Abilene Pain
INTERVENTIONAL PAIN MANAGEMENT

Understanding Nociplastic Pain

When the pain system itself becomes the problem

You have been told that some or all of your pain is **nociplastic**. This is a real, physical, medically recognized pain type — formally adopted in 2017 by the International Association for the Study of Pain as the third major category of pain, alongside nociceptive and neuropathic pain.

Nociplastic pain means your nervous system has become **amplified**. The alarm system that detects and reports pain has turned its volume up and left it there. The pain you feel is genuine. What has changed is not the tissue — it is how your spinal cord and brain process the signals coming from it.

> **The most important point**
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> Normal imaging and normal blood work do *not* mean your pain is imagined, exaggerated, or psychological. It means the problem is in signal processing rather than in the tissue — and signal processing does not show up on an MRI. We are not telling you the pain is in your head. We are telling you where in the nervous system it is being generated.

The Three Types of Pain

Type	What is happening	Typical examples	What usually helps
Nociceptive	Tissue is damaged or inflamed and correctly reports it	Arthritis, fracture, post-surgical pain, muscle strain	Anti-inflammatories, injections, treating the damaged structure
Neuropathic	A nerve itself is injured, compressed, or diseased	Sciatica from a herniated disc, diabetic neuropathy, shingles pain	Nerve medications, nerve blocks, decompression, neurostimulation
Nociplastic	The nervous system amplifies and sustains pain without ongoing tissue or nerve damage	Fibromyalgia, widespread chronic pain, some persistent back and pelvic pain	Exercise, sleep repair, specific nerve-modulating medications, brain-based therapies

These categories are not mutually exclusive. **Most of our patients have a mixed pain picture.** You can absolutely have a real disc herniation, real arthritis, or a real prior surgery *and* have a nociplastic component layered on top. Recognizing that layer is what allows us to stop chasing the wrong target.

What Is Physically Happening: The Pathophysiology

Several measurable changes occur in the nervous system in nociplastic pain. These are not theoretical — they have been demonstrated in laboratory testing, functional brain imaging, and spinal fluid studies.

1. Central sensitization

Pain-carrying nerve cells in the spinal cord become hyperexcitable. Receptors on these cells (particularly NMDA receptors) are switched on and stay on, so a signal that should produce a small response instead produces a large one. Over time these connections physically strengthen, the same way a memory is consolidated. Your spinal cord has, in effect, learned the pain.

2. Loss of the body's built-in pain brakes

Your brainstem normally sends signals *down* the spinal cord that suppress incoming pain traffic — this is why a soldier can be injured and not feel it until later. In nociplastic pain, this descending inhibitory system underperforms and the descending *facilitating* system dominates. Less brake, more accelerator. This is measurable in the laboratory and is one of the most reproducible findings in this condition.

3. Neuroinflammation and glial activation

Glial cells — the support cells of the nervous system — shift into an activated, inflammatory state and release chemical messengers (cytokines) that keep neighboring pain neurons excitable. This is inflammation inside the nervous system, not inflammation in your joints or muscles, which is why anti-inflammatory pills do so little for it.

4. Altered brain chemistry and connectivity

Imaging studies in people with nociplastic pain consistently show elevated glutamate (the main excitatory chemical) and reduced GABA (the main calming chemical) in pain-processing regions, along with altered communication between the insula, anterior cingulate cortex, and the brain's resting network. This is the biological reason the pain feels constant and intrusive even at rest.

5. Amplifiers

Poor sleep, physical deconditioning, sustained stress, and untreated mood disorders all measurably lower pain thresholds and sustain the changes above. This is also why treating them is not a consolation prize — it is direct treatment of the mechanism.

Symptoms You May Recognize

- **Widespread or shifting pain** — multiple body regions, or pain that moves and does not respect a single nerve or joint pattern
- **Pain out of proportion to the findings** on imaging or examination
- **Allodynia** — ordinary touch hurts: bedsheets, waistbands, a bra strap, a light hug
- **Hyperalgesia** — painful things hurt far more and far longer than expected, including after minor procedures
- **Sensory sensitivity beyond pain** — to bright light, loud sound, strong odors, cold, or heat
- **Non-restorative sleep** — you sleep hours but wake unrefreshed and stiff
- **Fatigue** that is not proportional to activity
- **Cognitive symptoms** — word-finding difficulty, poor concentration, short-term memory lapses
- **Flares** triggered by weather change, stress, overexertion, illness, or poor sleep

Commonly associated conditions include irritable bowel syndrome, interstitial cystitis or bladder pain syndrome, temporomandibular (jaw) pain, tension and migraine headache, restless legs, and pelvic pain. When several of these cluster in one person, it points strongly to a nociplastic mechanism.

> **About anxiety and depression**
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> These occur more often in people with nociplastic pain, and it matters that we address them — they share the same neurotransmitter systems and they measurably worsen pain amplification. But they are not the cause of your pain, and the relationship runs in both directions. Chronic pain reliably produces mood symptoms in people who never had them before.

