PAIN, CHRONIC PAIN, AND SUFFERING

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The impact of pain on our lives is staggering. Chronic pain affects approximately 116 million adults in the United States annually, and this figure does not include people in long-term care facilities, the military, or prison. It costs up to $635 billion a year in (largely ineffective) health care and lost wages (Committee on Advancing Pain Research, Care and Education 2011). This is more than the costs for cancer, heart disease, and diabetes combined (Pizzo and Clark 2012).

Back pain in particular is pernicious. The recent Global Burden of Disease Study estimates that back pain is one of the top ten conditions that cause disability or early death (Vos et al. 2012). The chance that a citizen in an industrialized nation will suffer low back pain in his or her lifetime is somewhere between 60% and 70% (Kaplan et al. 2013). This pain causes more time to be taken off from work than any other disease or injury, which in turn exacts a high economic cost on the individuals, their dependents, industry, and governments (Andersson 1997, Taimela et al. 1997). In the United States alone, estimates are that 149 million workdays are lost each year due to back pain and its accompanying disability (Katz 2006, Rubin 2007).

But what is pain exactly? It seems to be something with which we all have intimate experience, but at the same time, trying to articulate its fundamental nature is challenging. Is it information about one’s body? Is it an imperative to nurse injured tissue? Is it akin to an emotion? The answers are not obvious—and, not surprisingly, more complicated that many might assume.

1. False Things Said about Pain (and What Is True about Them)
Philosophers used to take pain as an easy and obvious example of a simple sensation when reasoning about what mental states are and how we know about them (e.g., Kripke 1980). They did not think too much about what pain was exactly, except as an obvious thing that we have all experienced and know well. Daniel Dennett (1978) put those assumptions to rest, as he adroitly pointed out how complicated pain processing is. He argued that our “folk” conceptions of pain—how we think about it in everyday life—were confused. Our ideas were so confused, in fact, that they were contradictory. For example, we believe that pains are inherently unpleasant, yet there are cases of pain that typically do not bother the holder (e.g., sore muscles in athletes). And we believe that pains are causally connected to tissue damage, yet quite often we can experience severe trauma to our bodies, yet not feel pain; this is a common occurrence in the battlefield. Nothing like what we intuitively think pain is actually exists...
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(see also Hardcastle 1999). [As an aside, Kevin Reuter has done some interesting experimental work that suggests that philosophers are wrong in what they believe the “folk” think pain is (2011). His data demonstrate that people actually have sophisticated and complicated views about pain.]

In the intervening years, many philosophers have tried to analyze the fundamental nature of pain, attempting to identify a simple core underlying all of the complicated details. Most recently, Colin Klein (2015) has advocated that pain is just an internal command to protect a part of the body. Klein compares pain to other homeostatic drives we have, like hunger, thirst, or itch. These are all things that are directly motivating. To be hungry is to be motivated to eat. Similarly, to be in pain is to be moved to protect.

This view has the perhaps paradoxical implication that pains aren’t actually painful, at least not at their core. We might have a secondary reaction of “ouch,” but we often have pains of which we are not even aware, like those that force us to adjust our posture on an ongoing basis. Just as we can be hungry but not be bothered by it, so too can we be in pain but not be upset by it. (At least for pains that are not severe.) Pains are also uninformative. Having a pain does not tell you why the body part was damaged, how it was damaged, how it might be fixed, or sometimes (in the cases of referred pain) even where it was damaged.

The problem with this view is that not all of our homeostatic systems require a concomitant sensation. Think about the very important systems in us directed toward maintaining a constant temperature. When we get hot, we sweat. Our bodies do that automatically, without needing to run anything through our brains. If pain is a homeostatic system, should it be like being hungry, which does require our brains’ cooperation to get us back to equilibrium, or is it like being warm, which does not?

