

When Pain Persists Despite Treatment: A Lived-Experience Perspective on Chronic Pain Persistence

Author: Scott Muenzler, ICF Certified Coach (ACC)

Behavioral coaching for chronic tinnitus and pain, adjunctive to medical and
psychological care

Date: January 2, 2026

Contact for clinicians: Physicians@c3coaching.co



Clinical Relevance and Practice Context

Many patients with centralized pain continue to experience symptoms even when medical evaluation does not indicate ongoing tissue damage. In these cases, contemporary perspectives emphasize how learned threat responses, attention, and interpretation can shape the experience of pain and contribute to its persistence. This article integrates current research with lived experience to clarify how pain can remain distressing over time and to highlight communication and behavioral principles that may help patients regain a sense of safety and confidence.

Key points for clinical care:

- **Clear reassurance and explanation** may reduce uncertainty and perceived threat, which can make engagement with care easier. In this context, reassurance refers not simply to ruling out dangerous pathology, but to helping patients understand why symptoms can persist and what that persistence does and does not signify.
- **Attention, interpretation, and emotional meaning** can influence how intense and intrusive pain feels in daily life.
- **Behavioral re-engagement**, such as resuming normal activities at a tolerable pace, is included in some approaches to centralized pain and focuses on addressing avoidance while maintaining function.
- **Communication that blends validation with realistic hope** may help patients feel safer and more willing to engage in recovery-oriented care.

The goal is to help clinicians translate pain neuroscience into practical communication, support, and referral strategies that complement medical evaluation and are intended to improve patient understanding, functioning, and engagement.

Background and Scope

Before the framework described in this paper made sense to me, I spent roughly two years seeking care for severe, persistent pain. I saw multiple specialists, each offering different explanations and proposed solutions, none of which resolved the problem. Without clear medical findings, the experience was costly, destabilizing, and increasingly frightening, particularly in the absence of a coherent account of why the symptoms persisted. When I discovered an approach that emphasized psychological and behavioral mechanisms rather than structural damage, I was initially resistant. That resistance, and what ultimately helped me move past it, strongly shaped how I now think about what patients need when symptoms continue despite appropriate medical evaluation.

When my pain first began, I assumed the problem was the result of physical injury or disease. Over time, and after extensive medical evaluation that did not identify a plausible structural cause, I came to understand that my symptoms were not being driven by ongoing tissue damage. Instead, they were being maintained by learned emotional, cognitive, and behavioral patterns. This article outlines the perspective that helped me arrive at that understanding, how later research clarified why it was plausible, and how similar principles may help patients adapt and regain function alongside standard medical care. The aim is not to replace medical assessment, but to describe a framework that can help explain why pain may persist after danger has passed, and how meaningful recovery can occur even in cases that initially appear implausible.

I am not a physician or researcher, but as someone who lived through severe chronic pain and now helps others navigate similar experiences, I wanted to better understand what actually changed when my symptoms resolved. A version of this story was first shared publicly in 2025 for individuals struggling with tinnitus, where it helped illustrate how fear, attention, and interpretation can shape symptom persistence. This version is written for clinicians, to describe from the inside what patients may be experiencing when pain continues despite appropriate evaluation, and to outline how patterns involving attention, emotion, and learned threat responses can sometimes shift in ways that lead to outcomes that initially seem difficult to reconcile with standard expectations. The details of that change, and why it is often met with skepticism, particularly from patients, are addressed later in the paper.

Onset of pain

My ordeal began quietly, with a heaviness in my groin that within days became testicular pain that made it difficult to walk across my living room. An ultrasound left the physicians uncertain whether they were seeing a benign vascular finding or an inflammatory process, and I was prescribed a course of antibiotics. When nothing changed, another physician declared the first mistaken recommended a different antibiotic, which also had no effect.

I then saw a urologist who concluded that it was a benign, non-serious condition and told me to rest, elevate my legs, and use acetaminophen as needed. He assured me it was nothing serious. But the pain persisted, and reassurance did little to settle the unease that had begun to grow. What troubled me was not only the discomfort itself but the way each professional seemed to see something different. As their assessments diverged, my confidence eroded.

I began checking the pain constantly, from the time I awoke. I slept with a pillow between my legs. I walked only when necessary. Even short errands sometimes led to days of increased discomfort. I gained weight. What had started as a physical problem was beginning to reshape the way I saw myself and the way I moved through the world. The diagnoses accumulated, and over an 18-month period, I filled prescriptions for acetaminophen, diclofenac, several antibiotics, gabapentin, and tramadol. None brought

lasting relief. Each failed intervention strengthened the sense that something important was being missed.

I remember that period as one filled with fear, frustration, and a kind of simmering agitation that left me feeling drained and ashamed. I was often on the edge of snapping, and I can only imagine how exhausting I must have been for those closest to me. The pain was not just physical; it chipped away at my confidence, my identity, and my sense of feeling normal. Working from home made it difficult to keep my mind from spinning out into worst-case scenarios. I pictured myself limping through the next fifty years. These thoughts did not feel dramatic or exaggerated at the time; they felt like the most accurate projections I could make.

By the end of that year I felt increasingly isolated. I rarely spoke about the pain to anyone except physicians, because it was uncomfortable talking about it with those close to me, and I imagined it was awkward hearing about it.

I didn't know what was going on with me at the time, and neither did the physicians I saw. That's not their fault any more than it was mine. I had grown up believing and trusting in a biomedical model that treated the body as a machine with many parts, and although I had heard of psychosomatic processes, the reactions I received whenever I mentioned them made me feel naive or foolish. Eventually I stopped asking. What I did not understand then was how much the fear itself was shaping the experience, and how the absence of a clear diagnosis was reinforcing every anxious projection I made. My experience with chronic pain and everything I've read since paint a very clear picture of what was happening with me in that first year. The sections that follow outline the concepts I later learned that helped make sense of this experience.

Erosion of identity

Research has long shown that chronic pain can erode a person's sense of identity, confidence, and normalcy. Early qualitative work found that people living with persistent pain often describe losing parts of themselves and struggling to recognize who they used to be.¹ More recent studies show that emotional avoidance and early adversity can reinforce this process by narrowing attention toward bodily symptoms, creating a cycle in which physical pain becomes an anchor for managing overwhelming emotion and internal conflict.² Contemporary psychosocial research also illustrates how ongoing pain disrupts valued roles, activities, and relationships, contributing to withdrawal, disconnection, and shifts in how people understand themselves in the world.³⁻⁵ Taken together, these findings help explain why chronic pain can profoundly reshape a person's identity even when tissue healing is complete.

