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ARTICLE

Pain Asymbolia is Not Pain

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Abstract

We challenge the standard interpretation of pain asymbolia (PA), a neuropsychiatric condition that causes unusual reactions to pain stimuli. The standard interpretation asserts that PA subjects experience pain but lack important features of the experience. However, we argue that the clinical evidence for PA does not support this interpretation and that the arguments put forward by the defenders of the standard interpretation end up making self-contradicting claims. Finally, we suggest that the best interpretation of the available evidence is to take a deflationist stance toward PA, at least until further evidence becomes available.

1. Introduction

Pain asymbolia (PA) is a neuropsychiatric condition first described by Paul Schilder and Erwin Stengel (1928). The condition involves severely atypical or inadequate avoidance behavior, emotional reaction, and verbal reporting in response to the application of threatening or noxious stimuli. Asymbolics smile and laugh in response to severe noxious stimuli (to the point of drawing blood) and often offer other body parts for further stimulation. The philosophical "discovery" of PA can be attributed to Nikola Grahek,[1] who discusses it in *Feeling Pain and Being in Pain* (Grahek 2007). Grahek's interpretation of PA 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.

This general interpretation has been adopted by several philosophers (Fink 2011, de Vignemont 2015, Geniusas 2017, Bradford 2020, Gerrans 2020),[2] who are primarily

Both authors have contributed equally to the paper. Their names are presented in alphabetic order.

[1] Though it is discussed by Trigg (1970).

[2] Others have been less committed, but still express at least some sympathy for it. Corns (2014) claims that this is what *seems* to happen with asymbolics. Sapién (2020) claims that though there might be no case of pain asymbolia, the debate about it at least shows its *conceivability* which is sufficient for him to draw inferences about the nature of pain.

interested in PA because of its implications for understanding the nature of pain. Their common idea is that, if it is true that PA patients embody the experiential component of pain while lacking other aspects, then PA would be evidence in favor of the claim that typical pain experiences are inherently complex, always involving more than one distinct phenomenal or psychological element. Moreover, if the complexity of typical pain experiences can be demonstrated in this way, then, at least plausibly, further investigation of PA could help us isolate exactly what these patients are missing and therefore tell us what the dissociable components of pain are.

Exactly which experiential component is missing from PA differs between philosophers who share this interpretation of PA. In Grahek's initial proposal the missing element is "painfulness," so that PA represents an experience of "pain without painfulness" (Grahek, 2007, 51). This understanding is adopted by Fink (2011), Corns (2014), Geniusas (2017), and Bradford (2020).[3] For Klein (2015) and Gerrans (2020), the missing element is the ability to care about the painfulness of pain, a view argued against by de Vignemont (2015). Taking on some suggestions from Klein, Bain (2014) argues that both the painfulness and the caring about the painfulness are missing. We will look more closely at Klein's and Bain's views in section 6.

Despite the heterogeneity of these approaches, they appear to subscribe to the same basic assumption, grounding their use of PA as a case for pain being a compound phenomenon (with the exception of Klein): asymbolics experience pain, but lack some component of pain. We shall call this the *Standard Interpretation*. Additionally, Park (2023) holds that asymbolics experience pain, and that their pain *does* have the additional component of "unpleasantness," but that this unpleasantness is drastically diminished as compared with normal pain. Park thus appears to only partially endorse the standard interpretation.

Our challenge to the standard interpretation will focus primarily on Grahek's initial arguments. Section 2 provides a general overview of the standard interpretation. Section 3 reviews the clinical literature for PA and challenges the legitimacy of the standard interpretation of this literature. Section 4 argues that, even if the evidence did not pose a problem for the standard interpretation, this interpretation entails a mereological fallacy, at least on an essentialist understanding of pain. Section 5 argues that if we set aside the previous two objections, the standard interpretation is still ultimately a version of "subjectivism," the position that the standard interpretation was designed to avoid. In section 6 we examine two alternative interpretations of PA, proposed by Klein and Bain. Section 7 offers our own deflationist interpretation of the clinical evidence: PA should not be viewed as clinical *syndrome*, but only as a *symptom* accompanying a number of distinct neuropsychiatric conditions. Section 8 concludes by briefly summarizing the overall argument and its consequences for the philosophy of pain.

While the aim of this paper is to make a general case against PA, not all of our arguments will apply equally to all of the philosophers appealing to PA. On the one hand, all our arguments work against philosophers subscribing to the standard interpretation within an essentialist framework, i.e. assuming the existence of necessary and sufficient conditions for something to be a pain. On the other hand,

[3] The same is true for Corns (2014) and Sapién (2020) to the degree they follow the standard interpretation of PA.

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philosophers who reject the essentialist understanding of pain in favor of, e.g., a family resemblance theory which holds that there is no single set of necessary and sufficient conditions (e.g. Fink 2011, 2012; Coninx 2020), will only find themselves directly challenged by our criticism of the available empirical evidence (see Coninx 2022 for a more-than-usually-circumspect interpretation of the clinical evidence for PA). We think, however, that in each case the arguments applying to the particular background theory of the nature of pain are sufficient to support the overall conclusion that PA should be removed from the arsenal of case studies in the philosophy of pain.

