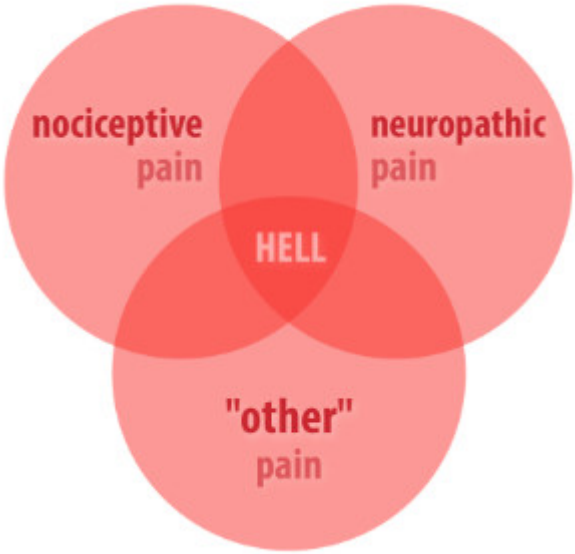


The 3 Basic Types of Pain

Nociceptive, neuropathic, and "other" (and then some more)

updated Nov 21, 2018 (first published 2016)
by Paul Ingraham

SUMMARY

There are two main classifications of pain: the common sensical sort that arises from damaged tissue (nociceptive pain), and the more exotic kind that comes from damage to the system that reports and interprets damage, the nervous system (neuropathic pain). This is the difference between engine trouble and trouble with that light on your dashboard that claims there's engine trouble. Oddly, there is still no official "other" category for the pain of conditions like fibromyalgia and irritable bowel syndrome, which involve dysfunction of the nervous system, as opposed to damage; names like nocipathic or algopathic are on the table.

Nociception and pain are not equivalent and there are no "pain fibres," just nerves that send data to the brain for consideration. All pain is technically a brain-generated experience. However, the illusion that pain is "in" our body is meaningful and functional.

Pain can also be classified as somatic (skin, muscle, bones, joints) and visceral (organs).

full article 4250 words

There are two well-recognized broad categories of pain: the common sensical sort (the pain of damage), and the somewhat more exotic kind that comes from damage to the system that *reports and interprets* damage, the nervous system. It's the difference between *engine trouble* and trouble with that light on your dashboard that *says* there's engine trouble. More specifically:

