



Neural Pathway Pain— A Call for More Accurate Diagnoses

In the treatment of chronic pain, the author argues that a patient's physical and emotional history may play a more significant role in long-term outcomes than expected.

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I gave a lecture on chronic pain at the Academy of Integrative Pain Management's 2017 annual meeting in San Diego. At the beginning, I asked the audience whether they thought pain care providers needed better treatments for chronic pain or better diagnoses of the causes of chronic pain. "Better treatments" was the unanimous response, and the following explains why I have come to the opposite conclusion.

Current Conceptions of Chronic Pain

The most widely accepted behavioral intervention model to treat chronic pain relies on the perspective that all pain is the same. If "pain is pain," treatments may be designed to reduce the pain, or to help patients cope with the pain.

This practice seems to blur the line between the two major causes of chronic pain:

- *inflammatory pain* due to ongoing physical injury, tumor, fracture (ie, nociceptive pain), or due to nerve damage (ie, neuropathic pain)
- *brain-induced pain* (ie, neural pathway, centralized, psychophysiological, or psychosomatic pain).

When physicians fail to make this distinction, they may be viewing chronic pain as a static, non-reversible process, for which the etiology does not matter.

Structural Distinctions

This lack of distinction is where pain practitioners may have it wrong. The most common reason that patients

visit physicians is due to neck or back pain,^{1,2} and the causes are almost always attributed to some kind of physical injury or degeneration.

For example, physicians typically rely on MRI scans to indicate the cause of the neck or back pain. When MRIs correlate fractures, tumors, inflammatory conditions, or severely herniated discs with evidence of nerve damage, a structural disorder may be indicated. Yet, MRIs of pain-free 30-year-olds have reflected degenerative disc dis-

clear benefit over placebo injections.⁵ Widespread opioid use for pain has been termed a national epidemic. Moreover, suggesting to a patient that the back may be irreversibly damaged may generate increased fear and anticipation of pain, thereby activating increased actual pain and disability.

Another misconception about chronic pain is that brain-generated pain is rare. Studies show that approximately 85% of patients with chronic neck or back pain do not have a clearly

clear changes in the brains of individuals with chronic pain.⁹⁻¹⁰ However, brain-generated pain is often conceived to be static and irreversible.

This conceptualization does not account for the dynamic nature of brain-generated pain. Emerging research on brain function may explain how our brains generate internal experiences, including pain.¹¹ There is a “danger/alarm” mechanism, for example, that creates pain when danger is sensed, either in the form of physical injury or emotional threat. The parts of the brain activated by emotionally upsetting events are identical to those activated by physical injury, thus demonstrating the mechanism by which emotional pain may lead to physical pain.¹²

We now know that children who suffer from the consequences of parental divorce, drug abuse, neglect, or outright abuse have much higher rates of chronic pain (and other difficulties) later in life.¹³ The experience of growing up feeling “unsafe” sensitizes the danger/ alarm mechanism that may then be triggered later in life through stressful life events or physical injuries, such as a car accident or a surgical procedure. In these situations, the brain may construct pain as a protective mechanism. Specifically, the brain activates neural circuits or pathways of pain that create real pain in the absence of tissue damage. These pathways are, however, reversible due to the brain’s neuroplasticity.

Identifying Neural Pathway Pain in the Clinical Setting

Physicians may use the above findings to guide their clinical practice by identifying patients who have brain-generated or neural pathway pain. After *ruling out* significant structural disorders, physicians may use clinical evidence to *rule in* neural pathway pain. Following are some guidelines that may help to link a patient’s symptoms to neural pathways:

- History of several neural pathway

The lack of distinction between inflammatory pain and brain-induced pain is where pain practitioners may have it wrong.... data suggest the majority of patients with chronic pain do not have a structural cause

ease in 50% of patients, and bulging discs in 40% of patients. Those statistics reach levels of 80% and 60%, respectively, in pain-free 50-year-olds, and are even higher in older patients.³ The vast majority of adults, therefore, have abnormal MRIs, suggesting that these “abnormalities” do not necessarily cause pain. Despite this, many clinicians continue to point to minor structural findings as the cause of chronic neck or back pain.

