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WHEN A PAIN IS NOT*

> Nature has placed mankind under the government of two sovereign masters, pain and pleasure.
>
> —Jeremy Bentham

Much to my surprise, a recent review article[1] in neuroscience began with the following quotation from Hilary Putnam: "The typical concerns of the Philosopher of Mind might be represented by three questions: (1) How do we know that other people have pains? (2) Are pains brain states? (3) What is the analysis of the concept pain?" Upon reflection, I decided that the quotation was entirely apt after all. Philosophers do take pain as their paradigm case in quite a number of projects, and the fact remains that we do not know exactly how pain processing works in the brain, so there is lots of room for wild and rampant speculation. A little digging into the philosophical literature uncovered a wide range of opinions and arguments regarding how we think about pain. Indeed, I daresay just about every conceivable position is currently held today by some leading thinker or other. (See table 1.) We find some philosophers and neurophysiologists arguing that pain is com-

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[1] A.V. Apkarian, "Functional Imaging of Pain: New Insights regarding the Role of the Cerebral Cortex in Human Pain Perception," *Seminars in the Neurosciences*, VII (1995): 279-93.

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Eliminativist		Averill Churchland Dennett
Completely Objective	Intrinsic to part of body	Armstrong Newton Pritcher Wilkes
	Behaviorist	Wittgenstein
	Functional state	Davis Lycan Shoemaker
	Perception	Hall Tye
Relation		Graham Stephens Conee Nelkin Kaufman
Purely subjective	Mysterious	Kripke McGinn
	Nonmysterious	Gillet Grahek

Table 1. Possible philosophical positions regarding the nature of pain, with the names of a few of the more prominent adherents listed. See the footnotes for bibliographic information.[2]

pletely objective; it is either intrinsic to the injured body part, a functional state, a set of behavioral reactions, or a type of perception. We also find some philosophers and psychologists arguing that pain is completely subjective; it is either essentially private and completely mysterious or it does not correlate with any biological mark-

[2] References for works not discussed elsewhere in this article follow: E.W. Averill, "Functionalism, the Absent Qualia Objection, and Eliminativism," *Southern Journal of Philosophy,* XXVIII (1990): 449-67; P.S. Churchland, "Consciousness: The Transmutation of a Concept," *Pacific Philosophical Quarterly,* LXIV (1983): 80-95; E. Connee, "A Defense of Pain," *Philosophical Studies,* XLVI (1984): 239-48; W.E. Dandy, *Bulletin from Johns Hopkins Hospital,* XLVIII (1933): 357-61; L. Davis, "Functionalism and Absent Qualia," *Philosophical Studies,* XLI (1982): 231-49; G. Graham and G.L. Stephens, "Are Qualia a Pain in the Neck for Functionalists?" *American Philosophical Quarterly,* XXII (1985): 73-80; R.J. Hall, "Are Pains Necessarily Unpleasant?" *Philosophy and Phenomenological Research,* XLIX (1989): 643-59; R. Kaufman, "Is the Concept of Pain Incoherent?" *Southern Journal of Philosophy,* XXIII (1985): 279-83; S. Kripke, "Identity and Necessity," in T. Honderich and M. Burnyeat, eds., *Philosophy as It Is* (New York: Penguin, 1979); W.G. Lycan, "Form, Function, and Feel," this JOURNAL, LXXVIII, 1 (January 1981): 24-50; N. Nelkin, "Pain and Pain Sensations," this JOURNAL, LXXXIII, 3 (March 1986): 129-48, and "Reconsidering Pain," *Philosophical Psychology,* VII (1994): 325-43; H. Putnam, *Renewing Philosophy* (Cambridge: Harvard,

ers but is completely nonmysterious. Finally, we find a few philosophers who disagree with both conceptions and hold that pain is not a state at all; either it does not exist as we commonly conceive of it or it is an attitudinal relation. Furthermore, each of these positions has become grist for someone's mill in arguing either that pain is a paradigm instance of a consciousness state or that pain is a special case and should not be included in any general theory of consciousness.

We are left with several significant questions and puzzles. Among them are Putnam's. I do try to answer Putnam's three questions here, but I also want to do more than merely carve out my niche among the myriad of positions. I aim to offer a diagnosis for why we have so little agreement concerning the nature of our pain states. In brief, I believe that there are two reasons. First, for many philosophers, there is a basic failure to appreciate the fundamental complexity of our neuronal processing. This is the less interesting cause. My claim is that philosophers are enamored with dissociation experiments, but fail to understand their purpose, which is to individuate the component pieces of our larger systems. I argue that our pain sensory system functions according to the same basic rules of all of our sensory systems.

Second, for many psychologists, neurophysiologists, and philosophers alike, there is an explicit or an implicit reliance on some sort of gate-control theory of pain. Although theories of this ilk can account for several "low-level" puzzling cases involving pain (why it is that stimulating our nociceptors under certain conditions can alleviate pain instead of causing more, for instance), they are notoriously vague when it comes to discussing the central gating mechanisms. This vagueness, I believe, obscures the fact that we actually have two separate systems involved in our perceptions of pain. One functions as a *pain sensory system* (PSS), quite analogous to our other sensory systems. The other—*pain inhibitory system* (PIS), which developed independently of our PSS—actively inhibits its functioning.

Differentiating between the two systems helps explain the remaining controversies surrounding the basic nature of pain. While a PSS

1992); S. Shoemaker, "Functionalism and Qualia," *Philosophical Studies,* XXVII (1975): 291-315, "Phenomenal Similarity," *Critica,* VII (1975): 3-37, and "Absent Qualia Are Impossible," *Philosophical Review,* XC (1981): 581-99; M. Tye, "A Representational Theory of Pains and Their Phenomenal Character," *Philosophical Perspectives, IX* (Atascadero, CA: Ridgeview, 1995), and *Ten Problems of Consciousness: A Representational Theory of the Phenomenal Mind* (Cambridge: MIT, 1995); K. Wilkes, *Physicalism* (London: Routledge, 1977).

supports a perceptual view of pain as a completely objective phenomenon, adding in a PIS (without explicitly recognizing that fact) accounts for the strongly subjective aspects of pain. I shall claim that a PSS functions according to the same basic rules of all of our sensory systems and that, insofar as the pain system is a simpler system than, say, vision or audition, it makes sense to take pain perception as a paradigm instance of a conscious experience. But insofar as we also have a PIS, pain also becomes a special case in our collection of conscious phenomena. Hence, contra Putnam, we should not be using the experience of pain as an intuitive and unproblematic example of consciousness.

As a final conclusion emerging from my understanding of pain, however, we shall see that the sensation of pain—what most philosophers of mind focus upon as absolutely central to being in pain—is neither a particularly fundamental nor a particularly important component of our pain processing. One current popular research question in the philosophy of mind is determining whether some philosophical approach or other (for example, identity theory, functionalism, weak supervenience) can capture in an appropriate way what sensations *feel like.* If I am right about how we should understand pain, then the fervor devoted to this project might better be spent elsewhere, for what something is like becomes less important in explaining our mind.

I. THE COMPLEXITY OF OUR SENSORY SYSTEMS

Let me begin by outlining a few facts regarding our other sensory systems. I do this as a preliminary to discussing pain not because we understand, say, visual processing, so much better than pain processing—we do not—but because many of the facts of perceptual processing regarded as commonplace (even among philosophers of mind) are the same sort of facts that seem to confuse philosophers and psychologists when theorizing about pain.

Our visual system is quite complex, spans many areas in the brain, and is comprised of several subsystems whose interactions remain a mystery. It is widely known that different aspects of visual processing occur in different processing streams. For example, color is processed in intralaminar pathway, while motion is processed in the magnocellular. The auditory system works in an analogous fashion (though the interactions of its subsystems are not as mysterious). The medial superior olive of our auditory system probably computes sound location using interaural time differences. The lateral superior olive, on the other hand, computes sound location by using differences in interaural frequency.

What is important to notice is that it is quite all right for there to exist more than one processing stream in each modality. We might be mystified how color gets joined with shape and motion so that we have unified visual experiences of particular objects.[3] But we are not confused about whether the neuronal paths involved in computing an object's color are visual, or whether computing interaural time differences is auditory. We are perfectly happy to have each modality be involved in several maybe ultimately unrelated computations. We say (or, at least, I say) the parts of the brain that normally respond to impinging photons are part of the visual system, and the parts of the brain normally sensitive to air compression trains are part of the auditory system.

Naturally, this is a gross oversimplification of how our sensory modalities are actually individuated: without unpacking what is meant by "normal functioning," the definitions are virtually unworkable. By way of partially rectifying this gloss, let me briefly touch upon the top-down and bottom-up investigative methodologies in neuroscience (and in psychology, to some degree), for these analytic tools help disambiguate what counts as normal functioning. More importantly, they allow us to make claims about which computational algorithms and cell assemblies are and are not included in our brain systems and subsystems.