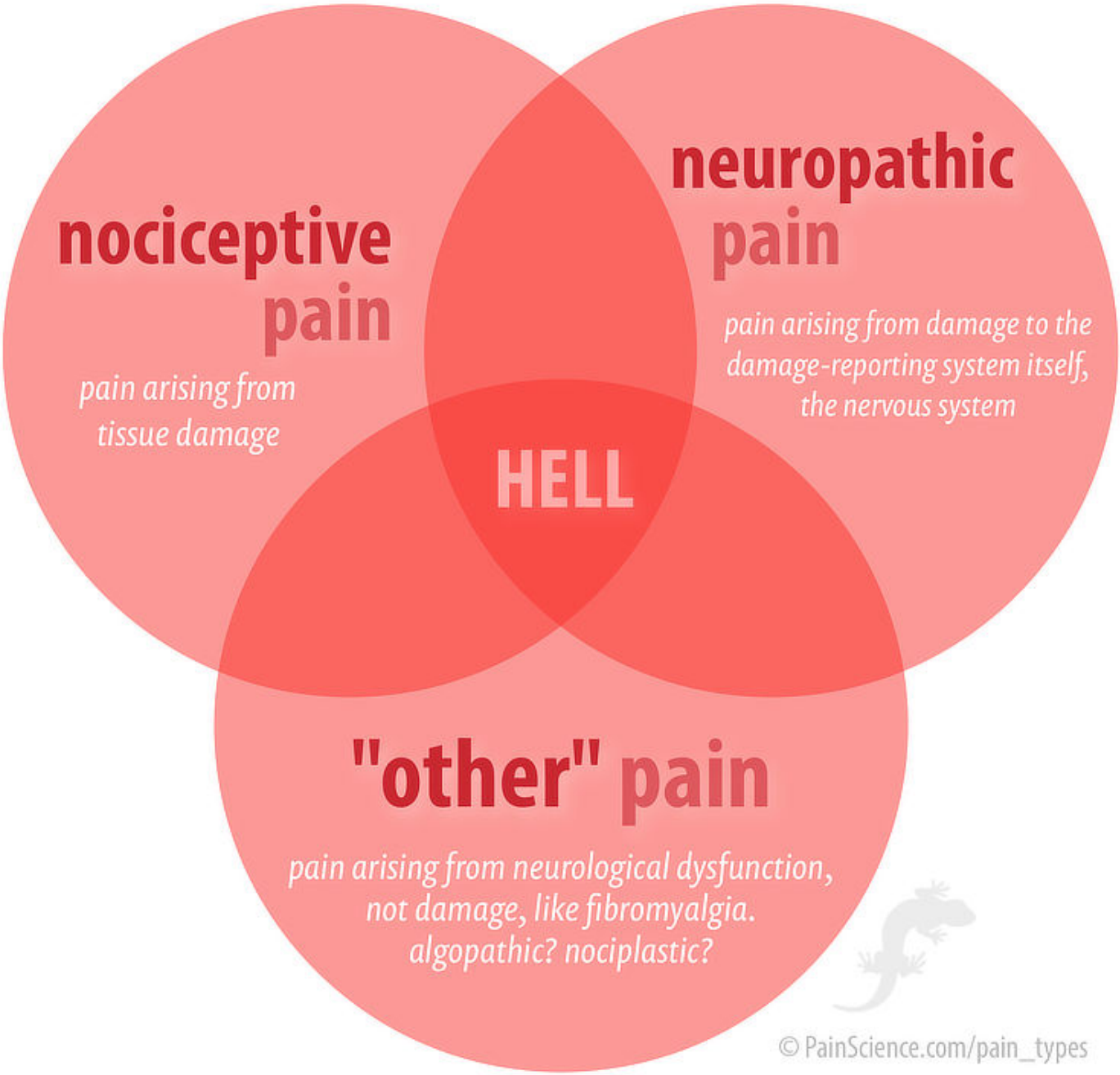
1. **Nociceptive pain** arises from various kinds of trouble in tissues, reported to the brain by the nervous system. [1] This is the type of pain everyone is most familiar with, everything from bee stings and burns and toe stubs to repetitive strain injury, nausea, tumours, and inflammatory arthritis. Nociceptive pain typically changes with movement, position, and load.

2. **Neuropathic pain** arises from damage to the nervous system itself, central or peripheral, either from disease, injury, or pinching. [2] The simplest neuropathies are mechanical insults, like hitting your funny bone or sciatica, but this is a big category: anything that damages neurons, from multiple sclerosis to chemotherapy to alcoholism to phantom limb pain. It's often stabbing, electrical, or burning, but nearly any quality of pain is possible. Unfortunately, it's also more likely to lead to *chronic* pain: nerves don't heal well. [3]

Migraine? Still tough to classify! Some experts consider it a major category of its own, parallel to nociceptive and neuropathic pain, [4] but it's probably just a complicated neuropathy. [5]

Obviously these kinds of pain can and do overlap. Some medical problems, like injuries, can affect *both* nerves themselves *and* other tissues, causing both kinds of pain. However, it's surprising how little obvious overlap there is: look at any list of the most painful conditions [NHS], or the great variety of pain causes, and they mostly fit into one category or the other. But "under the hood," most pain does involve elements of both types. [6]

Pain is predictably unpredictable, thanks to brains. Regardless of type, all pain is weird in some typical ways, because it's all under the total control of our brains, [7] *and brains have complicated and conflicting priorities for us that we are oblivious to.* [8] *The result is that pain is often weird, a somewhat paranoid guess about how much danger we're in, and that's when everything's working correctly. If the nervous system is damaged (neuropathic pain), then the brain is getting bad information, and pain gets even weirder. But when the nervous system misbehaves, pain can get so wonky that a whole new category of pain might be needed.*



Although the different kinds of pain certainly can overlap, mercifully it's relatively rare to end up in "hell" at the centre of this Venn diagram. Unfortunately, the "other" category often does arise from a history of other kinds of pain.

