CLOSING THE GAP ON PAIN

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A widely accepted theory holds that emotional experiences occur mainly in a part of the human brain called the amygdala. A different theory asserts that color sensation is located in a small subpart of the visual cortex called V4. If these theories are correct, or even approximately correct, then they are remarkable advances toward a scientific explanation of human conscious experience. Yet even understanding the claims of such theories—much less evaluating them—raises some puzzles. Conscious experience does not present itself as a brain process. Indeed experience seems entirely unlike neural activity. For example, to some people it seems that an exact physical duplicate of you could have different sensations than you do, or could have no sensations at all. If so, then how is it even possible that sensations could turn out to be brain processes?

One answer is that it is not possible. According to this line of reasoning, the puzzle of how brain states could be experiences is a genuine contradiction. Facts about brains do not settle

questions about conscious experience because experience is of a different sort. Our ability to imagine like-brained but differently-experienced critters reveals a deep metaphysical truth.[1]

A more modest but still troubling conclusion is that our imaginative successes are indicative of explanatory failures. It may be that sensations are brain processes. However that does not mean that we can explain facts about conscious experiences with facts about brains. It may be that emotions occur in the amygdala and color sensations in V4. But there is nothing special about the amygdala that explains why emotions occur there rather than in V4 or some other part of the brain, or in things that aren't brains. The problem is not a metaphysical fissure but rather an epistemological one. Between minds and brains there is an *explanatory gap*.[2]

The conclusion that there is an explanatory gap, however modest, is still worrisome. First, it places limits on any possible science of the mind, and on the more general ideal of a natural science of our world. This is particularly frustrating because consciousness could be a unique holdout in a world that has otherwise succumbed to natural explanation. Second, the modest conclusion may be exploited to argue for the more radical conclusion that there is a *metaphysical gap* between mind and brain.

In light of these concerns, it would be an achievement to show that there is no explanatory gap. We don't suppose that we can achieve that much, but we think we can make some progress. This paper has four parts: First we sketch a general approach to the explanatory gap challenge. Next, we argue that our approach counts as a response to the gap problem. Third, we provide an example of how the approach can be implemented in the case of pain experience.

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[1] Though no one probably would endorse this quick sketch as is, versions of this line of reasoning are meant to be familiar, e.g., from the works of Descartes, Kripke, and Chalmers.
[2] This is the expression made famous by Levine. Similar reasoning can be found in Nagel and McGinn. Jackson and (especially) Chalmers exploit this line of reasoning to drive the stronger conclusion, mentioned above.

Finally, we argue that the example fits the model provided earlier. If we are right, there is reason to doubt that there is an unbridgeable explanatory gap.

1. The Explanatory Gap and Identity Conditions

The idea that there is an explanatory gap between mind and brain—particularly between conscious experiences and neural processes—can be developed in many ways. The basic idea, however, is simple. It is hard to understand how any physical theory of brain processes can explain how or why a particular brain process must be correlated with a particular sensation. Because this puzzle can be repeated for every brain process and every sensation, it is hard to understand how any brain process could be associated with any sensation at all. Believers in the explanatory gap argue that it is not possible for physical explanation to tell us what we really want to know about conscious experience, namely, why any sensation depends necessarily on any particular neural activity. Why couldn't that sensation depend on different neural activity, or that neural activity mediate a different sensation? As Joseph Levine puts it: "What is left unexplained by the discovery of C-fiber firing is *why pain should feel the way it does!* For there seems to be nothing about C-fiber firing which makes it naturally 'fit' the phenomenal properties of pain, any more than it would fit some other set of phenomenal properties" (1983: 357). Because of the lack of natural fit between pains and C-fiber firings—or any particular neural process—no explanation that appeals to neural processes will tell us what we want to know about the nature of pains. Specifically, no account will tell us why pain sensation depends on C-fiber activity in such a way that the sensation is necessitated by the neural activity. The explanatory gappists do not deny that there are correlations between sensations and brain processes, and that neuroscience can discover these correlations. But they doubt that the

correlation is other than contingent, and thus that the brain sciences are discovering anything about the nature of conscious experience. To do that, they maintain, we have to know not only that pain is correlated with C-fiber firing but moreover that it must be so. [3] The demand that we explain the "fit" between sensations and brain processes is a demand for an account that ties them together as a matter of necessity. This is the challenge of the explanatory gap.

Here, then, is the situation. Advocates of the explanatory gap (call them *mysterians*, adapting the terminology of Flanagan 1991) argue that it is impossible to explain all the features of conscious experience in entirely natural or neuroscientific terms. There is one sure-fire way to show that something is possible, and that is to show that it is actual. So it goes for explanations of experience. Philosophers, psychologists, and neuroscientists have offered numerous examples wherein some phenomenon of consciousness is given a naturalistic explanation (e.g., Hardin 1988; Damasio 2000; Flanagan 1995, 2000; Gustafson 1998; Ledoux 1998). If some kinds of conscious experience have been explained naturally, this surely demonstrates that conscious experience can be explained naturally.[4] And if so, then the mysterians are wrong.

As a matter of fact, we think that some of the naturalistic responses to the gap should be counted as successful. But for the most part mysterians have been unmoved by the neuroscientific accounts. And why should they be when the explanatory gap reasoning concludes that no such data can close the gap? So despite the availability of naturalistic explanations of

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[3] As it happens we will discuss the example of pain in great detail, and even say a few things about C-fibers. But we think the best way to understand talk about "C-fibers" in most philosophical discussions is as a placeholder for some future neuroscientific account. For the most part, where you read *C-fiber* in a philosophical discussion you should interpret that at something like, *neural process Φ*. This is why it will not defuse the question of the explanatory gap to simply observe that it is in fact false that all pains are C-fiber firings.

[4] Strictly speaking, this leaves the possibility that some kinds of consciousness still cannot be explained naturalistically. But the explanatory gap reasoning concludes that no kind of consciousness can be explained naturalistically, and that is the problem we are concerned with at present.

conscious experience, in one way the mysterians are right to be unimpressed. What is missing is an account of why some neuroscientific model should count as explaining a kind of consciousness in the way that the mysterian demands. (One option, of course, is to deny that the mysterian is making reasonable explanatory demands. Perhaps that is right. But for now we'll play along.) The key to satisfying the mysterian is to take seriously the requirement that explanations of conscious experience explain the "fit" between brain processes and conscious mental occurrences. What is demanded is that we show how some brain states and experiential states go together necessarily.[5] Anything less would leave unanswered the question of why some brain activity, e.g., C-fiber firing, must feel the way it does.

Nearly everyone agrees that there is at least one kind of explanation of the relationship between experiences and brain processes that can satisfy the mysterians' demands. If conscious experiences are identified with brain processes then there is a necessitating dependency between brain processes and experiences. Identity is a relation that holds with necessity. This is as decisive an answer to the question of fit between sensations and brain processes as one would want. Now we can see the beginning of an answer to why some neuroscientific data should count against the explanatory gap argument. It will count when it is evidence that some sensation can be identified with some neural process. It may be that a less robust relation could close the explanatory gap, but identity will do the job.

Mysterians typically agree that mind-brain identification, if correct, would cut off the explanatory gap reasoning.[6] But they each believe that the mind-brain identity theory is, in fact,

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[5] This is where we would get traction if we wanted to dispute the mysterians' criteria. We would argue that it is too much to require a necessary connection, or that a contingent connection would be sufficient.

