The Experience of Pain

Neurophysiology of Pain Perception and its Bearing on Core Issues in the Philosophy of Mind

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“The aim of the wise is not to secure pleasure, but to avoid pain.”
- Aristotle

1. Introduction

As trite as the above quote may seem, there is a sense it which is highly relevant in a meta-philosophical way. The concept of pain is a notoriously confusing to physiologists, psychologists, and philosophers alike. Its place in the conversation within the philosophy of mind is unique—pain experiences are often trotted out when trying to advance claims about the famous “explanatory gap”, and the hard problem of consciousness. There are ways in which this is an attractive and convenient move to make—everyone (with the exception of congenital analgesics) experiences pain in their lifetime, and can easily reflect on these experiences. Our first-hand experience with pain seems to tell us a great deal: the circumstances in which it occurs, the way it feels to the body, and the emotions and actions it elicits. In fact, the manifest image of pain experiences is that the way they present themselves to us exhausts all there is to be a pain experience. This premise is what gives pain experiences such force in the relevant thought experiments.

However, despite its pervasive (ab)use, the notion of pain as used by philosophers is divorced from the full neurophysiological and neuropsychological picture. The favorite shorthand is that “C-fibers” are the neural correlates of pain experiences (sometimes, even just “pain fibers”). This gross simplification belies the complex, multifaceted nature of pain experiences, and, as I will argue, does actual philosophical harm to the arguments in which it is used. Yet, such arguments are still common, and rarely accompanied by deeper exploration into the neuroscientific framework that pain is housed in. In this way, contemporary philosophers are being “wise” in the Aristotelean sense—by avoiding [detailed explorations of] pain. Is this truly a wise move? I will answer this by introducing the plight of the congenital analgesics I mentioned earlier.

Congenital analgesic patients cannot feel pain—mechanical disruption of bodily tissue by stabbing or cutting, scalding hot temperatures, and even direct chemical injections with histamine do not bother them. Injuries that would be traumatic for anyone else barely prompt these patients’ attention or consideration. At first reading, this may sound like some sort of real-life superpower—
akin to the invulnerability of Superman or the Terminator. However, life is incredibly difficult for congenital analgesics. Consequences of not noticing normally painful events include: suffering third-degree burns after leaning on a hot radiator, infections in the foot from embedded nails and glass that were not detected, and permanent joint damage from failing to shift posture or positions when standing or sleeping. Indeed, these patients have a shorter life expectancy, and it is common for them to die in childhood due to untreated injury or illness. Pain, as it turns out, is a vital gift that we should not reject.

So it goes with philosophy: contra Aristotle, it would be very unwise to avoid pain, or at least to avoid talking meaningfully about it. Instead, we ought to delve deeply and thoroughly into the study of pain, integrating what knowledge is available from neuroscience and psychology. This will be my goal in this paper: to develop a more nuanced concept of pain, informed by the relevant science, and show that it has a profound bearing on big issues in philosophy of mind. In particular, it bears on arguments for the existence of an “explanatory gap”, and shows that a divergence between mental reality and mental appearance is possible. Before going straight to how I believe we ought to treat pain, I will present pain as it is commonly viewed, and introduce the arguments it is present in.

2. The manifest image and gaps

How should we characterize the manifest image of pain? One way to start is by asking “what it is like” to be in pain. This involves consulting our conscious experiences of pain and reflecting on their character. Upon introspection, pain presents itself as a simple, unified kind of experience. If we feel like we’re in pain, we are in pain! If I tell you I am in pain, and I am truly reporting my felt experience, it would be bizarre and rude for you to deny my claims. The way pain appears to us, then, is that it is self-verifying—the very act of thinking\(^1\) the proposition “I am in pain”, makes that proposition true.

With this account in mind as the manifest image of pain, I will now introduce the “modal argument.” This thought experiment appears first in Kripke’s *Naming and Necessity*, and can be paraphrased as follows\(^2\). Consider two identity statements:

1) \(\text{Water} = \text{H}_2\text{O}\)

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\(^1\) By ‘thinking’ here I mean genuinely and organically believing it to be the case, so as not to include instances where I deliberately think the sentence “I am in pain” with no real force behind it.