File under "other": pain problems for which we might need a new word

Some common kinds of pain are not a great fit for either of the two official categories. The canonical example is the pain of fibromyalgia. [Mayo] Other major examples:

- complex regional pain syndrome (CRPS) [Mayo]

- nonspecific chronic low-back pain
- irritable bowel syndrome [Mayo] and other functional visceral pain disorders
- conditions that begin as nociceptive pain, like osteoarthritis, but then go into a hellish downward spiral of sensitization [9]

Fibromyalgia is probably a pain system *dysfunction*, a poorly understood multi-system failure causing widespread body pain (and more [10]), but "dysfunction" of the nervous system is *specifically excluded from neuropathic pain*, by decree, as of 2011. [11] Dysfunction means that fibromyalgia isn't caused by any (known) damage to the nervous system, but by its *misbehaviour*, and so it's not welcome at the neuropathy club. It was before 2011! But not anymore.

Maybe there are unknown lesions? Maybe someday we'll know that fibromyalgia is caused by some kind of subtle damage to the nervous system. [12] There are at least two theories of subtle lesions of this type. [13] That would make it just another neuropathy after all, ho hum. But for now it's still more plausible that it's a dysfunction, arising from widespread problems in a complex system, and no clear point of failure will ever be discovered. [14] [15] But who knows. Science is not finished with fibromyalgia.

Meanwhile, what do we call it? And other miserable pain problems that arise from what seem to be neurological *dysfunction*?

Possible names for this other kind of pain

"Other" is a bit vague, so experts have proposed some more descriptive names for this category. There are issues with all of them. (The first three here all come from the same source. [16])

- **Nocipathic pain**: abnormal nociception. This is basically a fancy way of saying "other" pain, and I don't think it adds much.
- **Nociplastic pain**: like nocipathic, but a little more descriptive, implying specifically that the problem arises from *changes* in how nociceptive pain works.
- **Algopathic pain**: pathological perception/sensation. This is the Greek way to say "pain disease."
- **Centralized pain** [17] or **maladaptive central processing** [18]: pain driven by the spinal cord and brain, regardless of what's going on in the tissues.

The "noci" terms seem a little misguided to me, because the pain of a misbehaving nervous system is so much closer kin to neuropathic pain. [19] Centralization is an important concept and element, but clearly not the whole story [20] (and "maladaptive" might be too blamey [21]). Algopathic gets my vote: suitably neutral and formal, while saying just enough to be better than "other."

Do sensitization and centralization belong in the "other" category?

Sensitization is the amplification of danger signalling in any part of the system (which can be driven by both peripheral and central neurology, while *centralization* is referring only to the latter). It seems like a slam dunk to put these in the "other" category, but not so fast: the nervous system is not being dysfunctional just because it overestimates some danger. Many of us experience normal, healthy centralized sensitization *every time we go to the*

Many of us experience normal, healthy centralized sensitization every time we go

dentist, because our nervous system is not an idiot and is very suspicious of dentists based on past experiences. Our brains think the dentist is a threat, and although they are technically wrong — the dentist is actually there to help — the judgement is not "dysfunctional."

to the dentist, because our nervous system is not an idiot

Sensitization is clearly part of the *normal* function of the pain system — it' s not a dumb system where pain is always exactly proportionate to tissue danger. It's more subtle. Sensitization/centralization is only dysfunctional when it's chronic and seriously disproportionate. And of course there's a grey zone a mile wide between normal and dysfunctional. It's a spectrum.

File under "unknown": pain problems that can't be categorized *yet*

Sometimes we just don't understand a problem well enough to classify it. The best example of this is the sensitive spots in soft tissue that so many people have — "trigger points," the things that motivate most massage therapy appointments. There is a popular hypothesis that they are caused by a problem with muscle tissue, which would make it a clear case of nociceptive pain from a fairly subtle lesion … but it's just an hypothesis, and some experts have suggested that trigger points are caused by a problem with nerves themselves, which would make them neuropathic. **22** No one actually knows, and it's not likely to be settled for a long time.

A key deep thought: nociception isn't pain, and there are no "pain nerves," but muscles can still be sore

Nociceptors are the nerves that detect noxious stimuli, and *nociception* is what they do for a living: they send reports about tissue state, *not pain*.

Pain is a brain-generated *experience* based on many factors, *including* but not limited to nociception. If nociception is the major trigger that leads to a pain experience, it's reasonable to call it "nociceptive pain." But nociception is one thing, and pain is another.

Example: If you step on a Lego piece, the nerves send that information to the brain (nociception), and brain *always* decides that this information means you're in a *lot* of danger. Stepping on Lego is something the brain takes very seriously! So it always hurts. But it isn't pain until the brain says so.

So, nociception and pain are not the same thing, and nociceptors are not "pain fibres."