[6] Recently Chalmers and Jackson (2001) have argued that even identity is not enough. They demand not just that the relationship be metaphysically necessary but that it also be

false. We are quite a bit more optimistic about the identity theory, as you'll soon see. The more salient point at the moment is that what it takes to close the explanatory gap, on our model, is just whatever it takes to justify mind-brain identity claims for conscious experiences. If we are justified in asserting mind-brain identities, then we are justified in denying that there is an explanatory gap. To make this a substantial claim we need to provide an answer to the question: How can we justify mind-brain identity claims?

2. A Model for Mind-Brain Identification

These days there is widespread agreement that we can empirically discover the truth of some scientific identities. It has been discovered that temperature in a gas is mean molecular kinetic energy, that the evening star is the morning star, that gold is the substance with atomic number 79, and that water is H_2O. None of these examples are uncontroversial, however the controversies are generally over whether these particular identifications are correct rather than over the very possibility of such identifications. In this way explanatory gap concerns differ from other disputes about identity. The mysterians' argument purports to show that no mind-brain identifications can be correct. Yet mysterians do not reject the possibility of scientific identifications in general.[7] The question, then, is: How can we establish or justify scientific identity claims?

The basic answer is that if *x* is identical to *y* then *x* and *y* have the same identity conditions. Identity conditions are, as Alan Sidelle puts it,

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"epistemically" necessary. We're not convinced, so we're going to neglect that objection for present purposes.

[7] The explanatory gap is supposed to be a special problem for consciousness, and not for a posteriori identities in general. (See, for example, Block and Stalnaker 1999 and Chalmers and Jackson 2001) There may be other problems about such identities. We don't mean to play down those difficulties, but they are not the explanatory gap.

> the sorts of things that are represented by statements saying, for any possible object, what features it must have in order to be, or those which suffice for it to be (identical to) some particular thing (or, for kinds or properties, for something to be a member of that kind, or possess that property). …a specification of identity conditions need not state with full precision—need not mention—what the relevant features are; for certain purposes, 'this chemical microstructure' or 'this thing's origin' will do as well as 'H_2O' or 'sperm S and egg O'. (1992: 291)

Sameness of identity conditions is at least good evidence for identity and may be sufficient for identity. After all, since identity conditions determine (to wit) the identity of a kind of thing, if two kinds of things have exactly the same identity conditions then they are the same kind of thing. We shall assume for present purposes that establishing that two things have the same identity conditions is sufficient for establishing their identity.[8]

So the general answer is that sensations and brain processes are identical if they have the same identity conditions. The next question is what their respective identity conditions are, and how their sameness can be established. The answer to this question cannot be a platitude about identity. We will have to provide a substantial model for establishing certain scientific claims about sensations. Earlier we suggested that the key idea in the explanatory gap argument is the demand that we be able to explain the necessary "fit" between sensations and brain processes. Meeting that demand is central to establishing identities and closing the gap. Robert Van Gulick writes,

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[8] In assuming that sameness of identity conditions is sufficient for identity, we do not take ourselves to be making a move that would not be acceptable to mysterians. The explanatory gap argument is comfortably construed as working from the premises that conscious experiences and brain processes have different conditions of identity. Some may wish to argue that sameness of identity conditions is not sufficient for identity, but this is a different argument. See the previous footnote.

> The more we can articulate structure within the phenomenal realm, the greater the chances for physical explanation; without structure we have no place to attach our explanatory "hooks." There is indeed residue that continues to escape explanation, but the more we can explain relationally about the phenomenal realm, the more the leftover residue shrinks toward zero. (Van Gulick 1993; in Block, Flanagan and Güzeldere 1997: 565)

This tactic is what we call the Structure of Experience strategy (see Polger and Flanagan 1999).[9] The idea is that once we have a thorough description of experiential phenomenology and its structure, and of the correlated structures of neural events and processes, then we will be able to see that the two "fit" together after all. We will be able to see the fit, because we will see that the experiential and neural processes have the same identity conditions (Polger 2004). This still seems right to us. But we have come to see that the Structure of Experience approach needs to be further elaborated if it is to be more than a platitude about identities.

When we talk about phenomenal and physiological structures fitting together, there is a temptation to think of the relation as a kind of flat mapping (Figure 1). But this is too simplistic, for the phenomenal and physiological structures are not one dimensional. Rather, we need an account of how phenomenology and its neural bases fit into a multilevel mechanistic explanation of activity and experience. Machamer, Darden, and Craver have provided a useful model for thinking about mechanistic explanation (2000; Craver 2001). Figure 2 illustrates their proposal. Providing a full mechanistic explanation of a system involves not only describing what a thing does, but also showing how it fits into a broader context and how its behavior is realized by its constituents. Different phenomena may be explained at different levels—some by explaining

[9] Actually, we called it the "Structure of Consciousness" reply back then, but we now think that "Structure of Experience" sounds better. This is not meant to be a substantive change.

how the system contributes to the activity of a containing system, and others by reference to the behavior or its parts.

Moreover, even this sort of multilevel mechanistic explanation does not stand on its own. It must be situated in the context of general background theories that unify scientific explanations (Polger and Flanagan 1999). When a multilevel mechanistic explanation is anchored in a background theory, theoretical identifications are the natural consequence. As Lewis argued, "theoretical identifications *in general* are implied by the theories that make them possible—not posited independently" (1972, in Block 1980: 207).[10] Take, for example, the atomic theory of chemical elements. Could one adopt the atomic theory and still wonder whether gold is the element with atomic number 79? It does not seem so. Once one adopts the atomic theory the identity of gold falls out. It looks like this is because the atomic theory fixes the identity conditions for chemical elements. Furthermore, we cannot ask whether gold could have different properties that it does, for the atomic theory explains the properties of elements in terms of their identity constituting atomic structure. The periodic table is a systematized representation of the basic identities and property explanations implied by this theory of chemical elements.

The case of sensations and brain processes is no different, in principle. In practice the case is not so simple, for we presently lack anything like a general background theory of neuroscience against which to set our mechanistic accounts of experience. Yet there is a background theory for biological and psychological sciences that can be of some use, namely, evolutionary theory. Providing an evolutionary account of experience can help us secure the mechanistic hooks in our account. Such an explanation may (but need not) take the form of an adaptation explanation. That is, it need not account for some aspects of experience as products of

[10] Lewis may have thought, and thereby encouraged others to think, that theories analytically entail identity claims. That is not our position.

evolution by natural selection favoring experience because it is fitness enhancing. William James (1896) asserts that conscious experience is necessary for flexible behavior, a claim that is often repeated. But Owen Flanagan (1995, 2000) argues that the phenomenal experience of dreams is a spandrel, and we have argued the same for the phenomenology of chronic pain (Sufka 2000). If experience is a natural feature of biological organisms then it must have a place in the general theories of how organisms come to be as they are. One such theory is evolutionary theory. But it is not the case that every evolutionary explanation will be an adaptation explanation.