2. The argument for asymbolia being pain

The claim that PA constitutes a genuine pain experience is motivated by two distinct empirical premises and an important methodological commitment. The first empirical premise is simply that there are clinical reports attesting to PA being a pain experience. The second is that there is compelling neurobiological evidence that brain lesions associated with PA disrupt pain perception. In other words, the argument runs: patients with asymbolia say that they experience pain, they show evidence of recognizing when a stimulus becomes painful for normal subjects, and there is independent evidence which suggests that this is not impossible, in spite of their bizarre reactions to noxious stimuli. The conclusion Grahek draws is that the experience of PA is a pain experience and, as such, has important implications for how we should understand pain in general, even though this experience is, as he says, "without painfulness." Though Grahek's commentators have not agreed with him that it is necessarily *painfulness* which is missing from PA experience, they do agree that PA is pain, and that it is missing something.[4]

Grahek draws on clinical evidence from six separate studies conducted by Schilder and Stengel (1928), Pötzl and Stengel (1937), Rubins and Friedman (1948), Hemphill and Stengel (1940), Berthier, Starkstein, and Leiguarda (1988), and Ramachandran (1998). We will be examining these studies more closely in the next section, so for now we will only outline what is important for Grahek's initial point—the claim that pain asymbolics say they are experiencing pain and seem to understand when a stimulus has become painful. Berthier et al. offer the most stringent diagnostic criteria for PA: a patient was considered to have PA "only if they were alert and cooperative; had no evidence of dementia, confusion, or previous history of psychiatric disorder; had no deficit in pain perception; and had absent or inadequate motor and emotional responses to painful stimuli applied anywhere on the body surface" (Berthier et al. 1988, 42).

Of the patients examined by Berthier et al., only six met these strict criteria, and these six were subjected to an extensive series of tests designed to evaluate their responses to various noxious stimuli. These stimuli included noxious thermal stimulation, deep pain induced by heavy pressure and hyperextension, squeezing the calf muscles and the Achilles tendons, pinpricks, pinching, and the threat of slaps,

[4] It is worth mentioning that Grahek's choice of "painfulness" being the missing component in PA is perhaps unfortunate, as it tends to lead to confusion. Others have proposed the terms "hurt" or "suffering" as alternatives. Bain (2014) suggests that there is "pain" and "unpleasant pain"; we only tend to assume that pain must be unpleasant because most pains actually are. For our purposes, we will use "suffering" when referring to the unpleasantness of pain.

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punches, and needles to the eyes. In all cases the patients demonstrated a distinct lack of appropriate response to noxious stimulus. The tendency to flinch and withdraw was significantly diminished or non-existent. The patients did not groan or complain of the stimuli, and some even laughed during noxious stimulation. None of the patients became angry or irritated with those administering the stimuli, and some were remarkably cheerful and courteous. In short, for those diagnosed with PA, noxious stimuli do not appear to elicit even mildly unpleasant experiences.

Nevertheless, there *is* evidence that patients are indeed experiencing something which at least resembles pain. This evidence comes in two essentially distinct forms. First, and most importantly, some of the patients testify explicitly to having a pain experience. According to Schilder and Stengel, a patient pricked on the hand and asked if it hurt said, "It hurts indeed" (Schilder and Stengel 1928, 151). Likewise, Pötzl and Stengel report their patient's testimony as: "I feel it indeed; it hurts a bit, but it doesn't bother me; that is nothing" (Pötzl and Stengel 1937, 180). Even more tellingly, another of Schilder and Stengel's patients "laughs contentedly, jerks the palm lightly, says 'oh hurts, that hurts,' smiles on it, but stretches the hand further toward the examiner and turns on all sides" (Schilder and Stengel 1928, 147).

The second source of empirical evidence for asymbolics having a pain experience comes in the form of Berthier et al.'s pain threshold experiments. For three (out of six) of their patients, Berthier et al. tested pain threshold, where the patient identified when a stimulus first became painful, pain tolerance, where the patient identified when a stimulus finally became intolerable, and pain endurance, which was a function of the difference between these two values. As expected, the pain asymbolics had significantly higher pain tolerance and, as a result, significantly higher pain endurance than the control group. Importantly, however, their pain *threshold* was not significantly different from pain threshold values identified by the control subjects. That is, a stimulus was identified as painful at roughly the same intensity for both normal subjects and the asymbolics. One natural interpretation of this experiment is to say that, indeed, asymbolics know when an experience is normally painful and, therefore, that they are having pain experiences. We ought to point out however, for the sake of completeness, that the average pain threshold measured over five control subjects was 3.9 mA,[5] while the average pain threshold over three pain asymbolic patients was 5.1 mA; this difference could be interpreted as significant, as the average asymbolic pain threshold was over 25% higher than the control, though this pales in comparison with the average asymbolic pain endurance measurement, which was over 600% greater than the control.[6]

Additionally, there is neurobiological evidence to suggest that pain systems are involved with PA. The first thing to note is that Berthier et al. reported insular and parietal opercular lesions in all of their patients. It is clear then (according to Berthier et al.) that the insular cortex and/or parietal operculum play a crucial role in pain asymbolia and, by extension, in the appropriate appreciation of pain perception. However, since pain perception itself is not interrupted by lesions in these cortical

[5] The noxious stimuli involved with this experiment was an electric shock of increasing intensity.

[6] According to Gray (2014), asymbolics do still have an upper threshold because the experience, whether painful or not, remains intense. Still, the relation of intensity to pain as such remains an open question.