First, scientists use the method of *double dissociation* to isolate the processing streams that comprise our subsystems. If we can get *X* to occur without *Y* and also *Y* to occur without *X*, then scientists take this as grounds to claim that *X* and *Y* function as independent units. For example, explicit priming tasks in psychology demonstrate that we can record the meaning of a word or phrase without storing its syntax; implicit priming tasks show that the syntax of word or phrase can influence later linguistic processing while the meaning remains inert. I call this a *top-down strategy* because we start with a crude parsing of our system writ large (for example, linguistic processing) and then divide that system into its component pieces (syntactic processing, semantics). This method of investigation forms the backbone of Daniel C. Dennett's[4] functional decomposition.

Second, scientists rely on a *teleological analysis* to unite the various and sundry parts into wholes. Breaking down larger pieces into smaller ones is not enough to get the explanatory job done, espe-

[3] This is known as the binding problem in psychology; for discussion, see my "Psychology's Binding Problem and Possible Neurobiological Solutions," *Journal of Consciousness Studies*, 1 (1994): 66-90.

[4] *The Intentional Stance* (Cambridge: MIT, 1987).

cially when several of our systems overlap inside the head. Our brain houses lots of individual processors; knowing all the pieces does not identify the larger puzzles. Why do scientists believe that color and motion processing belong to the same system but that echolocation belongs to something else? This is not a trivial question since each of the subsystems is (mainly) anatomically and physiologically distinct from the other, and since individual neurons do not know the sort of signal to which they are responding. The information contained in an atmospheric compression wave or a photon wave triplet is transmitted as electrical and chemical energy once one moves inside the body.

Scientists use three converging strategies to isolate and construct systems from the component dissociable subsystems. First, they look for correlations between neural firing patterns and events in the external world, very much what Fred Dretske[5] has in mind with his informational semantics. Neurophysiologists take the smallest pieces of the puzzles, usually individual neurons, or the extracellular spaces around small groups of neurons, and record what they do under a variety of circumstances. They conclude that our color and shape detectors belong together because they are active under similar circumstances, namely, when the organism's retinas are bombarded by photons. Auditory cells are active in different contexts. Luckily for the scientists and their correlation project, true polymodal cells are relatively rare.

Second, scientists look at the neural connections fore and aft. Aside from knowing how a cell resonates with the environment, they also need to know to what this cell is connected—where the information the cell lights up to goes—and what is connected to the cell—how it gets the information to which it does respond. Determining the processing algorithm of any cell group is not as easy as it might sound; it is not a matter of merely recording all the stimuli it likes and then deciding what all the stimuli have in common. As S. R. Lehky and T. R. Sejnowski[6] remind us, even cells that we think we know well, such as D. H. Hubel and T. N. Weisel's[7] simple edge detectors, might not be involved in the computations we think they are. It is entirely likely, given Lehky and Sejnowski's simulation results,

[5] *Knowledge and the Flow of Information* (Cambridge: MIT, 1981), and *Explaining Behavior: Reasons in a World of Causes* (Cambridge: MIT, 1989).

[6] "Network Model of Shape-from-Shading: Neuron Function Arises from Both Receptive and Projective Fields," *Nature*, CCCXXXIII (1988): 452-54.

[7] "Functional Architecture of Macaque Monkey Visual Cortex," *Proceedings of the Royal Society of London B*, CXCVIII (1977): 1-59.

that the so-called "edge-detector" cells actually are involved in computing an object's axes of curvature.

For another, more striking example, consider synesthesia, a condition in which one gets a bimodal experience from monomodal inputs—one can see and hear colors or taste and hear words.[8] Although we would probably want to say that someone seeing blue columns when she hears a bell ringing is having a visual experience, we would not want to say that atmospheric compression waves are visual inputs, even for this person. The inputs are still auditory; they come in through the ears and pass through the traditional auditory centers. They just happen also to travel through some of the visual pathways (probably via the limbic system). If we only had access to single-cell recordings of synesthetic cells, we would obviously misidentify what those cells were doing. Knowing how things are connected prevents us from leaping to what would otherwise be an entirely rational (but also entirely false) conclusion.

Finally, scientists consider historical and evolutionary facts whenever possible. We are biological organisms equipped to move through our environment. We evolved that way because (roughly speaking) those who can move most effectively through their environment succeed in reproducing the most. When thinking about our perceptual systems, especially when worrying about various components' purposes, it is important to keep in mind how the hypothesized system or subsystem is supposed to function with regard to motor assembling. For most, if not all, information processing in the brain is related to the motor system in one way or another. For example, the visual areas all have at least some indirect contact with some motor structure or other, either the basal ganglia, or the motor cortex, or the tectum, or something.[9] Motor information needs to be "siphoned off" the visual pathways at all stages along the ascending route so that the visual input can be used for motor output.[10] Quite often what seems strange or curious from a psychological point of view seems quite natural from an evolutionary standpoint.

If we can group subsystems together into larger systems via their function—which is just what it is about that system that increases the reproductive rate of the organism that houses it[11]—then so much the better. The brain puts great emphasis on the priority of motor tasks, and we

[8] R.E. Cytowic, *Synesthesia: A Union of the Senses* (New York: Springer, 1989), and *The Man Who Tasted Shapes: A Bizarre Medical Mystery Offers Revolutionary Insights into Reasoning, Emotion, and Consciousness* (New York: Putnam, 1993).

[9] E.R. Kandel and J.H. Schwartz, *Principles of Neural Science* (New York: Elsevier, 1985, 2nd ed.).

[10] P.S. Churchland, "Epistemology in the Age of Neuroscience," this JOURNAL, LXXXIV, 10 (October 1987): 544-53.

[11] Cf. C. Wright, "Functions," *Philosophical Review,* LXXXII (1973): 139-82.

should pay attention to this emphasis. Whatever purpose we ultimately propose has to fit with our biological natures. (Often, however, such considerations are not possible or are little better than just-so stories, for the details of the advantages have been lost over evolutionary time. Why do we see in color, for example? What reproductive advantage would it have given our ancestors long ago? The answer is not easy, nor is it clear.)

I call this collection of research strategies *bottom up* because we begin with the smallest units in the brain and then arrange them into nested hierarchies. Based on gross similarities in response patterns, connections to other systems and organs, and putative selective advantages, we group the double-dissociated subsystems into hierarchically arranged classes. The process is not cut and dried by any means, but it is the best we have at the moment. Perhaps someday we shall be able to identify definitively to which system various cell assemblies belong in virtue of the inherent rhythms of the cells' firing patterns, or something like this.[12] Until then, though, the best we can do is to make educated guesses based on converging evidence.

Both approaches are required for a complete explanation of psychobiological phenomena. By breaking cognitive engines into interacting component pieces, the top-down strategy helps explain why organisms behave the way they do; and by categorizing and grouping the isolated parts, the bottom-up strategy helps explain what purpose the analyzed behavior serves. Reminding ourselves that we use both strategies in understanding our neural systems will rid us of the tendency to make our pain system into a cartoon; and reminding ourselves of biological heritage will aid in justifying a counterintuitive system that prevents our pains from occurring. Ultimately, I claim that our system for perceiving pain works in exactly the same fashion as our visual and auditory systems: it is a complex system with dissociable subsystems. Furthermore, it is a system that appears quite natural when considered against an evolutionary backdrop.

II. A SKETCH OF OUR PAIN SYSTEM

The classic view of our basic pain system is of two three-neuron subsystems.[13] (See figure 1.)[14] Each subsystem has a set of neurons which

[12] R. Emmers describes modality specific firing patterns in the thalamus in *Pain: A Spike-Interval Coded Message in the Brain* (New York: Raven, 1981).

[13] Even the classic story is becoming more complicated; see E.R. Kandel, J.H. Schwartz, and J.J. Jessel, *Principles of Neural Science* (New York: Elsevier, 1995, 3rd ed.), chapter 27.

[14] I owe this vision of the classic view to S.A. Cross's excellent review article, "Pathophysiology of Pain," *Mayo Clinic Proceedings*, LXIX (1994): 375-83. See also Kandel and Schwartz; and P. Roland, "Cortical Representation of Pain," *Trends in Neuroscience*, XV (1992): 3-5.