So what? Life is a reification fallacy

"Reification" is treating an abstraction as if it were a real thing, making it concrete. We do this constantly, of course. It's a really basic feature of being human.

As we have come to understand how pain is surprisingly independent of tissue state — you can hurt without tissue problem and vice versa, to an amazing degree — some experts have started to object to statements like "my muscles are sore." Why? Because, technically, it's your *brain* that hurts, not your muscles. Muscles can't feel! That's reification! "Reification fallacies" like this are pedantically asserted during shop talk on social media, often enough that it's starting to get on my nerves.

It is important to be *able* to deconstruct pain like this, even for patients — it's a big part of pain science — but it is not a requirement, and it's mostly way too clunky and technical for all but the most formal and delving discussions.

Literally everything that exists (not just what we sense) is a mental construct, a brain-made story. *Life is a reification fallacy* and refusing to tolerate reification is impractical. The entire *point* of sensation is to treat abstractions *as if they were real.*

Example: this delicious muffin I'm eating does not technically possess deliciousness or any blueberry flavour — those experiences really are all in my mind, like everything else — but "this blueberry muffin is delicious" is still a useful, meaningful statement.

And so is "my muscles are sore."

Another way of categorizing pain: visceral and somatic

Somatic pain is experienced in the skin, muscles, bones, and joints.

Visceral pain is the pain of organs, in the thoracic or abdominal cavities.

Both somatic and visceral pain can be nociceptive, neuropathic, or algopathic. The two classification systems have full overlap.

The somatic/visceral distinction isn't just about location and depth: visceral and somatic pain have very different characteristics, reflecting different priorities. For instance, much of somatic pain neurology is all about *localization*: the brain is very concerned with *where* somatic pain is occurring, the better to avoid external sources of danger. Visceral pain is notoriously "diffuse," difficult to isolate, and is often felt in remote locations, because the brain basically can't tell where the pain is coming from and starts making it up, referred pain. [23]

For much of medical history, no one even thought organs *could* hurt, which seems a bit quaint now — just what did they think was going on with the pain of a heart attack? Organs are largely impervious to the things that the somatic pain system is tuned to detect, like lacerations or burning, but they *are* very sensitive the kinds of things that are more likely to go wrong with organs, like stretch (a swollen gut), ischemia (a heart deprived of oxygen during a heart attack), and inflammation (inflammatory bowel disease). Hence the confusion. Now it's obvious not only that viscera can hurt, but that they can hurt *excessively and stubborn*: algopathic visceral pain, or *visceral hypersensitivity*, which is at the root of functional gastrointestinal disorders (gut trouble without an obvious cause, mainly irritable bowel syndrome).

Fast and slow pain: yet another way to categorize

Fast pain gets all the press, the kind of pain that makes you jerk your hand away from a hot stove. Pain is basically a threat detection system intended to get you to move away from danger, and you can't read about pain science anywhere without encountering this idea… which always seems a bit nuts to people with cpj, because where's the threat there? Which way do you run? What do you jerk away from when your back has been killing you for six months?

Pain protects from two kinds of threats in two very different ways. And it's the *other* way that matters to most people with persistent pain.

Of course, acute pain drives rapid movement away from immediate, external threats, mediated mainly by nociception via speedy "A" type nerve fibres.

But *chronic* pain — with threat information delivered via the sluggish "C" fibres — is trying to protect us by *discouraging* movement, by forcing us to hunker down for rest and healing! That's a really different scenario.

These modes are strongly analogous to fight/flight and rest/digest. One system is optimized for emergencies, the other is optimized for recuperation.

Most chronic pain is not trying to tell us about external threats we need to get away from, but about the need to stay put until we feel better. Just as with acute pain, the warning is not necessarily accurate, and in most cases of longer term chronic pain we are *too* shut down and need to get moving despite the warning.

If you found this article useful, consider making a small contribution to my income — like paying a busker.

About Paul Ingraham

I am a science writer, former massage therapist, and I was the assistant editor at ScienceBasedMedicine.org for several years. I have had my share of injuries and pain challenges as a runner and ultimate player. My wife and I live in downtown Vancouver, Canada. See my full bio and qualifications, or my blog, Writerly. You might run into me on Facebook or Twitter.