\(^2\) Kripke himself uses “Heat = Mean molecular kinetic energy”, but water is easier to think about and less of a mouthful to continue referencing, so I will use water.
2) Pain = C-fiber firing

Let us assume that the words “water”, “H₂O”, “pain”, and “C-fiber firing” are all rigid designators. In our world, we take identity statement 1) to be necessarily true—water and H₂O are the same thing by definition. One may even say that H₂O is just the “scientific name” for water. And for the identity theorist, i.e. anyone who wishes to reduce mental states to brain states, pain in the same way just is C-fiber firing (or, quoting Dennett, “whatever brain state ‘turns out to be’ associated with pain”), and is likewise necessarily true (Dennett 1978, 450).

But if these terms are all rigid designators, then these identity relations must hold in every world where such objects exist. However, although these identity statements are true in our world, in a sense we can see them as only contingently true. For instance, it seems that we can imagine a world where water exists that is not H₂O, or pain that is not C-fiber firing. And, since the terms are rigid, if water and H₂O are distinct entities in one world, they are distinct in every world they exist. This results in a paradox: how can these identity statements be both necessarily true and contingent at the same time?

According to Kripke, we can resolve this paradox in the water case by noticing what it is we are imagining when we imagine water that is not H₂O. What we are doing in this case is not imagining water, but just something that has the appearance of water. In this way, it only seems contingent that water = H₂O. In reality, it is inconceivable that we could have water that did not have the molecular structure it does. Really, what happens is we imagine “fool’s water”, which has the observable properties we are familiar with: a clear, non-viscous fluid with certain macroscopic properties that nonetheless lacks the molecular structure H₂O. However, Kripke argues, we cannot do the same with the pain identity statements. This is because, recalling our earlier characterization of the manifest image of pain, if we appear to be in pain, we really are in pain. So, we cannot appeal to appearances to explain away the apparent contingency. For saying that we can conceive of “the appearance of pain” as being present without C-fiber firing is just to say we conceive of pain itself without C-fiber firing.

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3 Kripke defines a word as a rigid designator if “in every possible world it designates the same object” (Kripke 1972, 48). In this way, the name “Benjamin Franklin” is a rigid designator, whereas “the inventor of bifocals” is non-rigid. This is because, in another world, the inventor of bifocals could have been some other person, whereas in every world Benjamin Franklin exists, he is necessarily Benjamin Franklin.

4 As it turns out, what we consider “water” in a folk sense is not really 100% H₂O, and pure H₂O is rare in natural settings. I use this identity statement, though, for convenience and ease of reference.
The fact that the contingency of \( 2 \) cannot be explained away is taken to mean that such an identity statement is false, and that in general, mental states cannot be identical with brain states. This is a powerful conclusion and has shaped many discussions around the nature of qualitative states and physicalism in general. However, say we want to question that the move from conceivable to possibility is a valid one (this is a reasonable reservation, as I will revisit in a later section). Let’s suppose then, that it truly is necessary that pain = C-fiber firing. Levine argues that even if this identity is necessarily true, we can’t understand how it could be (Levine 1993, 548). This is in contrast to the water identity, because “Once all the standard superficial properties are explained by reference to the structure of \( \text{H}_2\text{O} \) molecules and general chemical laws, there seems to be no substantive cognitive significance to the question of how water can be \( \text{H}_2\text{O} \).” The argument here is that, even if we had a suitable neuropsychological theory of qualitative states, it would remain unintelligible why this identity holds. This “explanatory gap” is the epistemological version of the metaphysical disjunction claimed by Kripke’s modal argument.

I think it is interesting that pain experiences are taken to be the token qualitative mental states in question when talking about these particular thought experiments. In a conversation where visual experiences, especially, color, serve as the favorite representative of qualitative states, it is intriguing that pain experiences are so heavily favored here. The reason for this, I think, is that pain experiences are taken to be a more vivid illustration of the coincidence between mental appearance and mental reality. Pain seems to us to be a simple, unified experience whose appearance is essential to its existence. Since that is all there is to know about pain, we end up with a concept is entirely nebulous, and divorced from the neuropsychological processes that identity theorists are supposed to couch it in. There is, as I will argue, considerable evidence that we should not accept this. Given a suitably detailed conception of pain, I hope to alleviate the threats from both metaphysical and epistemological “gaps.” The first step to outlining a better concept of pain is to acknowledge that it involves multiple kinds of experiences.