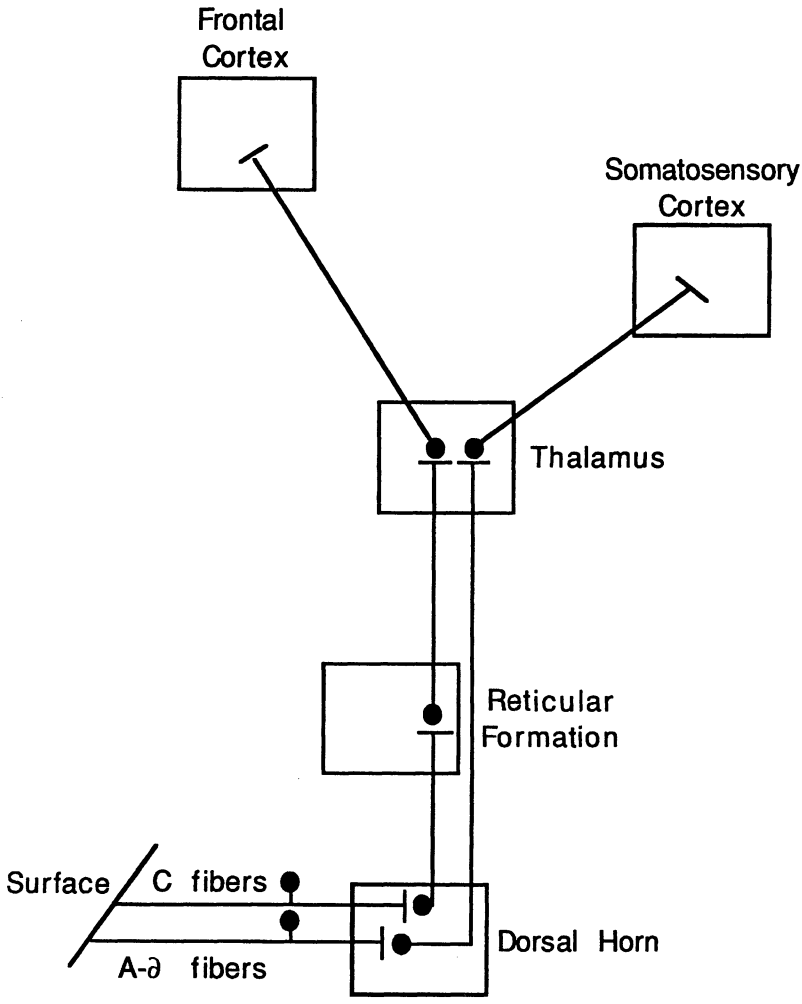

Figure 1. Diagram showing our pain sensory system. The first set of neurons take in information from the periphery and then synapse with a second set of neurons in the dorsal horn. These neurons ascend, some terminating in the reticular formation of the brain stem, others traveling to the thalamus. Axons that terminate medially in the thalamus synapse with a third set of neurons that project to the frontal cortex. Those which terminate laterally synapse with neurons that project to the somatosensory cortex.

resides in the dorsal root ganglion of the spinal column. These neurons extend their axons to whatever tissue they innervate and receive external input there. They also have a second axon that projects across to the dorsal horn. The axon in the dorsal horn connects with

a second set of neurons housed in the dorsal horn whose axons run out of the spinal column and up to the thalamus. The third set of neurons projects from the thalamus to the postcentral gyrus in the cerebral cortex.

In 1911, H. Head and G. Holmes[15] proposed a dual system of afferent projections in our pain sensory system: an *epicritic* system that processes information regarding intensity and precise location, and a *protopathic* system that delivers the actual pain sensations. Eighty-six years later, we still believe they were fundamentally correct. We now know that we have a "sensory discriminative" subsystem that computes the location, intensity, duration, and nature (stabbing, burning, prickling) of the stimuli. This subsystem is subserved by the A-∂ fibers. These mechanoreceptive neurons are mylinated, so information can travel quite quickly along them (approximately 5-30 m/sec, as opposed to .5-2 m/sec for information traveling along unmylinated pathways[16]). Consequently, they transmit what is known as "first pain" or "fast pain." The threshold for activation is constant from person to person (threshold here), and this subsystem remains active (assuming no other defects in the organism) only as long as the raw nerve endings are stimulated.

We also have an "affective-motivational" subsystem that supports the unpleasant part of painful sensations. This system feeds directly into our motor response systems and is considered to be phylogenetically older than other aspects of our multifaceted pain system. This polymodal subsystem begins with the well-known unmylinated C fibers. Once they are activated, they will continue to fire for some time, even after the noxious event has ceased. This subsystem gives rise to what is known as "slow pain" or "second pain," so-called because this is what we feel second whenever we are injured—a diffuse and persistent burning pain. When someone has chronic pain, a protracted second pain is what is being referred to.

Similar to the color and form processors in the visual system, the A-∂-fiber and C-fiber pathways remain largely segregated. For example, generally speaking, they terminate in different layers on the dorsal horn. But there is more interaction than what we find in either the visual or auditory system. The dorsal horn contains *wide-dynamic range* (WDR) neurons that respond to both A-∂ and C neurons, as well as to other peripheral stimuli. WDR neurons are also sensitive to

[15] "Sensory Disturbances from Cerebral Lesions," *Brain,* XXXIV (1911): 102-254.

[16] Information traveling at the slower speeds would take about 8 seconds to reach a horse's spinal column from its hoof.

visceral stimuli. It is possible that referred and sympathetic pains depend upon this sort of visceral-somatic convergence.[17]

Once pain information exits the dorsal horn, it travels either to the reticular formation in the brain stem or to the thalamus. Laminae I and V project to the lateral nuclei in the thalamus,[18] and laminae I, V, and VI project to the medial nuclei. Each type of nuclei underwrites a different sort of information; the lateral nuclei process discriminative information (fast pain), while the medial nuclei and reticular connections process affective-motivational information (slow pain). The two thalamic streams remain separate on their trip to the cortex as well. Pain neurons in the lateral nuclei synapse in the somatosensory cortex, which then can compute the location and characteristics of the pain; those in the medial nuclei synapse in the anterior cingulate gyrus in the frontal lobe, which figures in our emotional reactions to pain. The frontal lobe (and its connections) process our actual suffering.

III. PHILOSOPHY'S ERROR

Now we can see how and why several philosophers are mistaken in their conclusions that there are no such things as pains,[19] or that pains are located in our limbs,[20] or that pains are purely subjective,[21] or that pains are reactive behaviors.[22] Each of these positions identifies pain with one of the neuronal groups within the pain system, while failing to recognize that our pain system is complex and contains at least a duality[23] of sub-

[17] For contrary evidence, though, see G.D. Schott, "Visceral Afferents: Their Contribution to 'Sympathetic Dependent' Pain," *Brain,* CXVII (1994): 397-413. Other neurons that show this sort of convergence are the "nociceptive-specific" neurons in lamina I and the "complex" neurons in laminae VII and VIII of the dorsal horn. The interaction of autonomic information with somatic appears to be quite common throughout our pain system.

[18] A.D. Craig, M.C. Bushnell, E-T. Zhang, and A. Blomqvist, "A Thalamic Nucleus Specific for Pain and Temperature Sensation," *Nature,* CCCLXXII (1994): 770-73.

[19] P.M. Churchland, "Reduction, Qualia, and the Direct Introspection of Brain States," this JOURNAL, XXXVIII, 1 (January 1985): 8-28; Dennett, "Why You Can't Make a Computer That Feels Pain," *Synthese,* XXXVIII (1978): 449.

[20] D.M. Armstrong, *The Nature of Mind and Other Essays* (Ithaca: Cornell, 1981); N. Newton, "On Viewing Pain as a Secondary Quality," *Noûs,* XXIII (1989): 569-98; G. Pritcher, "Pain Perception," *Philosophical Review,* LXXIX (1970): 368-93.

[21] G.R. Gillett, "The Neurophilosophy of Pain," *Philosophy,* LXVI (1991): 191-206; N. Grahek, "Objective and Subjective Aspects of Pain," *Philosophical Psychology,* IV (1991): 249-66; C. McGinn, *The Subjective View* (New York: Oxford, 1983).

[22] L. Wittgenstein, *Philosophical Investigations,* G.E.M. Anscombe, trans. (Cambridge: Blackwell, 1953).

[23] R. Melzack and P.D. Wall, "Pain Mechanisms: A New Theory," *Science,* CL (1965): 971-79, and E.R. Hilgard and J.R. Hilgard, *Hypnosis in the Relief of Pain* (New York: Brunner/Mazel, 1994, rev. ed.) both argue that there are three components to pain processing: the discriminative, the affective-emotional, and the evaluative. I find little physiological evidence to support these claims. Here, I discuss only two components.

systems, each of which processes a different sort of information. (See figure 2.) In general, philosophers make these mistakes because they misunderstand the double dissociation methodology. We can, either through purposeful intervention or accidents of nature, dissociate our discriminative pain processing from our affective-motivational pain processing. Ingestion of morphine (or other opiates), lesions to the medial thalamus, and prefrontal lobotomies all result in sensations of pain without a sense of suffering and without producing characteristic pain behaviors (wincing, moaning, complaining, and so on) (*ibid.*). In these cases, patients can localize their pains but are not upset by the