Admittedly evolutionary theory is a historical theory, rather than a structural theory like the modern atomic theory of chemical elements. As such, it does not imply mind-brain identities on its own, but it is a grounding framework for structural theories that do that job. To that extent we may be seen as offering something akin to what Machamer, Darden, and Craver (2000) call a mechanism sketch or schema, rather than a completed mechanistic explanation. Yet that should be enough. A mechanism sketch is probably all that is had in the case of an explanation of life in inorganic terms. Sometimes the problem of explaining life is compared to the problem of explaining consciousness. This comparison is apt in that both share evolutionary theory as part of their background. But if there is no explanatory gap in the case of life (as even some mysterians have supposed, e.g., Chalmers 1996, Chalmers and Jackson 2001) then it must be that mechanism sketches are sufficient for showing that an explanatory gap can be closed. For it is clear that we do not yet have a complete mechanistic explanation of life.[11]

[11] Here, as above, we emphasize that the explanatory gap is supposed to be a special problem about consciousness. We welcome to the comparison of the problem of explaining consciousness to that of explaining life. We are puzzled as to why Chalmers and others think this case favors the explanatory gap reasoning. If the mysterian falls back to general concerns about identities, reference, and natural kinds, then that will be a rather different debate that is not peculiar to consciousness.

The explanatory model that we are urging, which we argue has the resources to justify identity claims and thereby bridge the explanatory gap, is quite general. Consider the standard case of the identification of water with the molecule H_2O. How does this theoretical identification work? We begin with a qualitative analysis of water. Water is the clear, wet, potable stuff that rains from the sky, is found in lakes and rivers, boils at 100°C, and so forth. Some qualitative facts about water are readily observed and others take more work, for example, facts about the reactions into which water can enter. This gives us a picture of the structure of the phenomenon, in this case, water. These qualitative facts about water are then matched with a qualitative facts about molecules. These are harder to ascertain but no less crucial. When enough is known about the structure and activities of water and the structure and activities of certain molecules, we can explain how the molecular structure of water accounts for its qualitative chemical properties.[12] The multilevel mechanistic explanation of water identifies it with a certain molecule type, H_2O, and explains its behavior in terms of the behavior and composition of H_2O molecules. Water is H_2O. But, of course, one doesn't just make the identification of water and H_2O. That identity only makes sense if other substances are also identified with other molecules, and if elements are identified with atomic kinds, and so forth. In short, the identity of water and H_2O works because it is set in the context of molecular and atomic chemistry. Those background theories entail the identifications. This is just the point we made earlier with respect to the atomic number of gold. One could, we suppose, wonder whether water is correctly identified with H_2O rather than some other molecule; but within the context of chemistry as we know it, one cannot

[12] In fact we believe available explanations to be incomplete, and thus only mechanism schema, even in the case of the properties of molecular substances. For example, as far as we know there is no complete mechanical explanation of fluid properties (e.g., viscosity and turbulence) in terms of atomic and molecular features.

sensibly wonder whether water might fail to be any kind of molecular substance at all. There is no explanatory gap about water and H_2O.

This is the model that we urge in general, and for conscious experience in particular. In the remainder of this paper we show how to apply the model to the case of pain experience. Pain experience has distinctive sensory and affective structures. There is a well integrated multilevel account of the physiological mechanisms that explain the qualitative structures of pain. This mechanistic explanation is set against the background of (admittedly nascent) theories of neurons and neuronal systems. There are also evolutionary explanations of pain experience, but these are not always adaptation explanations. Together these explanations show how the mechanisms of pain fit the phenomenology of pain. Once pain mechanisms are identified, there is no room for an explanatory gap.

3. Case Study: Pain

Pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey and Bogduk 1994: 207-213; see also Merskey 1986). Like other sensory systems, the pain sensory system is designed to carry out feature detection processes for a given sensory modality. To accomplish this task, the pain sensory system contains (1) specialized receptors that respond only to certain kinds of noxious stimuli, (2) a multitude of dedicated pathways that carry specific features of this sensory modality (i.e., line-labeling) to the central nervous system, and (3) numerous distinct subcortical and cortical areas that process and coordinate the various sensory and emotional features and behavioral responses of this sense modality. Functionally, the pain sensory system is responsible

for detecting stimuli that have the potential for damaging tissue and for causing the organism to engage in escape and avoidance responses.

It will be useful to keep in mind an outline of the neuroanatomy of the pain sensory system (Figure 3). Noxious stimuli, which have the potential to damage tissue, are detected by a special class of receptors called nociceptors which are located at the distal ends of primary afferent fibers (PAFs) or simply put, first order sensory neurons. Following transduction, the process by which physical energy is converted to a neural code, information in the form of action potentials is carried to the central nervous system via two classes of PAFs: Aδ and C fibers. These Aδ and C fibers terminate in several of the ten distinct laminae or layers of the spinal cord dorsal horn and synapse onto nociceptive specific, wide dynamic range second-order neurons. The best way to think of these second-order neurons is that their feature detection capabilities are maintained through the principle of line labeling. Line-labeling preserves receptor individuation throughout a pathway. For example, adjacent receptors project to adjacent locations in the thalamus, and so onward. Thus the whole pathway can be thought of as coding for the feature detected by the receptor, and distinct from that of the adjacent receptor or pathway. The line-labeled second-order neurons project via both monosynaptic and polysynaptic parallel pathways to numerous supraspinal centers—predominately the thalamus, but also the hypothalamus, the amygdala, and the brainstem periaqueductal gray and reticular formation. Projection neurons from these centers extend their axons to distinct regions of the cortex including primary somatosensory, secondary somatosensory, anterior cingulate, insula, and medial prefrontal cortical areas. As we will discuss below, electrophysiological, regional cerebral blood flow, positron emission tomography and functional resonance imaging studies all demonstrate that activity in these areas follows the presentation of noxious or tissue damaging stimuli. This

activity can be linked with various conscious sensory and emotional states. The more we are able to describe the phenomenology of pain and map these experiences onto neurobiological processes, the closer we come to eliminating the explanatory gap.

3.1. The Phenomenal Structure of Pain Experience

With this outline of neurophysiology in mind, let us examine the phenomenological structure of pain experience. Pain is a multidimensional experience that involves both sensory and affective dimensions. The sensory dimension of a pain experience typically refers to the quality or type, intensity and spatiotemporal dynamics characteristics. Melzack and Torgerson (1971), in an attempt to develop a valid and reliable pain questionnaire, compiled, categorized and scaled a list of descriptors of pain phenomenology (Figure 4). Interestingly, these verbal descriptors not only clustered in predictable ways around the multidimensional phenomenology, the individual descriptors within a single dimension revealed a scaling with respect to an intensity dimension (using anchor words: 1 = mild, 2 = discomforting, 3 distressing, 4 = horrible, 5 = excruciating). For example, a thermal pain experience might be described as ranging from hot to burning to searing while a mechanical (pressure) pain experience might be described as ranging from pinching to cramping to crushing. For the temporal feature of a pain experience, one might describe it in terms ranging from flickering to pulsing to pounding. We argue that these sensory characteristics of type, intensity and location can be mapped to neurobiological processes in the pain sensory system.

Another dimension of a pain experience that can be mapped onto neurobiological processes is its affective/emotional component. The affective dimension of a pain experience has two components: primary and secondary affect (Price, 2000; Wade, Dougherty, Archer and Price

1996). Primary affect refers to the initial unpleasantness one experiences as a result of a threat. It comprises "the moment by moment unpleasantness, distress and possible annoyance that are often closely linked with the intensity of the painful sensation and the accompanying arousal" (Price 1999: 59). Secondary affect refers to one's "elaborate reflection and relates to memories and imagination about the implications of having pain, such as how pain may interfere with different aspects of one's life" (Price 1999: 59).