Related Reading

- Why Does Pain Hurt? — How an evolutionary wrong turn led to a biological glitch that condemned the animal kingdom — you included — to much louder, longer pain
- 25 Surprising Causes of Pain — Trying to understand pain when there is no obvious explanation
- Central Sensitization in Chronic Pain — Pain itself can change how pain works, resulting in more pain with less provocation
- Insomnia Until it Hurts — The role of sleep deprivation in chronic pain, especially muscle pain
- Nerve Pain Is Overdiagnosed — A story about nerve pain that wasn't really nerve pain
- Pain & Injury Survival Tips — Dozens of ideas (and links) for evidence-based rehabilitation and self-treatment for common pain problems and injuries
- Chronic, Subtle, Systemic Inflammation — One possible sneaky cause of puzzling chronic pain

Appendix: Neuropathic versus nociceptive pain

If you have unexplained pain, can at least diagnose the type? Can you put it in one of the two major pain categories, neuropathic or nociceptive? Sometimes! And often you can't. These types of pain overlap and often the only clues are the quality of the pain and the messy stories and ideas we have about it: how it started, what makes it worse or better, and so on. We can't just say that neuropathic pain is "electrical," because not everyone's sciatica feels that way.

But we can usually make an educated guess, at least.

Neuropathic pain is mostly more distinctive and specific than nociceptive pain, so it's best to think in terms of whether pain is or is not neuropathic.

It is *usually* burning, electrical, or stabbing. The better these words seem to fit, the more likely it is to be neuropathic pain.

Neuropathic pain is sometimes associated with other sensory disturbances like tingling (parasthesia) and numbness, or weakness.

More exotically, neuropathic pain may cause odd effects like exaggerated pain (hyperalgesia), or even pain from stimuli that shouldn't hurt at all (allodynia), or pain "echoes." If any of these other non-pain neurological symptoms are present, it's nearly a diagnostic slam dunk: the pain is probably neuropathic.

If any non-pain neurological symptoms are present, it's nearly a diagnostic slam dunk: neuropathic!

Unfortunately, plenty of neuropathic is not conveniently packaged with other obvious neurological effects. So how else can we judge it?

- Extent. Neuropathic pain is often more widespread. A damaged nerve can cause symptoms in many locations "downstream" from the damage. Nociceptive pain is mostly more isolated.

- Location, location, location. Although it's hard for patients to judge this, neuropathic pain often occurs in specific locations associated with nerves or nerve roots. And yet this can fool pros too: these patterns aren't nearly as clear-cut as the tidy diagrams we learned from in school.

Neuropathic pain is also more likely to occur in some locations. For instance, a very broad rule of thumb is that neuropathic pain is more common in the limbs, and the further you go out towards the tips the more likely it gets, simply because the limbs of are full of long and relatively exposed nerves.

- Response to medication. Neuropathic pain is rarely responsive to over-the-counter pain killers; nociceptive pain is much more so, especially when it's still fresh.

- Duration. This is perhaps the roughest rule of thumb of them all — there are lots of exceptions to it — but neuropathic pain tends to last longer and is strongly associated with chronic pain.

So, if you have stubborn, widespread, burning pain in your limbs that defies acetaminophen … that's probably neuropathic pain. And yet it's still not a sure thing!

What's new in this article?

Eight updates have been logged for this article since publication (2016). *All* PainScience.com updates are logged to show a long term commitment to quality, accuracy, and currency.

2018 — New section: “Fast and slow pain: yet another way to categorize.”

2018 — New section: “Another way of categorizing pain: visceral and somatic.”

2018 — New section: “A key deep thought: nociception isn’t pain, and there are no ‘pain nerves,’ but muscles can still be sore.”

2017 — New section — “Appendix: Neuropathic versus nociceptive pain.”

2017 — Added an important point to the definition of neuropathy: it tends to be more chronic.

2016 — Added a nice new Venn diagram depicting the intersection of all three kinds of pain in “hell.” Added section “Do sensitization and centralization belong in the “other” category?”

2016 — Added sidebar about migraine, and converted section about the influence of the brain to a sidebar. Elaborated on overlapping pain problems.

2016 — Extensive next day revisions.

2016 — Publication.