Treatments for neck or back pain commonly include surgery, injections, and/or opioid medications. However, there are no studies demonstrating that surgery for axial back pain is superior to nonsurgical interventions.⁴ Meta-analyses of injection therapies show no

identifiable, structural cause for their pain.⁶ Of the millions of individuals experiencing tension and migraine headaches, only about 5% have an identifiable structural cause. Very few people with irritable bowel syndrome, fibromyalgia, and many chronic pelvic pain syndromes have tissue damage to account for their pain.⁷ These data suggest that the majority of patients presenting with chronic pain do not necessarily have a structural cause.

Brain Distinctions

As pain management evolves, practitioners are turning to mechanisms in the brain to explain chronic pain.⁸ Proof exists in MRI and functional MRI studies (fMRI) that demonstrate

induced syndromes over a lifetime, such as headaches, migraine, irritable bowel syndrome, interstitial cystitis, neck or back pain, pelvic pain, fatigue, insomnia, anxiety, and depression

- Early-life trauma
- Personality traits of perfectionism, people pleasing, self-criticism, lack of self-compassion, lack of assertiveness, perfectionism (increased pressure upon oneself may further activate the danger/alarm mechanism)
- Pain that:
 - worsens over time
 - spreads to new regions of body
 - shifts from region to region
 - turns “on and off”
 - has an onset upon awakening
 - worsens with increased stress / improves with less stress
 - radiates to areas that do not conform to structural norms
 - is often bilateral in nature.

Once aware of these patterns, clinicians may be able to determine which patients have neural pathway-related disorders as opposed to purely structural causes. Some patients may have a combination of the two.

Step-by-Step Approach

With a clear and accurate diagnosis, treatment of neural pathway pain may involve components of: cognitive-behavioral therapy; mindfulness/meditative processes; and acceptance and commitment therapy. There is, however, one caveat. While these modalities may often be used to treat chronic pain, practitioners may tend to apply them under a premise that the pain is caused by physical problems in the body. However, these types of interventions take on an entirely different meaning when the premise is *change*. With the understanding that there is no structural disorder—ie, the pain is generated by neural pathways in the brain, the goal changes from pain management to pain elimination. Primary steps in this process include:

- *Educating the patient* about the nature of pain and the role of the brain in generating neural pathway pain (ie, understanding that there is no physical damage and that recovery is possible)
- *Reducing activation of the danger/alarm mechanism* in the patient’s brain by using cognitive methods

to reduce fear of pain (eg, pain may be reframed as “unharmful sensations produced by the brain”)

- *Encouraging increased activity* and resumed normal activities without fear of pain or of injury; making life changes that are necessary to promote safety and well-being; and increasing enjoyment in daily life
- *Helping the patient to process emotions* that may have led to the painful syndromes (eg, engaging in exercises that allow the recognition, experience, expression, and processing of emotions that may have been avoided in the past (eg, anger, guilt, sadness and compassion)).¹⁴

A Case Example

When Casey was 14-years-old, he developed severe abdominal pains that progressed to the point of him being unable to attend school or participate in daily activities. Over the next three years, he underwent numerous medical tests [CAN YOU LIST EXAMPLES OF SPECIALISTS SEEN], yet there was no evidence to support any physical injury to account for the severe pain. His pain was constant and, at times,