In addition to the dimensions of pain outlined above, there are a number of alterations in pain sensation that can follow an injury or illness and include allodynia and hyperalgesia (see Willis 1992). Allodynia is pain due to a stimulus that does not normally provoke pain. Hyperalgesia is an increased response to a stimulus that is normally painful. The difference between allodynia and hyperalgesia is in the relationship between the stimulus and the painful experience. Allodynia involves two modes of processing where a non-noxious stimulus is perceived as being noxious, and hyperalgesia involves a single mode of processing where a noxious stimulus is perceived as being more noxious. Allodynia and hyperalgesia are hallmark signs of chronic pain and can be mapped onto changes in both peripheral and central nervous system functioning.

3.2. A Multilevel Mechanistic Model of Pain

We claimed that mechanisms that explain pain phenomenology have been identified. Now we need to deliver on this promise. In this section we illustrate how pain phenomenology (outlined in section 3.1) can be explained by specific neuroanatomical and neurophysiological processes (outlined in section 3.0.) We will restrict this discussion to coarse-grained phenomenology of pain sensation (type, intensity and location), pain affect, and the chronic pain symptoms of

allodynia and hyperalgesia. Other dimensions of pain phenomenology are open to such explanation, and progress continues to be made in understanding the neurobiological underpinnings of pain.

Type Coding

People are quite adept at differentiating the various types of noxious or tissue damaging stimuli. Our pains can be felt as coming from either mechanical stimuli (smashing a finger in a door), thermal stimuli (touching a hot oven), or chemo-inflammatory stimuli (getting lemon juice into an open cut.) The pain sensory system is capable of this level of feature detection through three types of nociceptors (Belemonte and Cervero 1996; Reichling and Levine 1999). Although least studied, mechanical nociceptors are probably the simplest in terms of the transduction process and consist of high threshold mechano-gated ion channels; low threshold mechano-receptors are for cutaneous or touch sensory system. Noxious mechanical stimuli open ion channels of this receptor leading to a depolarizing current in the primary afferent fibers. Thermal nociceptors are a bit more complex. High intensity thermal stimuli activate non-specific cation channels as well as both vanilloid and non-vanilloid-like receptors. The influx of positively charge ions leads to depolarizing current in the PAFs. Chemo inflammatory nociceptors are perhaps the most complex. A variety of exogenous substances, for example capsaicin, and endogenous neural and non-neural substances, for example bradykinins, prostaglandins and serotonin, can activate our pain sensory system through binding to their respective receptors (Reichling and Levine 1999). Some of these chemo-inflammatory nociceptors depolarize primary afferent neurons through iontropic mechanisms while others do so through metabotropic or 2nd messengers mechanisms. Adding to this kind of feature detection are non-specific nociceptors that seem to respond to

more than one kind of stimulus. The VR1 receptor is a good example in that it responds to thermal stimuli and changes in pH (Reichling and Levine 1999). In short, our pain sensory system, like other sensory systems, engages in feature detection processes due to, in part, a principle of receptor specialization. Receptor differences explain why pain is experienced as coming in different types that have different responses. But type is only one dimension of the structure of pain phenomenology.

Intensity Coding

The intensity of pain experiences seems to be initially coded at the receptor level as well. Elegant neural recording studies in PAFs show that the activity in these fibers parallel psychophysical assessment of pain intensity for both noxious mechanical and thermal stimuli (Handwerker and Kobal 1993; Gybels, Handwerker and Van Hees 1979; Bromm, Jahnke and Treede 1984). However, there is not a one-to-one correspondence between stimulus intensity and actual transduction processes. This is due to an ensemble of mechanisms that interact to modulate the eventual pattern of activity in PAFs. Among these mechanisms include multiple transduction avenues for a single stimulus, functional interactions between ion channels due to their spatial distribution, and the ability of 2nd messenger systems to modulate receptors, ion channels and other 2nd messenger systems (Reichling and Levine 1999). Nevertheless, there seems to be a strong relationship between a noxious stimulus' intensity and verbal ratings using the Visual Analogue Scale (VAS); this relationship yields a positive power function with an exponent of 3.0-3.5 (Price and Harkin 1987). Thus, a psychophysical relationship exists between level of the noxious stimulus presented and the perceived intensity of pain that is explained by the response characteristics of the nociceptor and the eventual activity along the PAFs.

Temporal Coding

One interesting phenomenon of pain is in its temporal dynamics. Some kinds of tissue damage initially produce a "sharp and highly localized" pain that is followed by a "dull and more diffuse" pain. The temporal ordering of these distinct pain experiences maps well to physiological properties of the Aδ and C PAFs.[13] These two fibers differ in axonal diameter and whether they are covered with myelin. Axonal diameter and myelin sheathing greatly affect signal conduction velocities. Aδ fibers have medium sized myelinated axons of around 2-6 μm in diameter with conduction velocities of 120-300 m/s. C fibers have thin unmyelinated axons of around 0.4-1.2 μm in diameter with conduction velocities of 5-20 m/s (Besson and Chaouch 1987; Laem, Willis, Weller and Chung 1993). While both fibers are responsive to noxious stimulation, Aδ fibers evoke the sharp, first phase of the pain experience whereas C-fibers evoke the dull, second wave of the pain experience (Price and Dubner 1977; Price 1996). For example, stubbing a toe leads to two distinct and sequentially-processed pain experiences: the first is a sharp and highly localized pain experience that is followed by a dull, throbbing and diffuse pain experience. These distinct phenomenal qualities nicely map onto the differences in signal processing capabilities found in the Aδ and C PAFs.

Location Coding

To understand how an organism is capable of spatially localizing noxious stimulation requires an understanding of receptive fields, dermatomes, and cortical somatotopy. A receptive field refers to an area of skin in which a stimulus will elicit a response in a receptor or, on a larger scale, a

[13] Finally, C-fibers!

single PAF. The receptive fields for C-fibers range in size up to about 10 mm in diameter depending upon the body location. Dermatomes are essentially very large receptive fields for one of the 31 pairs of spinal nerves. Cervical nerves 1-8 have dermatomes in head, neck, shoulder and arm regions, thoracic nerves 1-12 have concentric dermatomes down the midsection, and so forth for the 5 lumbar, 5 sacral and single coccygeal nerve pairs. Through line-labeling, the integrity of this spatially discriminated information is maintained up to the level of the cortex where in areas of the primary and secondary somatosensory cortex of the parietal lobe, several somatotopic maps are located (Figure 5). The discovery of human somatotopic maps dates back to Wilder Penfield's pioneering research from the 1930s-1950s (Penfield and Jasper 1954) and are essentially maps of the body surface projected onto the surface the post-central gyrus of parietal lobe. PET studies have revealed these somatotopic maps indeed map pain loci by showing that noxious stimulation to different body regions (i.e., receptive fields) give rise to increased activity in distinct areas of S1 that yield pain experiences to a particular body region (Andersson, Lilja and Hartvig 1997). This supports the conclusion that the spatial features of pain experience can be explained by the spatial features of somatosensory cortex.