Notes

1. IASP-pain.org [Internet]. International Association for the Study of Pain. IASP Taxonomy; 2012 May 22 [cited 16 Sep 1]. The formal definition: “Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors [nerve endings that detect tissue damage].” The next definition is also based on this reference. BACK TO TEXT

2. Formally: “Pain caused by a lesion or disease of the somatosensory nervous system.” You get a gold star if you notice that “dysfunction” isn’t included there. More on this below. BACK TO TEXT

3. Cohen SP, Mao J. Neuropathic pain: mechanisms and their clinical implications. *BMJ*. 2014;348:f7656. PubMed #24500412. BACK TO TEXT

4. Chakravarty A, Sen A. Migraine, neuropathic pain and nociceptive pain: towards a unifying concept. *Med Hypotheses*. 2010 Feb;74(2):225–31. PubMed #19765908. “Migraine, neuropathic pain and nociceptive pain are the three commonest pain syndromes affecting human.” BACK TO TEXT

5. Biondi DM. Is migraine a neuropathic pain syndrome? *Curr Pain Headache Rep*. 2006 Jun;10(3):167–78. PubMed #18778570. BACK TO TEXT

6. Cohen SP, Mao J. Neuropathic pain: mechanisms and their clinical implications. *BMJ*. 2014;348:f7656. PubMed #24500412. There’s so much overlap between the mechanisms of neuropathic and nociceptive pain that “many experts view them as different points on a chronic pain continuum, rather than distinct entities.” As a simple example, all acute nociceptive pain — every toe stub — involves an immediate ramping up of sensitivity of nerve endings in the area. That sensitizing process shares a lot of biology with neuropathic pain. BACK TO TEXT

7. Pain is an *output* of the brain.

> Pain is the end result. Pain is an output of the brain designed to protect you. It's not something that comes from the tissues of the body.
>
> ~ Lorimer Moseley, from his surprisingly funny TED talk, Why Things Hurt YouTube 14:33

The formal definition of pain emphasizes its subjective, experiential nature: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP Taxonomy). This experience is based on many "inputs," *not* just nerve signals about tissue damage.

BACK TO TEXT

8. Although the brain takes data from tissues seriously, it also regularly minimizes, exaggerates, and misinterprets. The brain thinks minor things are serious, or serious things are minor. It's poor at locating the sources of internal pain. If the nervous system itself is damaged, the brain can *really* get thrown off (as with phantom limb pain). The brain's pain policies are warped by moods, anxieties, fatigue, and much more. For much more about this, see Pain is Weird: Pain science reveals a volatile, misleading sensation that is often more than just a symptom, and sometimes worse than whatever started it. BACK TO TEXT

9. Ingraham. Central Sensitization in Chronic Pain: Pain itself can change how pain works, resulting in more pain with less provocation. PainScience.com. 5428 words. Pain itself often modifies the way the central nervous system works, so that a patient actually becomes more sensitive and gets *more pain* with *less provocation*. This is called "central sensitization." (And there's peripheral sensitization too.) Sensitized patients are not only more sensitive to things that should hurt, but also to ordinary touch and pressure as well. Their pain also "echoes," fading more slowly than in other people. BACK TO TEXT

10. Poor quality sleep, fatigue, memory and mood issues — the infamous "fibrofog." BACK TO TEXT

11. Jensen TS, Baron R, Haanpää M, *et al*. A new definition of neuropathic pain. *Pain*. 2011 Oct;152(10):2204–5. PubMed #21764514.

> IASP has recently [2008] published a new definition of neuropathic pain according to which neuropathic pain is defined as 'pain caused by a lesion or disease of the somatosensory system.' This definition replaces the 17-year old definition that appeared in the Classification of Chronic Pain published by IASP in 1994, which defined neuropathic pain as 'pain initiated or caused by a primary lesion, dysfunction, or transitory perturbation of the peripheral or central nervous system'. Even though the definition has not been changed dramatically, there are two important changes in the new version: (1) the word 'dysfunction' has been removed and (2) a lesion or disease affecting the nervous system has been specified to be a lesion or disease of the somatosensory system.

The whole paper is excellent, but skipping to the cogent conclusion:

> A definition of neuropathic pain is only useful if it distinguishes conditions in a clinically meaningful way. If the definition does not provide additional benefit in terms of understanding and treating the condition(s), then there is no reason to keep it. Hopefully, the new definition of neuropathic pain will act as a stimulant to discuss the definition in more detail and provide input for studies that can be used to test the value of the definition.