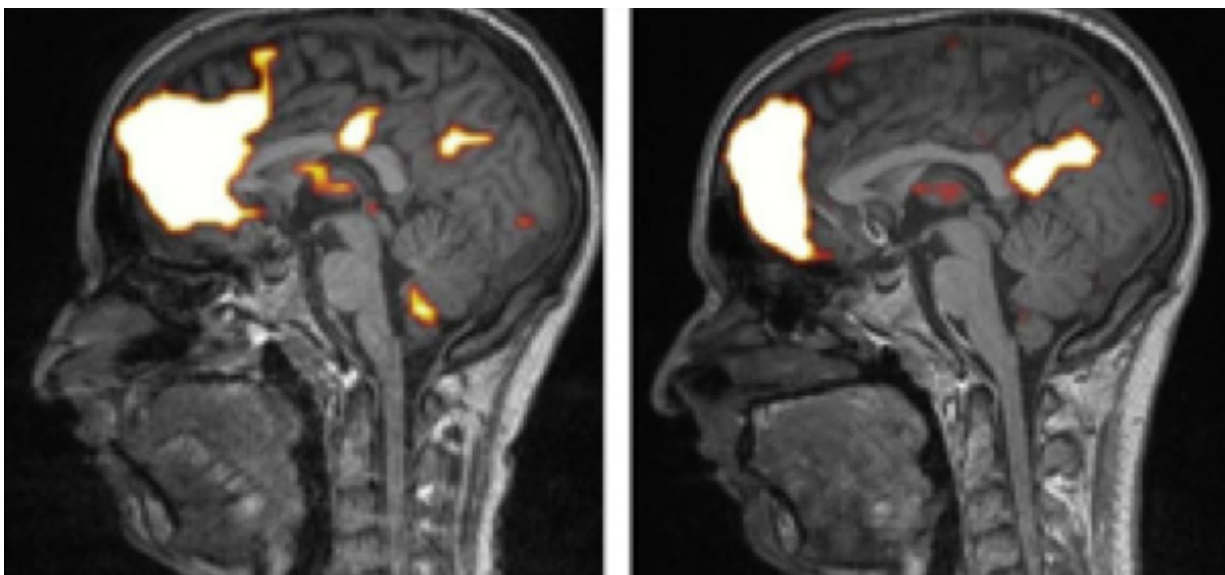


Figure 1: Case example of patient’s brain before (at left) and after (at right) interventional neural pathway therapy.

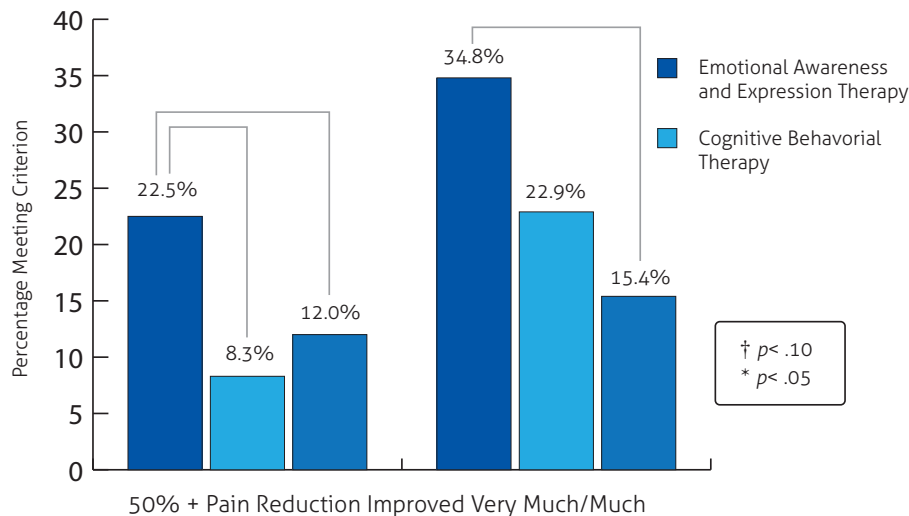


Figure 2: Reports from study groups after completion of education, cognitive behavioral therapy, and emotional awareness therapy. Reprinted with permission from Wolters Kluwer Health, Inc (Reference 19).

flared to levels high enough to cause syncope episodes.