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regions, it seems that pain perception and the experience of painfulness can come apart, and that the insular cortex and/or parietal operculum make the difference. This hypothesis is backed up by the electrophysiological work of Robinson and Burton on monkey brains (Robinson and Burton 1980). According to their research, the secondary somatosensory area, contrary to what one might expect, contained very few neurons which responded *specifically* to noxious stimuli; rather, the granular insula and opercular area 7b contained neurons which were highly sensitive to noxious stimuli. Further, a subpopulation of these neurons responded to visual stimulation in particular. These findings suggest that damage to the insula and/or parietal operculum may result in pain asymbolia if the secondary somatosensory area is left intact; i.e., pain perception (which happens primarily in the secondary somatosensory area) is unaffected, while pain *appreciation* (which seems to happen primarily in the insula and/or parietal operculum) can be radically disrupted. Berthier et al.'s conclusions find further support in the fact that specifically visual tracking of potentially threatening stimuli seems to involve the granular insula; this is one of the regions universally affected in Berthier's patients, none of whom showed sufficient appreciation of visually presented threatening stimuli. These findings are even further supported by Dong et al. (1994) and Price (2000). It is not necessary to go too deeply into the details of the neurological evidence for the possibility of PA, since it is not our intention to challenge the validity of this evidence itself.

We ought finally to point out a minor methodological premise, highlighted by Fink (Fink 2011, 2018). Grahek denies the validity of using one's own introspection to arrive at the necessary conditions of phenomena such as pain. Our own introspection gives us access, at best, to only those qualities which are universal over our own experiences. Moving by inference from what is universal in our own experiences to what must be universal in all experiences of the same kind is invalid. Thus, the fact that I have always experienced pain as unpleasant is not sufficient inferential grounds to claim that all pain experiences are (necessarily) unpleasant. This methodological premise can be challenged, but in order to remain charitable to Grahek (and Fink), and to avoid simply begging the question against them, that is, to remain innocent of "phenomenological foot-stomping," as Fink calls it (following Kriegel 2007), we must respect this methodological premise. Thus, any argument against Grahek's interpretation which simply proceeds by *defining* the essential features of pain misses the mark. Nevertheless, it is our contention that Grahek's interpretation of the cases in question is mistaken. We will show this first with a closer look at the cases themselves.

3. Interpreting the clinical evidence

The standard interpretation of PA is that it represents a genuine pain experience, but that this experience is lacking some essential component. Grahek's main support for this interpretation comes from the clinical evidence, which he claims "unequivocally" (Grahek 2007, 139) and "incontestably" (108) demonstrates the existence of pain without painfulness. This, however, is false; the actual clinical evidence is far from unequivocal or incontestable on this point. It is of course obvious that asymbolics are experiencing *something*, but whether or not what they are experiencing is pain remains highly debatable, as we will show in this section.

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When we look closely at the clinical evidence, we see that one of the general criteria for identifying pain, namely, verbal report, is neither consistent nor reliable when it comes to asymbolics.

A quick look at the lines from Schilder and Stengel (1928), which Grahek quotes only partially, very explicitly calls the nature of the experience into question: "Stich to the left hand. (Did that hurt?) 'Well it hurts, but I don't know what that really is. Maybe my heart hurts or something like that.' Touches her Chest. Sometimes the patient says 'It hurts a little bit'" (151). Accepting this last locution at face value appears too hasty. The patient is not sure how to characterize her experience and Schilder and Stengel have compromised any credibility her report might have had as a spontaneous description by asking specifically "Did that hurt?" rather than a less leading question. She is not able to correctly locate the pain, which is in seemingly direct contradiction to the standard diagnostic criteria of being able to demonstrate normal pain perception (see, e.g., Berthier et. al. 1988).

In Schilder and Stengel (1931) the findings are markedly less consistent. Of ten patients, five recovered from their asymbolia in parallel with their recovery from general sensory aphasia.[7] In these cases, pain asymbolia seems to be tied to the sensory aphasia. Other cases involved apraxia which became more severe with motor action concerning self-preservation. And, most tellingly, "Two of the patients said that they could remember that the pain had been inflicted, but that they did not feel it" (Schilder and Stengel 1931, 599). This study leads the authors to conclude "that a lesion in a particular region of the left parietal lobe makes it impossible to build up a full perception of pain" (600). Pain asymbolia, then, for Schilder and Stengel at least, should not be considered a pain experience, but rather an inability to integrate certain perceptual information into higher-level cognitive processes. The testimony of both of (1928, 1931) present problems for interpreting patient testimony as evidence of "pain without painfulness."

In connection with this, it is worth pointing out the relation between PA and pure word deafness described by Hemphill and Stengel: "[I]t is necessary to note that in the majority of cases asymbolia for pain is combined with some form of word deafness. All types of word deafness represent, after all, a loss of relation to the outer world, confined to the lack of understanding of spoken language" (Hemphill and Stengel 1940, 260). Obviously, it is difficult to gauge the validity of a verbal response to a question concerning a topic for which the subject suffers pure word deafness. The exact connection between PA and word deafness involving pain concepts remains unclear, as Hemphill and Stengel stress, but the regular co-appearance of these symptoms is enough to warrant caution when interpreting patient testimony.

To reiterate this point, sensory aphasia and pure word deafness are two of the most commonly co-occurring symptoms associated with asymbolia. Both represent linguistic and cognitive difficulties which must be taken into account when assessing the significance of patient testimony. The clinical literature, to the degree that its interest has been in understanding patient experience, has been sufficiently wary of the implications of these co-occurrences. The philosophical literature, we are suggesting, has not. The inability to comprehend pain and self-preservation concepts

[7] Sensory aphasia, also called receptive aphasia, is an inability to understand spoken, written, or tactile speech symbols as a result of brain damage (see Brookshire 2007).