3. What makes for a pain?

I wish to distinguish, as Nikola Grahek does, between the sensory-discriminative aspect and the emotional-motivational aspect of pain. Because our pain experiences almost always have components of both of these at once, it is easy to think they are all necessary components of a single, unified experience. However, understanding pain experiences as an interaction of these two parallel processes makes for a much clearer model. Not only is this distinction supported anatomically and
functionally, but there is a double dissociation of the two aspects as evidenced by the various pain disorders I will turn to soon.

To appreciate both aspects of pain as distinct but complementary parts, it is helpful to consider what adaptive role pain plays. As seen in the unfortunate case of congenital analgesia, pain serves an indispensable position in our well-being and self-preservation. Both the sensory-discriminative and emotional-motivational parts of pain contribute to the ability to effectively preserve one’s health. Sensory-discrimination of pain provides information about the location, intensity, temporal profile, and nature of stimuli that do actual or potential harm to body tissue. This amounts to the qualitative character of the pain, or the particular way pain physically feels—as being in the arm or leg, sharp or dull, burning or stinging, etc. The emotional-motivational aspect of pain complements this by eliciting unpleasantness, reflexive escape responses, and avoidance/protection behavior. Such affective and behavioral responses are in appropriate to the scope and intensity of damage as indicated by the sensory-discriminative sense. It is easy to see how the emotional-motivational component of pain serves well the goal of preserving well-being and protecting the body. If all we got was the discriminative information neutrally presenting to us “you’re going to be injured”, we would not necessarily spring to action or respond in the appropriate time or manner.

A vivid example is the failed attempt to make an external “pain sensor” for leprosy patients. Leprosy patients suffer from peripheral nerve damage to C- and A-delta fibers, and in the same vein as congenital analgesics suffer grave injuries because they do not notice when they occur. Because of this, a project called “A Practical Substitute for Pain” was created by Paul Brand that aimed to equip patients with artificial “pain sensors” that registered potentially damaging conditions to the extremities. This information would then appear to the patient in a non-painful way, like a blinking of lights or annoying sounds. The product was (jokingly) referred to as “a pain system that warns of danger but doesn’t hurt. In other words, we’ll have the good parts of pain without the bad!” (Brand and Yancey 1997, p. 192). The project was a spectacular failure. Patients viewed the flashing lights and buzzing noises as a nuisance interfering with their activities, and would ignore the signals or turn the device off. Without the “hurt” of pain, there was no motivational force to protect the endangered extremities. Fingertips were still lost even though the device accurately represented the danger at hand.5

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5 The engineers on the project realized that only pain that hurts motivates the right adaptive response. Since some pain perception was still present in the armpits and groin areas, the engineers wired up the device to
Another piece of evidence highlighting the role the emotional-motivational aspect plays comes with patients with pain asymbolia. These patients, unlike the leprosy patients, have intact sensory-discriminative capacities for pain, and can identify painful stimuli from non-painful stimuli. However, they are not bothered by painful stimuli, showing no negative affectual response—in fact, the common reaction to severe injury is for these patients to laugh, smile, or even approach the source of pain. While a more fleshed-out account of pain asymbolia appears in the next section of the paper, suffice it to say that pain asymbolia patients indeed register the information from sensory-discriminative sources, acknowledge this information as a sensation of pain, yet lack completely the negative affect that usually accompanies it, and have no desire for it to stop. As a consequence, pain asymbolics often suffer the same untreated injuries as congenital analgesics—burns, bruises, and broken bones, despite being detected, are no cause for alarm or action and therefore turn into more serious health issues.


