbolia is actually symptomatic of a larger issue about taxonomization (one that is ultimately far larger than just asymbolia), rather than something specific to asymbolia itself.

4. Conclusion

The core question about pain asymbolia, we have argued, is really about how to categorize it. Categorization in turn affects use, which affects convincingness: as we emphasized above, the differences in what one thinks of such reports in turn affect how they are used as an evidential base.

We end with a few words about G&K's claim that the current empirical evidence we have supports the view that the experiences of asymbolics are *not* pain (hence their title *Pain Asymbolia is Not Pain*). We think the foregoing analysis raises doubts about this claim. We have argued above that none of G&K's criticisms genuinely undermine the standard interpretation, which entails that asymbolics do experience pain. We've also pointed out that alternative interpretations that equally entail that asymbolics' experiences *are* pain are left intact by G&K's skepticism about the case reports. So, for all we know, pain asymbolia might still be pain.

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