At age 17, Casey presented at the author's practice, which included a licensed clinical social worker that specialized in the treatment of neural pathway or mind-body pain [EDIT OK?]. A functional MRI found severe abnormalities in the resting state of Casey's brain. An evaluation further confirmed that Casey had no physical cause for his pain complaints, leading the clinicians to conclude that Casey's pain was most likely due to neural pathways. It was explained to Casey that his pain was very real, but also that the most important aspect of his recovery had already taken place: he had been accurately diagnosed as having brain-generated, rather than tissue damage-generated pain.

By changing his understanding of the cause of the pain, Casey was able to reduce his fear of the symptoms. The intervention allowed his danger/alarm mechanism to turn off, which in turn, led to elimination of pain.

After three months, Casey reported a

complete recovery from the abdominal pain and a repeat fMRI showed normal function (see Figure 1). He returned to school and resumed his usual activities.

Supporting Research

The author's clinical experience, combined with the research noted herein, suggests that the neural pathways responsible for the majority of chronic pain may be reversible.¹⁵⁻¹⁶ Emerging data supports this view. Two outcome studies of individuals with chronic back pain and fibromyalgia demonstrated dramatic pain reductions using the approach described above (ie, cognitive-behavioral therapy; mindfulness/meditative processes; and acceptance and commitment therapy).¹⁷⁻¹⁸

Additionally, a small randomized, controlled trial showed that a mind-body approach was more effective than treatment as usual.¹⁹ In the study,²⁰ 230 patients diagnosed with fibromyalgia were placed in one of three groups:

- Group 1: received education only
- Group 2: underwent cognitive behavioral therapy

- Group 3: practiced exercises.

The exercises utilized by the third group were designed to help patients recognize, experience, express, and process emotions such as anger, guilt, sadness, and compassion that may have been avoided in their lives. The exercise group reported significantly higher rates (> 50%) in pain reduction compared to the first two groups. The emotional therapy group also provided descriptions of improving "very much" or "much" in comparison to the other two groups (see Figure 2 for full results).²⁰

Discussion and Conclusions

The combination of new research on how pain is generated and processed in the brain, along with data on improved outcomes, suggests that pain care providers may have an opportunity to change the paradigm of pain management and offer hope of recovery to patients living with chronic pain. However, there are several barriers to the widespread recognition of chronic pain disorders and the implementation of effective treatments.

First, the concept that pain may be generated by the brain—in the absence of tissue damage—may seem counterintuitive. Medical training focuses on structural abnormalities and many patients have evidence of mild structural anomalies that may actually be normal variants. A great deal of education and repetition, along with significant inquiry into the clinical evidence for neural pathway-generated pain, is necessary for this concept to be fully understood and accepted.

A second barrier is that pain generated by the brain is typically considered to be "not real." Stigma often surrounds conditions deemed to be psychological, and many patients are understandably sensitive to being told that their pain is "in their head." It is crucial, therefore, for practitioners to explain to their

patients that neural pathway pain is not only real but also common.

[EDITS TO FOLLOWING PARAGRAPH OK?] When treating neural pathway disorders, pain specialists are also encouraged to consider mental health providers as part of the patients' treatment team. Providers should ask patients about psychological issues that may be at the root of neural pathway disorders, and, in turn, mental health providers should take patients' medical histories to determine the causes of physical symptoms. Together, they may find that long-term patient outcomes improve when focusing on eliminating pain, versus coping with pain.

The final barrier to accepting and properly diagnosing brain-induced pain lies in standard practice. Many providers rely on standard medications and procedures for treating chronic pain, as opposed to exploring applying alternative therapies. It has been

estimated that more than \$600 billion is spent in the United States on pain care annually.²¹

If the model for diagnosing and treating neural pathway pain expressed herein were widely adopted, there may be significant economic repercussions in the healthcare system. However, this approach has the potential to dramatically reduce the suffering of millions of people suffering from chronic pain and may substantially reduce medical costs. ■

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