1) To distinguish sensory pain from nonphysical pain or mental suffering
2) To set pain apart from other unpleasant sensations by its distinctive quality
3) To differentiate it from other, phenomenally similar sensations and give unity to the sensations that pertain to the sensory modality of pain

The first two of these roles are relatively straightforward. The first distinguishes pain from the unpleasantness from things like hearing a dissonant chord on the piano or seeing a harsh color contrast. While we may describe the latter cases as “painful”, we do so only in a metaphorical way, likening these purely mental unpleasant events to the genuine cases of physical pain as an analogy. Similarly, the second role distinguishes pain from other unpleasant experiences, but these unpleasant experiences are physical or sensory in nature. To list a few such examples: nausea, the smell of rotting flesh, and the taste of limburger cheese. These are all unpleasant physical sensations, but we would not call them pains, since they lack the distinctive, unifying pain quality. The fact that they are

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send a shock to these areas instead of just blinking a light. However, this failed too as the leprosy patients would just turn off the device when it shocked them in the middle of a task.
accompanied by unpleasant affect and desire for them to stop demonstrates that emotional-motivational features alone are not sufficient for a true pain experience.

Rather, pain is a distinct sensory modality, or as Grahek calls it, an “experiential determinable that gives unity to sensations that carry its mark and sets them apart from sensations of other modalities that are qualitatively similar to pain” (ibid. 97). Note that we can discriminate between sharp and dull pressure within the modality of touch, or between stinging or burning within the modality of temperature. However, the qualitative character is discontinuous between the former and latter cases—touch and temperature sensation belong to two modalities. However, sharp, dull, stinging, and burning pain are all experiential determinates of the single modality of pain, just as different hues are to color vision. For this reason, it is possible to feel a mechanical sensation as sharp, but not painful, because it lacks the relevant pain quality. This is bolstered by dissociation syndromes and clinical evidence in the following section.

It is because of this former observation about the unifying nature of the sensory pain modality that I contend that we take the “mental reality” of what the sensation of pain is to be the sensory-discriminative aspect. This is the aspect of pain that constitutes its qualitative character. It is in this aspect of the pain experience where we have the distinctive “pain quality” that accounts for our ability to discriminate between different locations, types, intensities, and temporal profiles of the pain experience. This information serves as input to the emotional-motivational aspect of pain, and is therefore prerequisite to any pain experience that plays the proper functional role. Just having the negative affect and relevant desires cannot make a genuine case of pain—it needs to be determined by the presence of pain quality, otherwise nausea would be pain, or more generally, pain with no spatiotemporal profile would be possible. While it is true that lacking the emotional-motivational component of pain renders the sensation such that it fails to protect the well-being of the body (as seen in pain asymbolics), it is the sensory-discriminative information that qualifies the experience as belonging to the pain modality, whether it succeeds in producing the intended behavior or not. Above all, it is the fact that the qualitative character of pain—the distinctive pain quality that makes us feel a dull burning pain feel uniquely different than a sharp stinging pain—that motivates the decision.

To make this a little bit more precise, I believe that when talking about the sensory modality of pain, we should refer only to the sensory-discriminative aspects, and when talking about pain experience in terms of the functional role it plays, we should include both the sensory-discriminative aspect and the emotional-motivational aspect as necessary. So far, I have been dancing around the
details of the relevant neurophysiology and dissociation disorders. These are the clearest evidence for this distinction, and outlining the scientific information will make the relationship between the sensory-discriminative and emotional-motivational systems much clearer. First, a brief note about C-fibers.

3. Neurophysiology of sensory-discrimination

Far from being a mystery, much is known about the individuated structures and functions of nociceptive (pain-sensing) nerve fibers. While C-fibers are famous among philosophers, there are actually three kinds of pain-sensitive fibers: C-fibers, Type I A-delta fibers, and Type II A-delta fibers. These are primary afferent nerve fibers, meaning they have diffuse nerve endings found underneath the skin, and are found all over the body. They transfer signals from subcutaneous regions (some deeper than others) to “relay stations” in the spinal cord, which continue on to the brain. The anti-identity theorist may object here, saying something like, “so what if there are three or four or seventeen types of fibers? This is merely a minor detail that has no philosophical bearing on the modal argument.” Perhaps this is true, but I think the structure and function of these different kinds of nerve fibers enriches the relevant theory that is supposed to explain the relationship between the mental state and the physical state. With respect to Levine’s arguments about intelligibility, once we appreciate the relevant scientific facts, it becomes much less of a “mystery” how such neural patterns lead to the associated mental states.