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in general, which was observed in patients with this form of pure word deafness, and the inability to consistently describe pain experiences, which was observed in patients with this form of sensory aphasia, severely limits what can be taken as philosophical evidence from the testimony of these patients regarding their experience of pain. At present, the clinical literature is inconclusive about the exact or necessary relation between these comorbidities; in rare cases (e.g., Rubins and Friedman 1948) asymbolia appears to occur with little or no indication of aphasia or word deafness, while in most others (e.g., Berthier, Starkstein, and Leiguarda 1988, and those already mentioned) the presence of something like aphasia or word deafness seems undeniable—even if the researchers themselves were not looking for this connection. Relying on the pain-experience testimony of those suffering from a condition which is often coextensive with cognitive impairments which themselves may prevent the comprehension or expression of pain concepts is unfeasible.

Rubins and Friedman (1948) report even more contradiction in patient testimony:

> The predominant feature shown by our patients was the pain asymbolia, as described by Schilder. The degree of response to the stimulus varied from complete denial of the pain to verbal exclamation after stimulation and finally to some partial movement of escape. *Two patients stated repeatedly that the pinprick did not hurt even after prolonged application and to the point of drawing blood.* Another would sometimes produce a very inadequate "ouch" when stuck but never any withdrawal or effective reaction of defense. Other stimuli, such as heat or cold, produced a similar result. One patient would hold a lighted match until her fingers would almost burn without dropping it. Special pain-producing tests, including the intravenous injection of histamine and artificially produced muscle ischemia, also provoked the same inadequate response. (Rubins and Friedman 1948, 565; emphasis added)

As we emphasize in this quotation, two patients repeatedly denied being hurt, even upon being stuck with a needle to the point of bleeding, while two did not. This shows the heterogeneity in patient testimony which is typical in PA research. Naturally, the nervous activity produced by this kind of stimulation generates feelings of pain in normal subjects. The testimony of these patients pretty unequivocally indicates that, whatever they are experiencing, they are not experiencing pain. The point here is that some PA patients report feeling pain but not caring, while many other PA patients report not feeling any pain at all.

Regarding the patient who says "ouch" but does not withdraw the hand, we must admit that there is a suggestion of something like an incomplete or partial pain experience. The proponent of the standard interpretation might argue that this indicates a condition of partial PA, wherein the patient would be left with real but inadequate sensations of pain. The existence of partial asymbolia would not help the standard interpretation, however, because it would seem that the extent to which the patient felt pain would be the extent to which they did not suffer from PA. This is because it would seem, then, that partial asymbolia would have left the experience of pain partially intact, and by implication that asymbolia is a disruption of pain perception itself; this is directly the opposite of the standard interpretation. Regardless, cases of partial asymbolia would not offset the importance of testimony

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like the following, also given by Rubens and Friedman: "She gave this impression even when she was told that 'this is going to hurt you.' She then answered 'didn't hurt me' when the examiner pricked her to the point of drawing blood" (Rubens and Friedman 1948, 561). As they point out, this patient was completely capable of discriminating between sharp and dull noxious stimulation and seemed to have otherwise normal tactile perception.

In concluding this section, we can say that pain asymbolia is a rare condition and the literature on it is not extensive. More clinical work needs to be done here. However, none of the literature which exists unequivocally or incontestably demonstrates that what pain asymbolia patients are experiencing ought to be identified as a pain experience. The fact that patients with pain asymbolia can discriminate between experiences which are normally painful and those which are not has indeed been sufficiently demonstrated, but the testimony of these very patients inconsistently supports the claim that they are experiencing pain and, in some cases, directly contradicts it. It is more faithful to the extant clinical evidence to say that asymbolics experience noxious stimuli without painfulness (i.e., suffering), than to say that they experience *pain* without painfulness. From a clinical perspective, this is a mere verbal difference, but from the perspective of the philosophy of pain, this difference is crucial since the standard interpretation implies an unjustified and, indeed, potentially question-begging conflation (or even straightforward identification) of noxious bodily stimulation and the experience of pain. But this is the very thing which cannot be assumed in any philosophical discussion of the nature of pain. The main empirical support for Grahek's interpretation of PA, then, is not sufficient to justify the standard interpretation.

4. The argument from mereological fallacy

If, for the sake of the argument, we assume that we could present incontestable evidence that PA experiences are meaningfully similar to normal pain experiences, it can still be shown that understanding their experience as one of pain is incoherent; identifying PA with pain in this way entails what could be called a mereological fallacy, though, as noted above, the standard interpretation only clearly falls into this fallacy with the assumption of an essentialist framework about pain.

A mereological fallacy is a form of faulty reasoning which results from a violation of the rules of mereology. Typically, the term mereological fallacy refers to the violation of one specific injunction: do not attribute something to a part which can only properly be attributed to the whole. This mistake has been discussed extensively for the application of psychological predicates. However, psychological predicates are not the only kinds of predicates which can be the subject of a mereological fallacy; ontological predicates, such as "*is* pain," are equally subject to the rules of mereology.

The standard interpretation is committed to two distinct and incompatible concepts of pain. The first of these is the "pain" experienced by asymbolics and is supposed at once to be pain and to lack some component of pain. The second concept of pain, which slips in when a transition from pathological pain to typical pain is needed, and which is supposed to track our common sense understanding of pain, is the concept of pain as an *essentially complex* phenomenon, consisting of a pain-specific sensory phenomenal component, an affective component, and a motivational component.