Catastrophization

Research shows that catastrophizing is closely associated with greater pain intensity and emotional distress, and it is also linked to broader pain-related difficulties in clinical populations. Foundational work using the Pain Catastrophizing Scale identified three

components (rumination, magnification, and helplessness) and showed that higher overall catastrophizing scores predict greater pain severity and emotional distress across both experimental and clinical contexts.⁶ Neuroimaging work further demonstrated that higher catastrophizing is associated with increased activation in brain regions involved in anticipation, attention to pain, and emotional pain processing.⁷ Clinical findings also show that catastrophizing moderates the relationship between sleep efficiency and central sensitization in chronic musculoskeletal conditions such as knee osteoarthritis, indicating a pathway through which catastrophizing contributes to more difficult pain experiences.⁸

Relational pressures

Persistent pain often becomes a shared experience within families, affecting partners and relatives as well as patients. Research shows that spouses commonly report increased stress, reduced quality of life, and significant emotional strain as they work to interpret and respond to a loved one's pain.⁹ These pressures often contribute to role changes within the household and to ongoing emotional distress among partners and caregivers.¹⁰ Studies also indicate that certain partner responses, such as providing frequent assistance or discouraging activity, are associated with more pain behaviors and reduced activity levels in people with chronic pain.¹¹ Taken together, this research highlights pain as an interpersonal process that affects the broader family system and underscores the clinical importance of understanding its impact on partners and caregivers.

A crisis leads to a turning point

By the end of that first year, the solution to my pain had already landed in my lap. I had picked up *The Mindbody Prescription* by John E. Sarno, which I found intriguing, but I couldn't take it seriously enough at the time to act on. It ended up sitting on my bookshelf for the next six months. I continued seeing specialists, trying new medications, and being told I might need surgery that I could not afford after having already spent large amounts of money pursuing a cure.

Then something unexpected happened. A physician prescribed prednisone, and for three weeks I felt like I'd been reborn: completely pain-free. I became confident enough to take a short trip, but midway through it the prednisone stopped working. My pain returned, and I had to cut the trip short and fly home. The emotional crash was severe.

Back at home, I felt cornered. My options seemed limited. I could pursue surgery, which I could not afford. I could try to return to the United States for treatment, which I also could not afford. Or I could take another look at the book I had set aside months earlier. I had already ruled out every serious and life-threatening condition as a cause. That removed the barrier of "what if it is something serious" that had kept my fear alive, and it made room for the possibility that something else was happening. The conditions were right for me to take a serious second look at the book and consider that it might actually apply to me.

Reading it again was different. Believing it was about my pain was something else entirely. The book made the case that chronic pain can occur even when nothing is structurally wrong, and that emotional conflict, fear, and unresolved stress can play a central role in keeping symptoms alive. I found the ideas interesting, but accepting them on an emotional level was harder than understanding them in the abstract.

Still, the concepts stayed with me. A year later, after another crisis, I opened the book again and committed to trying the approach it described. That decision marked the beginning of the first real change I had seen since the pain began.

Long after I'd recovered, I began to wonder why I had been so resistant to Sarno's message, and why so many others continue to be. What I learned was both surprising...and not surprising at all. Understanding why this approach was so difficult for me helped clarify why many patients struggle with similar explanations, even when those explanations are accurate.

Why patients often resist psychological explanations

Intuitive Dualism

Many adults, including many patients, hold intuitions consistent with mind–body dualism (the view that the mind and body are separate entities). Research shows that these dualistic intuitions are common¹² and, in some patients, are associated with greater skepticism toward non-biological explanations for their illnesses.¹³ These beliefs shape how people explain and understand health-related experiences and can contribute to skepticism when clinicians offer psychological or behavioral mechanisms, because such explanations conflict with underlying assumptions about how the mind and body relate.

Stigma & Moral Meaning

Second, pain carries moral and social meaning. Research on social pain demonstrates that experiences of social rejection activate some of the same neural regions involved in physical pain.^{14,15} When psychological contributors are mentioned, many patients worry that their symptoms will be seen as less legitimate or misunderstood, especially if they have previously felt dismissed or if no clear structural pathology has been found.

Predictive-Processing & Sensory Intensity Mismatch

Perceptual and interpretive processes help explain why patients struggle with explanations that emphasize neural mechanisms. In chronic pain, the brain often generates threat predictions that outweigh or misinterpret sensory input, leading to a mismatch in which the perceived intensity of symptoms no longer tracks with tissue status.¹⁶ Neuroimaging studies show that chronic pain is associated with a shift from sensory to emotional–salience network processing, meaning the brain prioritizes predicted danger over incoming signals.¹⁷ This helps explain why pain can feel like

compelling evidence of ongoing physical harm, even when neural mechanisms such as prediction and expectation contribute significantly to the experience.¹⁸

Biomedical & Cultural Conditioning

Decades of biomedical conditioning also shape how patients interpret psychological explanations. Many people have been taught through public health messaging, clinical encounters, and imaging practices that pain must reflect structural damage, even though common degenerative findings frequently fail to correlate with symptoms.^{19,20} Within this model, the idea that pain could arise from neural processes rather than tissue injury feels counterintuitive, not because it threatens identity, but because it contradicts the medical framework patients have been repeatedly exposed to. Engel's critique of the biomedical model highlighted this narrow focus as early as 1977, and subsequent work has shown how structural labels and diagnostic language reinforce expectations of physical causation.^{21,22} For patients accustomed to such explanations, shifting to a neuroplastic or predictive-processing model represents a major conceptual change in how the body is understood.

The Self-Relevance Barrier

Psychological explanations can also conflict with a patient's self-concept. When people hold strong beliefs about what is happening in their body, information that contradicts those beliefs can evoke defensiveness. Classic work shows that self-relevant challenges activate protective processes that reduce openness to disconfirming evidence,²³ and newer research demonstrates that when new information threatens a person's sense of self, it can trigger strong emotional reactions that shape how people evaluate conflicting information.²⁴ Under those conditions, people tend to downplay or dismiss evidence that conflicts with their existing beliefs. For many patients, a psychological explanation does not feel like a clarification; it feels like a threat to their understanding of themselves.