The three types of nociceptive fibers have been extensively studied in both monkeys and humans. Thanks to the ever-expanding scope of technology available to neuroscientific research, individual intracellular recordings have been taken of all the different kinds of primary afferent neurons involved in mechanoreception, whether nociceptive or non-nociceptive. What this means is that we have a reliable source of data about precisely what kinds of stimuli elicit a response in each kind of fiber. We have mapped the size and location of the receptive fields of each kind, the thresholds required for action potential, and the exact neural pathway taken for each kind of fiber. This is done not only by passively recording activity while providing real stimuli, and recording the physiological and psychological response, but also by actively stimulating individual nerve endings to see what response occurs independent of a real stimulus. Below is a table showing the main denominations of primary afferent neurons (there exist more subtypes, but that is irrelevant for our purposes).
As seen in the table, the three nociceptive nerve fiber types differ in terms of what stimuli they are responsive to, as well as their anatomical structure. For instance, C-fibers are sensitive to potentially damaging chemicals, heat across a wide range of temperatures, and the chemical capsaicin. In contrast, A-delta fibers seem to respond to more mechanical disruption, and the more extreme ranges of temperature. Crucially, C-fibers are responsible for the long-lasting, duller, more diffuse kind of pain, while A-delta fibers are responsible for the sharp, acute, and stinging pain. These pain qualities are reliably elicited by these specific kinds of nociceptive fibers. Notice, that what we are talking about here when talking about “dull” or “stinging” are different determinate qualitative characters of pain! Finally, I also want to point out the clear anatomical differences between the fibers: C-fibers are the thinnest, and have little to no myelin surrounding the nerve, whereas A-delta fibers are thicker, and are surrounded by a wide sheath of myelin. In addition, the distribution of C-fibers is more widespread, with larger receptive fields than A-delta fibers. Thickness of the nerve and degree of myelination are both factors that affect conduction speed—the thicker the nerve, and the more myelin, the faster the signal travels from the afferent site to the spinal cord, and thus to the brain.

The reason I am mentioning the anatomical structure the different kinds of cells is because I believe there is a non-trivial connection between them and the qualitative character of the pain sensation they produce. There is a clear electrochemical reason for the fact that C-fibers tend to produce the pain quality “dull”, “diffuse”, “throbbing”, while A-delta fibers tend to produce the pain quality “sharp”, “stinging”, “stabbing.” The pain quality perceived is a direct consequence of the physical structure of the nerve fibers. The pain quality we feel actually resembles, in a way, a

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6 Myelin is a fatty, compressible sheath of tissue that surrounds nerve fibers (not just nociceptive ones). It is thought that they serve a multitude of functions, but one of the most important is the regulation of conduction speed. Demyelination can have devastating effects—one famous demyelinating disease is multiple sclerosis.
physical feature of how the signal is being conducted. Suppose, within the sensory-discriminative pain modality, we construct the identity statement “dull, diffuse pain P = spatiotemporal pattern of firing for C-fiber F.” Is this really any more unintelligible or incoherent than “water = H₂O” or “Heat = mean molecular kinetic energy”? Here, the relevant physical state actually has a very transparent and relevant similarity to the qualitative character of the mental state. Can the same be said for the way microscopic and macroscopic properties of water line up? The collective action of polar covalent bonds is, to me, an even less transparent, intelligible explanation for the observable surface tension we see in water. But, I accept the latter because it is explained by a robust, descriptive scientific theory that has been widely studied. Why not treat the neurophysiological data as such a theory? The basic neuroanatomy of afferent nerve fibers is well-studied and not considered part of the “cutting-edge” of current neuroscience, but part of the biological canon. Granted, there is much still to discover, but treating the connection between the current physical correlate and the mental state here as completely without rational basis, as Levine does, seems wrong.