If we consider the pain reflex, which is what jerks our hands off of a hot stove, we can see that that sort of pain processing—the sort that forces you to protect your tissues—happens outside of consciousness awareness. Indeed, the movement does not even require the brain to be enacted. Nociception, the dedicated peripheral response to noxious stimuli, travels to the spinal column, which then can direct the muscles to withdraw from the heat source. Only later do we feel any sensation, after we have moved our hand. Why does that happen? Why would we feel pain after the fact?

Even more confusing is the fact that if we “listen” to our pain, we appear to get into more trouble than if we don’t. In a set of experiments conducted 30 years ago, Wilbert Fordyce took patients with back pain and divided them into two groups. One group was told to take some muscle relaxants and rest in bed (the common “cure” from back then, which we now know is very bad advice) until they felt better. The other group was told to take the same medication and rest in bed, but these participants were given a specific length of time to do both. In other words, in the first instance, patients were to use their pain as a guide for protecting themselves; in the second instance, patients were to ignore their pain in favor of following a doctor’s orders (Fordyce et al. 1986). Fordyce learned that the second group fared much better, not just over the next few weeks, but even six months later. The second group felt better, was able to move with greater agility, and worked more effectively than the first. Letting your pain tell you what to do might not be a good idea. Indeed, we now know that often moving our bodies during healing, while increasing pain, also promotes recovery.

Klein appears to be right that pains motivate us to protect our bodily integrity (in some sense of “motivate” that we still need to flesh out), but he appears to be wrong that this is all pain is. His story does not explain all facets of pain processing. In particular, it does not explain why pain can and does lead us astray. We need a more nuanced view.

A second, very popular, view of pain in philosophy agrees that there is no single core to pain. Instead, pain is a duality; it has two cores, as it were. David Bain (2011) suggests that pain
provides us information about damaged tissues (namely, that it is damaged) and is something that is inherently unpleasant. In contrast to Klein, who thinks that the unpleasantness of pain is a secondary reaction, Bain thinks that being motivated to protect the body is the secondary reaction; it is not part of pain proper. This view of pain lines up nicely with scientific perspectives on pain since the turn of the 21st century.

Historically, pain processing also was divided into two fundamental components: a processing stream that encodes for the emotional response to pain and a processing stream that analyzes location and intensity. This division goes back to Sir Charles Scott Sherrington’s and Sir Henry Head’s work in the early 1900s on the function of neurons and the somatosensory system (Sherrington 1906, Head and Holmes 1911). The importance of this division was underscored in the 1960s, as neuroscientists discovered two different spinothalamic tracts involved in pain processing: a medial (in the middle) pathway involved in processing affect and a lateral (on the outside) pathway involved in sensory encoding. This two-dimensional structure for pain is ubiquitous in the scientific literature, as well as in philosophical works (e.g., Aydede 2006).

But, important for this discussion, scientists have not made much progress in uncovering the details of the putative duality of pain, even though they have been working on it for over half a century. Indeed, as Vania Apkarian maintains, evidence for division remains “fairly weak” (2012: 6). There are three challenges to the hypothesis. First, there is a lot of crosstalk in our nervous system, starting in the spinal tracts and continuing on up through the cortex (Giesler et al. 1981). Such crosstalk prevents any particular area from being either just affective or just sensory and suggests that various areas in the spine and brain process both types of information together. Second, verbal reports (or other behavioral indicators) of pain intensity (a sensory parameter) and the magnitude of pain’s unpleasantness (an affective parameter) are tightly correlated, which makes differentiating the two very difficult. And finally, some brain regions, like the insula, do not match up with either type of processing, for it is seemingly concerned only with magnitudes of responses and not with the emotional or sensory dimensions per se (Apkarian 2012).

The insula integrates magnitude information about a wide range of sensory input, including pain, and then feeds those calculations to the nucleus accumbens (NAc). The NAc calculates the potential reward values for its input and then sends its estimation to other limbic areas involved in analyzing rewards. These areas talk to our cortex, which concerns itself with planning and promoting actions (Cauda et al. 2011). This process, by the way, is not only what happens when we experience pain, but it is also what our brains do when we seek some intuitively pleasurable outcome. So what is going on? Are pains and pleasures two sides of the same coin?