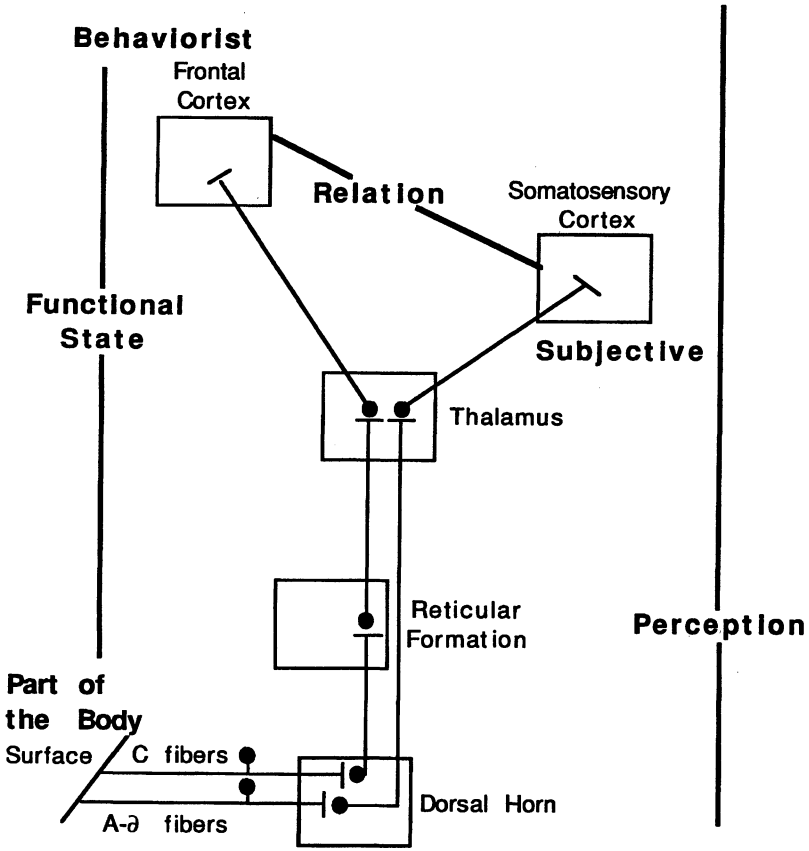

Figure 2. Part of the pain system identified with different philosophical views regarding the basic nature of pain. Most views identify pain with one subsystem or with one sort of neural processor. Two exceptions are functional state and perceptual views of pain, though perceptual views of pain often overlook or underestimate the motor component of pain processing.

fact that they are in pain. We can also get reverse effects, to a degree. Fentanyl causes one to react in pain, yet inhibits our discriminatory abilities for the pain.[24] Lesion studies and studies using hemispherectomies show that even with the cortex completely missing, we can still have a pain sensation; we simply lack fine localization and intensity discrimination.[25] Patients with Parkinson's disease and Huntington's chorea often have pain sensations but are unable to indicate where they feel the pains.[26]

We also find instances of the pain centers in the thalamus and cortex being activated without corresponding activations of A-∂ or C fibers ("nociception"). Fully 80% of lower back pain sufferers present no external or internal injury.[27] Phantom limbs and phantom pains in phantom limbs are quite common experiences in new amputees.[28] Stimulating the medial periaqueductal gray region, tectum, or thalamus directly can also result in painful experiences.[29] Finally, our emotional states heavily influence the degree of pain we feel, quite independent of actual injury. Indeed, psychogenic pains, pains without any corresponding injury or peripheral stimulation, have been documented for quite some time.[30]

[24] R.H. Gracely, R. Dubner, and P.A. McGrath, "Fentanyl Reduces the Intensity of Painful Tooth Pulp Sensations: Controlling for Detection of Active Drugs," *Anesthesia and Analgesia,* LXI (1982): 751-55.

[25] K.D. Davis, R.R. Tasker, Z.H.T. Kiss, W.D. Hutchison, and J.O. Dostrovsky, "Visceral Pain Evoked by Thalamic Microstimulation in Humans," *Neuroreport,* VI (1995): 369-74; L.A. French, S.N. Chou, and J.L. Story, *Clinical Orthopedics,* XLVI (1966): 83-86; Head and Holmes; and Roland.

[26] E.H. Chudler and W.K. Dong, "The Role of the Basal Ganglia in Nociception and Pain," *Pain,* LX (1995): 3-85.

[27] Wall, "Introduction," in Wall and Melzack, eds., *Textbook of Pain* (New York: Churchill Livingstone, 1989, 2nd ed.), pp. 1-18.

[28] See discussion in Hilgard and Hilgard; T.S. Jensen and P. Rasmussen, "Phantom Pain and Related Phenomena After Amputation," in Wall and Melzack, eds., pp. 508-21.

[29] Davis et alia; K.A. Keay, C.I. Clement, B. Owler, A. Depaulis, and R. Bandelr, "Convergence of Deep Somatic and Visceral Nociceptive-Information onto a Discrete Ventrolateral Midbrain Periqueductal Gray Region," *Neuroscience,* LXI (1994): 727-32.

[30] R. Roy, "Engel's Pain-Prone Disorder Patient: 25 Years After," *Psychotheraphy Psychosomatics,* XLIII (1985): 126-35. In *Illustrations of the Influence of the Mind upon the Body in Health and Disease Designed to Elucidate the Imagination* (Philadelphia: Henry C. Lee's, 1884, 2nd ed.), D.H. Tuke reported the case of a butcher who got fouled up on a meat hook and appeared to be in agony. When examined by the local chemist, however, it was discovered that the meathook had only penetrated his jacket sleeve and, even though the butcher was screaming in "excessive pain," he was completely unharmed (p. 168, as reported in A. Gamsa, "The Role of Psychological Factors in Chronic Pain, I: A Half Century of Study," *Pain,* LVII (1994): 5-15).

Correlatively, there are also examples of our peripheral pain processors being activated without this information proceeding on to the thalamus or cortex. About 37% of emergency-room visitors felt no pain at the time of their injury.[31] Athletes and soldiers can continue performing free of pain, even though they have been severely injured.[32] Hypnosis allows some subjects to engage in what would otherwise be painful activities without being in pain.[33] Placebos are notoriously helpful in relieving pain. (Interestingly enough, they relieve pain at half the rate of the real drug, regardless of the supposed strength of the drug.[34]) Of course, some lesions to the thalamus and cortex can result in the cessation of pain experiences, even though the peripheral neurons continue to operate normally.

Each of these double dissociations, however, individuates neuronal groups or subsystems only *within* our overarching pain system. I make this claim by analogy with our other perceptual systems. Blindsight patients can discriminate shapes and figures they claim not to be able to see consciously. Sufferers of Anton's syndrome insist that they can see perfectly well, even though they are completely blind and have severe bilateral damage to their visual association areas. No one uses these facts to argue, however, that vision does not exist, or that vision is located in our eyeballs, or that vision is purely subjective, or that vision is behavioral. We may not know exactly what to say about blindsight or Anton's syndrome, but no one claims that blindsight is not a disorder of the visual system or that patients with Anton's syndrome are not having a visual experience of some sort.

By misunderstanding what a perceptual system in the brain encompasses, many philosophers miss the boat regarding the basic nature and structure of pain. Double dissociation alone does not individuate our basic systems; that is used to isolate the subsystems that operate within the larger system. We then need to build our different systems out of the component pieces. Teleological considerations help us to do so. To wit: the neurons in our pain system all

[31] Melzack, Wall, and T.C. Ty, "Acute Pain in an Emergency Clinic: Latency of Onset and Descriptor Patterns Related to Different Injuries," *Pain*, XIV (1982): 33-43.

[32] H.K. Beecher, "Relationship of Significance of Wound to the Pain Experience," *Journal of the American Medical Association*, CLXI (1956): 1609-13.

[33] Evoked potential recordings of painful stimuli under hypnosis indicates that at least activity in the frontal lobe is affected (Helen Crawford, personal conversation).

[34] F.J. Evans, "The Placebo Response in Pain Reduction," in J.J. Bonica, ed., *International Symposium on Pain, Advances in Neurology, Volume IV* (New York: Raven, 1974), pp. 289-96.

respond to roughly the same sort of information; they increase their rate of firing in the presence of noxious stimuli on skin or deep organs. Moreover, the connection among the six-neuron tract is a stable, common, and isolable pathway. Connections fore and aft show a stream of information flowing from the nociceptors on the skin up through the cortex.[35] Finally, a pain sensory system tied to the somatosensory processors makes good evolutionary sense. As creatures eking out lives in a hostile environment, having a system which could warn us when damage occurred and which could force us to protect damaged parts until they healed would be tremendously beneficial. (Indeed, persons who cannot feel any pain at all often live a nasty, brutish, and short life.[36])

Neither our conscious experience of pain, the damaged tissue itself, nor our bodily or emotional reactions are fundamental to pain processing. Each is but one component of a larger processor. Hence, it is a mistake to try and claim one or the other as pain *simpliciter.* It is equally erroneous to conclude that since we cannot identify one or the other with pain, there is no such thing. The entire pain sensory system functions largely the same as any of our sensory systems. Their pieces are united by our best guess of their function, based on the three types of converging evidence discussed above. Hence, we have concluded that the components of our visual system take the information contained in photons bouncing around in the world and use it to compute the location, orientation, texture, color, and movement of objects in the environment. The components of our auditory system take the information contained in atmospheric compression waves and use it to compute the placement of things. And the components of our pain system take pressure, temperature, and chemical readings of our surface (and interior) and use this information to track what is happening to our tissues. The A-∂ cells and the C fibers do this, as do the spinothalamic tract and its connections to the cortex. In sum, we have a complex but well-defined sensory system that monitors our tissues to promote the welfare of our bodies.[37]

[35] Exactly what processing algorithms are being executed is a more complicated story, and one heavily influenced by the pain inhibitory system. Accounting for the details of the computations is beyond the scope of this paper.

[36] Critchley, "Congenital Indifference to Pain," *Annals of Internal Medicine,* XLV (1956): 737-47; R.A. Sternbach, "Congenital Insensitivity to Pain: A Critique," *Psychological Bulletin,* LX (1963): 252-64; and *Pain: A Psychophysiological Analysis* (New York: Academic, 1968).