Together, these factors mean that psychological explanations, if delivered without precision and sensitivity, can unintentionally heighten threat rather than reduce it. Clinicians can mitigate this by validating the reality and intensity of the pain, framing psychological mechanisms as neural processes rather than emotional weakness, and integrating these explanations gradually and collaboratively.

Sarno Was Early, Not Fringe

Sarno makes a strong case in his writings that the brain can create real pain in response to repressed emotion. But that idea has been exceptionally difficult for the modern medical establishment to accept.

Modern pain neuroscience has moved away from the language of 'repressed emotions,' but the underlying principle is now well supported: adverse life experiences, trauma-related emotional distress, and the persistent avoidance or inhibition of difficult emotions

are consistently associated with chronic centralized pain conditions and can contribute to their development and maintenance.²⁵⁻²⁷

Emotional avoidance is associated with increased physiological arousal, greater pain-related fear and anxiety, and difficulties resolving conditioned threat responses, all of which can contribute to heightened pain sensitivity.²⁸⁻³¹

Treatments that directly engage avoided emotional experiences, rather than simply managing pain-related thoughts or behaviors, have demonstrated meaningful clinical benefits, and in some conditions have been associated with changes in brain activity linked to emotional distress resolution.³²⁻³⁴

Emotional Awareness and Expression Therapy (EAET) targets these deeper emotional and interpersonal mechanisms, and multiple randomized trials demonstrate meaningful clinical impact.^{31,35-37} In the largest fibromyalgia RCT, EAET produced superior pain outcomes compared to education and showed advantages over CBT on key pain measures.³⁵ In a 2020 RCT of older adults with chronic musculoskeletal pain, 42 percent of EAET patients achieved at least 30 percent pain reduction, with one-third exceeding 50 percent, while only a single CBT participant reached the 30-percent threshold.³⁶ In a 2024 JAMA Network Open trial of older veterans, 63 percent of EAET patients achieved clinically significant improvement compared with only 17 percent receiving CBT, with EAET also outperforming CBT on anxiety, depression, PTSD symptoms, and life satisfaction.³⁷

These findings suggest that emotional processes such as unresolved conflict and avoidance are not merely correlated with chronic pain but can help sustain it, and that interventions targeting these emotional mechanisms can lead to meaningful symptom improvement.^{25,31,32}

But accepting all of that in the abstract is very different from saying, “That’s what’s happening in me.” Trusting in Sarno’s recovery process was the hardest part, and the most rewarding. Here’s what I did next:

My Recovery Process

I started journaling daily.

The goal wasn’t to vent, but to uncover repressed emotions, past and present. It wasn’t easy work, but doing it really paid off.

Emotional-processing approaches that help patients engage with difficult emotions, rather than relying on suppression or intellectualization, have been associated with symptom improvement in multiple studies.²⁶ Emotional avoidance has been discussed in the chronic pain literature, but the specific links to autonomic arousal, threat appraisal, and conditioned threat responses are not established in the cited studies. Expressive writing

interventions use a structured format in which participants write about stressful or emotional experiences.³⁸ In a randomized controlled trial, participants in expressive writing conditions showed significant and sustained reductions in physical and mental symptoms for at least four months after the intervention.³⁸ Emotional-awareness and expression-based interventions have shown improvements in chronic musculoskeletal pain and target emotional processes related to trauma or conflict.³⁹ These findings parallel results from emotional-processing therapies showing that addressing emotional conflicts, rather than avoiding them, can reduce pain intensity and interference.^{33,40} Together, this evidence suggests that written emotional-processing exercises may offer a useful pathway for improving symptoms.

I began resuming activity.

I had to believe that I wouldn't break anything by doing things as I had before. That meant walking even when it hurt. I talked to my brain while I walked: "I know what this is. There's nothing physically wrong with me, and I'm not afraid anymore."

Contemporary models of primary and centralized pain describe how cognitive-affective processes, including fear and avoidance-related beliefs, can contribute to the maintenance of chronic symptoms.⁴¹ These models also describe positive feedback loops in which threat-related appraisals can amplify pain and sustain chronicity, though the specific role of behavioral avoidance in these loops is not directly tested in the cited studies.^{35,41} Re-engaging in daily activities is incorporated into several multimodal or reconceptualization-based treatments and is associated with improvements in factors such as kinesiophobia and physical function.⁴² In a large real-world trial of fibromyalgia treatment, a multicomponent program based on pain neuroscience education produced medium to large improvements in pain, kinesiophobia, and physical function.⁴² Pain Reprocessing Therapy helps patients reinterpret pain sensations as non-dangerous, and in a randomized trial, about two-thirds of participants were pain-free or nearly pain-free after four weeks, accompanied by reductions in pain-related brain activity.⁴¹ These findings are consistent with integrative models describing chronic pain as a "danger alarm" that can be reduced by helping patients confront feared sensations or situations and reinterpret them as safe.³⁵

I stopped monitoring my symptoms.

Every time I checked in on the pain, I was reinforcing the belief that something was wrong. That kept me focused on the physical instead of the psychological. When I finally stopped monitoring every twinge, I started trusting the process, and that's when things started to really change.

Some patients with chronic pain show heightened attention to bodily sensations, particularly those who report high somatic awareness. Processes such as somatic focus can make those sensations feel more salient. Research shows that when pain is appraised as threatening, it tends to capture attention and draw focus toward bodily sensations.⁴³ Experimental work shows that interpreting identical sensations as potentially harmful can

increase reported pain intensity, and clinical research links somatic focus with higher pain reports.^{44,45} In clinical settings, these cognitive and emotional processes may appear as increased attention to symptoms. Somatosensory amplification, which includes a tendency to appraise benign sensations as more intense or pathological, is associated with greater psychological distress.⁴⁶ This pattern suggests that sustained attention to bodily sensations may make them feel more intrusive and harder to disengage from.

I discontinued pain medications.

As long as I kept trying to “fix things” with meds, I was telling myself I didn’t really believe in the process.