It appears that our brain does in fact process both affective and sensory information when processing pain, as duality theories suggest, but these signals intermingle with one another instead of being divided into two separate circuits. In addition, when we look at which brain areas are involved in pain, we see much more than just these two types of information involved. Perhaps we should stop trying to make pain into something that has a simple nature and take a second look at what we know about the neurobiology of pain processing. As we shall see, doing so will also connect pain with pleasure directly.

Once we stop trying to divine pain’s ultimate nature or divide pain into a few simple components, we make room for a new way of looking at nociceptive and pain processing data. For example, Apkarian’s lab has found particular temporal sequences in the activation of particular brain areas that correspond to anticipating a painful stimulus, the perception of the pain itself, and relief after the pain is over (Baliki et al. 2010). The NAc and the anterior portion of the insula are most active just at the start of a thermal painful stimulus, but when subjects
indicated that they actually felt pain, these regions quieted and the posterior portion of the insula and the anterior cingulate became the most active. Finally, as the stimulus was returning to baseline, the peri-acquiductal gray region became active and the posterior insula and anterior cingulate returned to baseline as well. We can thus identify three distinct and different networks that are sequentially activated during acute pain processing. Moreover, all of these regions are part of our motivational system that calculates and experiences risk and rewards.

This suggests a different approach to understanding pain processing: the pain system as a complex system of motivation. The basic idea is that normal acute pain, which usually indexes tissue damage, first prompts us to escape or avoid our current situation in order to minimize physical harm and then, when the pain stops, provides us with a sense of relief. Together, these two reactions—avoidance and the feeling of relief—not only protect our bodies but also contribute to our being able to predict the utility and costs of competing behavioral goals. That is, we learn from past pain experience how to evaluate the severity of threats, which then informs which goals we pursue and which behaviors we choose.

One consequence of adopting this perspective on pain processing is that it removes, or at least seriously diminishes, the strong distinction we normally assume between pain and pleasure. Intuitively, we believe that pain is unpleasurable and pleasure is, well, pleasurable. But if our pain system works because it ultimately gives us a sense of relief, then it too could be considered, perhaps, part of our pleasure-inducing system. The pain motivates us to do something, not just because we think it will hurt, but also because when we successfully do the something required to stop the pain, we feel more than the absence of pain. We also feel good. Taking this perspective means altering one’s views on what a pain is. From this perspective, pain becomes a series of brain states, each of which has a different function in our cognitive economy.

A second consequence of adopting this perspective is that it denies that there is a brain circuit specific to pain processing (Iannetti and Mouraux 2010), contrary to popular theorizing (see, e.g., Brooks and Tracey 2005). Pain processing is simply part of our ongoing risk-reward calculations. Indeed, it might turn out that Melzack’s (1989) original idea for a nonspecific, widely distributed network of neurons that crosses many areas of the brain and underlies pain perception might be correct after all. We have one reward calculator, as it were, and some actions of this calculator are what we normally consider to be pain processing. Again, the line between pain and pleasure becomes quite fuzzy, at least from the brain’s perspective.

But things get even more complicated, for not all pains are created equal. So far, we have been discussing acute pains, which are triggered by a noxious event and then resolve. But some pains, like some cases of chronic back pain, do not readily (or ever) resolve. These pains, it turns out, are quite different from regular acute pains.