How It Is Diagnosed

There is no blood test and no scan for nociplastic pain. It is a **clinical diagnosis**, made by pattern recognition: the distribution and quality of your pain, your examination, your associated symptoms, your response to prior treatments, and the absence of a structural finding that adequately explains the picture.

Validated questionnaires may be used to quantify how widespread your pain is and how severe the associated symptoms are.

Importantly, we still evaluate for other causes. Thyroid disease, inflammatory arthritis, vitamin D deficiency, sleep apnea, and anemia can all mimic or worsen this picture and are worth ruling out.

Treatments That Work

Foundation — the highest-value interventions

- **Graded aerobic exercise.** This has the strongest evidence of *any* treatment for nociplastic pain, including every medication. It restores descending inhibition — it repairs the brakes. Start below your flare threshold and increase slowly and consistently. Walking, stationary cycling, and pool-based exercise are all effective. Tai chi has produced results equal to or better than aerobic exercise in controlled trials.
- **Sleep restoration.** Fragmented sleep directly lowers pain thresholds. Screening for obstructive sleep apnea and treating insomnia (cognitive behavioral therapy for insomnia is first-line, not sleeping pills) often produces more pain relief than adding another analgesic.
- **Pain neuroscience education.** Understanding these mechanisms measurably reduces pain and disability. That is why you are reading this document.
- **Cognitive behavioral therapy or acceptance and commitment therapy.** These retrain the processing pathways involved. This is neurological treatment, not psychiatric referral.

Medications with the best evidence

- **Duloxetine and milnacipran** (SNRIs) — FDA-approved for fibromyalgia. They restore the descending inhibitory pathways described above.
- **Pregabalin** (FDA-approved for fibromyalgia) and **gabapentin** — reduce excitatory neurotransmitter release.
- **Low-dose tricyclics at bedtime** — amitriptyline or nortriptyline, and low-dose cyclobenzaprine. Doses used for pain are far below antidepressant doses; they improve both pain and sleep quality.
- **Low-dose naltrexone** — an off-label option targeting glial activation, with promising early trial evidence and a favorable safety profile.

Expect to trial more than one agent. Response is individual, benefit builds over weeks, and combination therapy at modest doses often outperforms a single drug pushed to maximum.

Useful adjuncts

- Aquatic therapy, yoga, and mindfulness-based stress reduction
- Correcting vitamin D deficiency; weight management where relevant
- Treating the coexisting conditions — migraine, IBS, jaw pain, sleep apnea

Treatments That Do Not Work — and Can Make This Worse

We want you to understand our reasoning here

- **Opioids.** These do not work for nociplastic pain, and with continued use they produce *opioid-induced hyperalgesia* — the drug itself sensitizes the nervous system further, increasing pain over time. Long-term opioid therapy in this population is associated with worse function and worse pain. Declining to prescribe them is a clinical decision about mechanism, not a judgment about you.
- **Anti-inflammatories and acetaminophen.** Minimal benefit against the nociplastic component, because there is no tissue inflammation driving it. They may still help a coexisting arthritic or inflammatory problem.
- **Benzodiazepines and Z-drugs.** No pain-relieving effect, and they degrade the deep sleep stages you most need to restore.
- **Steroids.** No established role in nociplastic pain.
- **Repeated procedures and surgery aimed at incidental findings.** Operating on a structure that is not generating the pain reliably fails, and in a sensitized nervous system each new incision or intervention carries a real risk of worsening the amplification.

Where Procedures Fit In

We are an interventional practice, and interventional treatment still has a place — but the target matters. Injections, nerve blocks, radiofrequency ablation, and neurostimulation treat **nociceptive and neuropathic generators**. When you have a genuine facet-mediated pain, a specific inflamed nerve root, or a discrete peripheral nerve problem, treating it is worthwhile, and reducing that ongoing input can help settle the amplification.

What a procedure cannot do is reverse central sensitization on its own. If we recommend an injection, we will tell you specifically which pain generator we are targeting and what portion of your pain we expect it to address. If your relief is partial or short-lived, that is often expected and informative — not a failure, and not a reason to escalate to a bigger procedure.

Setting Realistic Expectations

Be direct with us and we will be direct with you. Nociplastic pain is usually **managed rather than cured**. The realistic and achievable goal is meaningful reduction in pain intensity and flare frequency, with substantial gains in sleep, function, and independence.

Progress is measured in months, not visits, and it is rarely linear — expect good stretches interrupted by flares. Patients who do best are the ones who work several levers at once: consistent movement, protected sleep, an appropriate medication regimen, and active engagement with the therapy side. Any one of these alone underperforms.

Call our office if you develop

- New weakness in an arm or leg, or a foot that drags
- New loss of bowel or bladder control, or numbness in the groin or inner thighs
- Fever, unexplained weight loss, or night sweats with your pain
- A sudden, severe change in pain that feels different from your usual pattern

These are not features of nociplastic pain and require prompt evaluation.

YOUR CARE TEAM
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