Affect Coding

Numerous studies demonstrate that noxious stimulation activates several distinct regions of the cortex including primary somatosensory (S1), secondary somatosensory (S2), anterior cingulate (ACC), insula, and medial prefrontal cortical areas. Clever studies by Catherine Bushnell and her colleagues have been able to dissociate the sensory and affective dimensions of pain perception and to localize these dissociated processes to activity in distinct brain areas. In one PET study, rCBF levels were obtained during a thermal grill illusion task. This is an illusion in which

spatially alternating non-noxious warm and cool thermal stimuli leads to reports of pain and unpleasantness despite neither stimuli producing pain or unpleasantness when presented alone (Craig, Reiman, Evans and Bushnell 1996). Using a subtraction technique for the two conditions, only the increase in activity in the ACC seemed to account for the pain experience.

In another study, researchers used hypnotic suggestion to manipulate subjects' pain affect while holding their perception of pain intensity constant while subjects' hands were submerged in hot water. Verbal reports confirmed that hypnotic suggestion was successful in dissociating pain unpleasantness and intensity. Again rCBF results after subtraction (hand in warm water) show a significant increase in ACC activity that actually parallels subjects' level of pain unpleasantness ratings (Rainville, Duncan, Price, Carrier and Bushnell 1997). A similar study that held subjects' perception of pain unpleasantness constant while manipulating perception of pain intensity produced strikingly different results. Here, the strongest change in pain-intensity evoked activity was localized to S1 (Rainville, Carrier, Hofbauer, Bushnell and Duncan 1999).

These findings parallel the case of a 57 year-old male who suffered a stroke that damaged the hand region of his right S1 and S2 cortex (Ploner, Freund and Schnitzler 1999). Noxious cutaneous laser stimulation presented to his left foot produced a well-localized pain sensation; however, the same stimulus presented to his left hand at three times the intensity failed to evoke a pain sensation. Interestingly, the patient "spontaneously described a 'clearly unpleasant' intensity dependent feeling emerging from an ill-localized and extended area 'somewhere between the fingertips and shoulder' that he wanted to avoid" (Ploner, Freund and Schnitzler 1999: 213). Further, this patient was "completely unable to describe quality, localization and intensity of the perceived stimulus." Case studies like this, as well as the PET studies described

earlier, provide compelling evidence for the differential involvement of cortical structures in our sensory and affective dimensions of pain experiences.

By now our pattern of argument is familiar. We conclude that the quantitative aspects of pain experience including location, type and intensity is explained by activity in S1 while the qualitative or affective dimension of pain experience is explained by activity in the ACC (Sufka and Lynch, 2000).

Altered Sensory Coding

Finally, allodynia and hyperalgesia are two primary symptoms of chronic pain and reflect perceptual alterations to non-noxious and noxious stimuli, respectively. Considerable work over the last decade has elucidated the neurobiological processes that underlie the development of allodynia and hyperalgesia both of which result from neuroplastic changes in the peripheral and central nervous systems. More specifically, allodynia seems to result from of a phenotypic switch in Aβ PAF (Neumann, Doubell, Leslie and Woolf 1996). Aβ fibers are normally responsible for processing non-noxious cutaneous information. However, prolonged tissue trauma can cause a subset of these Aβ fibers to switch phenotypes to one that resembles Aδ and C fibers. This phenotypic switch is at the level of the neurotransmitter released onto second order neurons. The result is a cross-talk between the touch system and the pain system and it occurs in spinal cord relay neurons.

Hyperalgesia results from several related processes that ultimately increase the rate of firing in 2nd order pain neurons in the spinal cord, a phenomenon known as central sensitization (see Sufka 2000). Four neurobiological changes have been identified as being responsible for central sensitization induction. These include activation of silent nociceptors, increased release

of glutamate from PAFs, increased numbers of and signal conduction in glutamate receptors on 2nd order pain transmission neurons, and loss of inhibitory interneurons in the spinal cord dorsal horn (Woolf and Salter 2000).

3.3. Background Theories

As more and more details of pain processing are revealed, more pain phenomenology can be identified with neurobiological processes. With each new discovery the explanatory gap grows smaller. But we have also suggested that although empirical progress inevitably proceeds bit by bit, the identification of pain experience with neural processes is not piecemeal. We do not discover one identity at a time and then add them together. Rather, what justifies our claims to have identified the mechanisms of pain is the way the whole multilevel mechanistic story hangs together. Different aspects of pain experience are explained by different parts of the mechanism—some at the level of synapses, some neurons, some neural systems. No one of the identifications makes sense without the others, nor without a background understanding of neurons and organisms. So in order to make good on the identity claims in the previous section, we must articulate the background theories that are relevant to the case pain. To satisfy this requirement, we provide a multilevel, theoretical consideration of neuronal communication, sensory system organization and functioning, and evolutionary explanations of pain.

To our knowledge there is no widely accepted background theory that applies to all sensory systems in terms of organization, processing and functioning. This does not mean that nothing can be said. Neural communication is an electro-chemical process. Given the appropriate input from a receptor or other neurons, a cell transitions from a resting membrane potential to an action potential. The mathematical model that describes neuronal membrane potentials is the

Goldman Equation (Goldman 1943; Hodgkin and Katz 1949). Basically, the determination of the membrane potential is heavily influenced by a given ion concentration and its membrane permeability. Further, when membrane permeability is exceptionally high for a given ion, the Goldman Equation reduces to the Nernst Equation for that particular ion. Communication between neurons occurs via electrical and chemical transmission, although the latter being far more common in the nervous system. There are two competing theories (i.e., Classical Heuser-Reese model versus Ceccarelli Kiss and Run model) that describe neurotransmitter release both of which entail a calcium-dependent exocytosis of vesicle contents at the active zone (Holt and Jahn, 2004). For one wanting yet a lower level theoretical explanation, a decade of elegant studies by Roderick MacKinnon detail the structure and necessary requirements for the opening and closing of neuronal ion channels responsible for cellular communication (see MacKinnon, 2003).

Moreover, sensory systems, including the pain sensory system, seem to be designed in ways to allow them to carry out feature detection processes. How these sensory systems accomplish this task is, in most ways, quite similar across the various sensory modalities. Each sensory system has specialized receptors that respond only to certain kinds of stimuli. Further, these receptors typically respond in a manner that parallels, up to a point, the intensity of that stimulus. Each sensory system contains a multitude of dedicated, line-labeled pathways that carry specific information to the central nervous system. Finally, each sensory system routes information to various distinct cortical areas that are dedicated to the conscious processing of the various features of that sensory modality. How each sensory system accomplishes the complex processing task of feature detection is not fully understood. However, one theory of visual processing for form perception (viz., the Multichannel Model) holds that neuronal cells

throughout the visual system (from the retina to the cortex), which vary in their receptive field size, naturally extract information by a process analogous to spectral/Fourier analysis of a scene's four grating properties: spatial frequency, contrast, orientation and spatial phase (Campbell and Robson 1968).