[37] See also Wall. This view, too, accounts for why our C-fiber systems might be so slow. General monitoring of bodily conditions should not often require a quick response.

IV. STRANGE FACTS ABOUT PAIN

As I stated in the introduction, the above is an easy and less interesting reason why philosophers are wrong. More to the point: this way of looking at our pain system does not clear up all of the confusions, mysteries, and conflicts in our account of pain. There are several important empirical facts that any theory of pain needs to be able to explain. These are facts that, by and large, do not have analogues in our other perceptual systems. Moreover, they are facts that lead otherwise intelligent people to make prima facie bizarre statements regarding what pain is and is not. Here, I can only touch upon a few such facts, but it should be enough to motivate the challenge to theories of pain as well as to justify the approach I shall advocate in understanding pain phenomena toward the end of this paper.

(1) There is, in fact, a poor correlation between nociception and pain perception.[38] That is, the relationship between stimulating the A-∂ and C fibers and actually feeling or reporting a pain is not at all straightforward. Several tribal rituals give vivid illustrations of the dissociation. In parts of India, for example, men chosen to represent the gods have steel hooks inserted under the muscles of their back. They then swing above the crowds, suspended on these hooks by ropes, blessing children and crops. They exhibit no pain.[39] I mentioned that about 40% of all emergency-room patients reported feeling no pain at the time of injury; 40% more report greater pain than one would expect, leaving only 20% of all emergency-room visitors having pains appropriate to their injuries.[40] It is not the case that we can dissociate nociception from discriminative and affective-motivational reaction; it is that they regularly and frequently dissociate.

Our other perceptual systems are not like this. There is a highly reliable correlation between having the rods and cones in our retina being bombarded by light photons and our having some visual experience or other.[41] There is also a highly reliable correlation between

[38] Wall, "The Dorsal Horn," in Wall and Melzack, eds., pp. 102-11; Wall and S.B. McMahon, "Microneuronography and Its Relation to Perceived Sensation," *Pain*, XXI (1985): 209-29.

[39] D.D. Kosambi, "Living Prehistory in India," *Scientific American*, CXVI (1967): 105-14.

[40] See Melzack et alia.

[41] It is a live debate whether there is a good correspondence between the pattern of activity in the retina and a particular *type* of experience. How you answer that question depends upon how elastic you believe our visual "module" to be. P.M. Churchland and R.L. Gregory, for example, think that our visual system is cognitively penetrable from above; J. Fodor does not.

the vibration of our tympanic membranes at a certain frequency and hearing sounds. Any theory of pain is going to have to explain why our peripheral sensors for noxious stimuli do not appear to be well-connected to our sensations of pain. Indeed, relation between external events and internal indicators is part of what individuates our systems. Without better correlation between an external event and internal activity, we cannot claim that our putative pain system is, in fact, a pain system.

(2) Chronic pains are, by and large, a mystery.[42] Some people have pains which last for years, with no discernible cause, and which are completely resistant to treatment. These are cases above and beyond things like phantom pains (which also have no discernible cause and are resistant to treatment), for one might argue that phantom pains are simply abnormal instances of human suffering. (Who knows what is supposed to happen if you lose something as large as a limb? Surely, many areas of the topographically arranged somatosensory system will be thrown off track; maybe pains that appear to be in the limb that no longer exists are not so strange after all.[43]) But with chronic pains, otherwise perfectly normal people—with no serious (or even superficial) injury—live their lives in constant pain. Moreover, removing bits of the spinal column, the dorsal horn, the thalamus, the reticular formation, the somatosensory cortex, or the frontal lobe concerned with pain have no effect on the patients being in pain.

Again, there are few parallels with other perceptual systems. Rarely do otherwise normal individuals have ongoing visual or auditory experiences without some determinate cause and explanation. Chronic hallucinations by themselves are quite rare.

(3) Low-level stimulation of our thermoreceptors (the larger A-β fibers), which are not supposed to be connected to pain perception, inhibits the experience of pain,[44] while a higher level stimulus exac-

[42] K.L. Casey, S. Minoshima, R.A. Koeppe, J. Weeder, and T.J. Morrow, "Temporo-Spatial Dynamics of Human Forebrain Activity During Noxious Heat Stimulation," *Society of Neuroscience Abstracts,* XX (1994): 1573; C.A. Pagni, "Central Pain Due to Spinal Cord and Brian Stem Damage," in Wall and Melzack, eds., pp. 634-55.

[43] Some phantom pains may be attributable to memories of recent pains in the amputated limb, for phantom pains bear an uncanny resemblance to pre-amputation experiences of pain. See J. Katz, "Psychophysiological Contributions to Phantom Limbs," *Canadian Journal of Psychiatry,* XXXVII (1992): 282-98; E.J. Krane and L.B. Heller, "The Prevalence of Phantom Sensation and Pain in Pediatric Amputees," *Journal of Pain Symptom Management,* X (1995): 21-29; Melzack, "Phantom Limbs, the Self, and the Brain," *Canadian Psychology,* XXX (1989): 1-16.

[44] Wall, "Presynaptic Control of Impulses at the First Central Synapse in the Cutaneous Pathway," *Physiology of Spinal Neurons: Progress in Brain Research XII* (New

erbates pain.[45] Here, there are some parallels with our other systems, if we take the A-β interference to be a type of hard-wired pain illusion. Similarly, we see a straight stick as bent when placed half way in water, and we arrange groups of dots into rows and columns.

Facts such as these led the International Association for the Study of Pain (IASP) Subcommittee on Classification to conclude: "Pain is always subjective.... Many people report pain in the absence of tissue damage or any pathophysiological cause; usually this happens for psychological reasons. There is usually no way to distinguish their experience from that due to tissue damage if we take the subjective report... [P]ain...is always a psychological state)."[46] Could they be correct? Are the connections between actual tissue damage, or some other injury, and our sensation of pain so weak that it is better to discount nociception entirely when defining pain? The IASP subcommittee clearly thinks so in their definition of pain: "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, *or described in terms of such damage*" (*ibid.*; italics added).

This sort of position is not preferable, for a variety of reasons. First, if pains are not correlated with actual injury, or the potential for damage, then we lose our intuitive evolutionary story about why we have a pain-sensing system. If our pain system has somehow become detached from the job it is supposed to perform, then its existence and poor performance no longer have clear explanations.

Second, pains become very peculiar phenomena indeed, quite unlike our other qualitative experiences. We can have visual or auditory hallucinations—we can be mistaken about what we think we are perceiving—but if pain is purely subjective, then there is no way for us to have an illusion of being in pain. Phantom pains become just regular pains instead of some special case demanding special consideration and treatment. Of course, saying something is strange is not a reason for saying that it does not exist, but it is a reason, I believe, to be cautious in making such metaphysical commitments. Indeed, I do

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York: Elsevier, 1964), pp. 92-118; Wall and Cronly-Dillon, "Pain, Itch, and Vibration," *Archives of Neurology*, II (1960): 365-75; Wall and W.H. Sweet, "Temporary Abolition of Pain in Man," *Science*, CLV (1967): 108-09; see also W.D. Willis and R.E. Coggeshall, *Sensory Mechanisms in the Spinal Cord* (New York: Plenum, 1978); T.L. Yakshe, *Spinal Afferent Processing* (New York: Plenum, 1986).

[45] J.C. Willer, F. Boureau, and D. Albe-Fessard, "Human Nociceptive Reactions: Effects of Spatial Summation of Afferent Input from Relatively Large Diameter Fibers," *Brain Research*, CI (1980): 465-70.

[46] International Association for the Study of Pain (IASP) Subcommittee on Classification, "Pain Terms: A Current List with Definitions and Notes on Usage," *Pain*, Supplement 3 (1986), p. 217.

think that the IASP subcommittee (and Colin McGinn and Saul Kripke) are quite wrong in their understanding of what pain is.

V. GATE THEORIES OF PAIN

In general, gate theories of pain argue that the robust feedback loops in our pain system serve to inhibit, enhance, or distort incoming nociceptive information. So, in addition to the basic six-neuron structure of our pain system I outlined above, there are additional ascending connections from the spinal cord to the brain stem, circular pathways from the spinal cord to other areas in the spinal cord itself, and descending feedback loops from the cortex, hypothalamus, and brain stem back to the spinal cord.[47] Some portions of this theory have been worked out in considerable detail. Some of the mysterious pain phenomena can be explained in terms of well-confirmed portions of Melzack and Wall's[48] gate theory of pain.

Take the example of altering our sensation of pain by stimulating the larger A-β fibers. The A-∂ and C neurons in the dorsal horn are connected via inhibitory interneurons. These interneurons are stimulated by low-threshold A-β fibers. This means that stimulating A-β cells dampens the activity of the A-∂ and C neurons in the dorsal horn, which in turn means that less pain information would travel up to the brain.[49] (See figure 3.) Or, another example: gently pressing a grid with alternating cool and warm bars on the skin often causes the sensation of a strong burning pain.[50] Neurophysiological recordings now show that there are several ascending neurons that are sensitive to both pain and temperature. A bit of central disinhibition plus these bimodal neurons explain how this illusion can occur. It can also explain why cold things burn.[51] The relationship between tactile stimulation and pain is not completely clear here, however. Recent evidence indicates that painful stimuli can substantially decrease our sensations of touch.[52]

[47] See also Apkarian.