As a segue into my next topic—dissociation disorders, I will briefly mention the overall path of information after stimulation of the C- and A-delta fibers. The nociceptive fibers travel to the dorsal root ganglion, upon which they cross the midline, and ascend in the spinal cord to the thalamus, in the brain. Notably, this pathway is entirely distinct from other ascending pathways up the spinal cord that encode non-nociceptive information. The non-nociceptive pathway traverses the midline much later, in the medulla. This provides further evidence of a distinct sensory modality of pain that differs from the modality of pure non-nociceptive mechanical touch. Once the nociceptive information reaches the brain in the thalamus, it makes numerous connections with somatosensory cortex, insula, the forebrain, and the limbic system. These areas are associated with spatial mapping of the body, judgment and reasoning, representation of self, and emotion.
4. Implications of dissociation disorders

I would like to closely compare the two pain disorders mentioned so far: pain asymbolia and congenital analgesia. To frame the difference clearly, it may help to phrase these disorders in terms of the primary distinction I am making between sensory-discriminative and emotional-motivational aspects of pain. Pain asymbolics retain sensory-discriminative abilities, while exhibiting no emotional-motivational response to painful stimuli. Congenital analgesics, on the other hand, lack both sensory-discriminative abilities and emotional-motivational responses. As such, the outward expression of these two disorders is very similar—we normally know whether someone is in pain because they cry aloud, make a jerking motion, complain, seek help, or otherwise act as if they are injured. Because both types of patients lack these responses, they seem behaviorally similar. They also suffer similar health problems as a result, typically accumulating injuries that go unnoticed which lead to chronic problems like infection and joint damage. For these reasons, pain asymbolia and congenital analgesia are easily mistaken for one another, and they have caused much confusion in the world of medicine.

However, there are behavioral, psychological, and neuroanatomical reasons to believe they are distinct disorders. First and foremost, we have the report of the patients themselves. It is important to preface this by saying that it is necessary for the diagnosis of both these diseases that the patient show normal cognition, memory, and sensation other than their ability to feel pain, and have no history of mental illness or pain disorder. What congenital analgesics report is that they do feel pain when noxious stimuli are applied, saying things like “It hurts indeed, but I do not know what that really is”, or “I feel it indeed; it hurts a bit, but it doesn’t bother me; that’s nothing” (Pötzl & Stengel 1937, 180). In contrast, congenital analgesics deny feeling any pain whatsoever when noxious stimuli are applied. But, this is not due to anesthesia (total loss of sensation)—congenital analgesics can sense mechanical and heat stimuli just fine, and are able to distinguish, for instance, “…the sharp and blunt ends of a pin and had no difficulty localizing the pinprick” (Barber 1959, 443).

Further behavioral differences arise in the responses patients give to what would normally be incredibly painful stimulation. Congenital analgesics show a resounding indifference to the stimuli, showing not just the absence of wincing, crying, recoiling, or verbal report of pain, but no behavior at all. Pain asymbolics, on the other hand, show positive behavior when administered noxious stimuli. One patient “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” (Shilder and Stengel
1928, 147). Smiling and laughing is not an anomalous manifestation of pain asymbolia, but rather the norm. Upon noxious stimulation, patients often started to laugh and would stop abruptly and immediately when stimulation ceased. That this is a reliable and highly correlated behavior with noxious stimulation indicates that it is more than a meta-appraisal of the situation. Pain asymbolics are usually unaware that their response to pain is abnormal or that they are impaired at all (Hemphill & Stengel 1940, 257). The “approach” behavior exhibited by pain asymbolics is also bizarre and not anomalous. Rubins and Friedman, among other researchers, specifically note a “tendency toward pain-induced approach behavior”, often described by patients going out of their way to offer their hands, arms and bodies for more painful stimulation (Rubins & Friedman 1948, 559).