The general theoretical framework for the biological and psychological sciences is that of evolutionary theory. Evolutionary theory posits that organisms evolved through gradual change over time, subject to the influences of natural selection. Natural selection typically favors those biological forms that have an ecological or adaptive advantage in their environments. Pain has ecological value in that it alerts the organism to injury or threat of injury and elicits an escape/avoidance responses to terminate tissue trauma and/or a state of quiescence to prevent further injury and permit haling and recovery processes to ensue (Sufka and Turner, in press). Further evidence of the adaptive value of pain comes from a cluster of rare medical conditions known as Hereditary and Congenital Pain Insensitivity Syndromes (Nagasako, et al., 2003). People with these syndromes fail to experience the normal subjective and objective responses to noxious stimuli. Sufferers of these syndromes do not express aversion to noxious stimuli nor attempt to prevent injury from recurring. Further, they fail to notice injuries and illnesses and often die in childhood; rarely do these individuals live beyond his or her 30s. Syndromes such as these suggest that pain normally serves an adaptive function (Melzack and Wall 1983; Nagasako et al., 2003). However it is not necessary that all pains are adaptive. Chronic pain typically leads to states of withdrawal, anhedonia (i.e., loss of pleasure), and environmental indifference—in essence, depression. For some individuals, their chronic pain persists long after resolution of the initial tissue trauma and likely involves possibly permanent maladaptive changes in nervous system functioning (Scholz and Woolf 2002; Sufka and Price 2002). Whereas acute pain has

clear protective functions, Millan (1999) and Sufka (2000; Sufka and Turner, in press) have argued that chronic pain has no ecological value and is simply a spandrel or byproduct of the highly adaptive phenomenon of neural plasticity.

As we conceded earlier, evolutionary theory does not by itself entail mind-brain identity claims. (Mysterians, after all, do not reject evolutionary theory.) Rather, evolutionary theory provides some background constraints on structural theories of neuroscience. These latter we only have the beginnings of at the moment. And we only have them in a piecemeal way—as with the theories of electro-chemical communication and feature detection pointed to above. But these are the kinds of theories that warrant identity claims, and more are sure to come. Already we know enough to know what form the yet-to-be-discovered theories will take. This is not the blank check strategy, often lamented of naturalistic philosophy. The cash value of our proposal is our claim about the logical structure of mechanistic mind-brain explanations: they identify mental occurrences with neural processes. We have argued that we already have evidence of this, and we have given examples. The missing details are just that. An analogous case would be the situation, which we suppose actually occurred, in which one has sufficient evidence that the atomic theory of chemistry is correct but has not yet identified all the elements. In that situation you would already be justified in asserting that, say, uranium is identical to some atomic type that is individuated by its atomic number, even if you did not yet know what the atomic number of uranium is. You would also be justified in asserting that the chemical properties of uranium are explained by its atomic structure. This is what we claim about sensations and brain processes. If we are right, we are already justified in concluding that sensations are identical to brain processes.

4. Closing the Gap on Pain

As we have described them, the explanatory gap worries successfully point to a lacunae in the naturalistic explanations offered to date. But that kind of gap is not distinctive to explanations of consciousness. Nor is it located, as it were, between the phenomenology and the neurophysiology. Rather, the missing information involves enriching the detail of both the phenomenology and neurophysiology, and situating them together against the background of general theories of perception and sensation. We have indicated how this missing information can be provided in the case of pain experience. And we illustrated how the multi-level mechanistic explanation of pain fits the model that we endorse. Of course there is always more information that could be added and further detail that could be articulated. This is as much the case for water and H_2O as for pains and neural processes.

The explanation that we sketched for pain experience fits the model that we offered in section 2. We showed how features of the phenomenology of pain can be identified with neural properties. Our account identifies pain types with receptor types, pain intensity with PAF activity, pain "speed" with PAF type, and so forth. These identities do stand on their own. They are supported by a general view of transduction, coding, and processing that applies not only to pain but to other sensory systems, and in some cases to neural systems in general. These, in turn, can be understood and situated in and constrained by the background assumption that biological systems are evolved systems.

Our model is quite general. Indeed it helps us to see why some identifications are more intuitively compelling than others. The identification of water and H_2O is convincing because we know quite a bit about the general framework from which it derives, that of molecular chemistry. The identification of sensations and brain processes remains somewhat mysterious because the

mechanistic explanations and background theories are still under development. We have urged that this is a mundane fact about the status of neuroscientific theories rather than a special fact about conscious experiences. Good empirical theories, after all, are hard to come by. But when we have them, we have all that we need to close the explanatory gap.

5. Acknowledgements

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Figure Captions

Figure 1. The flat mapping notion of “fit”.

Figure 2. The multilevel mechanistic notion of “fit,” adapted from Craver (2001).

Figure 3. The pain sensory system.

Figure 4. The phenomenology of pain, adapted from Melzack and Torgerson (1971).

Figure 5. Organization of the somatosensory cortex (from Penfield and Jasper, 1954)