[48] Wall, "Introduction"; Melzack and Wall, "Pain Mechanisms," and *The Challenge of Pain* (New York: Penguin, 1986); see also N.F. Britton and S.M. Skevington, "A Mathematical Model of the Gate Control Theory of Pain," *Journal of Theoretical Biology*, CXXXVII (1980): 91-105. Several aspects of the original theory have been shown to be incorrect; for discussion, see P.W. Nathan, "The Gate-Control Theory: A Critical Review," *Brain*, XCIX (1976): 123-58; and Kandel and Schwartz. Most accept the general outline of the view, however.

[49] Wall, "Introduction."

[50] Known as Thunberg's thermal grill illusion, this was first demonstrated in 1896.

[51] A.D. Craig and M.C. Bushnell, "The Thermal Grill Illusion: Unmasking the Burn of Cold Pain," *Science*, CCLXV (1994): 252-55.

[52] Apkarian, R.A. Stea, and S.J. Bolanowski, "Heat-Induced Pain Diminishes Vibrotactile Perception, A Touch Gate," *Somatosensory Motor Research*, XI (1994): 259-67.

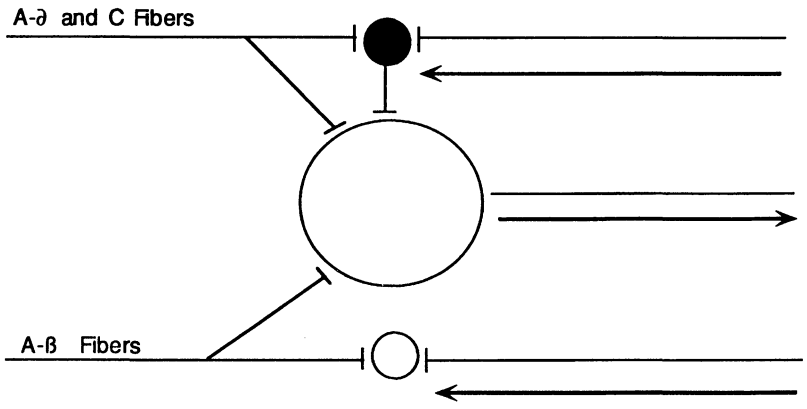

Figure 3. Low-level gate control. Cells in the dorsal horn and trigeminal nuclei which respond to A-∂- and C-fiber input have excitatory (filled circle) and inhibitory (open circle) interneurons associated with them. A-β fibers activate the inhibitory neurons as well as input to the nociceptive cells. Descending controls input to the interneurons.

Moreover, though more research needs to be done, the alternate routes in the central nervous system might help explain why cutting the primary dorsal horn pain channel, or parts of the spinal cord itself, may not remove chronic pain.[53] Indeed, it looks as though the failure at curing chronic pains by lesions is overstated anyway.[54] Some neuropathic pains are controllable using subarachnoid opioids, as they interfere with the pain centers in the dorsal horn, which are probably responsible for deafferantation pain.[55] Other data indicate that most chronic or prolonged pains are the result of changes in central nervous functions that have been caused by the neural firing patterns of early nociception.[56]

A Melzack-Wall type of gate-control theory of pain cannot be the entire story, however, for three reasons. First, we do not have anything approaching a complete theory at this stage in the game. Al-

[53] S. Canavero, "Dynamic Reverberation: A Unified Mechanism for Central and Phantom Pain," *Medical Hypotheses,* XVII (1994): 203-07; see also A. Dray, L. Urban, and A. Dickenson, "Pharmacology of Chronic Pain," *Trends in Pharmacological Science,* XV (1994): 190-97.

[54] See Canavero.

[55] R.P. Iacono, M.V. Boswell, and M. Neumann, "Deafferentation Pain Exacerbated by Subarachnoid Lidocaine and Relieved by Subarachnoid Morphine: Case Report," *Regional Anesthesiology (United States),* XIX (1994): 212-15.

[56] See discussion and review in A.L. Vaccarino and D.A. Chorney, "Descending Modulation of Central Neural Plasticity in the Formalin Pain Test," *Brain Research,* DCLXVI (1994): 104-08.

though the theory does explain low-level phenomena, nothing has been worked out in particular regarding the clearly "psychological" influences.[57] Melzack and Wall themselves simply gesture toward a central gating mechanism that presumably would explain hypnosis effects, any remaining chronic pains, the dismal correlations between emergency-room injuries or back injuries and pain, and so on. But without providing more details—where this central gating mechanism is located in the brain, how it functions to alter our pain perceptions, why we have such a powerful mechanism, for example—then they have said little more than that "some other stuff happens in the head that explains everything else." As Wall himself remarks of the many areas implicated in inhibiting our sensations of pain: "unfortunately we know little of their relative importance and nothing of the actual circumstances in which they come into action."[58]

Second, and more importantly, even if we could get the details on some sort of central gating mechanism, this would not mean that pains are not largely subjective (which is but a step away from being purely subjective). If top-down cortical processes (which is what I take 'purely subjective' to mean here) are mainly responsible for our sensations of pain, then the IASP subcommittee would be right and pains would be deeply peculiar. They would be the only perceptual experiences we have which are normal (that is, normal functioning creatures have them), natural (that is, not the product of tweaking something in a laboratory, but occurring in the wild, as it were), commonplace (we have them all the time), believed to be giving us information about the external environment (external to the brain, that is (but internal to our bodies)), but in fact not. Dreams might be the only exception, but, unlike dreams, we do not realize when a pain is over that it really was not about the external world after all.

Finally, "the body of psychological research into pain has failed to yield compelling evidence for a direct causal relationship between psychological factors and pain in the general population of pain patients."[59] In general, if physicians cannot find a physical cause for a pain and it continues despite medical interventions, then it is attrib-

[57] The influence of the psychological on the perception of pain has a long and venerable history; see Whytt (1786), Brodie (1837), Carter (1853), as described in H. Merskey and F.G. Spear, *Pain: Psychological and Psychiatric Aspects* (London: Baillière, Tindall, and Cassell, 1967).

[58] Wall, "Introduction," p. 12.

[59] A. Gamsa, "The Role of Psychological Factors in Chronic Pain, II: A Critical Appraisal," *Pain,* LVII (1994): 23.

uted to something "psychological" (*ibid.*).[60] This can be at best an unsupported hypothesis, however. At worst, it is a euphemism for our own ignorance. If pains are driven by psychological factors, then we should be able to isolate what those factors are. Thus far, we have been unsuccessful in discovering any psychological trait or ailment that is correlated with sensations of pain. In sum: those who claim that pain is subjective or psychological are being too facile.

Most philosophers, psychologists, and neurophysiologists who do not fall prey to the less interesting mistake do hold some version of a gate-control theory. Some advocate it explicitly (for example, Dennett, Melzack, Wall); others maintain it implicitly (Hilgard, Tye). Either way, I believe this commitment to be premature. If there is any way for us not to hold that our pain system is purely, or largely, or even significantly subjective, then we should not do it. I propose that instead of acquiescing to the IASP subcommittee's conclusion, we should take the mysterious and unexplained pain phenomena as evidence that the six-neuron view (even with the additional bells and whistles) is wrong. I suggest that we should reexamine the data from a different perspective.

VI. A PAIN-INHIBITING SYSTEM

If standard (higher level) gate-theory approaches are misguided, then we still have a chance at understanding pain from a solid neurophysiological and biological perspective without making pain into something peculiar. As will be clear in a moment, I advocate dividing what Melzack et alia have lumped into one cortically-driven pain system into two separate systems: a nociceptor-driven pain sensory system (PSS) and a largely top-down pain inhibitory system (PIS). First, though, let me sketch some data that I believe are relevant to this perspective, data that the more traditional gate theories either overlook or minimize; I shall then outline some theoretical considerations that point toward a two-system model.