Patients with chronic pain may receive biomedical explanations that frame symptoms in terms of structural damage or tissue vulnerability. These explanations often do not match the clinical course of centralized pain conditions, where symptoms can persist despite normal imaging and the absence of progressive pathology.⁴⁷⁻⁴⁹ When patients expect rapid improvement from medical interventions but do not experience it, this mismatch can heighten distress and amplify the sense that pain signals a continuing threat. Research shows that threat-related interpretations draw attention toward bodily sensations and make them feel more intense and harder to disengage from.⁴³⁻⁴⁵ In some conditions, such as whiplash-associated disorders, psychosocial and perceptual factors predict long-term outcomes more strongly than initial physical findings, highlighting the importance of how symptoms are understood and appraised.⁵⁰ Treatments that address these cognitive and emotional contributors, including interventions that help patients work with avoidance, worry, and unresolved emotional conflict, often produce greater improvements than usual medical care alone.^{35,40} Together, these findings suggest that helping patients revise threat-based appraisals and build a more accurate understanding of centralized pain mechanisms can reduce distress, improve function, and interrupt the processes that sustain chronic pain.

A recovery that’s difficult to believe

It only took about three weeks of doing all of the above to go from “can’t walk across a room” to 95% pain-free. Within another month, I was at 100%. That was 18 years ago. It’s still the most astonishing thing I’ve ever experienced...and I never would’ve believed it was possible if I’d had any other way out.

Rapid and substantial improvement has been observed in conditions driven by central mechanisms. Across multiple randomized trials, a meaningful proportion of patients experience major symptom reductions within weeks, particularly in treatments that target cognitive, emotional, or perceptual contributors to pain. In the Pain Reprocessing Therapy trial, two-thirds of participants became pain-free or nearly pain-free after four weeks, with benefits sustained for at least one year.⁴¹ Emotional Awareness and Expression Therapy shows similar patterns of rapid improvement. In recent trials, substantial proportions of patients achieved clinically significant pain reduction over brief treatment courses.^{36,37} and even a single emotional-processing session reduced pain

severity and interference in primary-care patients.⁴⁰ Meta-analytic evidence shows that therapies targeting emotional processes produce large and persistent improvements across somatic symptom presentations.⁵¹ These findings also align with research showing that patients' expectations are a powerful predictor of recovery; individuals who believe improvement is possible recover far more quickly than those who expect chronicity.⁵² Taken together, the evidence shows that rapid recovery is well documented in these interventions, particularly when treatments address emotional and cognitive contributors to pain.

The real test came when the pain returned

In 2010, I'd been pain-free for over three years when I got emails from two different guys on the same day. Each had found my story on a blog. Both described having the exact same problem I'd had and wanted to know how I got better. It felt good knowing that maybe my story could help someone.

The very next day, the pain came back. At first, I panicked. It felt like a cruel joke. But within a few hours, I managed to get quiet enough to think clearly: I've beaten this before. I can do it again. It took about a month. But it worked. Again.

Clinically, brief returns of symptoms can occur even after substantial improvement in conditions driven by central mechanisms. One way to understand this pattern is through models of threat learning, which describe how attention and vigilance can increase the salience of pain-related cues.^{43,53} From this perspective, a small degree of uncertainty or threat sensitivity may persist in the predictive system, even when overall symptoms have improved. When attention to bodily sensations increases, these threat-related associations can feel more prominent, consistent with research showing that threat value enhances attentional capture and intensifies pain experience.⁴³⁻⁴⁵ Therapeutic studies reflect similar dynamics. In Pain Reprocessing Therapy, changes in pain beliefs and threat expectations accompany clinical improvement,⁴¹ and emotion-focused approaches help patients work with distressing emotional material in ways that support symptom reduction.⁵¹ From a predictive-processing standpoint, occasional symptom fluctuations can be viewed as the system briefly expressing older threat associations rather than signaling new pathology or treatment failure.

What this taught me about recovery

For a long time, I believed I'd never walk without pain again. But I've been doing it for 17 years now. That recovery didn't just take the pain away; it changed how I respond to anything physical that worries me. It helped me avoid turning temporary issues into lifelong ones. I still get checked when something concerns me, but I don't catastrophize. I learned how much my thoughts, emotions, and assumptions influence my body, and that changing those patterns is not only possible, but sometimes necessary. Pain used to feel like the enemy. Now I think of it more as an alarm that sometimes signals danger, but just

as often reflects stress or fatigue. Learning to listen without panicking gave me back my life.

What this means for physicians

This section focuses on how these findings may inform clinicians' understanding, priorities, and framing when working with patients experiencing persistent pain.

Most physicians take chronic pain seriously, and that was true in my case; every doctor I saw tried to help. But each focused on a different anatomical possibility, and no one asked about stress, fear, or what was happening in my life. Looking back, it's clear that the episode that finally disabled me wasn't isolated; I'd had years of unexplained symptoms in other parts of my body that were treated one by one, never as part of a pattern. Like many patients with chronic pain and illness, my symptoms moved and changed over time, but without a framework for understanding that behavior, each episode was interpreted as a new medical problem. I've often wondered whether, if modern training routinely included the science of centralized and emotion-influenced pain, my problem might have been recognized far earlier. Instead, I had to discover that framework myself, which took 18 months, and even now, I sometimes get skeptical looks when I explain what ultimately helped me. Research shows that when patients leave without a clear explanation of what their symptoms mean, uncertainty increases and distress rises. In qualitative studies, patients describe continuing to search for explanations and elaborating their symptom narratives in an effort to make sense of the problem.^{54,55}

Clear, validating explanations and compassionate communication can play an important role in shaping how patients interpret symptoms and engage with care, even when symptoms persist. Letting patients know that their experience is real, shared by others, and potentially understandable can help orient clinical conversation away from distress-driven explanation-seeking. Clinical experience and treatment models suggest that when patients adopt a clearer and less threatening understanding of their symptoms, those symptoms can feel less overwhelming, a pattern that aligns with improvements reported in emotional-processing and psychodynamic treatments.^{26,36,51} A single well-structured session that helps patients explore connections between stress and symptoms can meaningfully reduce distress and symptom burden.⁴⁰