BACK TO TEXT

12. Multiple sclerosis attacks nerves in quite a dramatic way, which we could see in autopsies, but the lesions were invisible in living patients until the invention of MRI. It's plausible that there are still plenty of biological "lesions" that we haven't yet learned to detect, because they are just too small and transient. Despite all of our modern technological wizardry, most of the action in biology happens at the nanoscale, cells moving molecules and atoms around at dazzling speeds through the chaos of the molecular storm (brownian motion). There are probably nanoscale lesions. BACK TO TEXT

13. Both *small fibre peripheral neuropathy* and *positional cervical cord compression* are candidate neuropathic etiologies: both hard to detect, both capable of explaining at least some of the symptoms of fibromyalgia, both associated with people who have diagnosed with fibromyalgia. See the main fibromyalgia article for more information about these. BACK TO TEXT

14. Lyon P, Cohen M, Quintner J. An evolutionary stress-response hypothesis for chronic widespread pain (fibromyalgia syndrome). *Pain Med*. 2011 Aug;12(8):1167–78. PubMed #21692974. "Drawing on diverse findings in neurobiology, immunology, physiology, and comparative biology, we suggest that the form of central sensitization that leads to the profound phenomenological features of chronic widespread pain is part of a whole-organism stress response, which is evolutionarily conserved, following a general pattern found in the simplest living systems." BACK TO TEXT

15. BetterMovement.org [Internet]. Hargrove T. A Systems Perspective on Chronic Pain; 2014 Oct 23 [cited 16 Sep 12].

    This deep but beautifully readable article explains, with many pictures and apt examples, how "chronic pain is often driven by dysregulation of a 'supersystem' that coordinates defensive responses to injury. The supersystem results from dynamic interaction between different subsystems, most notably the nervous system, immune system, and endocrine system." It's hard to believe, but the article also manages to make this information seem quite practical.

    BACK TO TEXT

16. Kosek E, Cohen M, Baron R, *et al*. Do we need a third mechanistic descriptor for chronic pain states? *Pain*. 2016 Jul;157(7):1382–6. PubMed #26835783. PainSci #53276. BACK TO TEXT

17. Clauw DJ. Fibromyalgia: a clinical review. *JAMA*. 2014 Apr;311(15):1547–55. PubMed #24737367. BACK TO TEXT

18. Topical Issues in Pain (https://giffordsachesandpains.com/topical-issues-in-pain-relaunch-2013/) BACK TO TEXT

19. Even if this kind of pain does involve an exaggeration of nociception (which it probably does), it still seems to me that nociception itself is innocent. It's not nociception's fault! The focus should be on the *mechanism of sensitization* — that is what makes the problem what it is. BACK TO TEXT

20. Quintner, J. "Why "Centralized" Is Unacceptable As A Descriptor For The Pain Of Fibromyalgia." Aug 23, 2016. "Not only does the word itself not imply a mechanism but also it creates potential for confusion with conditions such as 'central post-stroke pain' (which is technically 'neuropathic') and quite different from the phenomena that underlie fibromyalgia." BACK TO TEXT

21. This term has some strong negative connotations. I think this term could strongly stigmatise the patient's condition, implying that they have "adapted badly" to things that other patients had no problem with. Even if that is technically true from one perspective, it's not where the emphasis should be. Most patients with these problems have already had lot of difficulty being heard, understood, and respected — let's not make it harder with a blame-y label. BACK TO TEXT

22. Quintner JL, Bove GM, Cohen ML. A critical evaluation of the trigger point phenomenon. *Rheumatology (Oxford)*. 2015 Mar;54(3):392–9. PubMed #25477953. Quintner, Cohen, and Bove argue that the common picture of trigger points as lesions in muscle and soft tissue, spelled out most formally in Gerwin 2004, is "flawed both in reasoning and in science." But not even these critics of trigger points deny that people have pain that *seems* to come from their muscles. But if it's not coming from the muscle, where *is* it coming from? They briefly discuss the possibility of *inflamed nerve fibres*, which would be a clear case of neuropathy. Neuritis is undoubtedly worth investigating, but it requires us to believe that nerve axons are routinely

inflamed for no apparent reason. And the evidence cited to support it is actually much more limited than the evidence for a lesion in muscle. BACK TO TEXT

23. The brain is somewhat inept at precisely locating internal pain and thus experiences muscle pain in a broad area around or *near* a trigger point (exactly like heart attacks are felt in the arm as well as the chest). The pattern for each muscle is somewhat distinctive, and most referred pain spreads away from the centre of the body. BACK TO TEXT