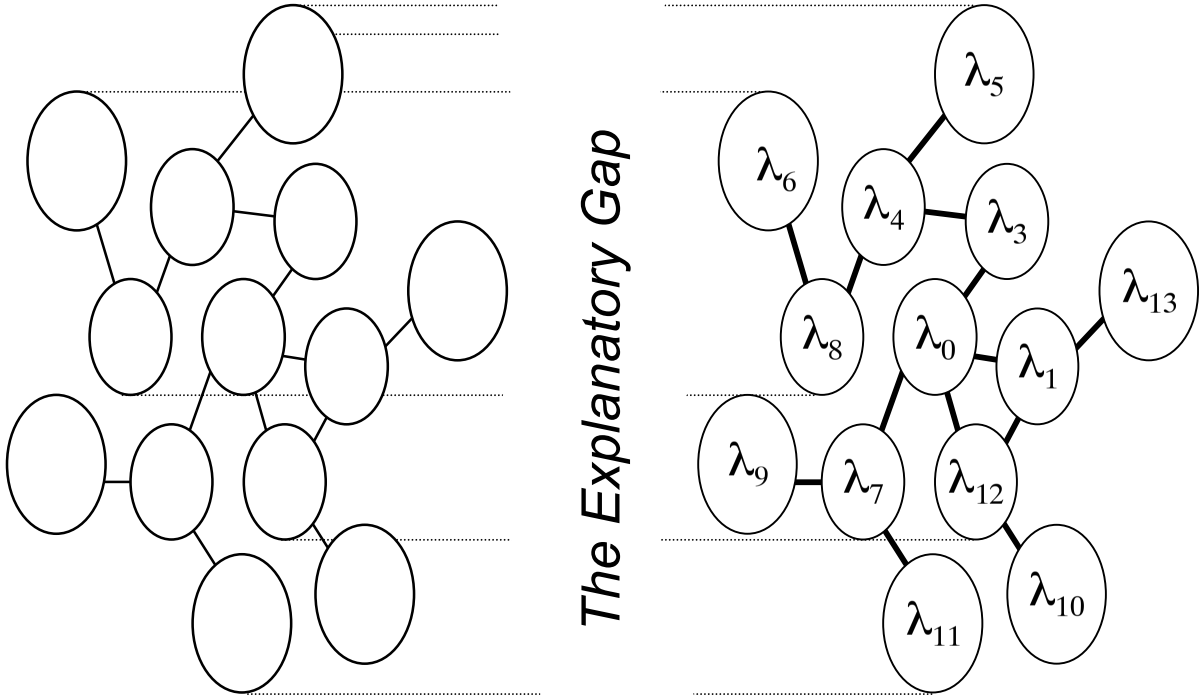

Figure 1

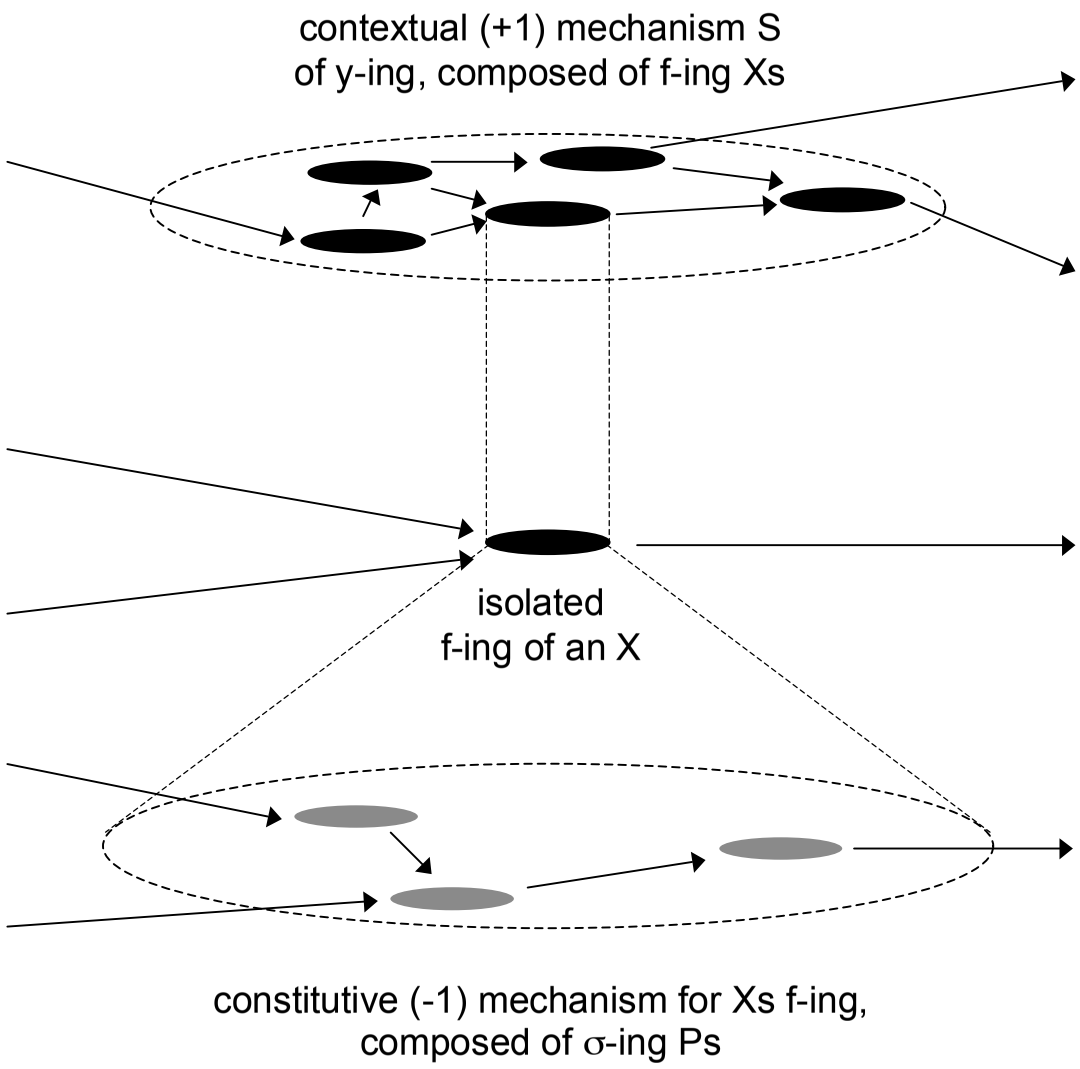
contextual (+1) mechanism S
of y-ing, composed of f-ing Xs
isolated
f-ing of an X
constitutive (-1) mechanism for Xs f-ing,
composed of σ-ing Ps

Figure 2

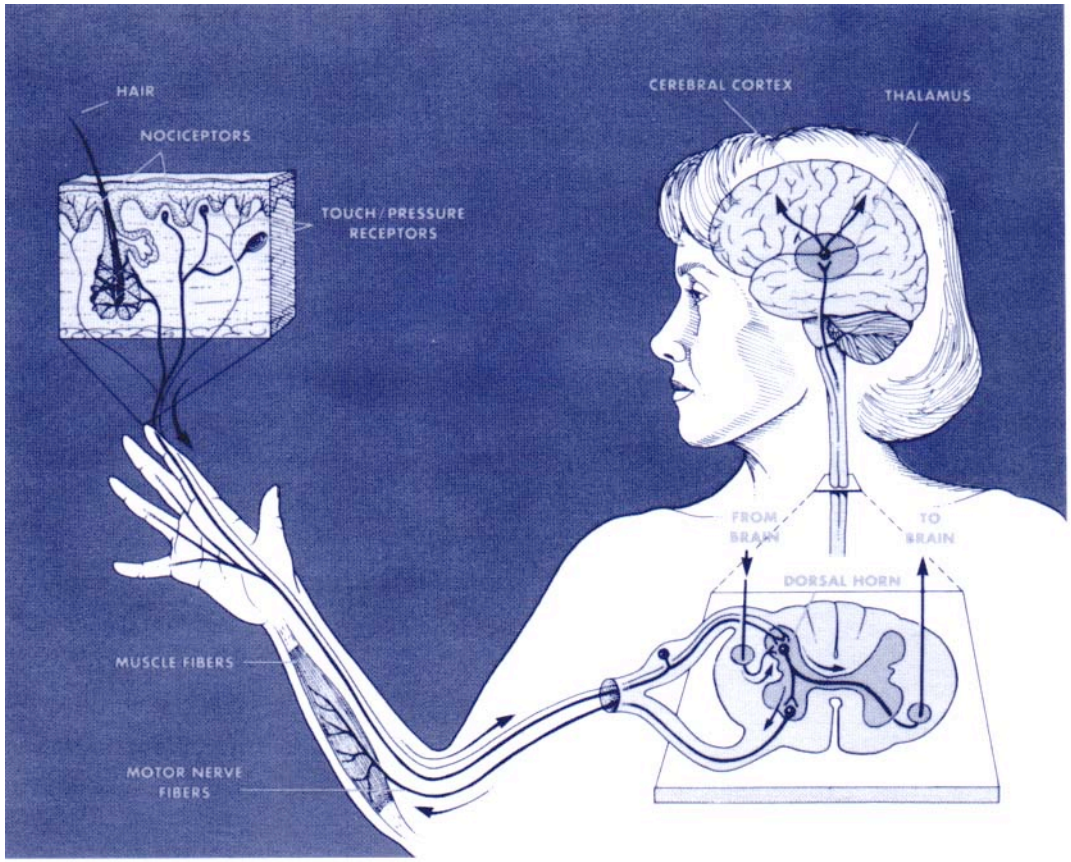
HAIR
NOCICEPTORS
TOUCH/PRESSURE RECEPTORS
CEREBRAL CORTEX
THALAMUS
FROM BRAIN
TO BRAIN
DORSAL HORN
MUSCLE FIBERS
MOTOR NERVE FIBERS