2. Chronic Pain Is Not Acute Pain That Does Not Stop

One might reasonably think that chronic pain is just an acute pain that does not go away, but this is not the case. Acute pains and chronic pains are quite distinct kinds of bodily events, with different impacts on the body and on one’s psychology. To take one example of this: chronic pain is represented in different areas in the brain from acute pain, in part because the brain rewires itself when pain becomes chronic. For example, as discussed above, we know that the insula indicates the appearance and magnitude of acute pain. But activity in the medial prefrontal cortex (mPFC), a part of the limbic system, is correlated with chronic back pain. Interestingly, when chronic back pain patients also experience an acute pain, such as a burn on their arm, their insula lights up just as normal subjects’ would under similar conditions (Baliki et al. 2006). Hence, people with chronic pain can experience two distinct types of pain—chronic and acute—and these differences are reflected in their patterns of brain activity.
The way the NAc is connected to the rest of the brain is different in chronic pain patients as well. In normal subjects, the NAc and the insula are highly interconnected, but in chronic pain patients, the NAc shifts its functional connectivity to the mPFC. That is, in normal subjects, when the NAc lights up, the insula does as well, but in chronic pain patients, when the NAc is activated, the mPFC responds (Baliki et al. 2010). And, the more chronic pain, the stronger the correlation between activity in the NAc and mPFC. Chronic pain shifts what would be a normal pain reaction to a more emotional one.

In addition, the nearly continuous activation of these areas caused by ongoing chronic pain has specific effects on the brain’s firing patterns: the baseline level of activity in the insula and anterior cingulate is much higher in chronic pain patients than in normal controls. Such is not the case for other areas of the brain, not associated with pain, like the sensory cortices (Malinen et al. 2010). One hypothesis is that the near-continuous activation of the limbic areas shifts reward valuation, and these shifts in turn modulate learning and memory (Apkarian 2012). In other words, being in chronic pain fundamentally changes how one thinks, learns, remembers, and feels.

As a result of this rewiring, NAc activity differs between healthy subjects and chronic pain patients for instances of acute pain, especially during the “relief” phase of pain processing. Normal subjects’ brain activity signals quite reliably that a reward is coming, but chronic pain patients’ brains show activity that reflects a lack of predicted reward and perhaps even disappointment. Normal subjects would be happy and relieved that their pain is ending, but chronic pain patients would still have their chronic pain when the acute pain stimuli ends. Indeed, quite often an acute pain relieves or at least covers over the chronic pain. Under those circumstances, the chronic pain patient would be disappointed that the acute pain is ending.

This change in brain connectivity is a functional rewriting not specific to pain processing, for we see similar effects for monetary rewards in chronic pain patients—their brains show no real response to reward or loss (Apkarian 2012). In other words, chronic pain puts stress on our protective and adaptive motivational systems such that our motivational system fundamentally changes how it operates. And this change in functionality is so large that it distinguishes between normal subjects and chronic pain patients with an accuracy of more than 90% (Baliki et al. 2012). The evidence surrounding chronic pain processing indicates that it is intimately tied to our reward circuitry; chronic pain appears to be a disorder of our motivational/affective system.

Disorder is the operative word, for additional symptoms are associated with chronic pain, beyond the pain itself. Chronic pain patients also experience neuroendocrine dysregulation, fatigue, dysphoria, diminished physical performance, and impaired cognition and executive function (Chapman and Gavrin 1999). Chronic pain clearly is about much more than a simple sensation or bodily imperative.

A clever series of experiments from the 1970s demonstrates this point. Fordyce and his colleagues examined the ability of chronic back pain patients to exercise. At first, the patients were given a repetitive exercise to do and were instructed to do it “until pain, weakness, or fatigue causes you to want to stop” (Fordyce 1979). The researchers discovered that these patients, more often than not, stopped exercising on a number of repetitions that was divisible by five. If the capacity for exercise were really being controlled by pain, then one would expect to find a random distribution of repetitions. Fordyce hypothesized that when movements are easily countable, multiples of five comprise convenient mileposts. They become cues that indicate continuing to exercise risks increasing pain. So patients were stopping not because they could do no more, but because they anticipate (and dread) forthcoming pain.