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As Grahek writes, "pain, although seemingly homogeneous, is actually a complex experience. The sensory-discriminative, emotional-cognitive, and behavioral components typically occur together, but they can exist separately" (Grahek 2007, 73). When it comes to the individual components taken in themselves, and as they plausibly are separated for PA patients, they are at times characterized as "mock pains": "the pure juice or essence of pain experience thus extracted [in PA] has turned out to be a blunt, fleshless, inert sensation pointing to nothing beyond itself, leaving no traces in the memory and powerless to move the mind and body in any way. [. . .] But then one would be strongly inclined to say that this is not real pain, that it is only mock pain" (Grahek 2007, 76).

Even though Grahek takes PA to be deprived of essential elements of pain, he clearly wants (and his arguments against subjectivism demand it) to maintain that PA is a real kind of pain. He asks if we should drop the idea that painfulness (i.e., suffering, hurt, unpleasantness) is an essential part of what it is to have a pain experience and answers: "I think that we should not [. . .] asymbolia should instead be viewed as a phenomenon which clearly shows us what pain comes to when it is deprived of unpleasantness—that is, it reminds us that the bare sensation of pain comes to nothing and serves no biological purpose" (Grahek 2007, 139–40).

According to Grahek, pain, *in its essence*, is complex; there can be no pain which is not complex; complexity is a necessary condition for something to be pain. And yet, PA must still count as an instance of pain, for then the essential link between PA and pain would be severed and we would have no reason to think PA tells us anything interesting about pain. It must be supposed (contrary to much of the clinical evidence) that PA represents a modality of pain, specifically. But, we would like to point out, PA cannot deviate from pain so radically that we are no longer inclined to associate the phenomena at all. This is why Grahek introduces terms like "mock pain," in order to avoid, as best as he can, the tension in the inconsistent triad: 1) pain is complex, 2) PA is not complex, 3) PA is pain. But of course pain cannot be both simple and complex in the same way at the same time; if pain is *essentially* complex, as the standard interpretation holds, 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.

At first glance, the debate concerning the nature of PA experiences might seem like a merely verbal dispute; it could be argued that we are merely quibbling over whether or not to give the name "pain" to PA experiences. One might say, for instance: "Granted, PA experiences are not 'pain.' But that is precisely because they are simple; were they complex in the relevant ways, i.e., if they had added to them the various motivational-affective components they are said to lack, then they would be pain. This demonstrates that pain is complex, and this is what is important and interesting."

Setting aside the fact that this is not really how the standard interpretation is spelled out, we have to observe that the objection is actually invalid. The idea behind this objection is to say that PA experiences fail to be pain because they are simple, therefore proper pain must be complex. But this inference does not follow; even if the clinical evidence supported the conclusion that PA is a simple psychological phenomenon, then it would still be an invalid inference to move from the simplicity of PA and its failure to be pain to the conclusion that pain must therefore be complex.

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Really (as we will argue in the next section), one ought to conclude just the opposite: if PA is simple, and PA is pain, then pain is simple (Klein is more consistent than Grahek on this point). But it was the part–whole relation between pain and its components which was to be established by the argument from the standard interpretation. Thus, this relation cannot also serve as a premise. So, far from being verbal, the question of whether PA is or is not pain undergirds and informs the entire discourse surrounding the standard interpretation and its importance for the philosophy of pain. We agree that it is not (necessarily) very important which entities are called "pain" and which are not. What is important, however, is that we do not mistakenly identify heterogeneous phenomena. Since the empirical evidence often contradicts the claim that PA patients are experiencing pain, and since claiming that they *do* experience pain involves a mereological fallacy, it is crucially important that these phenomena not be confused.

5. The argument from collapse into subjectivism

In this section we will argue that even if you set the previous two objections aside, the standard interpretation entails a version of subjectivism, which is independently fatal for the view. Specifically, as Grahek writes, the position of subjectivism holds that:

> the sensation of pain with its distinctive phenomenal content or quality—the "what-it-is-likeness" of pain—is the essential component of our total pain experience and plays the central or fundamental role in it. Allegedly, when this component is absent, there is no pain or pain becomes ersatz pain, despite the presence of all other components of pain experience. (Grahek 2007, 76)

There are many notable proponents of subjectivism (e.g., Kripke 1980; Jackson 1982; Campbell 1983; Chapman and Nakamura 1999) and the view has merit. However, as Grahek argues, subjectivism seems committed to the difficult position that PA represents the best and purest instance of a pain experience, given that PA appears to isolate the subject from every element of a typical pain experience other than what the subjectivist claims is the essence of pain. And yet, we have to say that PA is a pale and distorted shadow of pain if we want to remain anywhere close to the common sense idea of pain, which must at least include that it is bothersome. Grahek himself is deeply unsatisfied with subjectivism. Worse, subjectivism is incompatible with the standard interpretation and if the standard interpretation entails subjectivism, then it is incoherent. This is because the standard interpretation, whatever else it holds, must hold that pain is complex, while the subjectivist view holds explicitly that pain is ultimately simple.

First, recall that the standard interpretation of PA holds that PA is a pain experience, but that it lacks some typically associated experiential or motivational component. Subjectivism holds that *the* essential component of pain is its phenomenal character, divorced from any affective or emotional components. PA seems to present us with just such an experience; patients can report on when a stimulus becomes painful, what kind of pain it is, and how painful it is, and yet have no negative reactions. The standard interpretation wants to maintain that PA is missing some component of pain, so since subjectivism must claim that PA is *not* missing any components, the standard interpretation and subjectivism are strictly incompatible.