Proponents of gate theories write as though just about any psychological event or any area of the cortex has the potential of influenc-

[60] R.C. Kupers, H. Konings, H. Andriaensen, and J. Gybels, "Morphine Differentially Affects the Sensory and Affective Pain Rating in Neurogenic and Idiopathic Forms of Pain," *Pain*, XLVII (1991): 5-12; see also discussion in S. Benjamin, D. Barnes, S. Berger, I. Clarke, and J. Jeacock, "The Relationship of Chronic Pain, Mental Illness, and Organic Disorders," *Pain*, XXXII (1988): 185-91; M. Grushka, B.J. Sessle, and R. Miller, "Pain and Personality Profiles in Burning Mouth Syndrome," *Pain*, XXVIII (1987): 155-67; H. Merskey, "Symptoms that Depress the Doctor, Too Much Pain," *British Journal of Hospital Medicine* (January 1984): 63-66, and "Psychiatry and Chronic Pain," *Canadian Journal of Psychiatry*, XXXIV (1989): 329-35; R.A. Sherman, C.J. Sherman, and G.M. Bruno, "Psychological Factors Influencing Chronic Phantom Limb Pain: An Analysis of the Literature," *Pain*, XXVIII (1987): 285-95.

ing the perception of pain, what Melzack calls a *neuromatrix.*[61] Although there are lots of feedback loops and other sorts of pain connections, not every area in the brain is sensitive to pain information. Recent advances in functional imaging technology—which includes magnetic resonance imaging (MRI), xenon gamma emission detection, single photon computer emission tomography (SPECT), and positron emission tomography (PET)—allow us to map active areas of the brain with greater precision than ever before.[62] Imaging studies of pains clearly show that chronic pains are correlated with increased activity in the cingulate and frontal cortex, as well as sometimes with the insular cortex, hypothalamus, and periaqueductal gray.[63] Phasic pains are keyed to increased activity in the anterior cingulate, frontal cortex, thalamus, and primary and secondary somatosensory cortex.[64] Although lack of activity does not prove con-

[61] Melzack, "Phantom Limbs and the Concept of a Neuromatrix," *Trends in Neuroscience,* XIII (1990): 88-92; "Central Pain Syndromes and Theories of Pain," in E.L. Casey, ed., *Pain and Central Nervous Disease: The Central Pain Syndromes* (New York: Raven, 1991), pp. 59-64, and "Phantom Limbs," *Scientific American,* CCLXVI (1992): 90-96; see also discussion in Canavero.

[62] See Apkarian.

[63] Y. Hosobuchi, "Treatment of Cerebral Ischemia with Electrical Stimulation of the Cervical Spinal Cord," *Pace,* XIV (1991): 122-26; Y. Katayama, T. Tsubokawa, T. Hirayama, G. Kido, T. Tsukiyama, and M. Iio, "Response of Regional Cerebral Blood Flow and Oxygen Metabolism to Thalamic Stimulation in Humans as Revealed by Positron Emission Tomography," *Journal of Cerebral Blood Flow Metabolism,* VI (1986): 637-41; E.C. La Terre, A.G. De Volder, and A.M. Goffinet, "Brain Glucose Metabolism in Thalamic Syndrome," *Journal of Neurosurgery and Psychiatry,* LI (1988): 427-28; Y.R. Tran Dinh, C. Thurel, A. Serrie, G. Cunin, and J. Seylaz, "Glycerol Injection into the Trigeminal Ganglion Provokes a Selective Increase in Human Cerebral Blood Flow," *Pain,* XLVI (1991): 13-16.

[64] Apkarian; Apkarian, R.A. Stea, S.H. Manglos, N.M. Szeverenyi, R.B. King, and F.D. Thomas, "Persistent Pain Inhibits Contralateral Somatosensory Cortical Activity in Humans," *Neuroscience Letters,* CXL (1992): 141-47; M. Backonja, E.W. Howland, J. Wang, J. Smith, M. Salinsky, and C.S. Cleeland, "Tonic Changes in Alpha Power During Immersion of the Hand in Cold Water," *Electroencephlography and Clinical Neurophysiology,* LXXIX (1991): 192-203; E.L. Casey, ed. *Pain and Central Nervous Disease;* K.L. Casey, S. Minoshima, K.L. Berger, R.A. Koeppe, T.J. Morrow, and K.A. Frey, "Positron Emission Tomographic Analysis of Cerebral Structures Activated Specifically by Repetitive Noxious Heat Stimuli," *Journal of Neurophysiology,* LXXI (1994): 802-07; R.C. Coghill, J.D. Talbot, A.C. Evans, E. Meyer, A. Gjedde, M.C. Bushnell, and G.H. Duncan, "Distributed Processing of Pain and Vibration by the Human Brain," *Journal of Neuroscience,* XIV (1994): 4095-108; V. Di Piero, S. Ferracuti, U. Sabatini, P. Pantano, G. Cruccu, and G.L. Lenzi, "A Cerebral Blood Flow Study on Tonic Pain Activation in Man," *Pain,* LVI (1994): 167-73; G.H. Duncan, C. Morin, R.C. Coghill, A. Evans, K.J. Worsley, and M.C. Bushell, "Using Psychophysical Ratings to Map the Human Brain Regression of Regional Cerebral Blood Flow (RCBF) to Tonic Pain Perception," *Society of Neuroscience Abstracts,* XX (1994): 1572; J. Hsie, Ö. Hägermark, M. Stahle-Bäckdahl, K. Ericson, L. Eriksson, S. Stone-Elander, and M. Ingvar, "Urge to Scratch Represented in the Human Cerebral Cortex During Itch," *Journal of Neurophysiology,* LXXII (1994): 3004-08; A.K.P. Jones,

clusively that an area is not sensitive to pain information, these sorts of studies should give advocates of global cortical influences on our perception of pain pause for thought.

Moreover, the neuronal areas sensitive to pain information are different from what we originally believed. Rat studies suggest that, in addition to the structures discussed above, areas of the limbic system are also involved.[65] Functional images of human brains indicate that homologous areas are involved in us as well.[66] Moreover, limbic activity in chronic pains becomes quite important as imaging studies also show a *decrease* in thalamic activity, instead of the increase one would expect. The decrease in thalamic activity, coupled with the increase in cingulate response, tells us that a spinothalamic pathway cannot be what is causing the increase. Scientists have discovered a direct connection between the spinal-cord projections and multiple limbic areas in both the rat and monkey.[67] The limbic system is then tightly connected with the cingulate and frontal cortex.

The limbic system also receives inputs from the reticular system (another pain juncture). As Apkarian concludes, "the brain imaging studies of pain...point to a very different emphasis in research regarding the central processing of pain.... [S]ystems outside of the spinothalamic system may control the type of processing taking place in the spinothalamic system. [Chronic pains]...seem to activate cortical areas outside the spinothalamic domain, which in turn inhibit the spinothalamic inputs to the cortex" (*op. cit.*, p. 290). That the thalamus and cortex are probably not important in processing our perceptions of pain stands in direct contrast to what many take to be common knowledge regarding how our pain system works.

Because new facts regarding the transmission of pain information in the brain are only slowly emerging, we should be fairly conservative in our conclusions. All I want to claim at the moment is that the

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W.D. Brown, K.J. Friston, L.Y. Qi, and R.S.J. Frackowiak, "Cortical and Subcortical Localization of Response to Pain in Man Using Positron Emission Tomography," *Proceedings of the Royal Society of London*, CCXLIV (1991): 39-44; J.D. Talbot, S. Marrett, A.C. Evans, E. Meyer, M.C. Bushnell, and G.H. Duncan, "Multiple Representations of Pain in Human Cerebral Cortex," *Science*, CCLI (1991): 1355-58.

[65] J. Mao, D.J. Mayer, and D.D. Price, "Patterns of Increased Brain Activity Indicative of Pain in a Rat Model of Peripheral Mononeuropathy," *Journal of Neuroscience*, XIII (1993): 2689-702.

[66] See Apkarian; and Roland.

[67] Apkarian; H.M. Newman, R.T. Stevens, C.M. Pover, A.V. Apkarian, "Spinal-Suprathalamic Projections from the Upper Cervical and the Cervical Enlargement in Rat and Squirrel Monkey," *Society of Neuroscience Abstracts*, XX (1994): 118.

six-neuron sketch of nociception transmission is probably over simplified to the point of being misleading. Accepting this conclusion opens the possibility of describing our pain systems differently than it has been in the past. More important for our purposes, it allows the possibility of dividing what we have been calling our pain system into two different and independent processing streams. I shall argue that doing so helps clear up the remaining puzzles in pain phenomena.

We have known for some time that many of the inhibitory streams are not merely feedback loops in our ascending pain fibers, for they are anatomically distinct from our pain processors. Three areas are primarily responsible for inhibiting pain information in the spinal column: the cortex, the thalamus, and the brain stem. The dorsal raphe is probably heavily involved as well.[68] In particular, the neocortex and hypothalamus project to the periaquaductal gray region (PAG), which then sends projections to the reticular formation (see figure 4). The reticular nuclei then work to inhibit activity in the dorsal horn.[69] This processing stream works by preventing a central cortical representation of pain from forming.[70] Endogenous opioids, stimulating the PAG, and morphine all dampen incoming information in the same way in the dorsal horn. That is, this pain inhibition stream does not merely disrupt the transmission of pain information, it actively prevents it from occurring.

In addition, this subsystem is not the only pain inhibitor at work. Stress-induced analgesia can occur without any opioids being released and it is not prevented from occurring when opioid-blockers are administered.[71] Moreover, different neural substrates are involved in inhibiting fast pains and slow pains. Stimulating the hypothalamus reduces tonic pain and is not related to stress-related analgesia.[72]

[68] Q-P. Wang and Y. Nakai, "The Dorsal Raphe: An Important Nucleus in Pain Modulation," *Brain Research Bulletin,* VI (1994): 575.