Explain that persistent pain is often understood not as ongoing injury but as a protective response that can remain active even after tissues have healed, and that contemporary models describe this protective state as closely tied to how the brain interprets signals of threat and safety. A large body of theoretical and empirical work shows that expectation plays a key role in shaping symptom intensity through predictive-processing mechanisms, and models of threat learning offer an additional framework for understanding how fear and attention can influence pain.^{17,56,57} Helping patients understand this mechanism gives them a framework for hope: the problem is real, the pain is real, and many contemporary models emphasize that distress can be shaped by

how sensations are interpreted in the moment, not only by the state of the tissues. Studies demonstrate that when patients reinterpret symptoms from “damage” to “overprotection,” fear decreases and pain becomes less disruptive.⁴¹

If time allows, going beyond reassurance and offering specifics can make a meaningful difference. Explain that persistent pain is not always a sign of ongoing injury; strong evidence shows that symptoms can feel threatening even when no tissue damage is present, because pain functions as a protective perception shaped by threat and meaning.^{58,59} Cognitive–emotional factors such as attention, interpretation, and prior experience play a key role in determining how intense and distressing sensations feel; threat-focused attention increases salience, and labeling sensations as harmful makes them more painful.^{43,44,46} Clinical trials of emotional-processing and reinterpretation-based interventions show substantial improvements in pain and distress, and some approaches demonstrate meaningful change even over very short time frames.⁴⁰ In chronic pain RCTs, emotional-processing therapies have achieved outcomes that match or exceed standard approaches to care.^{33,36,37} Mechanistic studies show that threatening interpretations of bodily sensations increase distress, and interventions that help patients reinterpret symptoms reliably reduce distress.^{26,36,40,43,46}

If scope or time prevents you from guiding patients through this process yourself, refer them to someone who can, such as a psychologist, therapist, or coach experienced in centralized pain and emotional–cognitive contributors. Evidence from meta-analyses shows that empathy, therapeutic alliance, and the sense of being understood have a measurable impact on clinical outcomes across a range of treatment modalities, including in chronic pain populations.^{60,61} A referral to a clinician or coach who understands these mechanisms, and ideally someone who has lived through similar symptoms, can be the lifeline that moves a patient from fear to recovery.

When clinic time is tight, a behavioral coach can help with the non-medical work that often determines whether reassurance sticks: pacing, sleep stabilization, attention retraining practice, and reducing safety behaviors. If you ever want a quick fit check on whether coaching is a reasonable adjunct for a particular patient, I am happy to talk it through briefly, no referral expected.

What patients with chronic pain (and other symptoms that lack a clear structural explanation) need from medicine is clarity, compassion, and a credible “how”: a roadmap that explains why their symptoms feel so overwhelming, why that does not necessarily indicate damage, and what specific steps they can take to get on a path to recovery. Clinicians who communicate belief, optimism, and a clear next step make measurable changes in patient outcomes, not by minimizing symptoms, but by helping patients better understand what they are experiencing.⁶² For clinicians who want a deeper clinical explanation of these patterns, I have a companion article that reviews the research behind [neuroplastic recovery](#) and the treatment of chronic pain and illness.

This way of understanding persistent pain may be especially useful when you’re seeing patients who continue to return distressed despite normal workups and repeated reassurance, and nothing you say seems to land.

The following section translates these principles into concrete language that can be used in clinical conversations.

In Practice: Communicating With Patients After Serious Pathology Has Been Excluded

The following examples illustrate how these principles can be translated into concrete, patient-facing language during clinical encounters.

- “Your pain is real, and what you’re feeling makes sense in light of how the body and brain process pain.”
- “Pain can feel more intense or persistent when the body and brain are more sensitive, and the encouraging news is that this state can change.”
- “Stress, sleep, attention, and emotional strain can all turn up the volume on pain. Improving even one of these areas can help reduce pain.”
- “Many people improve a great deal once they understand what their symptoms mean, feel less threatened by them, and gradually get back to normal activities.” People often show substantial improvements in pain and function in clinical trials of behavioral and emotional treatments for centralized pain.^{35,36,41} In some studies, improvements have been accompanied by shifts in pain understanding and reduced fear.^{63,64}
- “You don’t have to face this alone. Support exists, and there are clinicians and trained professionals who can help you navigate this kind of recovery.”

These kinds of messages combine validation, clarity, and hope. Supportive clinician communication of this kind has been associated with improved patient outcomes.⁶⁰

Clinical Takeaways

- Many patients with chronic pain are afraid their symptoms signal ongoing harm. Brief, clear explanations about centralized pain mechanisms can sharply reduce uncertainty and promote engagement with care.
- When imaging and clinical workup do not identify a dangerous or progressive condition, it can be appropriate and reassuring to explain that pain can persist even when tissues are not being damaged.

- Cognitive, emotional, and behavioral processes such as threat perception, expectation, attention, and avoidance can amplify pain in ways patients often do not recognize. Naming these processes without judgment helps patients see a path forward.
- Several behavioral and emotional treatments for centralized pain have shown substantial improvements in randomized trials. Patients often benefit from knowing that improvement is possible even after long periods of suffering.
- Supportive communication matters. A calm, validating explanation paired with realistic hope may reduce fear and help patients approach the recovery process rather than withdraw from it.
- When appropriate, referral to non-medical support such as psychology, therapy, or coaching may help patients engage with the behavioral and attentional work described above.
- Above all, patients who understand what is not happening and what *can* happen with the right support often feel more confident taking steps that help reduce pain and restore function.

For clinicians interested to see how coaching integrates with medical care, [there's an article about that as well](#).