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On the other hand, if PA experiences are like pain experiences in the way that the standard interpretation claims, it is reasonable to ask which qualities or characteristics unite them; if PA experiences are pain experiences, then they must meet the necessary and minimally sufficient conditions for something's being a pain experience and it is fair to ask what those conditions are. It is clear that the proponent of the standard interpretation has no alternative but to say that the very elements identified by the subjectivist as the essence of pain experience do after all constitute the necessary and minimally sufficient conditions in question. Denying this amounts to claiming that PA experiences are not pain experiences, and is therefore obviously not an option for the standard interpretation. This is because PA would no longer have any obvious connection to pain and therefore would not serve the argument that the standard interpretation needs. But accepting this amounts to endorsing subjectivism; because subjectivism claims that the essence of pain is the phenomenal experience of pain, this one aspect of pain must be both necessary and sufficient for a pain experience. And this cannot be accepted, because then pain is simple rather than complex, which contradicts the fundamental thesis of the standard interpretation. This is a bitter dilemma for a position which finds subjectivism "deeply unsatisfying" and is explicitly designed to avoid it.

It might be suggested here that we have not given Grahek the leeway he asks for when he writes:

> I do my best to resist the strong philosophical temptation to prejudge on the basis of preconceived ideas whether pain without painfulness and painfulness without pain should be treated as cases of pain at all. It is more interesting and important to learn something from these bizarre and puzzling cases about the true nature and structure of pain, than to relish the fact that another piece of evidence speaks in favor of one's cherished theory, or to despair if it does not. (Grahek, 2007, 5)

This is a wise move, and we are not challenging the standard interpretation on this point. What we would like to point out is that we have not engaged with the question of whether pain without painfulness should be considered pain. Rather, what we have argued is that PA cannot be used as an example of pain without painfulness. We also have not offered or endorsed any theory concerning the true nature and structure of pain, and what we have said about pain comes directly from what is uncontroversial among those endorsing the standard interpretation.

6. Two alternative models

Two models of PA which have been offered as alternatives to the standard interpretation are given by Colin Klein and David Bain. These are interesting in their own right, and deserve their own consideration here, not least because such consideration helps to clarify our arguments against the standard interpretation. These are the "Lost Capacities" model and the "Evaluative" model, respectively. We will present each in turn and offer some thoughts about how the present arguments bear on these models.

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For Klein, unlike Grahek and other composite theorists, pain, in its essence, is a homeostatic sensation (a sensation which signals when the body has gone out of balance in a certain way, like hunger and thirst) with a non-representational, imperative content. Klein's schematic for this imperative content is "Keep B from E (with priority P)!" (Klein 2015). Keep [your ankle] from [bearing weight] (with priority [less than starvation but greater than peckish])! This imperative is issued by the body, which we normally take to be a valid, though not unimpeachable, source of authority for this kind of imperative. Such imperatives are inherently motivating, just in virtue of their content. These imperatives can also generate a secondary motivation oriented toward the imperative itself; these secondary motivations are what we typically identify with "hurt" or "suffering." However, these secondary motivations are to be distinguished from the primary motivations which properly constitute pain; Klein's model is therefore, as he says, a "pure-imperative" model, in that pain really only has this one component. Thus, my pain consists in the imperative my body gives me to favor my right foot, for instance, while my suffering consists in certain negative feelings I have toward that imperative, including how unpleasant, disagreeable, inconvenient, etc. that imperative is. This allows for pain and suffering to come apart on Klein's account in a way not dissimilar to how they come apart on the standard interpretation of PA, but which seems secure from any charge of mereological fallacy. This is because Klein maintains that pain, in its fundamental essence, is simple; namely, pain simply is a protection imperative issued by the body.

This strikes us as an improvement over the standard interpretation. The apparatus is simpler and the terminology is clearer (admittedly, Klein does occasionally talk about the distinction between pain and painfulness, but he also substitutes "painfulness" with "hurt" and "suffering," which helps distinguish between different levels of motivation, as we mentioned above). It is, however, beyond the purposes of the present paper to compare Klein's general imperativist account of pain with Grahek's dual-aspect account of pain; here, we are interested only in the different interpretations of PA. In this respect, Klein's account strikes us as superior. For him, the asymbolic does indeed experience the imperative content of pains, but interprets these as being directed toward someone else, rather like someone who hears the policeman shout, "Stop or I'll shoot!" but doesn't realize the policeman is talking to them (Klein 2015). For Klein, this lack of recognition is not an accident, but results from the patient's having lost the capacity to care about bodily integrity. Fundamentally, for Klein, asymbolics have lost the ability to care about their bodily integrity and thus their bodies have lost the authority to issue serious imperatives. Klein calls this the "Lost Capacities" model (and Bain refers to it as the "care-lack" model). Moreover, Klein admits that this is speculative (as indeed it must be, given the scant evidence for asymbolia), but suggests that the neural substrate associated with care concerning bodily integrity is what has been damaged in asymbolics.

As we say, this is far from the worst interpretation that one could give of asymbolia. However, we wonder if Klein has sufficiently motivated his interpretation of PA. On the one hand, as we demonstrated above, while there is some evidence that (some) asymbolics recognize noxious stimulation as containing something like an imperative, there is at least as much evidence to suggest that (some) asymbolics don't. At the same time, as Klein recognizes, asymbolics also tend to have significant difficulties with language, so that even when first-person testimony might suggest the comprehension

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of something like an imperative, we still have reason to be skeptical. According to Klein, asymbolics experience the imperatives of pain, but experience them as devoid of authority. But is there really good evidence of this? The most basic evidence that normal pain experience consists in an imperative is just that we respond to pain as we would to commands, and that no simple representation seems able to command in this way. But asymbolics do not respond to pain as if they are being commanded; rather, they laugh, they extend their hand, they thank the experimenter. If anything tells against Klein's account of PA, it seems to be the wide inconsistency of the empirical evidence. Could we not tell a story about PA where the imperative content of pain is altered, rather than one in which the capacity to care about pain has been diminished? I compulsively wrench my hand away when I am burned, but I also compulsively laugh when I am tickled. Some asymbolics laugh when they are burned. Some say thank you. Some ask for more. Perhaps the structure of the imperative itself has changed in PA, so that instead of "Keep B from E (with priority P)!" the patient experiences something like "*Subject* B to E (with priority P)!" Or perhaps we could tell a story which is a mixture of both change in imperative content and a loss in capacity to care about bodily integrity.