To test this hypothesis, Fordyce then asked both chronic back pain patients and normal controls to exercise to tolerance in such a way that they could not count how much they were
doing. For example, in one trial, subjects were asked to ride a stationary bicycle that had no speed or distance indicators or gear controls in a room with no clock or windows. In these cases, the performance of chronic pain patients and normal subjects was virtually identical (Fordyce 1979). When the pain patients had no way to calculate their effort, they could not anticipate future pain as easily, which led to their being able to do much more physically.

Chronic pain heightens the affective aspects of pain, inducing stress, anxiety, and fear, as it diminishes the ability to think clearly and rationally. These affective dimensions, anticipating increasing pain and having no relief in sight, are key components in the suffering sometimes associated with pain. Although they may often go together, suffering, however, is something over and above pain processing. It is the secondary reaction.

### 3. Suffering and Chronic Pain

Suffering is not the same thing as pain, and in fact many instances of acute pain do not entail suffering, but suffering often accompanies chronic pain. What is suffering exactly? And how do we know?

Unlike pain, there are no peripheral sensory systems that respond to suffering. We only know that people are suffering when they report that they are. Surveys of advanced cancer patients, whom we often take to be exemplars of suffering, are revealing. The first thing we learn is that we drastically overestimate how much people with advanced cancer do suffer (Schulz et al. 2010). Indeed, only one-quarter of patients with advanced cancer agree that they are suffering at a moderate or extreme level (Wilson et al. 2007). The second thing we learn is that the symptoms associated with suffering resemble those of chronic pain: fatigue, dysphoria, disrupted sleep, hypervigilance, reduced appetite, impaired physical functioning, impaired cognition, and negative ruminations (Chapman and Gavrin 1999). The third thing we learn is that while complaints regarding suffering often revolve around pain, the emotions associated with suffering have a greater effect (Wilson et al. 2007). We might talk about pain, but our feelings impact us the most.

In a survey given to a wide range of people, when asked to indicate the “worst pain you have ever experienced,” many different types of answers were given, ranging from accidents and injuries to childbirth to illness to migraines. Only 4% of respondents answered that their greatest pain was emotional (Bendelow 2006). However, when respondents were asked individually to elaborate on their answers, the discussion quickly turned to feelings. One subject claimed a toothache was the worst pain he ever experienced, but talked about his anxiety attacks:

> It makes me totally separate from whatever was going on—it’s like an inner terror, it is like a physical pain at times, it’s like a vice on my temples and an incredible pressure on my head that does produce a headache but essentially it’s just a brooding feeling within the skull.

(2006: 66)

Another subject, who had experienced a horrible motorcycle accident in India while on vacation, talked about a psychological trauma instead:

> The emotional pain goes on longer—I think it’s somehow worse than a physical pain because that is usually comprehensible, logical and there’s a certain amount of control—you can get your head round it. I’d rather go through all the horror of smashing up my leg and all the being in hospital than two months of what I’ve just been through.

(2006: 66)
The point is that, while we might assume our greatest suffering is tied to pain, with a little prodding, we can see that often it is connected to emotional duress.

So what is suffering? Not surprisingly, there are a multitude of definitions (Kellehear 2009), but the consensus centers on suffering being a multidimensional experience of physical symptoms, psychological distress, existential concerns, and social-relational worries. It arises from the belief that one’s biological, social, or psychological being is being in jeopardy. Eric Cassell (2004) contrasts pain, which he sees as a threat to bodily integrity, with suffering, which he describes as a threat to the intactness of the self. It alters one’s sense of self, due to some kind of loss—pain, injury, deprivation, severe illness, some type of significant social or personal change.

Unlike pain, suffering is always a conscious experience. It is about mourning a loss, a sophisticated secondary reaction, dependent on complex interactions of cognition and affect. To suffer requires the ability to project one’s self into the future and to anticipate a continued loss. In general, those who are suffering are looking for healing, not for a cure; they are looking for a way to understand themselves in relation to the cause of their suffering (Cassell 1991).