Contact:

Scott Muenzler, C³ Coaching
Physicians@c3coaching.co

References

1. Osborn M, Smith JA. The personal experience of chronic benign lower back pain: An interpretative phenomenological analysis. *British Journal of Health Psychology*. 1998;3(1):65-83. doi:[10.1111/j.2044-8287.1998.tb00556.x](https://doi.org/10.1111/j.2044-8287.1998.tb00556.x)
2. Lane RD, Anderson FS, Smith R. Biased Competition Favoring Physical Over Emotional Pain: A Possible Explanation for the Link Between Early Adversity and Chronic Pain. *Psychosom Med*. 2018;80(9):880-890. doi:[10.1097/PSY.0000000000000640](https://doi.org/10.1097/PSY.0000000000000640)
3. Grünenwald I, Kaluza AJ, Schultze M, van Dick R. Stress Mindset and Social Identification in Chronic Pain Patients and Their Relationship to Coping, Well-Being & Depression. *J Clin Psychol Med Settings*. 2023;30(1):153-168. doi:[10.1007/s10880-022-09883-8](https://doi.org/10.1007/s10880-022-09883-8)

4. Voorhees HL. "I Was Literally Just Not Myself": How Chronic Pain Changes Multiple Frames of Identity. *Health Commun.* 2023;38(8):1641-1653. doi:[10.1080/10410236.2022.2025702](https://doi.org/10.1080/10410236.2022.2025702)
5. Yu L, McCracken LM. The self in pain. *Curr Opin Psychol.* 2025;62:101972. doi:[10.1016/j.copsyc.2024.101972](https://doi.org/10.1016/j.copsyc.2024.101972)
6. Sullivan MJL, Bishop SR, Pivik J. The Pain Catastrophizing Scale: Development and validation. *Psychological Assessment.* 1995;7(4):524-532. doi:[10.1037/1040-3590.7.4.524](https://doi.org/10.1037/1040-3590.7.4.524)
7. Gracely RH, Geisser ME, Giesecke T, et al. Pain catastrophizing and neural responses to pain among persons with fibromyalgia. *Brain.* 2004;127(Pt 4):835-843. doi:[10.1093/brain/awh098](https://doi.org/10.1093/brain/awh098)
8. Campbell CM, Buenaver LF, Finan P, et al. Sleep, Pain Catastrophizing, and Central Sensitization in Knee Osteoarthritis Patients With and Without Insomnia. *Arthritis Care Res (Hoboken).* 2015;67(10):1387-1396. doi:[10.1002/acr.22609](https://doi.org/10.1002/acr.22609)
9. West C, Usher K, Foster K, Stewart L. Chronic pain and the family: the experience of the partners of people living with chronic pain. *J Clin Nurs.* 2012;21(23-24):3352-3360. doi:[10.1111/j.1365-2702.2012.04215.x](https://doi.org/10.1111/j.1365-2702.2012.04215.x)
10. Ojeda B, Salazar A, Dueñas M, Torres LM, Micó JA, Failde I. The impact of chronic pain: the perspective of patients, relatives, and caregivers. *Fam Syst Health.* 2014;32(4):399-407. doi:[10.1037/fsh0000069](https://doi.org/10.1037/fsh0000069)
11. Kindt S, Vansteenkiste M, Loeys T, et al. When Is Helping your Partner with Chronic Pain a Burden? The Relation Between Helping Motivation and Personal and Relational Functioning. *Pain Med.* 2015;16(9):1732-1744. doi:[10.1111/pme.12766](https://doi.org/10.1111/pme.12766)
12. Forstmann M, Burgmer P. Adults are intuitive mind-body dualists. *J Exp Psychol Gen.* 2015;144(1):222-235. doi:[10.1037/xge0000045](https://doi.org/10.1037/xge0000045)
13. Burgmer P, Forstmann M. Mind-Body Dualism and Health Revisited: How Belief in Dualism Shapes Health Behavior. *Social Psychology.* 2018;49(4):219-230. doi:[10.1027/1864-9335/a000344](https://doi.org/10.1027/1864-9335/a000344)
14. Eisenberger NI, Lieberman MD, Williams KD. Does rejection hurt? An fMRI study of social exclusion. *Science.* 2003;302(5643):290-292. doi:[10.1126/science.1089134](https://doi.org/10.1126/science.1089134)
15. Eisenberger NI. Social pain and the brain: controversies, questions, and where to go from here. *Annu Rev Psychol.* 2015;66:601-629. doi:[10.1146/annurev-psych-010213-115146](https://doi.org/10.1146/annurev-psych-010213-115146)

16. Hashmi JA, Baliki MN, Huang L, et al. Shape shifting pain: chronification of back pain shifts brain representation from nociceptive to emotional circuits. *Brain*. 2013;136(Pt 9):2751-2768. doi:[10.1093/brain/awt211](https://doi.org/10.1093/brain/awt211)
17. Apkarian AV, Baliki MN, Farmer MA. Predicting transition to chronic pain. *Curr Opin Neurol*. 2013;26(4):360-367. doi:[10.1097/WCO.0b013e32836336ad](https://doi.org/10.1097/WCO.0b013e32836336ad)
18. Brown CA, El-Deredy W, Jones AKP. When the brain expects pain: common neural responses to pain anticipation are related to clinical pain and distress in fibromyalgia and osteoarthritis. *Eur J Neurosci*. 2014;39(4):663-672. doi:[10.1111/ejn.12420](https://doi.org/10.1111/ejn.12420)
19. Bedson J, Croft PR. The discordance between clinical and radiographic knee osteoarthritis: a systematic search and summary of the literature. *BMC Musculoskelet Disord*. 2008;9:116. doi:[10.1186/1471-2474-9-116](https://doi.org/10.1186/1471-2474-9-116)
20. Brinjikji W, Luetmer PH, Comstock B, et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. *AJNR Am J Neuroradiol*. 2015;36(4):811-816. doi:[10.3174/ajnr.A4173](https://doi.org/10.3174/ajnr.A4173)
21. Engel GL. The need for a new medical model: a challenge for biomedicine. *Science*. 1977;196(4286):129-136. doi:[10.1126/science.847460](https://doi.org/10.1126/science.847460)
22. Hadler NM. MRI for regional back pain: need for less imaging, better understanding. *JAMA*. 2003;289(21):2863-2865. doi:[10.1001/jama.289.21.2863](https://doi.org/10.1001/jama.289.21.2863)
23. Steele CM, Spencer SJ, Lynch M. Self-image resilience and dissonance: the role of affirmational resources. *J Pers Soc Psychol*. 1993;64(6):885-896. doi:[10.1037/0022-3514.64.6.885](https://doi.org/10.1037/0022-3514.64.6.885)
24. Wischniewski M, Krämer N. The Role of Emotions and Identity-Protection Cognition When Processing (Mis)Information. *Technology, Mind, and Behavior*. 2021;2(1). doi:[10.1037/tmb0000029](https://doi.org/10.1037/tmb0000029)
25. Lumley MA, Cohen JL, Borszcz GS, et al. Pain and emotion: a biopsychosocial review of recent research. *J Clin Psychol*. 2011;67(9):942-968. doi:[10.1002/jclp.20816](https://doi.org/10.1002/jclp.20816)
26. Lumley MA, Schubiner H. Psychological Therapy for Centralized Pain: An Integrative Assessment and Treatment Model. *Psychosom Med*. 2019;81(2):114-124. doi:[10.1097/PSY.0000000000000654](https://doi.org/10.1097/PSY.0000000000000654)
27. Afari N, Ahumada SM, Wright LJ, et al. Psychological trauma and functional somatic syndromes: a systematic review and meta-analysis. *Psychosom Med*. 2014;76(1):2-11. doi:[10.1097/PSY.000000000000010](https://doi.org/10.1097/PSY.000000000000010)