Figure 3

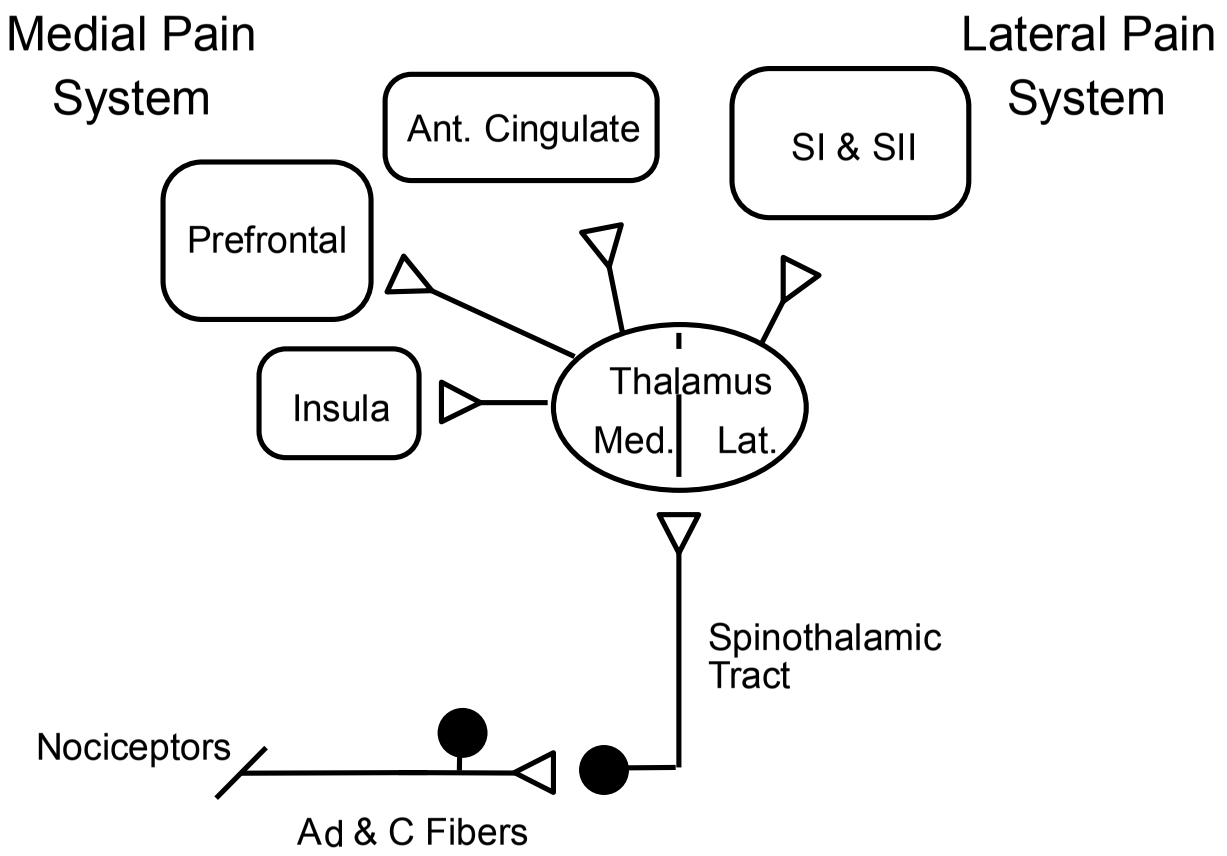

Figure 3 (alternative)

Figure 4

	SENSORY									
	Temporal	Spatial	Punctate	Incisive	Constrictive	Traction	Thermal	Brightness	Dullness	
1									Dull	1
2	Quivering		Pricking		Pinching			Smarting		2
3	Pounding	Shooting		Sharp		Tugging	Hot		Heavy	3
4			Stabbing	Lacerating	Crushing		Searing			4
5										5

	AFFECTIVE					EVALUATIVE	
	Tension	Autonomic	Fear	Punishment	Misc.	Anchor Words	
1						Mild	1
2	Tiring					Discomforting	2
3	Exhausting	Sickening	Frightful	Gruelling	Wretched	Distressing	3
4		Suffocating	Terrifying		Blinding	Horrible	4
5				Killing		Excruciating	5

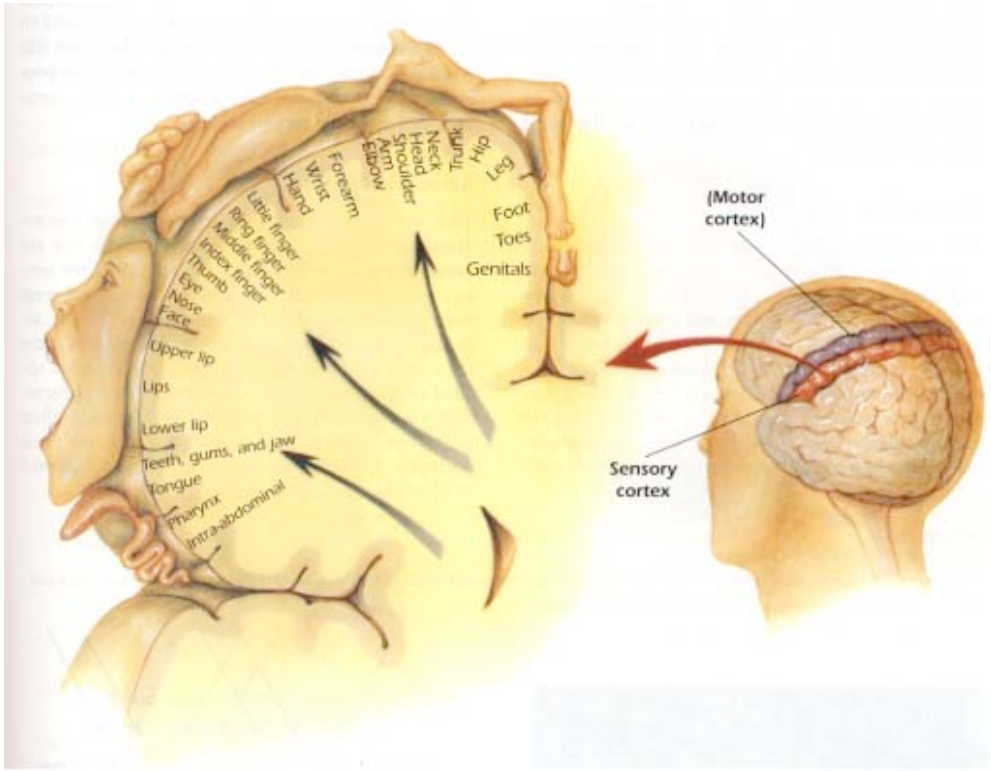
Hip
Leg
Trunk
Neck
Head
Shoulder
Arm
Elbow
Forearm
Wrist
Hand
Little finger
Ring finger
Middle finger
Index finger
Thumb
Eye
Nose
Face
Upper lip
Lips
Lower lip
Teeth, gums, and jaw
Tongue
Pharynx
Intra-abdominal
Foot
Toes
Genitals
(Motor cortex)
Sensory cortex

Figure 5