All that said, we think that the imperativist could really welcome this line of argumentation. As Klein says, "Pain asymbolia is arguably the cleanest apparent counterexample to motivationalism" (142). The imperativist interpretation of PA is more plausible than the standard interpretation, but we think it still struggles to account for the wide variety of symptoms associated with PA in the clinical literature. We would like to suggest that this is less the fault of imperativism and more the fault of the clinical literature. Would it not be more circumspect to pause and ask whether PA, as currently understood, represents a counterexample to anything? It seems to us that imperativism ultimately benefits from adopting a skeptical attitude towards PA.

Bain's interpretation of PA attempts in some ways to split the difference between the standard interpretation and Klein's "Lost Capacity" model. And we ought immediately to point out that Bain's arguments are less damaged by the inconclusiveness of the empirical evidence for PA because, explicitly recognizing this inconclusiveness himself, he is careful to couch his arguments as conditional; *if* something like PA is possible or actual, *then* the arguments he presents follow. This has the interesting effect of shifting the argument onto the (mostly) conceptual plane. Pointing out that one or two accounts of PA contradict the evidence he cites does not effectively challenge his point. This already strikes us as an improvement over the standard interpretation.

Bain accepts Klein's lost capacity model, but rejects Klein's unitary imperativism. Klein emphasizes again and again that one of the virtues of his account is that it makes do with only a single component—the imperative content of pain sensations—to explain all the interesting and curious features of pain. But according to Bain, Klein's unitary imperativism fails to make sense of why asymbolics do not grimace or withdraw from noxious stimuli, among other things. This is because, as we take it, the imperative content should still be present in the asymbolic's experience, even if the unpleasantness is not; that is, on Klein's view, according to Bain, the asymbolic should still grimace and withdraw, but then also report that, despite this behavior, the experience was not unpleasant. Instead, Bain proposes an evaluative model, wherein care for one's body generates a negative evaluative attitude toward the body's destruction; for Bain, unpleasantness is dependent upon an evaluative layer which

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identifies damage to the body as bad. In asymbolics, this evaluative layer has been eliminated, so the sensations (which represent bodily destruction) cannot be identified as bad, which means that they cannot be unpleasant.

This is a subtle difference. Klein, according to Bain at least, holds that asymbolic pain is unpleasant, but that this unpleasantness fails to motivate (as if the imperative were directed toward someone else), while Bain holds that they are not unpleasant to begin with. And while we cannot do justice to this difference here, it is worth pointing out that what is at stake is whether a unitary account of pain, one which claims that pain consists of only a single component, is capable of satisfactorily explaining all the various pain phenomena. It is the unitary-ness of Klein's account that distances him most of all from the standard interpretation, and the reintroduction of complexity (with *unpleasant* pains) brings Bain somewhat closer to the standard interpretation. But Bain is definitely not offering the standard interpretation. Against Grahek, he claims that, in itself, bodily damage is not sufficient as the cause of unpleasant pains. I can see that something is being damaged, including my own body, without therefore caring about the damaged thing or the damage being done. Some psychological level of evaluation must be at work, and this level appears to be disabled by PA. In many ways, Bain's account of PA strikes us as the most compelling, especially since his arguments are couched as conditional. Note also that our argument is not against composite theories of pain as such, but with the conjunction of a composite theory and the identification of PA with a pain experience. These two claims, we maintain, do appear to be in conflict, and maintaining both of them together constitutes the mereological fallacy which seems to trouble the standard interpretation.

However, Bain's evaluative model is not completely satisfying. While we see the qualification which conditionalizes his arguments as both valid and prudent, there is left open the question of what to make of the clinical evidence we do have. A story still needs to be told here. Additionally, Bain is a little unclear about the final status of PA. He writes that PA pain is abnormal because the asymbolic patient is abnormal. Surely this is correct, but some question remains as to how we should cash it out. Is PA pain so abnormal that it should no longer be recognized as pain? If so (which we think a charitable reading of Bain would grant), then the exact relation of PA to pain becomes obscured once again. But if not, then Bain seems open to the same charge of mereological fallacy we brought against the standard interpretation. Let us turn now to how we think PA should be interpreted.

7. Pain asymbolia deflated

So far we have argued that there are three distinct reasons for rejecting the standard interpretation. The first is that the available clinical evidence does not support that interpretation. The second is that the interpretation itself is guilty of the mereological fallacy. The third is that, even ignoring these, the standard interpretation is still reducible to the position of subjectivism, which it was explicitly designed to avoid. But at this point it would be fair to ask how we are supposed to interpret PA if the standard interpretation fails. The answer, it seems to us, is that we must deflate pain asymbolia, at least until further empirical data is available.

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If we take on board all the currently available evidence, the presumption that PA presents us with a homogeneous, independent clinical syndrome which specifically dissociates typically unified components of pain is, we believe, clearly premature. If we consider only what the evidence unequivocally shows, in conjunction with the fact that 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.