28. Quartana PJ, Burns JW. Painful consequences of anger suppression. *Emotion*. 2007;7(2):400-414. doi:[10.1037/1528-3542.7.2.400](https://doi.org/10.1037/1528-3542.7.2.400)
29. Rhudy JL, Meagher MW. Fear and anxiety: divergent effects on human pain thresholds. *Pain*. 2000;84(1):65-75. doi:[10.1016/S0304-3959\(99\)00183-9](https://doi.org/10.1016/S0304-3959(99)00183-9)
30. Rivat C, Becker C, Blugeot A, et al. Chronic stress induces transient spinal neuroinflammation, triggering sensory hypersensitivity and long-lasting anxiety-induced hyperalgesia. *Pain*. 2010;150(2):358-368. doi:[10.1016/j.pain.2010.05.031](https://doi.org/10.1016/j.pain.2010.05.031)
31. Lumley MA, Krohner S, Marshall LM, Kitts TC, Schubiner H, Yarns BC. Emotional awareness and other emotional processes: implications for the assessment and treatment of chronic pain. *Pain Manag*. 2021;11(3):325-332. doi:[10.2217/pmt-2020-0081](https://doi.org/10.2217/pmt-2020-0081)
32. Hsu MC, Schubiner H, Lumley MA, Stracks JS, Clauw DJ, Williams DA. Sustained pain reduction through affective self-awareness in fibromyalgia: a randomized controlled trial. *J Gen Intern Med*. 2010;25(10):1064-1070. doi:[10.1007/s11606-010-1418-6](https://doi.org/10.1007/s11606-010-1418-6)
33. Lumley MA, Schubiner H. Emotional Awareness and Expression Therapy for Chronic Pain: Rationale, Principles and Techniques, Evidence, and Critical Review. *Curr Rheumatol Rep*. 2019;21(7):30. doi:[10.1007/s11926-019-0829-6](https://doi.org/10.1007/s11926-019-0829-6)
34. Drossman DA, Ringel Y, Vogt BA, et al. Alterations of brain activity associated with resolution of emotional distress and pain in a case of severe irritable bowel syndrome. *Gastroenterology*. 2003;124(3):754-761. doi:[10.1053/gast.2003.50103](https://doi.org/10.1053/gast.2003.50103)
35. Lumley MA, Schubiner H, Lockhart NA, et al. Emotional awareness and expression therapy, cognitive behavioral therapy, and education for fibromyalgia: a cluster-randomized controlled trial. *Pain*. 2017;158(12):2354-2363. doi:[10.1097/j.pain.0000000000001036](https://doi.org/10.1097/j.pain.0000000000001036)
36. Yarns BC, Lumley MA, Cassidy JT, et al. Emotional Awareness and Expression Therapy Achieves Greater Pain Reduction than Cognitive Behavioral Therapy in Older Adults with Chronic Musculoskeletal Pain: A Preliminary Randomized Comparison Trial. *Pain Med*. 2020;21(11):2811-2822. doi:[10.1093/pmt/pnaa145](https://doi.org/10.1093/pmt/pnaa145)
37. Yarns BC, Jackson NJ, Alas A, Melrose RJ, Lumley MA, Sultzer DL. Emotional Awareness and Expression Therapy vs Cognitive Behavioral Therapy for Chronic Pain in Older Veterans: A Randomized Clinical Trial. *JAMA Netw Open*. 2024;7(6):e2415842. doi:[10.1001/jamanetworkopen.2024.15842](https://doi.org/10.1001/jamanetworkopen.2024.15842)
38. Baikie KA, Geerligs L, Wilhelm K. Expressive writing and positive writing for participants with mood disorders: an online randomized controlled trial. *J Affect Disord*. 2012;136(3):310-319. doi:[10.1016/j.jad.2011.11.032](https://doi.org/10.1016/j.jad.2011.11.032)

39. Burger AJ, Lumley MA, Carty JN, et al. The effects of a novel psychological attribution and emotional awareness and expression therapy for chronic musculoskeletal pain: A preliminary, uncontrolled trial. *J Psychosom Res.* 2016;81:1-8. doi:[10.1016/j.jpsychores.2015.12.003](https://doi.org/10.1016/j.jpsychores.2015.12.003)

40. Ziadni MS, Carty JN, Doherty HK, et al. A life-stress, emotional awareness, and expression interview for primary care patients with medically unexplained symptoms: A randomized controlled trial. *Health Psychol.* 2018;37(3):282-290. doi:[10.1037/hea0000566](https://doi.org/10.1037/hea0000566)

41. Ashar YK, Gordon A, Schubiner H, et al. Effect of Pain Reprocessing Therapy vs Placebo and Usual Care for Patients With Chronic Back Pain: A Randomized Clinical Trial. *JAMA Psychiatry.* 2022;79(1):13-23. doi:[10.1001/jamapsychiatry.2021.2669](https://doi.org/10.1001/jamapsychiatry.2021.2669)

42. Serrat M, Almirall M, Musté M, et al. Effectiveness of a Multicomponent Treatment for Fibromyalgia Based on Pain Neuroscience Education, Exercise Therapy, Psychological Support, and Nature Exposure (NAT-FM): A Pragmatic Randomized Controlled Trial. *J Clin Med.* 2020;9(10):3348. doi:[10.3390/jcm9103348](https://doi.org/10.3390/jcm9103348)