How might one measure suffering, if it is so multifarious? A start would be to look at the case of complicated grief. (“Complicated grief” refers to grief that continues unabated at least six months beyond the time of the loss.) Grief normally activates the anterior cingulate, the insula, and the peri-acquiductal gray areas—all regions associated with pain. In some cases, however, grief does not subside as it should. Like chronic pain, it persists long beyond its expected trajectory. Its principle symptom is a yearning for the missing loved one so intense that it crowds out other wants and needs. And, just like chronic pain, complicated grief activates the NAc, part of our reward system, in addition to the pain areas just mentioned (O’Connor et al. 2008). (The activity of the NAc is depressed in cases of normal grief, just like in cases of acute pain.) Activation of NAc appears to be correlated with a sense of yearning for the lost love, which Mary-Francis O’Connor compares to the craving one finds in addiction (O’Connor et al. 2008).

### 4. The Reward System and Its Disorders

Interestingly, we see the same changes in NAc and insula activation across chronic pain and complicated grief, as just discussed, but in addiction as well. Could it be that they are all of a piece and that they are all disorders of a single reward system? Many contemporary theories of addiction identify impulse control difficulties as well as compulsive behaviors. Patients with impulse control disorders feel an increasing sense of tension or arousal before committing an impulsive act, and then pleasure, gratification, or a sense of relief at the time of doing the act itself. These types of disorders are generally associated with positive reinforcement mechanisms (American Psychiatric Association 2013). In contrast, patients with compulsive disorders feel anxiety and stress before engaging in some compulsive behavior, and then a sudden release from the stress as they perform the compulsive behavior. These disorders are associated with negative reinforcement mechanisms. Already, we can see interesting comparisons with pain processing, which is associated with positive reinforcement at the cessation of acute pain sensations in normal subjects, but not for the cessation of acute pain in cases of chronic pain.

Impulsivity often dominates early in addiction, and impulsivity combined with compulsivity dominates later in the disease. As addicts move from impulsivity to compulsivity, the driving force motivating their addictive behaviors shifts from pleasure and positive reinforcement over to anxiety, stress, and negative reinforcement (Koob and Le Moal 2001, Edwards and Koob 2010). We see a similar pattern in the shift from acute to chronic pain: the patient shifts from being motivated to seek a pleasurable relief to being unable to experience such relief at all. We
also see a similar pattern in complicated grief: the yearning that accompanies the loss is not one of pleasant memories, but of sadness.

The transition from normal consumption to genuine drug or alcohol dependence involves circuitry in the forebrain, including the NAc and prefrontal cortex (Modesto-Lowe and Fritz 2005, Gilpin and Koob 2008, Gianoulakis 2009, Egli et al. 2012). Similar areas are involved in the transition from acute pain processing to a chronic pain syndrome and in comparing normal grief with complicated grief. It does indeed appear that pain, pleasurable consumption, and grieving (and their related disorders) all share the same underlying neural circuitry. All are very complex reactions that stem from our reward circuitry.

Let us return now to where we started: philosophers would like to describe the thing that pain is. We have seen that pain is not a thing, but a multifaceted process. The whole complex of somatosensory information, a representation of badness, and the affective anticipation and motivation comprise pain, of which the experience of “ouch, that hurt” is only a part. And this complex is not specific for pain, but perhaps reflects how the brain processes all incoming stimuli. This suggests that pain, chronic pain, grief, complicated grieving, and addiction (as well pleasurable experiences) are all effects of the basic reward circuitry in the brain. Our reward system normally gives us both pleasures and pains, but with chronic pain processing, unremitting grieving, or extended episodes of intoxication, our brain circuitry and functionality change—and change in very similar ways—such that we can become lost in our disappointment.

Understanding the neurobiological details behind pain can help philosophers analyze its nature. It turns out that their initial intuitions were misguided, and that pain appears to be related to other types of processing that at first blush seem to be categorically different. Moreover, appreciating the complexity and the details of these processes might help all of us to better help those who are in pain and suffering.

References


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Further Reading