[69] H.L. Fields, "An Endorphin-Mediated Analgesia System, Experimental and Clinical Observations," in J.B. Martin, S. Reichlin, and K.L. Brick, eds., *Neurosecretion and Brain Peptides, Implications for Brian Function and Neurological Disease* (New York: Raven, 1981); H.L. Fields and A.I. Basbaum, "Endogenous Pain Control Mechanisms," in Wall and Melzack, eds., pp. 206-19; for references and reivew, see also J.E. Sherman and J.C. Liebeskind, "An Endorphinergic, Centrifugal Substrate of Pain Modulation: Recent Findings, Current Concepts, and Complexities," in J.J. Bonica, ed., *Pain* (New York: Raven, 1980), pp. 191-204.

[70] See Vaccarino and Chorney.

[71] See Kandel and Schwartz; and Fields and Busbaum.

[72] R. Lopez, S.L. Young, and V.C. Cox, "Analgesia for Formalin-Induced Pain by Lateral Hypothalamic Stimulation," *Brain Research,* DLXIII (1991): 1-7.

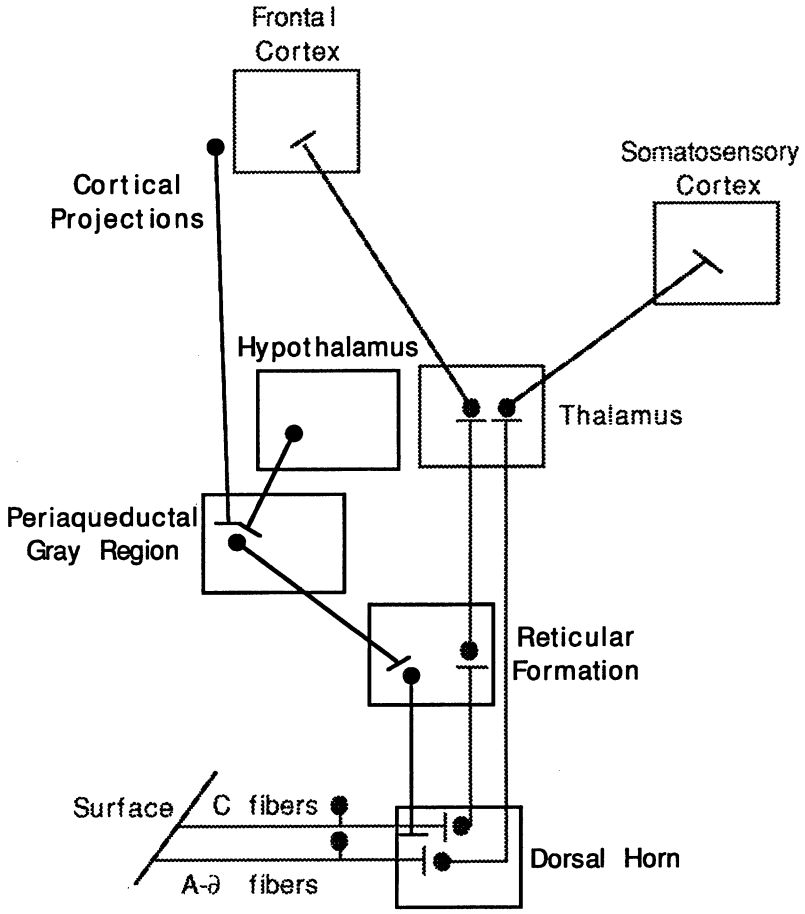

Figure 4. Diagram depicting the pain inhibitory system in contrast to the pain sensory system. Projections originating in the cortex and hypothalamus descend to the periaqueductal gray of the midbrain, the reticular formation, and then finally to the dorsal horn, where they inhibit the ascending nociceptive transmissions.

More important than being anatomically distinct and dissociable from nociception, the inhibitory streams are *teleologically* distinct from our PSS system as well. The inhibitory streams are not merely general purpose dampers; they are triggered by a very specific constellation of peripheral and cortical inputs. Otherwise, their removal would drastically increase our sensations of pain or make them all chronic. Such is not the case though. Lesioning the PAG does not appear to be tied to the onset of

chronic pains.[73] Moreover, we do know that the inhibitory systems are activated (or inhibited) by nociception plus cortical arousal (stress) on the organism.[74] Fear and learned hopelessness also affect their activity.[75] Hence, the correlates for the firing of these neurons differ in kind from what our ascending pain system is sensitive to.

In addition, if we look at the connections of the inhibitory streams, we can see that they differ substantially from the PSS. It would not be proper to call them sensory systems or subsystems for they have no connections to the periphery. The pain inhibitory streams halt at the dorsal horn. Also unlike our ascending pain streams, the hypothamalus and dorsal raphe nuclei receive massive inputs from cortical processors, which presumably could carry information about our goals and immediate plans, what else is occurring in the environment, and our emotional context.[76] They have immediate access to information that the ascending pathways do not.

Finally, a two-system theory of pain sensation explains our evolutionary confusions. When we are under stress, it is often more adaptive not to feel pain than to be incapacitated by pain.[77] If we are fighting or fleeing from an enemy, it would be preferable to do so unencumbered by the need to nurse or protect our limbs, even if this results in more nursing or protecting later (when we are presumably safe). It is important to know when damage is occurring in our bodies, but it is equally important to be able to shut that information out when circumstances demand. A dual system

[73] See Pagni.

[74] Fields and Basbaum; M.L. Mayer, "Periaqueductal Gray Neuronal Activity, Correlation with EEG Arousal Evoked by Noxious Stimuli in the Rat," *Neuroscience Letters,* XXVIII (1982): 297-301.

[75] S.F. Maier, R.C. Drugan, J.W. Grau, "Controllability, Coping Behavior, and Stress-induced Analgesia in the Rat," *Pain,* XII (1982): 47-56; J.W. Lewis, J.T. Cannon, and J.C. Leibeskind, "Opioid and Non-opioid Synapse Mediates the Interaction of Spinal and Brain Stem Sites in Morphine Analgesia," *Brain Research,* CCXXXVI (1980): 85-91; J.W. Lewis, J.E. Sherman, and J.C. Leibeskind, "Opioid and Non-opioid Stress Analgesia, Assessment of Tolerance and Cross Tolerance with Morphine," *Journal of Neuroscience,* I (1981): 358-63; J.W. Lewis, M.G. Tordoff, J.E. Sherman, and J.C. Liebeskind, "Adrenal Medullary Enkephalin-like Peptides May Mediate Opioid Stress Analgesia," *Science,* CCXVII (1982): 557-59; Fields and Basbaum.

[76] See Kandel and Schwartz.

[77] R.J. Bodnar, "Effects of Opioid Peptides on Peripheral Stimulation and 'Stress'-Induced Analgesia in Animals," *Critical Reviews in Neurobiology,* VI (1990): 39-49; Kandel and Schwartz; J.I. Szekely, "Opioid Peptides and Stress," *Critical Reviews in Neurobiology,* VI (1990): 1-12.

would allow just such a contingency; we could inhibit our pains as needed, but then feel them again when the danger is gone. A PSS and a PIS then serve two different goals: the PSS keeps us informed regarding the status of our bodies. It monitors our tissues to maintain their intactness whenever possible. In contrast, the PIS shuts down the PSS when flight or fleeing is immanent, and then enhances the PSS response in moments of calm. If our brains are geared for motor control, then the dual pain system makes good biological sense.

In sum: the bottom-up and the top-down investigative strategies support the conclusion that our pain inhibitors form a separate and distinct system. They react to different types of stimuli. They have different points of input and output from the PSS, and their existence is supported by evolutionary considerations.

VII. REPRIEVE: THE SENSATION OF PAIN

If my story is correct, then, contrary to how things seem to us, the sensation of pain is not what is most important in pain processing. It is but one minor aspect of our entire pain and pain-inhibitory systems, which themselves are geared to help us flee, fight, or nurse ourselves, depending upon the circumstances. They function just as any of our perceptual systems do: they help us get around in our environment as effectively as possible. From an evolutionary perspective, visual sensations are not the *raison d'être* of our visual system, and auditory sensations are not the ultimate goal for our auditory system. Sensations of pain are no different. Insofar as the PSS is simpler than other perceptual systems, then it makes good sense to use pain as a paradigm case of our conscious experiences.

We must issue an important caveat, however: pains are the product of a complex sensory system that has to struggle to get itself heard. Our conscious sensations are the product of both nociception and activity of a PIS.[78] Even though nothing about painful experiences is deeply mysterious—they are not random, nor inexplicable, nor tied to our psychological whims—they still are poorly correlated with actual tissue damage. What is interesting and different about our pains is the PIS. It is geared to suppress or enhance the activity of a single sensory system, and there is no other system quite like it in our nervous system. We do have general inhibitory and excitatory

[78] This is not to say that the PIS is directly connected to our sensations of pain. It is not. We can remove bits and pieces of the PAG, for example, and still feel pain. Nevertheless, the PIS heavily influences when and where we feel pain.

systems in the brain, but none of these is specific to a particular sensory system. The PIS entails that our sensations of pain are almost independent of nociception. Hence, insofar as we have a PIS, then our sensations of pain are special and are not a typical example of a phenomenological process. Putnam and others are simply wrong to think of a pain as the exemplar of consciousness.

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