43. Eccleston C, Crombez G. Pain demands attention: a cognitive-affective model of the interruptive function of pain. *Psychol Bull.* 1999;125(3):356-366. doi:[10.1037/0033-2959.125.3.356](https://doi.org/10.1037/0033-2959.125.3.356)

44. Arntz A, Claassens L. The meaning of pain influences its experienced intensity. *Pain.* 2004;109(1-2):20-25. doi:[10.1016/j.pain.2003.12.030](https://doi.org/10.1016/j.pain.2003.12.030)

45. O'Brien EM, Atchison JW, Gremillion HA, Waxenberg LB, Robinson ME. Somatic focus/awareness: Relationship to negative affect and pain in chronic pain patients. *Eur J Pain.* 2008;12(1):104-115. doi:[10.1016/j.ejpain.2007.04.001](https://doi.org/10.1016/j.ejpain.2007.04.001)

46. Barsky AJ, Wyshak G, Klerman GL. The somatosensory amplification scale and its relationship to hypochondriasis. *J Psychiatr Res.* 1990;24(4):323-334. doi:[10.1016/0022-3956\(90\)90004-a](https://doi.org/10.1016/0022-3956(90)90004-a)

47. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. *Pain.* 2011;152(3 Suppl):S2-S15. doi:[10.1016/j.pain.2010.09.030](https://doi.org/10.1016/j.pain.2010.09.030)

48. Kosek E, Cohen M, Baron R, et al. Do we need a third mechanistic descriptor for chronic pain states? *Pain.* 2016;157(7):1382-1386. doi:[10.1097/j.pain.0000000000000507](https://doi.org/10.1097/j.pain.0000000000000507)

49. Clauw DJ. Diagnosing and treating chronic musculoskeletal pain based on the underlying mechanism(s). *Best Pract Res Clin Rheumatol.* 2015;29(1):6-19. doi:[10.1016/j.bepr.2015.04.024](https://doi.org/10.1016/j.bepr.2015.04.024)

50. Scholten-Peeters GGM, Verhagen AP, Bekkering GE, et al. Prognostic factors of whiplash-associated disorders: a systematic review of prospective cohort studies. *Pain*. 2003;104(1-2):303-322. doi:[10.1016/s0304-3959\(03\)00050-2](https://doi.org/10.1016/s0304-3959(03)00050-2)
51. Abbass A, Town J, Holmes H, et al. Short-Term Psychodynamic Psychotherapy for Functional Somatic Disorders: A Meta-Analysis of Randomized Controlled Trials. *Psychother Psychosom*. 2020;89(6):363-370. doi:[10.1159/000507738](https://doi.org/10.1159/000507738)
52. Carroll LJ, Holm LW, Ferrari R, Ozegovic D, Cassidy JD. Recovery in whiplash-associated disorders: do you get what you expect? *J Rheumatol*. 2009;36(5):1063-1070. doi:[10.3899/jrheum.080680](https://doi.org/10.3899/jrheum.080680)
53. Crombez G, Van Ryckeghem DML, Eccleston C, Van Damme S. Attentional bias to pain-related information: a meta-analysis. *Pain*. 2013;154(4):497-510. doi:[10.1016/j.pain.2012.11.013](https://doi.org/10.1016/j.pain.2012.11.013)
54. Salmon P, Peters S, Stanley I. Patients' perceptions of medical explanations for somatisation disorders: qualitative analysis. *BMJ*. 1999;318(7180):372-376. doi:[10.1136/bmj.318.7180.372](https://doi.org/10.1136/bmj.318.7180.372)
55. Salmon P. The potentially somatizing effect of clinical consultation. *CNS Spectr*. 2006;11(3):190-200. doi:[10.1017/s109285290001436x](https://doi.org/10.1017/s109285290001436x)
56. Kiverstein J, Kirchhoff M, Thacker M. An Embodied Predictive Processing Theory of Pain. *Review of Philosophy and Psychology*. 2022;13. doi:[10.1007/s13164-022-00616-2](https://doi.org/10.1007/s13164-022-00616-2)
57. Song Y, Yao M, Kemprechos H, et al. Predictive coding models for pain perception. *J Comput Neurosci*. 2021;49(2):107-127. doi:[10.1007/s10827-021-00780-x](https://doi.org/10.1007/s10827-021-00780-x)
58. Moseley GL. Reconceptualising pain according to modern pain science. *Physical Therapy Reviews*. 2007;12(3):169-178. doi:[10.1179/108331907X223010](https://doi.org/10.1179/108331907X223010)
59. Moseley GL, Butler DS. Fifteen Years of Explaining Pain: The Past, Present, and Future. *J Pain*. 2015;16(9):807-813. doi:[10.1016/j.jpain.2015.05.005](https://doi.org/10.1016/j.jpain.2015.05.005)
60. Kelley JM, Kraft-Todd G, Schapira L, Kossowsky J, Riess H. The influence of the patient-clinician relationship on healthcare outcomes: a systematic review and meta-analysis of randomized controlled trials. *PLoS One*. 2014;9(4):e94207. doi:[10.1371/journal.pone.0094207](https://doi.org/10.1371/journal.pone.0094207)
61. Flückiger C, Del Re AC, Wampold BE, Horvath AO. The alliance in adult psychotherapy: A meta-analytic synthesis. *Psychotherapy (Chic)*. 2018;55(4):316-340. doi:[10.1037/pst0000172](https://doi.org/10.1037/pst0000172)

62. Street RL, Makoul G, Arora NK, Epstein RM. How does communication heal? Pathways linking clinician-patient communication to health outcomes. *Patient Educ Couns.* 2009;74(3):295-301. doi:[10.1016/j.pec.2008.11.015](https://doi.org/10.1016/j.pec.2008.11.015)
63. Moseley GL, Nicholas MK, Hodges PW. A randomized controlled trial of intensive neurophysiology education in chronic low back pain. *Clin J Pain.* 2004;20(5):324-330. doi:[10.1097/00002508-200409000-00007](https://doi.org/10.1097/00002508-200409000-00007)
64. Leeuw M, Goossens MEJB, van Breukelen GJP, et al. Exposure in vivo versus operant graded activity in chronic low back pain patients: results of a randomized controlled trial. *Pain.* 2008;138(1):192-207. doi:[10.1016/j.pain.2007.12.009](https://doi.org/10.1016/j.pain.2007.12.009)