To spell out what we mean by this distinction and why it is important, let us reiterate what the clinical evidence does unequivocally demonstrate: first, patients with certain similarly located brain lesions can discriminate between types of normally painful stimuli, e.g., sharp, burning, dull, etc.; second, that these same patients can perceive at what threshold these stimuli would become painful for normal patients; and third, that these patients do not exhibit normal pain avoidance behavior. Beyond these three points of agreement, cases of PA are highly heterogenous and seem always to be accompanied by different comorbidities which could account for the appearance of PA.

The diversity of PA cases is made especially clear in Schilder and Stengel. Of their ten patients, six had typical sensory aphasia, one "showed difficulty in finding the right words," two did not have aphasia or it disappeared rapidly, one had global speech difficulties, five recovered from their asymbolia while five did not and the disappearance seemed connected with the disappearance of sensory aphasia, some cases showed apraxic disturbances, two showed strongly increased postural reflexes, and two patients "showed preservational tendencies in connection with the asymbolia for pain concerning action" (Schilder and Stengel 1931). Interestingly, these last two patients were discovered to have lesions of the frontal lobe, rather than parieto-occipital or temporal cortices, as is typically the case with PA patients. An association between PA and pure word deafness has been documented (Hemphill and Stengel 1940) but is certainly not universal, and the exact relation between them remains a matter of speculation. As we have already shown, the verbal reports of patients subjected to noxious stimuli range from complete inability to articulate the nature of the experience, through complete denial of all painfulness, to breezy and smiling acknowledgement of the excruciating painfulness of the experience. With all this diversity taken together, it becomes more and more difficult to maintain that PA is a genuine clinical syndrome in its own right, rather than, as we have said, a mere symptom or byproduct of other clinical conditions.

One could draw an analogy here with muteness. There are many reasons why a patient might be mute; on the one hand, she may have cognitive impairments which prevent any language processing, and so prevent her from speaking, or, on the other hand, she may have had her larynx and vocal cords removed. In both cases, of course, the patient will demonstrate an inability to speak, but this is not a good reason to conclude that something similar is going on with these two patients. In the one case, the patient may want to speak but be unable, while in the other she may not even be able to want to speak in the first place. Similarly, with PA; inadequate or atypical responses to noxious stimuli may represent an inability to make sense of pain concepts, as they seem to in Berthier et. al. (1988), or they may represent a radical alteration of pain experience itself, as seems to be the case in

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Schilder and Stengel (1928). Do we have good reason to think that these two studies are tracking a fundamentally unified phenomenon? One might have their doubts. Rather, it seems more likely that the fundamental syndromes in question are distinct, that PA accompanies each as a symptom, and that there is no further PA experience beyond the syndromes themselves.

What does our deflationist interpretation of PA mean for the philosophy of pain in general? It is clear from the arguments for the standard interpretation that a belief in the existence of PA as a unified pain syndrome is a common presupposition held by many philosophers. It was developed by Grahek in part as an argument against subjectivism. If PA is not accepted as a unified pain syndrome, then some other argument against subjectivism will have to establish that pain is a complex phenomenon or that its essential element is something other than its experiential content. However, up to now it has remained an option to bite the bullet, to embrace subjectivism and accept that, however strange it seems, PA patients have the purest experience of pain. This might be motivated by observing that, after all, we know that most PA patients can still discriminate between typically "painful" and "non-painful" stimuli and between different types of noxious stimuli. But if we take a deflationist view of PA, even this move is not an option; if PA is merely a symptom that only occurs in the contexts of other neuropsychiatric disorders, the experiential components of which may differ radically, then even an essentialist, subjectivist account of pain does not succeed by appealing to PA.

On top of this, we must again point out that PA, whether syndrome or symptom, is an extremely rare condition and the relevant clinical literature is not only inconsistent, but also very limited. The scarcity of PA means that any discussion of this condition has to be somewhat speculative. Speculation is of course perfectly valid, as far as it goes, but if we want to move beyond speculation we must wait for more conclusive empirical data. As it is, the empirical evidence just does not support any conclusive interpretation. And since this is the case, it seems prudent to proceed in as deflationist a manner as we can, since this is most likely to avoid problematic commitments.

In any event, once the idea that PA is a type of pain is removed, the standard interpretation becomes unworkable and the arguments which depend upon the standard interpretation lose their force or have to be approached from a different angle. This is ultimately what we have wanted to show with our analysis of asymbolia and its relevant literature.

8. Conclusion

We have offered three independent arguments for why the standard interpretation of PA should be rejected. First, we have argued that the clinical evidence does not support the conclusion that PA patients experience pain. Second, we have argued that the standard interpretation is guilty of a mereological fallacy wherein it attributes an ontological predication to a part of pain which is only appropriate to pain as a whole. And, finally, we have argued that, given an essentialist framework for understanding pain, the standard interpretation is fatally reducible to subjectivism. Additionally, we have argued that whatever one believes about pain in general, we should be skeptical of PA's status as a genuine syndrome. Plausibly, PA is a *symptom* which merely

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accompanies a number of diverse neuropsychiatric conditions, each of which may have a very distinct experiential character. The status of PA's existence, however, must ultimately be decided by further empirical research. Further, we have not taken any position on whether pain in general is simple or complex. Rather we have only shown that PA cannot be used as ammunition in the philosophy of pain in the way that it has over the last two decades. Above all, we wish to motivate further clinical research in this area; PA is a fascinating phenomenon, with rich implications for the philosophy of pain (and perception and cognition in general), and we should not be satisfied with our current understanding of this phenomenon, or with its position in the philosophical discourse surrounding pain.

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