In addition to this slew of behavioral and psychological data suggesting that pain asymbolia and congenital analgesia are indeed two distinct disorders, there is also neuroanatomical evidence that conforms with my positing the differences in terms of sensory-discriminative ability and emotional-motivational response. Pain asymbolic and congenital analgesic patients show clear patterns of neurological damage:

“Lesion location was the most important factor associated with the development of the syndrome, as the insular cortex was invariably damaged in every patient. In fact, the development of the severe and persistent [pain asymbolia] syndrome in one of our patients, following a discrete ischemic lesion in the posterior insula and parietal operculum, strongly suggests that the involvement of such structures may be sufficient for the production of the syndrome.” (Berthier, Starkstein, and Leiguarda, 1988, 41)

Research in the years since has only confirmed that pain asymbolia is closely associated with posterior insula and parietal operculum damage. The significance of this is that these damage to these structures results in a sensory-limbic disconnection. Grahek summarizes it in the following way: “Since the somatosensory cortical areas responsible for the detection of sensory features of noxious stimulus are spared in asymbolia patients, they are able to recognize the modality, qualities, intensity, and location of noxious stimuli. However, the damage to the insula and parietal operculum may disrupt the connections between the sensory and limbic structures, impairing the subjects’ ability to attach appropriate emotional significance to the painful stimuli” (Grahek 52). That the posterior insula and parietal operculum, and the sensory-limbic connection they constitute, are essential for the assessment of the affective-motivational content of pain in monkeys and humans has been supported (Mesulam and Mufson 1985, 216; Robinson and Burton 1980; Dong et al. 1994). This is all consistent with the idea that pain asymbolia can be explained as an intact sensory-discriminative ability with an impaired emotional-motivational system.
Compare this striking neurophysiological data with what is known about congenital analgesics. As the name implies, it is heritable, and has been traced to the mutation of a specific gene whose functions are well known (Cox et al. 2006). The gene mutation in question is that of the SCN9A gene, which encodes the synthesis of the Na\textsubscript{V}1.7 sodium ion channel. Sodium ion channels in particular are important in neurons throughout the body, as they allow for the propagation of action potentials—the basic unit of excitation for a neuron. The specific species of sodium ion channel Na\textsubscript{V}1.7 is selectively expressed in nociceptive neurons—it is strongly and disproportionately expressed in C-fibers, A-delta fibers of both types, and dorsal root ganglion cells, which relay the signals from the fibers to the ascending spinal pathway. Na\textsubscript{V}1.7 is also present in lower concentrations in olfactory neurons (Zufall et al. 2012). Non-coincidentally, congenital analgesics usually have an impaired or absent sense of smell. Finally, other than some other symptoms related to autonomic processing (tear production, susceptibility to fever), no significant neurological defects are present. Lesions and abnormalities in the brain are absent, and intellect and emotional processing are normal (Zhang et al. 2016). What this all suggests is that congenital analgesia can be explained in terms of an absent sensory-discriminative ability, with an intact emotional-motivational system. However, since there is no output of the sensory-discriminative system, there is no input to the emotional-motivational system, and therefore no relevant expression in behavior.

Taking the neurophysiological accounts of both disorders together with the behavior and reported experience of the patients, we get strong support for the working model of pain set out so far. That is, that standard pain experiences happen when sensory-discriminative systems represent pain quality in response to stimuli, and relay this information neurally to emotional-motivational systems, where typical, externally expressible behavior and cognition are set into motion. This model of pain encompasses two constituent processes that work closely together in normal cases, and therefore appear to be a unified experience. However, as these non-standard cases show, the two processes are separable. So at this point, we have to make a choice as to what to consider a genuine instance of “pain”. As I mentioned in section 2, I am inclined to define pain strictly as the sensory-discriminative aspect of the experience. That is, the subject’s ability to discriminate among noxious stimuli via the pain qualities the modality presents, is the necessary and sufficient criterion for pain. This means that the pain felt by pain asymbolia patients is genuine. But, if one takes pain to be the manifest image of pain, i.e. the unified experience that includes emotional-motivational as well as sensory-discriminative aspects, then the pain felt by asymbolia patients is something mysterious or
ersatz. What this amounts to is that it is possible for the appearance of pain and reality of pain do diverge.

5. Return of the gap

What does this mean for the modal argument and the explanatory gap? Let’s reconsider the two identity statements from the beginning:

1) Water = H₂O
2) Pain = C-fiber firing

Now, we are taking pain to be a sensory modality that is defined by the ability to discriminate between noxious stimuli, and we are taking the neural correlate to be the nociceptive system of primary afferent nerves and the ascending spinal pathway, which includes both C-fibers and A-delta fibers of different kinds. Crucially, we don’t include the emotional-motivational aspect of the experience, nor do we include the neural systems that support it. After all that, we can reword the statements as:

1) Water = H₂O
2) Pain = C-fiber … and A-delta fiber firing

This incredibly anticlimactic reveal hopefully adds more than just a few words. When we “imagine” pain without the relevant neural state, I would argue that we cannot help but imagine the manifest image of pain, in all its vague and unified glory. Perhaps, in the same way that we imagine “fool’s water” that only has the superficial properties of water, we imagine this unified concept of pain, which, as stated in the beginning, amounts only to how the pain “appears” to us. If we are to really pass the conceivability test in earnest, we must imagine a genuine case of pain, as a bare sensory modality outside of the emotional baggage it carries, without the relevant neural states. Is this possible? To answer this, we would need to ask a pain asymbolia patient, because that is how they experience pain sensation (though, with a certain neural state). But suppose Kate the pain asymbolic is a proponent of Kripke, and says, “No! I’m still imagining the pain I feel, as a bare sensation, without C- or A-delta fiber firing!”

At this point, it is unclear how to evaluate such a statement. The modal argument relies on a move from conceivability to possibility. The best defense of this move’s credibility comes in cases when appearance and reality are the same thing. This is why it is supposed to work for the pain case.
But, if we deny that appearance and reality converge for pain, as I have spent the majority of this paper trying to demonstrate, this move becomes dubious. I could still say “No! I’m really imagining water isn’t H₂O, even if it’s not actually possible.” At this point, it becomes confusing what it really means for something to be conceivable, and how Kate’s claim is any different from mine. The water identity, when it comes down to it, is backed by a theory—the laws of chemistry. Once we accept that appearance and reality can diverge in the case of pain, we can construct an identity statement that is similarly backed by a robust and accepted theory—neurobiology. It is hard for me to see how a chemical basis for identity statements is any better than a neurobiological one.

At this point, we might retreat to the epistemological gap proposed by Levine. Suppose we assume that it is indeed a necessary identity that 2). Levine’s original claim is that even if the identity is necessary, we can’t understand how it could be. Claims equating mental and physical states are supposed to be incoherent, lacking a rational basis that is present in statements like 1). But I stress the point I made when discussing the neurophysiology of pain sensory-discrimination. There is arguably a more coherent reason to believe that pain quality can be reduced to a neural pattern, than there is for water = H₂O. We describe pain quality with words like “sharp”, “burning”, “dull”. The relevant neural patterns involved in representing these different qualities actually share some of these properties in an intelligible way. The way the stabbing, acute pain of a needle prick feels has everything to do with the fact that the means we have for sensing such a stimulus are structured in a way such that the information is conveyed intensely (high action potential frequency), briefly (corresponding to duration of stimulus), by receptors that only activate when acute pressure is applied sufficiently deeply. The pain quality we feel resembles or reflects the physical properties of the stimulus. To claim that it is unintelligible or incomprehensible that pain quality just is neural firing pattern, while it is perfectly rational that the brown, solid wood desk I’m sitting at is composed of tiny, colorless particles that are mostly empty space, seems wholly unjustified.

Both the epistemological and metaphysical “gaps” between pain and the relevant neural states result from a willfully vague, undetermined definition of both pain and the relevant neural states. The standard force behind the modal argument and explanatory gap comes from 1) the assumption that the way pain really is and the way pain appears to us are the same, and 2) the uninformative, underdetermined physical state that is supposed to be on the other side of the equation. The reason these are so attractive stems from the fact that pain experiences are unusual. Unlike other sensory modalities, like vision and hearing, pain experiences (unless you have pain asymbolia) seem to necessarily include certain affectual and behavioral factors. This is due to the
hidden interaction in standard pain experiences between two subsystems of sensory-discrimination and emotional-motivational response. This interaction is hidden because it is not disclosed by introspection about our own pain experiences. The standard response has been to enthusiastically embrace the mystery, and take mental appearances at face value. But, given an honest attempt at investigating the nature of our pain experiences using the neurobiological and neuropsychological evidence available to us, we get closer to a non-mysterious conception of what pain is.
Citations


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